Comparison of Gait with Ankle Foot Orthosis (AFO) and Functional Electrical Stimulation (FES) in patients following Stroke

Dissertation submitted to
The Tamil Nadu Dr. M.G.R. Medical University
in partial fulfilment of the requirement for
M.D. branch XIX – Physical Medicine and Rehabilitation final examination in May 2019

CERTIFICATE

This is to certify that the dissertation titled “Comparison of gait with Ankle Foot Orthosis (AFO) and Functional Electrical Stimulation (FES) in patients following stroke” is the bona fide work of Dr. Gourav Sannyasi, candidate number 201629052 towards the MD Physical Medicine and Rehabilitation Degree Examination of the Tamil Nadu Dr. M.G.R Medical University to be conducted in May 2019. This work has not been submitted to any university in part or full.

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DECLARATION

I hereby declare that this dissertation titled “Comparison of gait with Ankle Foot Orthosis (AFO) and Functional Electrical Stimulation (FES) in patients following stroke” is a bona fide work done by me under the guidance of Dr. George Tharion, Professor, Department of Physical Medicine and Rehabilitation, Christian Medical College, Vellore. This work has not been submitted to any university in part or full.

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CERTIFICATE – II

This is to certify that this dissertation work titled "Comparison of gait with Ankle Foot Orthosis (AFO) and Functional Electrical Stimulation (FES) in patients following stroke" of the candidate Dr. Gourav Sannyasi with registration number **201629052** for the award of MD in the branch of Physical Medicine and Rehabilitation. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows (6%) percentage of plagiarism in the dissertation.

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Contents

INTRODUCTION:

Stroke is sudden occurrence of permanent injury to an area of brain due to vascular aetiology. Stroke has been a major cause of disability. In 2013, stroke was the second leading cause of death comprising 11.8% of all deaths worldwide and third most common cause of disability. (1) According to the India stroke factsheet updated in 2012, the estimated age-adjusted prevalence rate for stroke ranges between 84-262/100,000 in rural and between 334- 424/100,000 in urban areas. (2) Hypertension, Diabetes, Dyslipidaemia, Atrial fibrillation and tobacco consumption are the most common modifiable causes of stroke. (3)

Hemiplegia is one of the most common impairments following stroke which significantly affects the normal gait pattern. The mobility of majority of stroke patients is limited and recovery of gait pattern is considered as top priority for rehabilitation. At 3 weeks of stroke 50-80% of patients can walk with some support and 65-85% of stroke patients start walking independently by 6 months following stroke with persisting gait deviation. (4,5) Walking endurance measured by 6 minute walk test remained the major problem among patients with chronic stroke. (6) Lower extremity weakness mainly hip extensor, knee extensor, ankle plantar flexor leads to decreased speed and asymmetry while walking. (7)

Footdrop, the decreased ability to dorsiflex the ankle during the swing phase of gait, is a significant lower extremity motor impairment following stroke which contributes to mobility related disability. Ankle dorsiflexor weakness, adaptive shortening of ankle plantarflexor result in foot drop. Ankle dorsiflexors help in clearing the foot during swing phase of gait cycle. As a result many stroke patients with foot drop use circumduction and hip hiking while walking. (8,9) The traditional mode of treatment provided for foot drop is ankle foot orthosis (AFO) which keeps the ankle in neutral position. AFO provides medio-lateral ankle stability in stance phase and achieves effective toe clearance during swing phase. There are few disadvantages of AFO such as restricted ankle mobility that may lead to development of contracture, difficulty to get up from a chair, reduced cosmesis and discomfort in donning and doffing. (10–12)

The newer modality of treatment is Functional Electrical Stimulation (FES) of the peroneal nerve. FES applies low intensity current to the intact nerves of the body to generate muscle contraction. (13) While walking FES can be used to generate ankle dorsiflexion by stimulating common peroneal nerve in foot drop patients. Ankle mobility is unrestrained with FES. Peroneal nerve stimulator has not been routinely recommended due to exorbitant cost.

Both these treatment options are well established for the management of foot drop and there is no conclusive evidence to suggest that FES is superior to AFO for correction of foot drop.(14) The current study was done for the comparison among FES and AFO among the patients with post stroke foot drop.

AIMS and OBJECTIVES:

Aim of the study:

To determine whether FES has any added benefits as compared to Ankle Foot Orthosis (AFO) in post stroke patients, by measuring gait parameters.

Objectives of the study:

-To compare spatiotemporal parameters between barefoot, Ankle-foot-orthosis (AFO) and Functional electrical stimulation.

-To evaluate ankle-foot kinematics in patients with stroke

HYPOTHESIS:

AFO (Ankle Foot Orthosis) is equally effective for the management of foot drop in post stroke patients compared to FES (Functional Electrical Stimulation).

REVIEW OF LITERATURE:

DEFINITION OF STROKE:

In 1970, stroke was defined by the World Health Organization as ‘a clinical syndrome consisting of rapidly developing clinical signs of focal (or global in case of coma) disturbance of cerebral function lasting more than 24 hours or leading to death with no apparent cause other than a vascular origin.’ (15) In 1960, stroke was considered to be sudden neurodeficits of vascular origin lasing for more than 7 days. Transient ischaemic attacks (TIA) were considered if neurodeficits persist for less than 24 hours. Neurodeficits that lasted between 24 hours to 7 days was considered as Reversible Ischaemic Neurological Deficits (RIND). RIND is an obsolete term as most of neurological events in RIND are associated with cerebral infarction on neuroimaging.(16) 50% of TIAs show brain injury (infarction) on diffusion weighted imaging which confers that arbitrary 24 hour time period of diagnosing TIA was inaccurate. The new guideline removed the time factor from definition of TIA. Transient ischaemic attacks are considered as a transient episode of neurodeficits due to focal ischemic lesions in the brain, spinal cord, retina without any acute infarction.(17) The updated definition of stroke for currently is based on neuropathological, neuroimaging, and/or clinical evidence of permanent injury (infarction) which also includes silent infarction and haemorrhages.(18)

EPIDEMIOLOGY OF STROKE:

In 2013, stroke was the second leading cause of death comprising 11.8% of all deaths worldwide and third most common cause of disability (4.5% of DALYs). According to Global Burden of Disease (GBD, 2013 study), prevalence of haemorrhagic stroke was 3,725,085 cases and ischaemic stroke was 7,258,216 cases among adults aged 20-64 years worldwide. Globally the prevalence of stroke has increased in the young and middle aged adults. The incidence and prevalence of stroke in 2013 was more in men than women.(19) According to the India stroke factsheet updated in 2012, the estimated age-adjusted prevalence rate for stroke ranges between 84-262/100,000 in rural and between 334- 424/100,000 in urban areas. (2)

RISK FACTORS OF STROKE: (20)

Non-modifiable risk factors:

1) Age: The Incidence of stroke doubles for each decade after 55 years.(21)
2) Sex: Premenopausal women have less risk of stroke compared to age matched men.(22)
3) Genetic factors: Parental and family history increases the risk of stroke.

Modifiable risk factors:(3)

The modifiable risk factors are very important. Intervention strategies are made to prevent or treat these factors to reduce the incidence of stroke.

1. Hypertension: It is the single most important risk factor for stroke for both haemorrhagic and ischaemic type.(3)
2. Diabetes Mellitus (DM): In diabetics, there is two-fold increased risk of stroke. The duration of DM also increases the stroke (ischemic) risk by 3% each year and triple after 10 years.(23)
3. Atrial fibrillation and atrial cardiomyopathy: Stasis of blood in a fibrillating left atrium resulting in thrombus formation which can cause embolic stroke. Paroxysmal supraventricular tachycardia (PSVT) also increases the embolic stroke without fibrillation.(24) Autosomal recessive atrial dilated cardiomyopathy is associated with dilatation of atrium and thromboembolic risk. These patients were found to have mutation of natriuretic peptide precursor A gene and severely low levels of ANP.(25)
4. Dyslipidaemia: The use of statin decreases the risk of total ischaemic stroke by reducing LDL and does not increase in haemorrhagic stroke.(26)
5. Sedentary lifestyle, Diet, Nutrition: Physical activity decreases the risk of stroke by reducing blood pressure, blood glucose and body weight. Salt intake increases

the risk of hypertension and stroke. Increased potassium intake and diet rich in fruits and vegetables reduces the risk of stroke.

6. Obesity and metabolic syndrome: Metabolic syndrome comprises of obesity, dyslipidaemia, hypertension, Diabetes. Each components of metabolic syndrome are individual risk factor of stroke. Increased waist-hip ratio increases the stroke risk.(3)
7. Cigarette smoking and alcohol: Cigarette smoking is a major risk factor of stroke.
8. Inflammation: High sensitive C-reactive protein (hsCRP) is a very sensitive marker of inflammation. Several studies showed the modest association between raised hsCRP and ischaemic stroke, coronary artery disease.(27) Atherosclerotic plaque contains macrophages and inflammatory mediators which could be reflected by high level of hsCRP.

Selected genetic causes of stroke:(28)

1. Cerebral autosomal dominant/recessive arteriopathy with subcortical infarcts and leucoencephalopathy (CADASIL/CARASIL): Mutation in NOTCH3 gene.
2. Familial amyloid angiopathy: Leading to rupture of cortical and subcortical vessels.
3. Ehlers-Danlos syndrome, Fabry disease, Marfan syndrome, Mitochondrial encephalopathy.

PATHOPHYSIOLOGY OF STROKE: (29) There are two types of brain injury in stroke patients.

1. Ischaemic: Decreased blood supply deprives the brain tissue from oxygen and nutrition.
2. Haemorrhage: Rupture of blood vessels causing extravasation of blood into the brain. Bleeding compresses the brain tissue and damages the neuronal pathways.

ISCHAEMIC STROKE: Ischaemia can occur due to thrombosis, embolism, and systemic hypoperfusion. (30)

Thrombosis:

It refers to occlusion of blood flow due to clot formation. In atherosclerosis, vascular lumen is encroached by plaque which acts as a nidus for deposition of thrombin, platelets and fibrin. Clot may be formed due to any systemic hyper-coagulable state. Fibromuscular dysplasia, Takayasu arteritis, giant cell arteritis, and dissection of the vessel wall are the less common causes of obstruction of blood flow.(30)

Embolism:

In embolic stroke, material (embolus) dislodges in an artery from its source and occludes the blood flow. Embolus is mostly formed in heart due to valvular defects, prosthetic valve, clots in the atrium due to atrial fibrillation. Clots which formed in systemic veins can cause stroke by travelling through the atrial septal defects or patent foramen ovale, which is called paradoxical embolism. Rarely fat (due to long bone fracture), air (decompression), particulate matter from injectable medicines, tumour cells can embolize to cerebral arteries. (30)

Systemic Hypoperfusion:

Most common cause of systemic hypoperfusion is heart failure (due to myocardial infarction or arrhythmia) and systemic hypotension (due to hypovolemic shock). It affects the brain diffusely in the terminal zones of major blood vessels resulting in watershed infarct.(30)

Effect of ischaemia to brain:

Ischaemia leads to depletion of energy production (ATP) due to lack of glucose. It causes dysfunction of membrane pump (Na/K ATP-ase pump) resulting in cytotoxic edema by accumulation of sodium and water inside the cell. Reactive Oxygen Species (ROS) are also produced which damage the vascular endothelium most. (31)

Ischaemic Penumbra:

Brain tissue which undergoes ischaemia has two layers: ischaemic core with very poor blood flow (10-25%) causing necrosis and outer layer (penumbra) of less critical hypoperfusion, supplied by collaterals. Tissue in the penumbra can be retrieved by intervention. (32)

Cerebral Oedema:

Cerebral oedema is of two types: a. Cytotoxic and b. Vasogenic.

Cytotoxic oedema occurs within minutes to hours and is reversible. There is swelling of neurones, endothelial cells due to failure of membrane pump system. Vasogenic oedema evolves within hours to days and is irreversible. There is increased vascular permeability of serum protein (albumin) leading to increase in extracellular fluid volume. Vasogenic oedema may lead to raised intracranial tension and midline shift. (33,34)

HAEMORRHAGIC STROKE: (30) It can be divided into two types.

Intracerebral haemorrhage:

The causes are hypertension, bleeding diatheses, trauma, amyloid angiopathy, illicit drugs (amphetamine, cocaine), anti-coagulant overdose. Most common site of hypertensive bleeding is putamen.

Subarachnoid haemorrhage:

Bleeding occurs due to rupture of aneurysm or arterio-venous malformation. Aneurysm most commonly is seen in the junction between anterior cerebral artery and anterior communicating artery.

CEREBRAL CIRCULATION: (35)

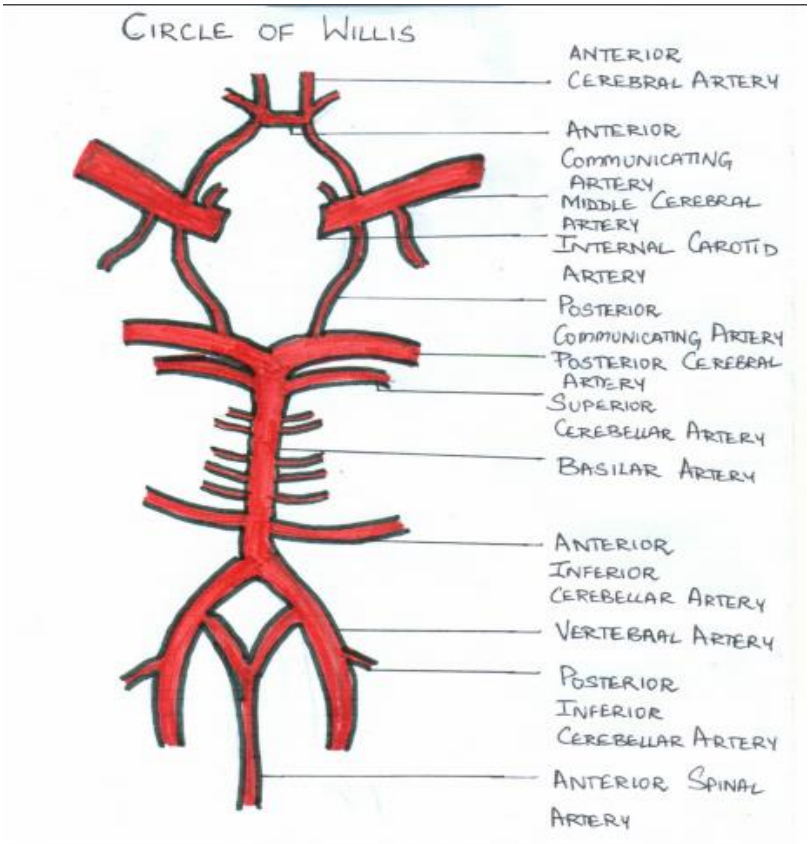
Arterial supply:

Cerebral circulation is divided into anterior and posterior circulation. Internal carotid artery supplies the anterior part of brain and vertebral artery supplies the brainstem and posterior part of brain.

Anterior Circulation:

Common carotid artery bifurcates in the upper border of thyroid cartilage into internal and external carotid artery. Internal carotid artery (ICA) enters the skull through carotid canal. Ophthalmic artery is the first branch of ICA. ICA gives rise to anterior choroidal and posterior communicating artery after penetrating the duramater. Anterior choroidal artery runs the area which lies between anterior and posterior circulation. Anterior cerebral artery and middle cerebral artery are the terminal branches of ICA.

Figure 1: Circle of Willis



Anterior Cerebral Artery(ACA):

ACA is the smaller of the two terminal branches of internal carotid artery. ACA supplies the medial surface of the cerebrum and the upper border of the parietal and frontal lobes. ACA is linked to the opposite ACA by anterior communicating artery anterior to optic chiasma.

Cortical branches of ACA:

- Orbital / Orbitofrontal artery
- Frontopolar artery
- Callosomarginal artery
- Pericallosal artery

Recurrent artery of Heubner: This is the largest of the deep branches of ACA. It supplies lower part of head of caudate nucleus, lower part of frontal pole of putamen, anterior limb (frontal pole) of internal capsule.

Middle Cerebral Artery (MCA):

The largest branch of the internal carotid artery is MCA. Lenticulostriate branches arise from the horizontal segment of MCA and supply the putamen except its anterior part, upper part of head of caudate nucleus and entire body of caudate nucleus, lateral part of globus pallidus, internal capsule (posterior part of anterior limb, genu and anterior third of posterior limb). Lenticulostriate branches are vulnerable for hypertension induced fibrinoid necrosis.

Cortical branches of MCA:

- Anterior temporal artery
- Orbitofrontal artery
- Perirolandic artery
- Rolandic artery

- Anterior parietal artery

- Posterior temporal artery

- Posterior parietal artery

- Angular artery (MCA terminates as angular artery)

Figure 2: Blood supply of the cerebral cortex, Lateral surface (36)

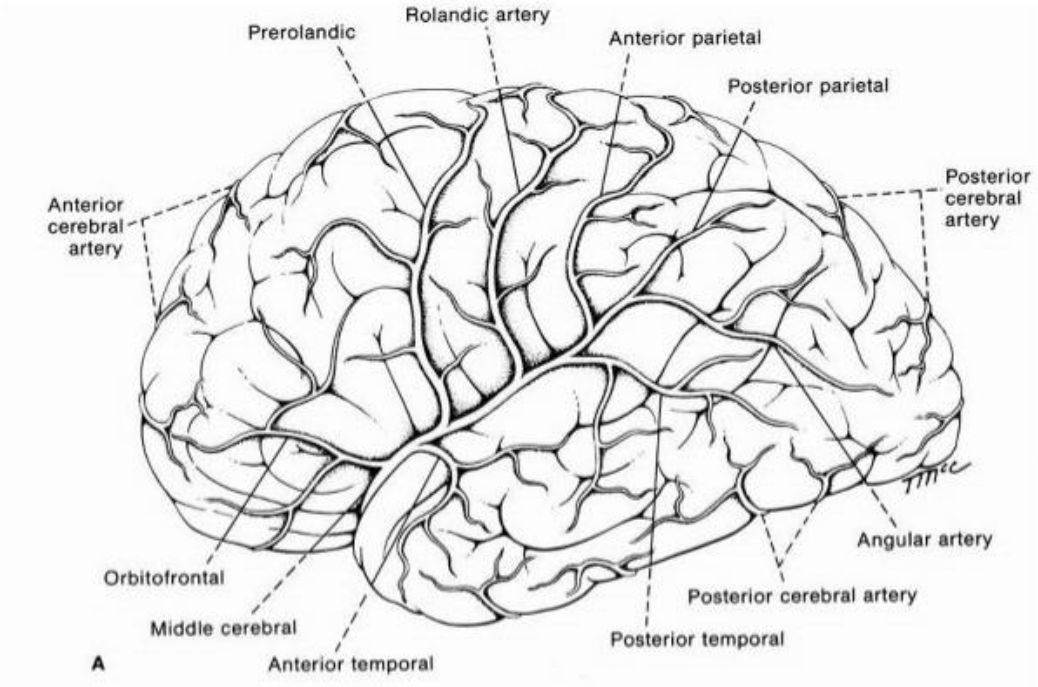
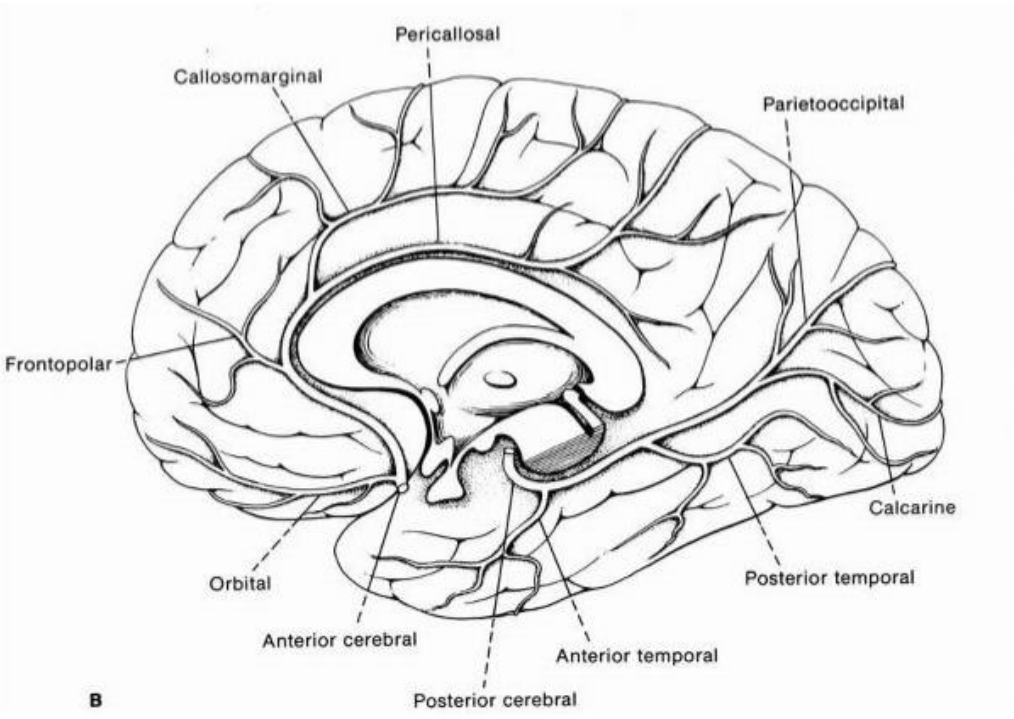


Figure 3: Blood supply of the cerebral cortex, Medial surface (36)



Posterior Circulation:

It is formed by vertebral and basilar artery.

Vertebral artery arises from subclavian artery and joins together at ponto-medullary junction to form basilar artery. Vertebral artery gives rise to anterior, posterior spinal artery and posterior inferior cerebral artery (PICA) which supplies the cerebellum.

Basilar artery supplies the pons by pontine branches, cerebellum and divides into posterior cerebral arteries (PCA).

Posterior Cerebral Artery (PCA):

Posterior cerebral arteries are formed with the bifurcation of basilar artery. PCA supplies midbrain, thalamus, and occipital lobe.

Branches of posterior cerebral arteries (PCA):

- Anterior temporal artery
- Posterior temporal artery
- Calcarine artery
- Parieto-occipital / Posterior occipital artery

Venous Drainage: (37)

a. Cerebral Veins:

Cerebral veins can be divided into two groups, external or superficial veins and internal or deep or central group.

External / Superficial Veins:

1. Superior Cerebral Vein: It drains the medial, lateral and superior surface of the hemisphere above the lateral sulcus. They are 8-12 in number and terminate in superior sagittal sinus.
2. Inferior Cerebral Vein: It drains the basal surfaces of the hemisphere and lower part of lateral surface. It terminates in superior sagittal sinus.

3. Middle Cerebral Vein: It drains the insula and opercular region. It terminates in either cavernous sinus or sphenoparietal sinus. It is linked with the superior sagittal sinus and transverse sinus by the great anastomotic vein of Troland and anastomotic vein of Labbe respectively.

Deep Cerebral Veins (Central): The great cerebral vein of Galen is formed by union of two internal cerebral veins. It drains into straight sinus.

b. Venous Sinuses:

Venous sinuses are located between the meningeal and parietal layers of duramater. Superior, inferior and straight sinuses are found in falx cerebri of the duramater. They come together at the confluence of sinuses (Trocula Herophili). From the confluence, transverse sinus continues bilaterally as sigmoid sinus and later as internal jugular vein. Straight sinus is formed by the union of inferior sagittal sinus and great cerebral vein of Galen. Cavernous sinus is located on lateral side of pituitary gland (sella). Superior petrosal and inferior petrosal sinus connects the cavernous sinus with transverse sinus and internal jugular vein respectively.

Figure 4:Venous drainage of cerebral cortex: Lateral surface (36)

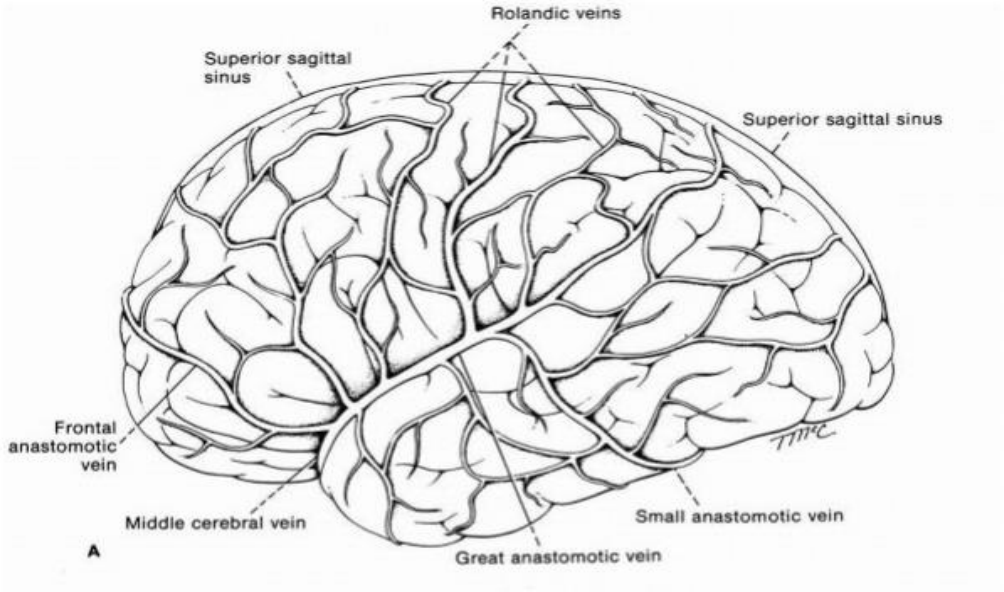
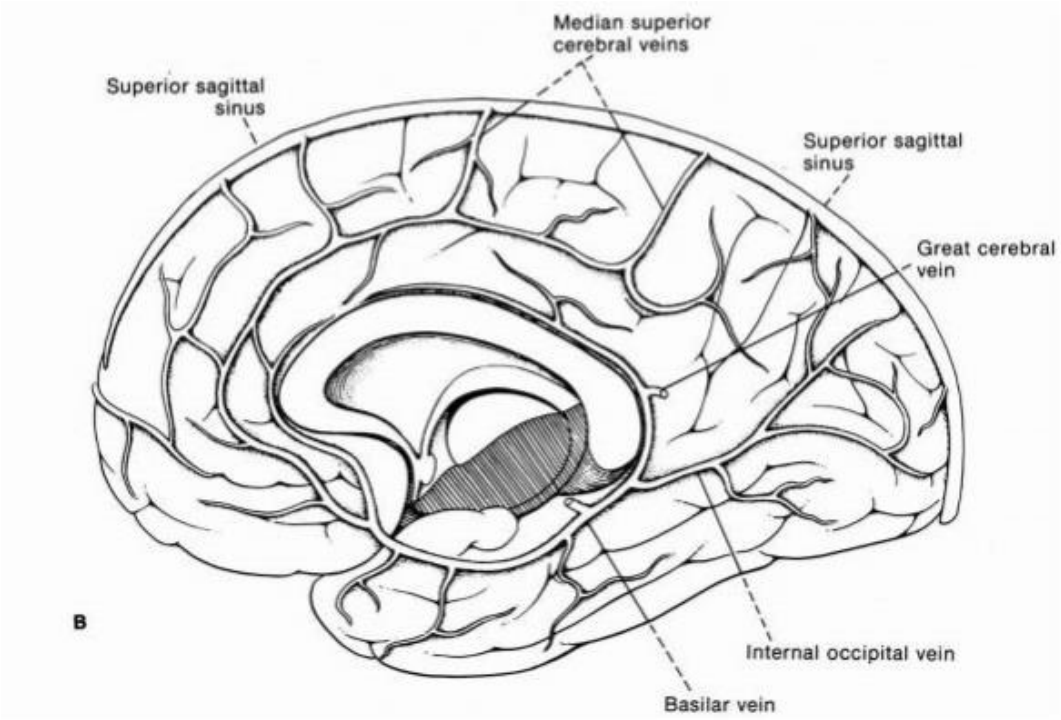


Figure 5:Venous drainage of cerebral cortex: Lateral surface (36)



LOCALISATION PATTERNS: (38)

Neurological signs and symptoms can help to detect the location of brain lesion.

1. Left hemisphere lesion: It can produce aphasia, right hemiparesis, right sided hemianaesthesia, right visual field defect, alexia, agraphia, acalculia.
2. Right hemisphere lesion: It can produce left visual neglect, left visual field defect, left hemiparesis, left sided sensory loss, difficulty in copying, drawing.
3. Left PCA lesion: Right hemianopia, alexia without agraphia, inability naming colours, objects presented visually, intact repetition, sensory loss of right side.
4. Right PCA lesions: Left limb numbness, left visual field defect occasionally with neglect.
5. Vertebrobasilar territory lesion: It will cause giddiness, diplopia, ataxia, vomiting, occipital headache, weakness or numbness of all four limbs, crossed hemiplegia/sensory loss.
6. Pure motor stroke: Hemiparesis with intact cortical function, sensory and visual function. The lesion usually located in internal capsule or basis pontis.
7. Pure sensory stroke: Numbness on one side of body with intact cortical, motor, visual function. The lesion is located in thalamus.

INVESTIGATIONS: (39)

1. Computed Tomography:

   CT scan can rapidly diagnose haemorrhagic stroke which appears hyperdense on CT scan. CT brain may show hypodense lesion or may remain normal in acute infarction. Acute brain ischemia can be identified by the following signs on CT scan: loss of gray and white differentiation, hypodensity, hyperdense artery may indicate thrombosis.

2. Magnetic resonance imaging (MRI):

   MRI is more sensitive to detect ischaemic changes in brain. Infarct appears hyper intense (bright) on Diffusion weighted imaging (DWI) and hypo intense (dark) on apparent diffusion co-efficient (ADC). DWI can detect ischaemia within first hour of stroke which could be reversible. Infarct appears bright on T2 image are irreversible. T1 weighted images showed hypo intense lesion.

   MRI can also readily diagnose intracranial haemorrhage. MRI signals changes vary depending on evolution of haemorrhage. Within first 12 hours of ICH, Oxyhaemoglobin is formed which is not paramagnetic. It appears isointense/hypointense (dark) on T1 weighted image and bright (due to water content) on T2 weighted image. Chronic haemorrhage appears dark on T1 and T2 weighted MRI.

3. Magnetic Resonance Angiography (MRA):

   MRA creates an image of blood flow in vessels. It is a functional imaging and does not delineate vascular anatomy like standard angiogram. If blood flow is reduced the vessels will appear narrowed or absent on MRA. In those cases contrast MRI is required for better image of arterial circulation. MRA is an excellent screening tool for occlusive diseases.

4. Computed Tomography Angiogram (CTA):

   It is a three dimensional computerised picture of blood vessels. Spiral CT scanning was done after injecting a bolus of dye. CTA has advantage over MRA as it is based on anatomic imaging even when blood flow is reduced. CTA has advantage over DSA in detecting steno-occlusive disease of posterior circulation. (40)

5. Lumbar puncture:

   It can help us to diagnose subarachnoid haemorrhage specially few days after bleeding when CT/MRI are not sensitive to detect SAH.

6. Transcranial Doppler (TCD):

   TCD helps to delineate intracranial arteries. 2MHz ultrasound probe is used. Probe is placed in three positions: orbital window which shows flow along the

ACA, temporal window which shows MCA, proximal PCA, ICA bifurcation and suboccipital window which shows vertebrobasilar system. TCD can detect atherostenotic intracerebral arteries and haemodynamic effect of extracranial obstruction on intracranial vessels. TCD can also detect embolus in cerebral circulation with sudden change in blood flow and high intensity transient signals.

7. Cardiac evaluation:

   Cardiac evaluation is required to rule out embolic stroke. ECG, Transthoracic ECHO, Transoesophageal ECHO, Holter monitoring are the investigations to look for arrhythmia, source of embolus.

<u>Indications of cardiac evaluation for CVA</u>:

a. Diagnosed to have heart disease
b. History of embolism in systemic vessels in limbs.
c. Young age with no risk factors of atherosclerosis and imaging is normal.
d. CT/MRI shows infarct in more than one vascular territories
e. History suggestive of embolism- sudden onset neurodeficits, maximum at onset, while active, no past history of TIAs.
f. History of embolism in systemic vessels in limbs.
g. Young age with no risk factors of atherosclerosis and imaging is normal.
h. CT/MRI shows infarct in more than one vascular territories

i. Normal CTA, MRA in a patient while neurodeficits and brain imaging are not matching

j. Haemorrhagic transformation of a cerebral infarct in one vascular territory.

8. MR spectroscopy:

Elevated lactate, decreased N-acetyl aspartate, creatine and choline are typical of MRS spectrum in the region of infarction. Lactate is a marker of anaerobic metabolism, therefore elevated in necrotic areas and infection. NAA is marker of neuronal viability. It is reduced in any process that destroys neurones.

Evaluation of haemorrhagic stroke:

Most common cause of ICH is hypertension. If imaging shows bleeding in atypical location for hypertensive bleed then investigations should be done to look for AVM or aneurysm.

Evaluation of ischaemic stroke:

Blood investigations should be carried out to rule out hypercoagulable state. The following blood tests should be done: Haemoglobin, haematocrit, WBC count, Platelets, PT, APTT, Serum Fibrinogen, Antiphospholipid antibodies, blood sugar, ANCA, protein C, protein S, serum calcium, homocysteine, CRP, ESR.

POST STROKE COMPLICATIONS: (41)

Post stroke complications are very common. Most of the complications are medical and not neurologic. Complications may occur during acute condition or may develop during rehabilitation phase.

Brain oedema:

Brain oedema becomes clinically obvious within 1-4 hours following stroke. Rooper and Shafran described the features and raised intracranial pressure due to increased brain oedema. The main symptom was drowsiness which was accompanied with one or more of the followings: pupillary asymmetry (0.5-2 mm), periodic breathing, sixth nerve impairment, extensor plantar on the normal side, papilledema, bilateral extensor posturing. Intracranial pressure persistently more than 15 mm of Hg carries poor prognosis. (42)

Seizures:

Patient with intracerebral bleeds have seizures more than infarcts. Bleed in cerebral cortex has a higher chance of seizure than subcortical lesions. Patients with embolic infarct due to cardiac origin are at more risk of having seizure than large artery thrombosis. (43) Early onset seizures are focal seizure with secondary generalisation. Late onset seizures are mostly generalised type.(44) Poststroke seizures are easily managed with single anticonvulsant (Carbamazepine or Phenytoin)(45) .

Medical complications: (41)

1. Deep vein thrombosis and Pulmonary embolism:

   Pulmonary embolism is most fatal complications following stroke. Majority of patient have deep vein thrombosis in paretic limb. Prophylaxis with low molecular weight heparin leads to significant reduction of DVT.(46) Pulmonary embolism should be suspected if patient develops breathlessness, chest pain, hypotension, hypoxia, altered breathing pattern, agitation, confusion. Investigations should be done on the degree of suspicion: arterial blood gas, chest x-ray, ECG, Pulmonary CT angiography.

2. Cardiac complications:

   Mortality related to cardiac abnormalities is second most common cause of death in acute stroke patients next to neurological complications. Elevated cardiac enzymes (creatine-phospokinase, troponin) and cardiac arrhythmia are commonly found in acute stroke survivors. (47) Myocardial infarction is common in stroke patients with past history of heart disease. (48)

The mechanism of secondary cardiac dysfunction in stroke patients are: (49)

a. Direct injury in structures like insular cortex, hypothalamus, brainstem nuclei- causes autonomic dysfunction.

b. Activation of hypothalamo-pituitary axis stimulates release of catecholamines and corticosteroids.
c. Mass effect causing compression of hypothalamus and brainstem.
d. Brainstem stroke (medullary involvement) can cause vagal discharge resulting in sinus bradycardia, arrhythmia, fall in diastolic blood pressure and elevation of systolic blood pressure. These changes are called as Cushing response.

3. Swallowing abnormalities and Pneumonia:

Dysphagia and aspiration are common complications following stroke. Dysphagia is mostly seen brainstem stroke and bi-hemispheric lesion. Pneumonia is seen in both acute and late periods. The common causes are older age, decreased alertness, difficulty to speak and, severe focal or global neurodeficits. (50) Nasogastric feeding does not seem to be protective against aspiration. (51)

4. Metabolic and nutritional disorder:

Prolonged undernutrition is very common among stroke survivors. It can be managed with multivitamin supplements, NG feeding or percutaneous endoscopic gastrostomy (PEG) tube feeding. (52) 15% stroke patients develop hyponatremia mostly due to SIADH.

5. Urinary tract infection (UTI): UTI is very common complications. Most common causes of UTI are-(53)

-Indwelling Foley catheter

-Alteration of behaviour of bladder wall and external sphincter dysfunction

6. Complications due to immobility:

-Pressure ulcer

-Contracture, shoulder pain

- Nerve injury

- Osteoporosis, osteopenia

-Fatigue, depression, insomnia

GAIT: The study of human walking

Task of gait:(54)

1. To maintain support of trunk, arms, head.
2. Maintain erect posture and balance of the body
3. Safe ground clearance and smooth heel or toe landing
4. To conserve energy during forward propulsion of body
5. Shock absorption and stability or reduce the forward velocity.

Phases of gait cycle: (55)

A gait cycle consists of two successive events of the same limb. Each gait cycle is divided into two phases: a stance phase, when a part of the foot is on the ground (60% of gait cycle) and a swing phase, when foot is in the air (remaining 40% of gait cycle). There are two events of double limb support in a gait cycle and it makes up 22% for a gait cycle. Hence body is supported by one limb approximately 80% of gait cycle.

Events in a gait cycle: (55)

A. Weight acceptance**:**

1. Initial contact: It refers to the instant foot touches the ground. The limb prepares to commence stance with a heel rocker.
2. Loading response: The phase starts with initial contact and ends until the contralateral foot is lifted. This is the initial double limb support.

B. Single limb support:

3. Mid stance: It starts when contralateral foot is lifted for swing and persists until body weight is transferred over the forefoot. This is the initial phase of single limb support.
4. Terminal stance: This phase starts with heel off and continues until the other foot touches the floor. It is the end of single limb support.

C. Limb Advancement:

5. Pre-Swing: It begins with initial contact of contralateral limb and ends with ipsilateral toe-off.
6. Initial swing: This first phase consists of one-third of swing period. It starts when the swinging foot lifts the ground and continues till it comes opposite the contralateral foot (stance foot).
7. Mid Swing: This phase begins as the swinging limb is directly beneath the body. It ends when the swinging limb crosses the stance limb and tibia is vertical.
8. Terminal swing: This is the final phase of gait cycle. It begins with vertical tibia and ends with initial contact.

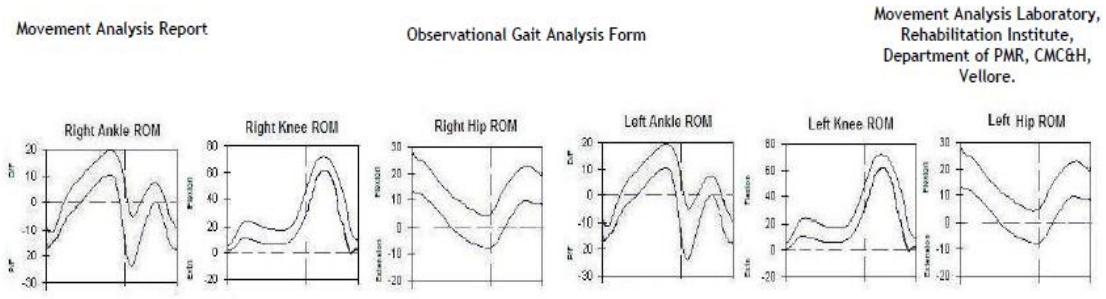
GAIT TERMINOLOGY: (54)

- Stance time: Time required during the stance phase of one limb in a gait cycle.
- Single limb support time: It is the time period when one limb is on the floor of a gait cycle.
- Double limb support time: It is the time that elapses when both the limbs are on the ground of a gait cycle. The percentage of double limb support increases in those with balance issues and decreases as the walking speed increases.
- Stride length: It is the linear distance between two consecutive events done by same limb during gait. It is the interval between two consecutive initial contacts by same lower extremity. Stride length includes two steps, a right step and a left step.
- Step length: It is the linear distance between two consecutive points of contact of opposite limbs. Gait symmetry is determined by comparing right and left step lengths.
- Cadence: The number of steps accomplished by a person per unit of time (per second, per minute). Shorter step length will increase cadence at a particular velocity. A typical cadence for men is 110steps /minute and female is 116steps /minute.
- Step Width: It is the distance between midpoint of the heel between two feet. Step width increases in balance problems.

- Kinematics: It describes the movements and does not consider any internal or external forces.
- Kinetics: It deals with forces acting on body causing the movement.

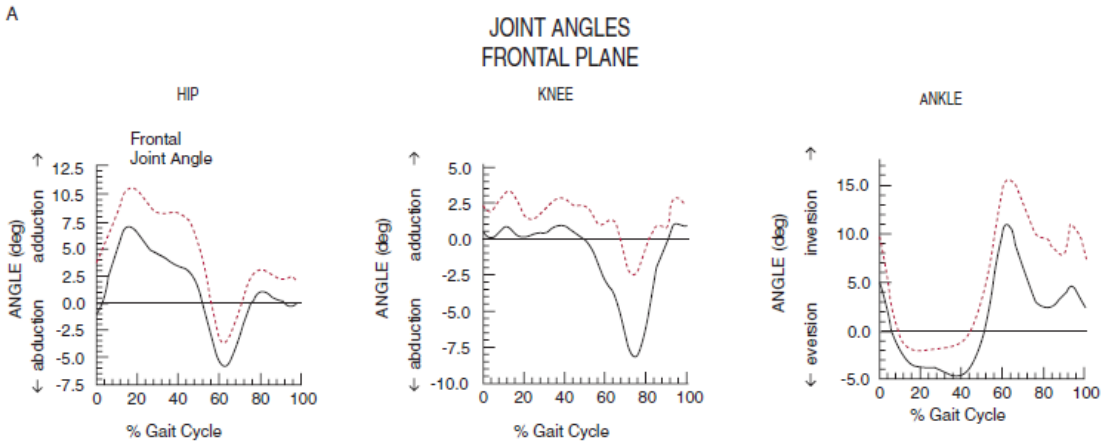
SAGITTAL PLANE JOINT ANGLES:(56)

Figure 6: Sagittal plane joint angles



FRONTAL PLANE JOINT ANGLES:(57)

Figure 7: Frontal plane joint angles



DETERMINANTS OF GAIT: (58)

These factors minimize the excursion of centre of gravity (COG) in both horizontal and vertical plane and reduce energy consumption while walking. The six determinants of gait are:

1. Pelvic rotation: Forward rotation of pelvis on the swinging leg side in the horizontal plane enables slightly longer step length and prevent sudden drop of the COG. During the swing phase, medial rotation of 5 degree at the stationary hip (stance phase) advances the swinging hip.

2. Pelvic tilt: The pelvis sags by 4-5 degree on the swinging side. The magnitude of pelvic tilt is controlled by the hip abductor of stance side. Pelvic tilt results knee flexion during swing to clear the ground.
3. Knee flexion in stance phase: The knee is in extension at the initial contact, and after that begins to flex. It is approximately 15-20 degree and occurs at mid-stance. The bending of knee reduces hip-to-ankle distance in mid stance. This lowers the COG.
4. Foot mechanism: Ankle plantar-flexion at initial contact lowers the trajectory of the COG.
5. Knee mechanism: After mid stance, there is extension of knee as the ankle plantar flexes.
6. Lateral displacement of the pelvis: During stance phase there is displacement of pelvis toward the stance limb to maintain balance. This brings the COG closer to the stance leg, making it easier for the hip abductors to lift the swing limb and prevent pelvic tilt. This factor reduces displacement on the horizontal plane.

CHARACTERISTICS OF GAIT IN HEMIPLEGIA:

Hemiplegia is one of the most common impairments following stroke which significantly affects the normal gait pattern. Post stroke hemiplegic gait is mixture of kinematic deviation from normal gait and adaptation. There are several patterns of gait deviations found in stroke patients. These are drop foot, equinovarus, stiff-knee gait and genu recurvatum.

Spatio-temporal factors of walking of the hemiplegic patient:

1. Hemiplegic patients have decreased stride and step length compared to normal, wide based gait, greater toe-out angles.(59,60)
2. Patients with hemiplegia have decreased walking speed, reduced cadence and increased stride times.(61,62)
3. Altered stance swing ratio has been reported in hemiplegic patients. Non paretic side shows increased stance duration and a reduced period of swing.(59–61)
4. Severity of motor impairment is the prime factor affecting stride length and walking velocity. Single limb support, total support, and step duration are indicators of severity of motor dysfunction.(63)

5. According to Holden et al. the degree of physical assistance for functional walking depends on walking velocity, stride length, step length, cadence, and the ratio of stride length to length of lower extremity.(64)
6. Most rapid motor recovery was noticed over first 6 weeks to 3 months, and slow improvement being seen till 1 year after stroke.(60,65)

Gait deviation during stance phase:

1. Decreased hip extension in late stance phase: (8,8,66) During normal gait hip extends from 16 degrees of flexion at initial contact to 11 degrees of extension. Peak hip extension occurs during late stance phase. Hip extension helps to move trunk segment forward over stance foot. The effect is decreased in contralateral step length.(9)

Causes:

Hip extensor weakness, compensatory shortening or excessive activity of hip flexors, increased plantar flexor moment by excessive tension or shortening of ankle plantar flexor muscles.(9)

2. Decreased peak lateral pelvic displacement: Lateral displacement of pelvis is accomplished by ipsi-lateral concentric hip adductor activity and contralateral eccentric hip abductor activity. (67) This deviation is compensated by rapid side flexing of trunk toward stance side.

Causes:

Insufficient active tension by the hip abductors and adductors in early stance phase.

3. Increased peak lateral pelvic displacement:

Causes:

Shortening or excessive tension in hip adductors, Insufficiency of hip abductor muscles

4. Knee hyperextension (decreased knee flexion) in stance phase: It is very commonly observed gait abnormality.(8,9)

Causes:

i. Knee hyperextension is the compensatory mechanism to achieve a stable limb for weight bearing. As the knee goes into extension beyond a neutral position, trunk goes forward due to hip flexion to achieve sable support on paretic limb. So the combined effect of hip flexion and knee hyperextension cause the centre of mass of trunk to move anterior to knee, resulting in large weight moment which extends the knee.(66,68)

ii. Excessive plantar flexor moment (due to early calf muscle activity or adaptive shortening of plantar flexor muscles) prevents forward rolling of tibia by impeding ankle rocker leading to knee hyperextension. Hence, the ground reaction force (GRF) passes anterior to knee leading to instability.(69)

iii. Excessive knee extensor moment throughout the stance phase may cause knee hyperextension.(66)

5. Increased knee flexion in stance phase: This type of gait deviation is commonly seen in hemiplegic patients. Weakness of knee extensors is one of the causes of excessive knee flexion.(8) In mid stance phase eccentric contraction of plantar flexor inhibit forward rotation of leg and keeps the body's centre of mass inside the support.(70) Decreased eccentric contraction of ankle plantar flexor can cause knee flexion in mid stance.(66)
6. Reduced ankle plantar flexion at toe-off: Ankle goes into rapid plantar flexion from about 9 degrees of dorsiflexion to 18 degrees of plantar flexion.(70) Hemiplegic patients have difficulty to activate ankle plantar flexors during pre-swing phase.(8) Sometimes if the body's centre of mass is not anterior to ankle, planter flexor moment in late stance phase may result in posterior displacement of body. These patients are not able to contract the ankle plantar flexor in toe off phase.(66)

Gait deviation during swing phase:

Normally, the important events occurring in swing phase is hip flexion, knee flexion followed by extension and ankle dorsiflexion. The gait deviation occurs as a result of motor dysfunction or as a compensatory strategy for the motor problem.

1. Decreased peak hip flexion: Hip reaches its maximum flexion of about 19 degrees by mid swing. This flexor muscle moment in swing phase is caused by a

concentric contraction of the rectus femoris and iliopsoas muscle.(70,71) The amount of hip extension in terminal stance also determines the kinematics of swinging leg.(72) Hip flexor insufficiency in pre swing phase and decreased hip extension in stance phase are the potential causes of decreased peak hip flexion which rsults to a decrease in step length. Some people incline the trunk and pelvis backward in late swing phase which moves the swing foot in front of body and step length increases.(73)

2. Decreased peak knee flexion during initial swing phase:

Causes:

a. Knee flexors are not able to generate sufficient tension in pre-swing.

b. Excessive contraction of the knee extensor in pre swing

c. Adaptive Tendoachiles shortening or excessive tension in the plantar flexor during pre-swing

d. Reduced hip extension in terminal stance phase

Hemiplegic patients, with decreased knee flexion compensates by shortening the lower limb. They tend to raise the pelvis on the swinging side and occasionally circumducts the swinging leg.(8,9)

3. Decreased knee extension in terminal swing phase: (8,9,74)

Causes:

a. Decreased contraction of knee extensor in early swing

b. Excess tension with hamstring and gastrocnemius in swing phase

c. Adaptive shortening of gastrocnemius

d. Reduced peak extension of hip in terminal stance

Hemiplegic patients, with decreased knee extension in heel strike results in decrease in step length. They commonly compensate by increasing cadence.

4. Decreased ankle dorsiflexion: It is very commonly reported gait deviation in hemiplegia due to stroke.(8,9,75)

Causes:

a. Lack of sufficient dorsiflexor muscle moment

b. Adaptive shortening of tenoachiles or excessive contraction of plantarflexors. Lower limb is effectively lengthened as ankle fails to dorsiflex during swing phase. It is compensated by raising the pelvis on the affected side, abducting the swinging hip, and laterally flexing the trunk to non-paretic side.

Energy expenditure of walking in hemiplegics:(74)

Hemiplegic patients spend 50% to 67% more mechanical energy compare to normal individuals at the same walking velocity. The total energy pattern was determined by head, arms, and trunk (HAT). Olney et al reported that total energy conservation in stroke patients was low (22-66%) due to three major types of disturbances in the head, arms, and trunk.(74)

1. Lack or little exchange of potential and kinetic energy.
2. Low amount of kinetic energy resulting in minimal energy exchange. This problem can be addressed by increase the walking speed.

3. Single rise and fall of potential energy curve of swinging limb due to hip hiking. It can be managed by reducing hip hiking.

Electromyography:

The magnitude and phasic contraction of the lower limb muscles in stroke patients differ significantly from normal individual. They are differences between paretic and non- paretic limb as well as inter-individual variation. Knutsson and Richards classified the EMG pattern in hemiplegic patients into three types.(8)

1. Type I pattern (mild gait disturbances) - Phasic EMG pattern in tibialis anterior and gastrocnemius. Premature activation of calf muscle was noted in stance phase.
2. Type 2 pattern– EMG patterns of two or more muscle groups (affected limb) were significantly low or absent.
3. Type 3 pattern– EMG pattern showed co-activation of different muscles in a disorganised fashion.

Waters et al observed EMG activity in 27 hemiplegic patients. Premature and Phasic contraction was noted in gastrocnemius, soleus during terminal stance and early swing. The tibialis anterior showed continuous activity in 59.3% patients.(76)

Several studies found abnormal EMG activity in the unaffected limb. Carlsoo et al reported excess period of contraction in the pretibial, gastrocnemius, quadriceps, and hamstrings of the normal limb.

REHABILITATION OF GAIT PATTERN FOLLOWING STROKE:

Majority of stroke patients start walking with some aid although many do not able to achieve walking level to carry out their daily activities.(77) Gait recovery is a one of the primary goals during rehabilitation.

ANKLE FOOT ORTHOSIS:

An orthosis is an externally applied device which is used to alter the structural and functional characteristics of musculoskeletal system. An Ankle-foot-orthosis encloses the ankle joint and entire or part of the foot.(78)

Prefabricated AFO:

These prefabricated plastic AFOs are of limited use. They are used for early mobilization until custom made orthosis is available. Most common type of prefabricated AFO is Posterior leaf spring AFO.

Specific Indication of prescribing leaf spring AFO:

-Isolated ankle dorsiflexor weakness

-No significant spasticity

-No significant joint instability

-Orthosis which does not require any effect on hip or knee.

So, these prefabricated AFOs are not always suitable in stroke patients who have spasticity, knee recurvatum, varus deformity of foot.

Fabricated (Custom-made) AFO:

Custom-made AFOs are used for the management of complex gait abnormalities. These AFOs are very effective in controlling ankle triplanar deformity.

IMPACT OF AFO IN HEMIPARETIC GAIT:

AFOs are prescribed to improve gait pattern of hemiplegic patients with residual weakness and spasticity following stroke.(79–81) Gait training with AFO was found to have increases in functional independent measure score (FIM) at discharge.(82)

The basic goals of prescribing AFO are:(9)

1. To provide medio-lateral stability during stance phase
2. To achieve sufficient toe clearance during swing phase
3. To reduce energy expenditure

In normal gait, toe clearance is achieved by functional limb shortening. The degree of limb shortening is decided by the amount of flexion of knee.(83) Hemiplegic patients have weakened lower limb function which requires compensatory mechanism like hip hiking, circumduction of the affected leg during swing phase.(84) AFO with its mechanical property limits ankle plantarflexion and achieve limb shortening. Hip hiking is reduced as a result of limb shortening due to wearing AFO.(79)

Cruz et al. reported that AFO decreases the pelvic obliquity which is a compensatory mechanism of ankle dorsiflexor weakness.(85) AFO with a 5 degree of dorsiflexion significantly increases the gait speed by increasing duration of heel-strike phase compared to without wearing AFO. AFO with 5 degree of ankle plantar flexion increases the duration of push off phase.(9) A systematic review by Tyson and Kent showed using an AFO can improve walking speed, step length, stride length and balance. There was no positive effect on Timed up and go test (TUG) and postural sway.(86) There are few disadvantages of AFO such as restricted ankle mobility that may lead to

development of contracture, difficulty to get up from a chair, reduced cosmesis and discomfort in donning and doffing.(10–12)

FUNCTIONAL ELECTRICAL STIMULATION:

Electrical stimulation for the treatment of disease is mainly classified as functional and therapeutic. Therapeutic electrical stimulation ameliorates the health by inducing physiological alteration which persists even after the stimulation is stopped. When electrical current is applied to activate a paralysed muscle to supplement or achieve the lost function, it is called as functional electrical stimulation. In FES, to gain the desired function stimulation must be 'on'. A neuroprosthesis utilizes neuromuscular electrical

stimulation to stimulate specific muscles in a precise sequence to move the limb to carry out functional tasks. (87)

Lower limb application of FES in Stroke:

Functional electrical stimulation (FES) devices are currently available for the management of foot drop. The concept of using electrical stimulation to the common peroneal nerve to activate the tibialis anterior in the swing phase of gait

was first proposed by Liberson et al. (13) Common peroneal nerve supplies the ankle dorsiflexors and evertors. Electrical stimulation to dorsiflexors of ankle, by placing electrodes over common peroneal nerve is the most common form of FES. There are three types of peroneal nerve stimulator devices available approved by FDA for the management of foot drop in hemiparesis. These devices are WalkAide System (Innovative neurotonics, Austin, TX), Ness L 300 Foot drop system (Bioness, Inc.) and the Odstock Dropped Foot Stimulator (ODFS). These devices utilize a tilt sensor or a heel switch as a control to stimulation during the swing phase of gait. (88) FES devise can stimulate muscles by single, dual or multi channel stimulation. Single channel stimulator stimulates common peroneal nerve and resulting in contraction of tibialis anterior, peroneus longus and brevis to achieve ankle dorsiflexion and eversion during gait. (89) Correction of foot drop by FES has two types of effects: orthotic and therapeutic. The orthotic effect is defined as the effect that occurs during stimulation and the therapeutic effect is the effect that remains even after the withdrawal of stimulation. (90) Robbins et al suggested from a meta-analysis that FES improves the walking speed in post stroke patients. (91,92) The electrical stimulation also reduces the spasticity, improves energy expenditure and slows muscle atrophy. (14) Peroneal nerve stimulator was not found to be effective than usual care in improving stroke specific quality of life. There was no evidence of motor relearning of lower limb muscle weakness with Peroneal nerve stimulator. (93)

Kottink et al reported positive orthotic effect but no therapeutic effect with FES.(92,94) FES of the dorsiflexors does not improve gait quality of the patients with insufficient knee and hip control. Springer et al conducted a study using dual channel FES over hamsting and dorsiflexors and showed improvement of gait speed which did not depend on initial gait velocity. (95–97) Surface-based FES has a drawback of difficulty in positioning electrodes correctly and skin allergy. To overcome this problem implantable electrodes are also available. Patients with cognitive impairment have difficulty in donning and doffing the device. Skin should be monitored for rash or abrasion regularly among the patients with sensory deficits. (14)

METHODOLOGY

Setting:

The present study was done in Christian Medical College, Vellore, situated in the state of Tamil Nadu, India. It is a tertiary care hospital with 2500 inpatient beds, and average out patient census of about 5000 patients per day. The Department of Physical Medicine and Rehabilitation in CMC, has 123 inpatient beds and an average of 150 outpatients per day. Every year, about 200-300 patients with stroke are admitted here for rehabilitation which includes medical and surgical management of complications arising from stroke.

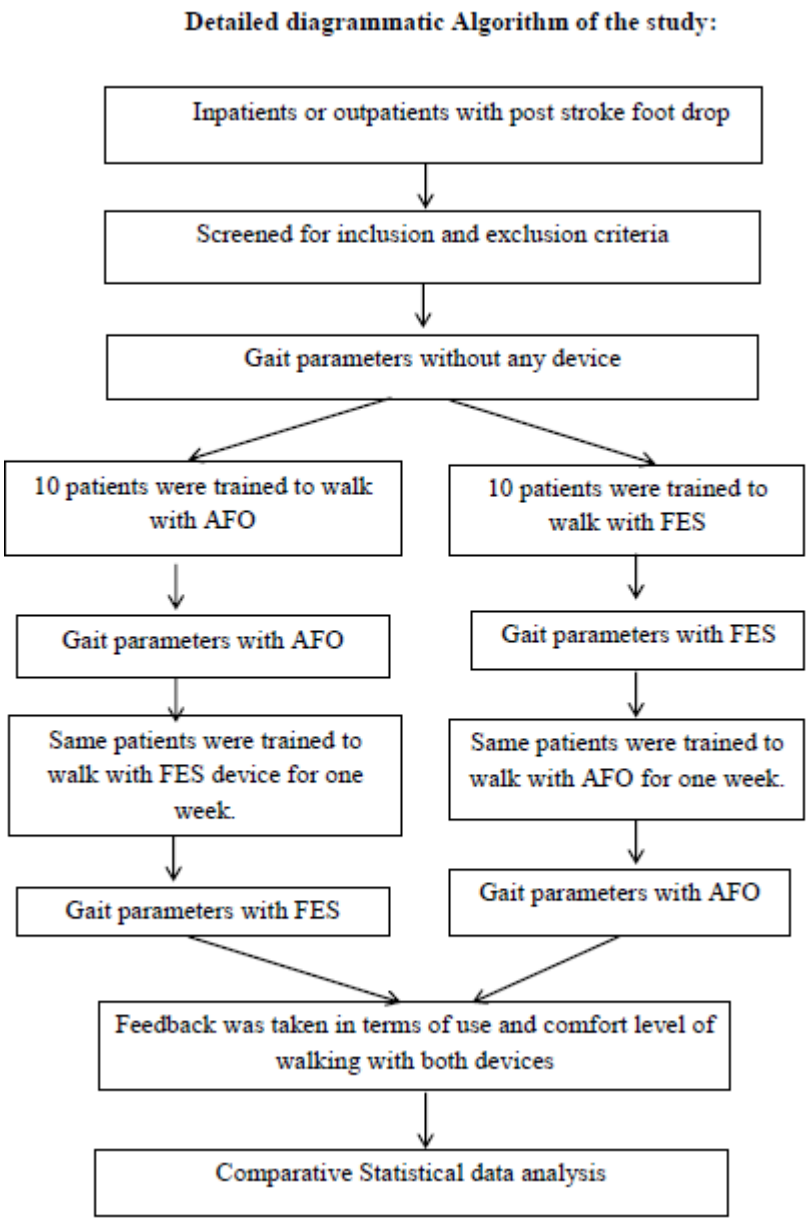
The study

The study was a non-randomized cross over trial to compare the gait in patients following stroke with Ankle-Foot-Orthosis (AFO) and Functional electrical stimulation (FES). The present study was approved by the Institutional Review Board of the Christian Medical College. Twenty patients with history of cerebrovascular accidents, who fulfilled the inclusion and exclusion criteria were enrolled from July 2017 to July 2018 after obtaining informed consent. Patients were recruited from the Stroke clinic, Physical Medicine and Rehabilitation outpatient and inpatient.

Patients were divided in two groups (group A and B) consisting of 10 patients in each group. Patients of group A were trained with Ankle-Foot-Orthosis (AFO) followed by Functional electrical stimulation (FES) and group B patients were trained first with Functional electrical stimulation (FES) followed by Ankle-Foot-Orthosis (AFO). They were divided in two groups to observe whether the order of trial with two devices has an effect on the outcome.

Baseline demographic parameters such as age, sex, type of stroke, risk factors, duration of stroke were collected from the patient and medical records.

Detailed diagrammatic Algorithm of the study:



Participants:

Inclusion criteria:

1. Age 18 years and above
2. More than three months from first clinical CVA
3. Hemiparesis
4. Able to walk 5 meters continuously with minimal assistance
5. Foot drop during ambulation
6. Adequate cognition and communication abilities
7. Ankle dorsiflexor strength of less than 2 on the MRC (Medical research council) scale.
8. Ankle dorsiflexion to at least neutral on electrical stimulation of common peroneal nerve.
9. Medically stable

Exclusion Criteria:

1. Any contraindication for using FES, e.g. epilepsy, pregnancy, Implants like cardiac pacemaker.
2. Local condition preventing wearing FES e.g. deep vein thrombosis of lower limbs, lower extremity ulcers.
3. Ankle contracture, LMN lesions, severe hemineglect.

The following tests were done:

A. Lower limb power according to MRC (Medical research council) grading:

MRC grading of muscle power: (98)

5 - Normal power

4 - Movement against moderate resistance over complete range of motion

3 - Full movement against gravity but not against any resistance

2 - Movement with gravity eliminated and full range of motion

1 - Visible or palpable flicker of contraction

0 - Total paralysis

All the recruited patients had ankle dorsiflexor power of MRC 1 or 0 at the time of recruitment.

B. Gait training with Ankle-Foot-Orthosis:

Ankle-foot-orthosis (AFO) is an orthosis, usually made of plastic or rigid substances, which is worn on the lower leg and foot to enclose the ankle joint and entire or part of the foot. All patients were prescribed polypropylene solid Ankle foot orthosis. They

were trained to walk initially inside the parallel bar and later progressed to walking outside the parallel bar. They were trained to climb stairs wearing an AFO. They had two sessions of therapy everyday each for a duration of two hours. After one week of training they were assessed for outcome parameters.

Figure 8: Ankle foot orthosis

C. Gait training with Functional electrical stimulation (FES):

The principle of FES is electrical stimulation to common peroneal nerve to achieve ankle dorsiflexion during swing phase of gait to correct foot drop. We used the WalkAide device for the study. WalkAide System (Innovative neurotonics, Austin, TX) approved by FDA is a type of peroneal nerve

stimulator. It is a battery-operated, single-channel stimulator used to correct foot drop with functional electrical stimulation by placing surface electrodes over peroneal nerve. This devise is an automatic devise which utilizes a tilt sensor to control stimulation during normal gait. WalkAide was programmed for each patient before gait training with FES. Gait training was done similar to Ankle foot orthosis for one week. Outcome measures were checked after one week of training. Physical and occupational therapy interventions were done based on the baseline functional status of each patient. Activities included lower extremity strengthening exercise, standing balance, passive and active range of motion exercise, weight shift training on paretic limb. Advanced ambulation training such as walking on various surfaces (ramp), stair climbing was done.

Figure 9: FES (WalkAide) stimulator and programmer

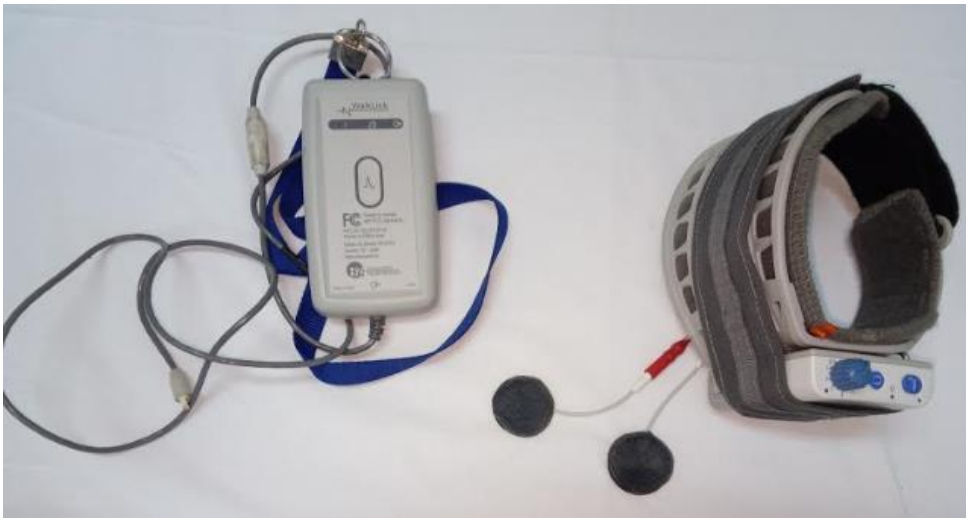
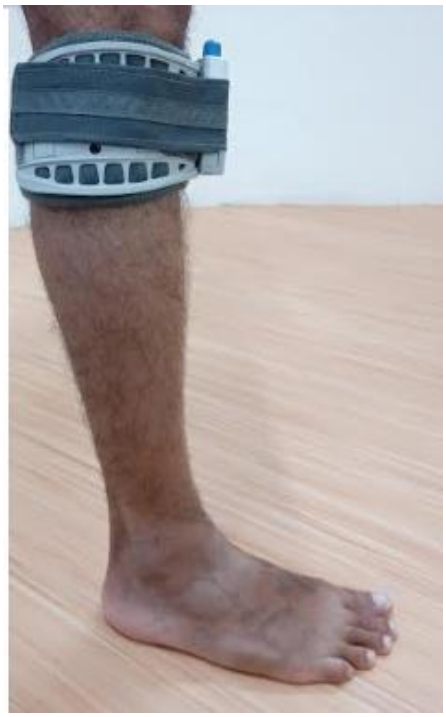


Figure 10: FES (Walkaide) device



Outcome measures:

Primary:

a. Gait velocity by measuring speed during 10 meter walk test expressed as meter/second

b. Endurance by distance covered during 6 minute walk test

c. PCI (Physiological cost index) -

[Heart rate during exercise – Heart rate at rest] / Walking speed

Secondary:

a. Step length
b. Stride length
c. Timed up and go test (TUG)
d. Step width
e. Stance and swing ratio
f. Effect on non-paretic limb
g. Single limb support
h. Walking speed
i. Feedback form (patient satisfaction)

10 Meter walk test:

The individual was instructed to walk 10 meters. The distance (10 meters) was divided by the time the individual took to walk 10 meters. Three trials were done and the average was calculated.

Normative value of 10 meter walk test was found to be 0.84 +/- 0.3 meter/sec. (99) Perry correlated ambulation ability with gait speed.(100)

- < 0.4 m/s – household ambulators
- 0.4 – 0.8 m/s – Limited community ambulators
- > 0.8 m/s – Community ambulators

Six-minute walk test:

This is a functional walking evaluation in which the distance subjects can walk for 6 minute is measured. Normative value of Six minute walk test was found to be 408 meter (133-700 meters).(101)

Physiological Cost Index:

Physiological cost index proposed by MacGregor is used to assess gait demand. At submaximal effort there is an association exists between heart rate and VO2. PCI and oxygen cost has good correlation in patients with stroke. PCI can be used as a substitute for oxygen cost of walking after stroke. (102) PCI is calculated as -

[Heart rate during exercise – Heart rate at rest] / Walking speed

PCI is expressed as beats per minute.

Timed Up and Go Test (TUG):

It is used to assess fall risk and measure balance sit to stand and walking. The patient starts in a seated position. On command the patients stands up, walks 3 meters, turns around, walks back to chair and sits down. This is an excellent parameter for evaluating gait performance in mild to moderate hemiparesis following stroke.(103)

Step length: Distance measured from the heel of one foot to the heel of the other foot.

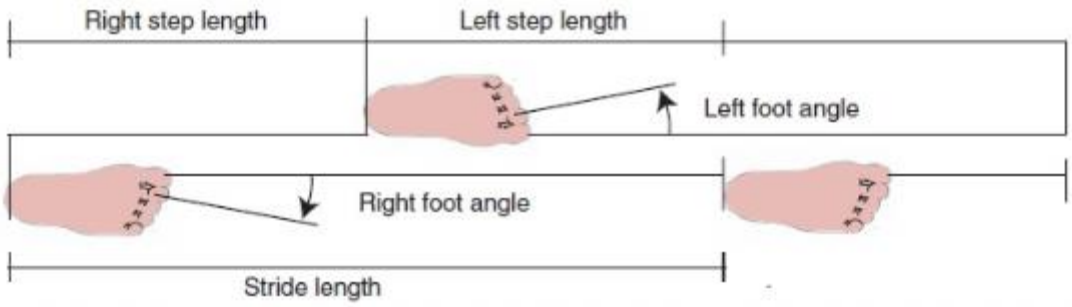
Stride length:

Distance between two successive initial contacts of the same foot. One stride in a gait cycles consists of two steps (left step followed by right). It is equal to the sum of two step lengths.

Step width:

It is the distance between midpoint of the heel between two feet. Step width increases in balance problems.

Fig 11: Stride and step length



Stance and swing ratio:

Each limb has a stance and swing phase in a gait cycle. Stance phase is 60% and swing phase is 40%. This 60:40 ratio is altered in pathological gait. In stroke patients, stance phase duration is short in hemiplegic limb as patient prefers to bear weight on non-paretic limb.(104)

Effect on non-paretic limb: Spatio-temporal data on non-hemiplegic limb.

Single limb support: Single limb support occurs when one foot is in contact with the ground. The percentage of gait cycle which is contributed by single limb.

Walking speed: measured by dividing the distance walked by ambulation time.

Feedback form: for satisfaction level.

GAIT ANALYSIS:

Video Gait Recording:

Patients were made to walk with self-selected speed. Video recording of anterior, posterior and lateral view were done.

Kinematic Data Collection:

The phase space apparatus provides a means of automatically recording of movement with the help of infrared cameras and Light emitting diodes (LEDs) attached to the bony prominences of both lower limbs. Calibration of the cameras was done using Phase Space collaboration software with a fixed point in the room with a set of light emitting diodes placed on a position reference structure before each gait analysis. It also gives information about the temporal-spatial gait

outcomes such as the walking speed, step width, stride length, percentage of stance and swing, single limb support.

Figure 12: Calibration instruments

LED Placements:

Fifteen LEDs were fixed to the following bony prominences.

LED 1 and 9 Head of the Fifth Metatarsal

LED 2 and 10 Lateral Prominence of the Heel

LED 3 and 11 Lateral Malleoli

LED 4 and 12 Head of Fibula

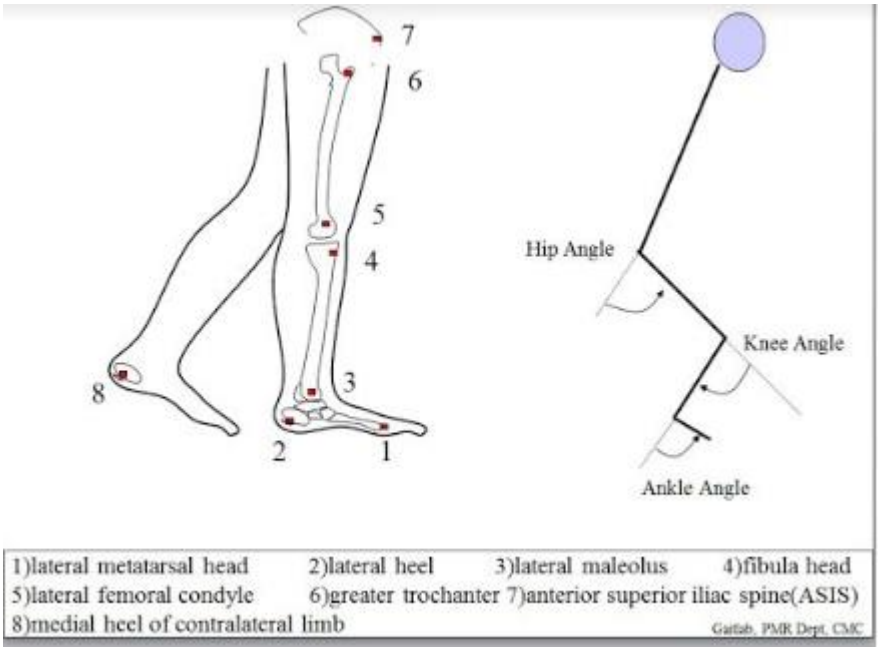
LED 5 and 13 Lateral Epicondyle of the Femur

LED 6 and 14 Greater Trochanter

LED 7 and 15 Anterior Superior Iliac Spine

LED 8 Sacrum

Figure 13: Showing placement of LEDs



8 special infrared cameras containing photocells were focussed on the moving subject. Movement of the light spot images over the photocell generates an electrical signal which is analysed by the computer. The output can also be displayed on a monitor as 3D moving stick figures.

Gait analysis was done with DAQ (Data Acquisition software) software, used to automatically detect and display the angle at each joint and compute angular velocities of motion. DAQ was developed by the Department of Bioengineering, CMC Vellore.

Kinetic Data Collection:

Kinetic gait recordings were made from a Force Plate (Kistler), which used strain gauges or piezoelectric crystals to measure the Ground Reaction Forces (GRF) i.e. vertical, forward/backward and medio/lateral forces. It was essential for the patient to produces a single strike at the force plate without his knowledge. The collected data was then processed through Gait analysis software.

Figure 14: Showing force plate strike

Dynamic Electromyographic Data Collection:

The Motion Lab system monitored the EMG activity during ambulation (Dynamic EMG) with the indigenous pre-amplifier unit connected with the wired EMG

module and the other end being strapped on the lower limb muscles of the patient / subject.

The following are the 8 muscles used for EMG recording:

1. Gluteus Maximus as the Hip Extensor

2. Rectus Femoris as the Hip Flexor

3. Tensor Fascia Lata as the Hip Abductor

4. Adductor Longus as the Hip Adductor

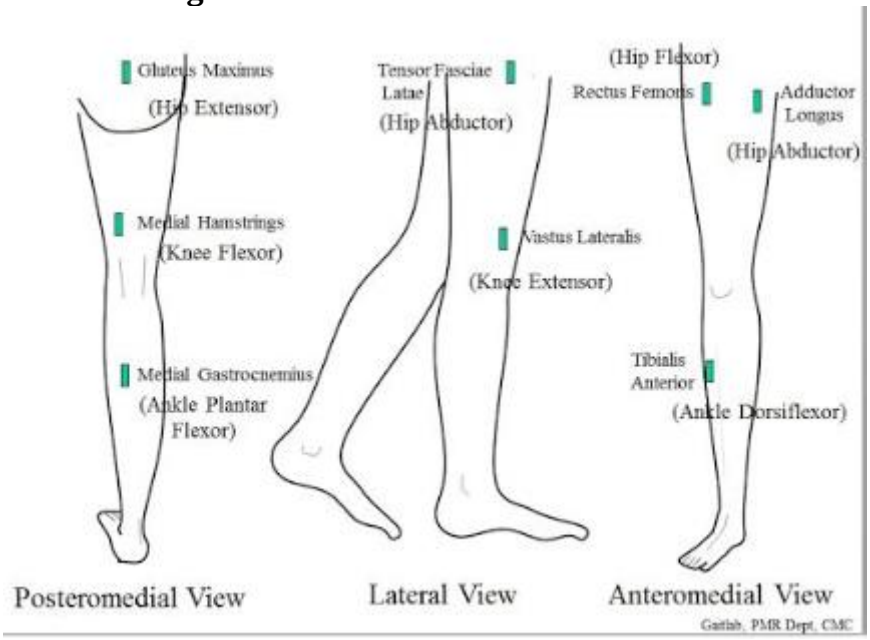
5. Vastus Lateralis as the Knee Extensor

6. Medial Hamstring as the Knee Flexor

7. Medial Gastrocnemius as the Ankle Plantarflexor and

8. Tibialis Anterior as the Ankle Dorsiflexor.

Figure 15: Placements of EMG



Energy Consumption Data Collection:

Heart rate at resting stage and after 25 meters walk was measured. A surface EMG electrode for measuring heart rate was placed on the apex. Energy expenditure was estimated by measuring the physiological cost index (PCI).

PCI = [Heart rate during exercise – Heart rate at rest] / Walking speed

Sample Size Calculation:

The minimum acceptable difference (gait velocity by 10 meter walk test) between the tools is 0.16 meter/sec, (105,106) assume this difference and an SD of 0.2 , we need a sample of 15 subjects with 80% power and 5% errors. We recruited 20 subjects for this study.

Formula:

$$N_{pairs} = \frac{\left(z_{1-\alpha/2} + z_{1-\beta}\right)^2}{\Delta^2} + \frac{z_{1-\alpha/2}^2}{2}$$

$$\Delta = \frac{(\mu_2 - \mu_1)}{\sigma} \qquad \sigma = \frac{\sigma_1 + \sigma_2}{2}$$

Where,

μ_1 = Mean of AFO

μ_2 = Mean with FES

σ_1 = Standard deviation of AFO

σ_2 = Standard deviation of FES

Statistical Analysis:

Data was entered in excel format and screened for outliers and extreme values.

Wilcoxon sign rank test was used to compare between AFO and FES.

RESULTS:

During the study period of 1 year, 20 patients with hemiplegia due to cerebrovascular accident who satisfied the exclusion and inclusion criteria were recruited.

Baseline Demographic Data:

Age distribution:

The mean age of the patients was 45.5 years ± S.D 9.45.

Gender distribution: Of the 20 patients, 19 patients were male and one patient was female.

Height and weight:

The mean height of the study population was 167.85 cm ± 7.10. The mean weight of the study population was 64.28 kg ± 8.91.

Table 1: Showing demographic data

Baseline Demographic Data	Total (n=20)
Male	19
Female	1
Age (years) Mean ± SD (Median)	45.5 ± 9.45(48)
Height (cms) Mean ± SD (Median)	167.85 ± 7.10 (168)
Weight (kg) Mean ± SD (Median)	64.28 ± 8.91(64.75)

Type of stroke:

Of the 20 patients, 16 (80%) patients had ischeamic stroke and 4 (20%) patients had haemorrhagic stroke. Among 16 patients 3 patients was diagnosed with cortical venous thrombosis. Rest 13 patients had left MCA territory infarct.

Figure 16: Type of stroke

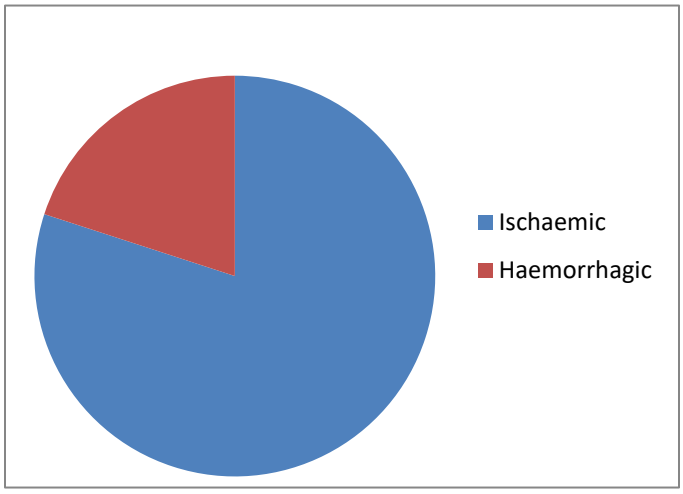
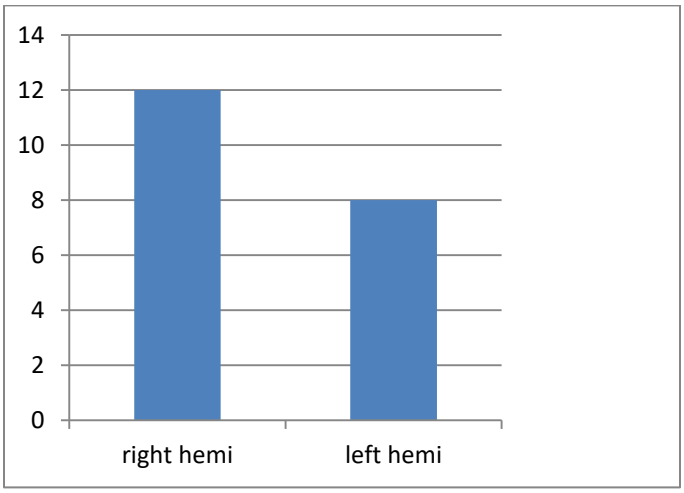


Figure 17: Hemiplegic side



Hemiplegic side:

12 (60%) patients had right hemiparesis and 8 (40%) patients had left hemiparesis.

Duration since stroke:

The mean duration since stroke was 12 months.

Table 2: Outcome measure analysis between barefoot, AFO and FES

Outcome	Mean ± SD			p value	Median		
	Barefoot	**AFO**	**FES**		**Barefoot**	**AFO**	**FES**
10 meter walk test (sec)	31.98 ± 12.06	28.96 ± 10.04	27.84 ± 10.52	0.0001	31.94	29.35	26.7
Speed (m/s)	0.36±0.17	0.40±0.20	0.42±0.22	0.0001	0.31	0.34	0.37
6 minute walk test (m)	140.6 ± 70.71	154.1 ± 81.36	162.3 ± 86.6	0.0001	124.5	131.5	131.5
TUG (sec)	28.29 ± 10.39	27.61 ± 11.27	25.19 ± 10.04	0.0001	27.67	26.05	24.25
PCI	2.47 ± 2.74	1.68 ± 1.23	1.5 ± 1.13	0.46	1.44	1.15	1.15

Table 3: Outcome measure analysis between barefoot, AFO and FES

Outcome	Mean ±SD			p value	Median		
	Barefoot	AFO	FES		Barefoot	AFO	FES
Stride length (paretic) (cm)	53.65 ± 23.69	54.3 ± 25.26	59.65 ± 19.29	0.086	49.5	50	60
Stride length (Non-paretic)	54 ± 23.61	60.85 ± 21.85	55.65 ± 21.46	0.109	53.5	57	55
Step Width (paretic)	7.60 ± 4.05	7.85 ± 4.41	7.6 ± 3.20	0.771	8	8	8
Step Width (Non-paretic)	8.5 ± 5.13	8 ± 4.19	8.3 ± 3.72	0.520	7	7	8
Single limb support (Paretic)	20 ± 7.64	20.7 ± 6.43	22.2 ± 6.45	0.033	19.5	20.5	24
Single limb support (Non-Paretic)	29.3 ± 9.22	27.65 ± 9.03	31.8 ± 8.63	0.018	30.5	28.5	31
Walking speed (Paretic)	18.95 ± 11.22	20.25± 13.24	20.2 ± 10.51	0.060	18	19	19.5
Walking speed (Non-Paretic)	18.95 ± 11.22	20.25± 13.24	20.2 ± 10.51	0.074	18	19	19.5
Stance swing ratio (Paretic) STANCE	70.7 ± 9.22	71.55 ± 8.93	68.3 ± 8.47	0.040	69.5	69.5	69
Stance swing ratio (Paretic) SWING	29.25 ± 9.26	28.45 ± 8.93	31.7 ± 8.47	0.040	30.5	30.5	31
Stance swing ratio (Non-Paretic) STANCE	80 ± 7.64	79.3 ± 6.43	77.8 ± 6.45	0.033	80.5	79.5	76
Stance swing ratio (Non-Paretic) SWING	20 ± 7.64	23 ± 13.22	22.2 ± 6.45	0.033	19.5	20.5	24

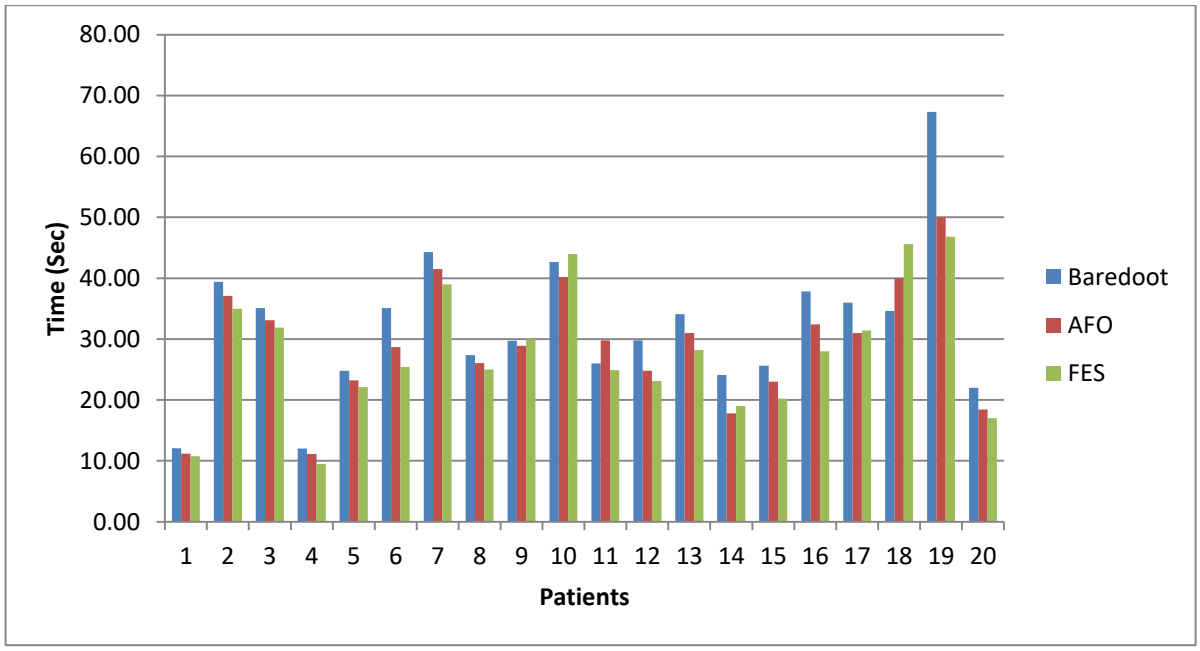
Table 4: Outcome measure analysis between AFO and FES

Outcome	Mean +/- SD		Median		
	AFO	FES	p value	AFO	FES
10 meter walk test	28.96 ± 10.04	27.84 ± 10.52	0.0365	29.35	26.7
Speed	0.40 ± 0.20	0.42 ± 0.22	0.000	0.34	0.37
6 minute walk test	154.1 ± 81.36	162.3 ± 86.6	0.004	131.5	131.5
TUG	27.61 ± 11.27	25.19 ± 10.04	0.0001	26.05	24.25
PCI	1.68 ± 1.23	1.5 ± 1.13	0.46	1.15	1.15
Stride length (paretic)	54.3 ± 25.26	59.65 ± 19.29	0.185	50	60
Stride length (Non-paretic)	60.85 ± 21.85	55.65 ± 21.46	0.3504	57	55
Step Width (paretic)	7.85 ± 4.41	7.6 ± 3.20	0.8509	8	8
Step Width (Non-paretic)	8 ± 4.19	8.3 ± 3.72	0.5360	7	8
Single limb support (Paretic)	20.7 ± 6.43	22.2 ± 6.45	0.0152	20.5	24
Single limb support (Non-Paretic)	27.65 ± 9.03	31.8 ± 8.63	0.0016	28.5	31
Walking speed (Paretic)	20.25 ± 13.24	20.2 ± 10.51	0.388	19	19.5
Walking speed (Non-Paretic)	20.25 ± 13.24	20.2 ± 10.51	0.653	19	19.5
Stance swing ratio (Paretic) STANCE	71.55 ± 8.93	68.3 ± 8.47	0.0078	69.5	69
Stance swing ratio (Paretic) SWING	28.45 ± 8.93	31.7 ± 8.47	0.0078	30.5	31
Stance swing ratio (Non-Paretic) STANCE	79.3 ± 6.43	77.8 ± 6.45	0.0152	79.5	76
Stance swing ratio(NonParetic) SWING	23 ± 13.22	22.2 ± 6.45	0.0251	20.5	24

Analysis of Spatio-temporal Data:

<u>10 meter walk test</u>:

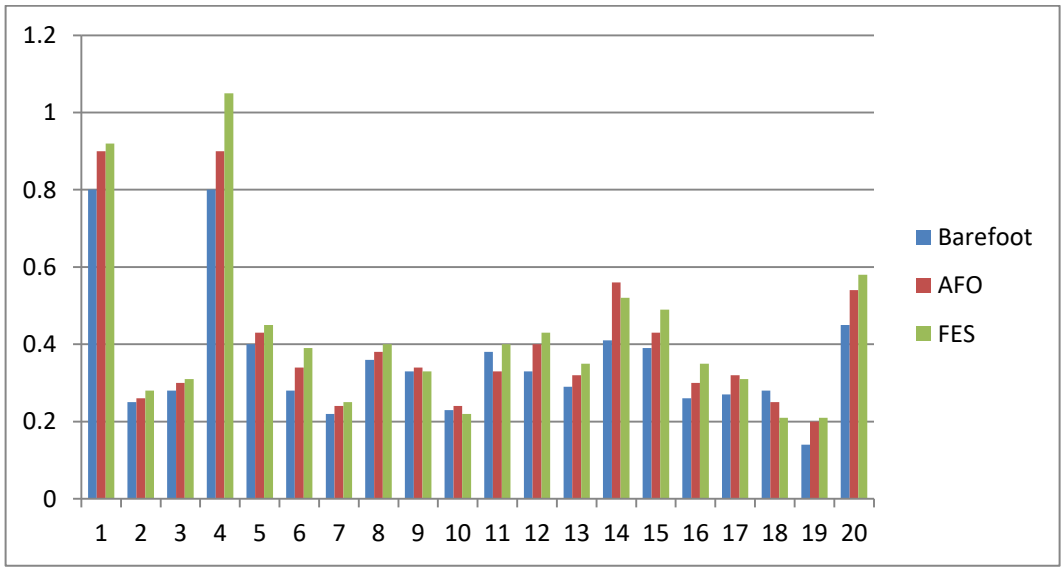
Figure 18: Comparison of 10 meter walk test in subjects walking barefeet, AFO and FES



The mean duration of walking 10 meters was 31.98 seconds (SD 12.06) barefoot, 28.96 second (SD 10.04) with AFO and 27.84 sec (SD 10.52) with FES. There was statistical difference in 10 meter walk test with FES compared to AFO (p value 0.0365).

Gait speed:

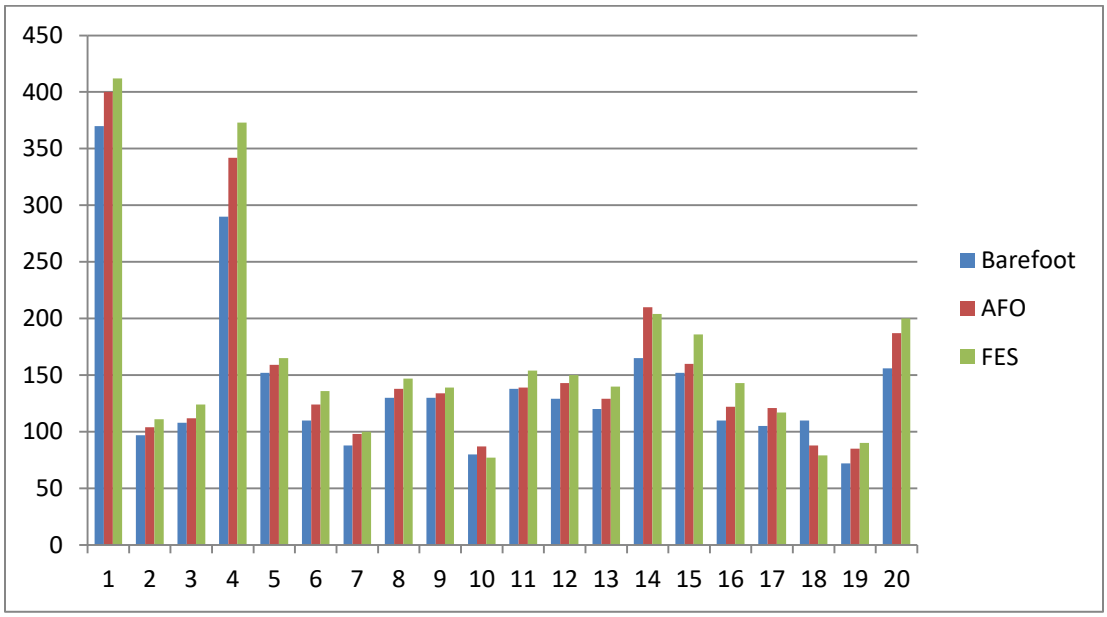
Figure 19: Comparison of gait speed in subjects walking barefeet, AFO and FES



The mean gait speed was 0.36 m/s (SD 0.17) barefoot, 0.40 second (SD 0.2) with AFO and 0.42 sec (SD 0.22) with FES. There was statistical difference in gait speed with FES compared to AFO (p value 0.0001).

6 minute walk test:

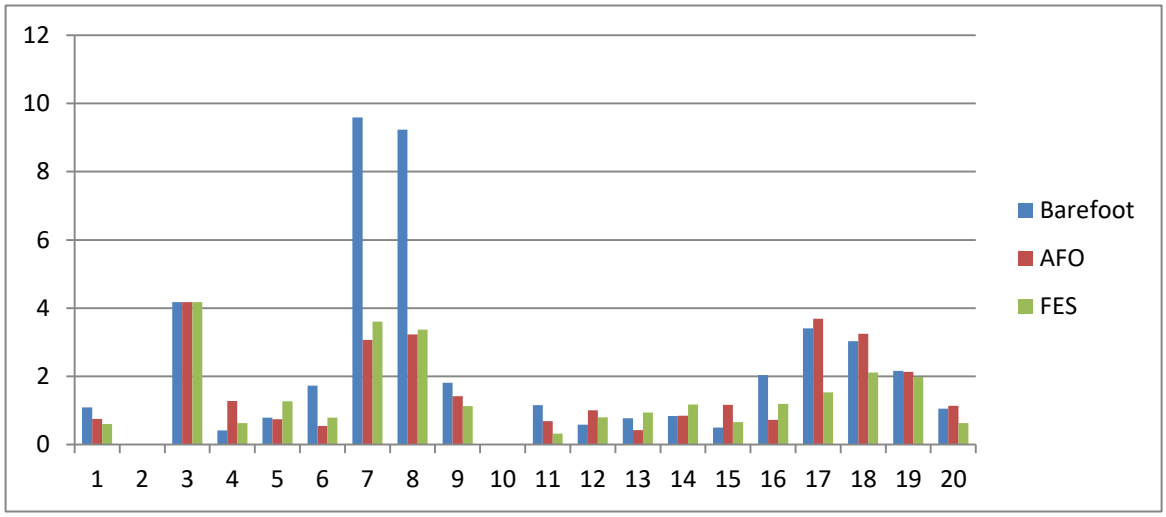
Figure 20: Comparison of 6 minute walk test in subjects walking barefeet, AFO and FES



The mean distance to walk in 6 minute was 140.6 meter (SD 70.71) barefoot, 154.1meters (SD 81.36) with AFO and 162.3 with FES (SD 86.6). There was statistical difference in 6 minute walk test with FES compared to AFO (p value 0.000).

Physiological cost index:

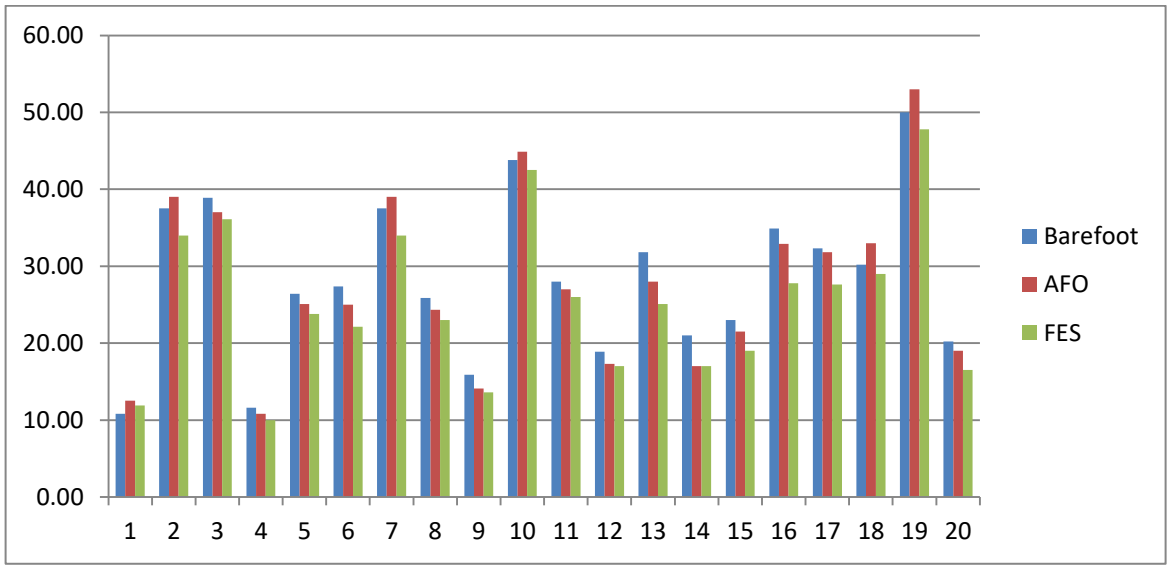
Figure 21: Comparison of physiological cost index in subjects walking barefeet, AFO and FES



The mean PCI was 2.47 beats/meter (SD 2.74) barefoot, 1.68 beats/meter (SD 1.23) with AFO and 1.5 (SD 1.13) with FES. There was no statistical difference in physiological cost index (PCI) with FES compared to AFO (p value 0.46).

Timed Up and Go test:

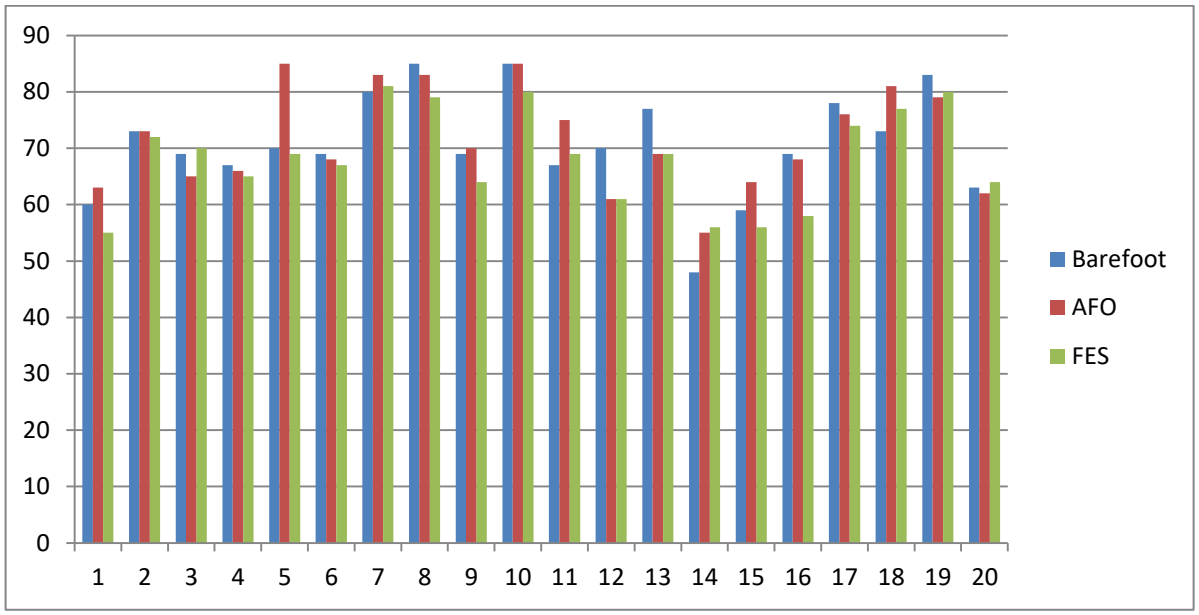
Figure 22: Comparison of Timed Up and Go test in subjects walking barefeet, AFO and FES



The mean duration of TUG was 28.29 seconds (SD 10.39) on barefoot, 27.61 second (SD 11.27) with AFO and 25.19 sec (SD 10.04) with FES. There was statistical difference TUG with FES compared to AFO (p value 0.0001).

Stance-Swing ratio (Paretic side): Stance phase:

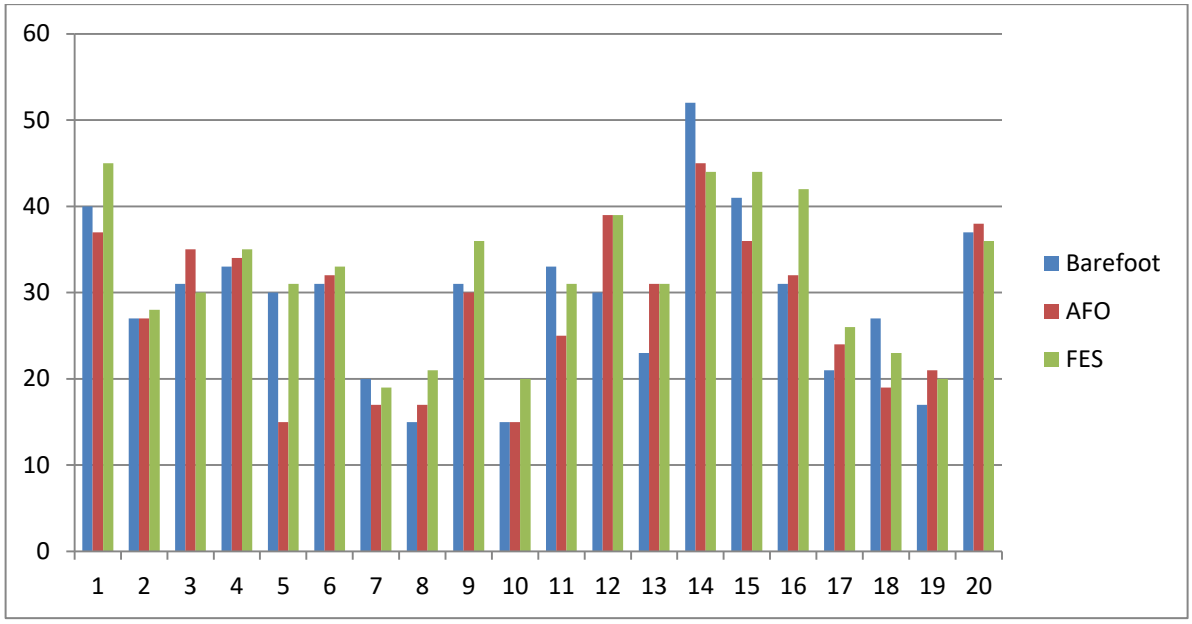
Figure 23: Comparison of stance phase on paretic side in subjects walking barefeet, AFO and FES



The mean duration of stance phase was 70.7 % (SD 9.22) barefoot, 71.55 % (SD 8.93) with AFO and 68.3 % (SD 8.47) with FES. There was statistical difference in stance phase on paretic limb among barefoot, AFO and FES (p value 0.04).FES showed statistically significant difference compared to AFO. (p value 0.0078).

<u>Stance-Swing ratio (Paretic side)</u>: **<u>Swing phase:</u>**

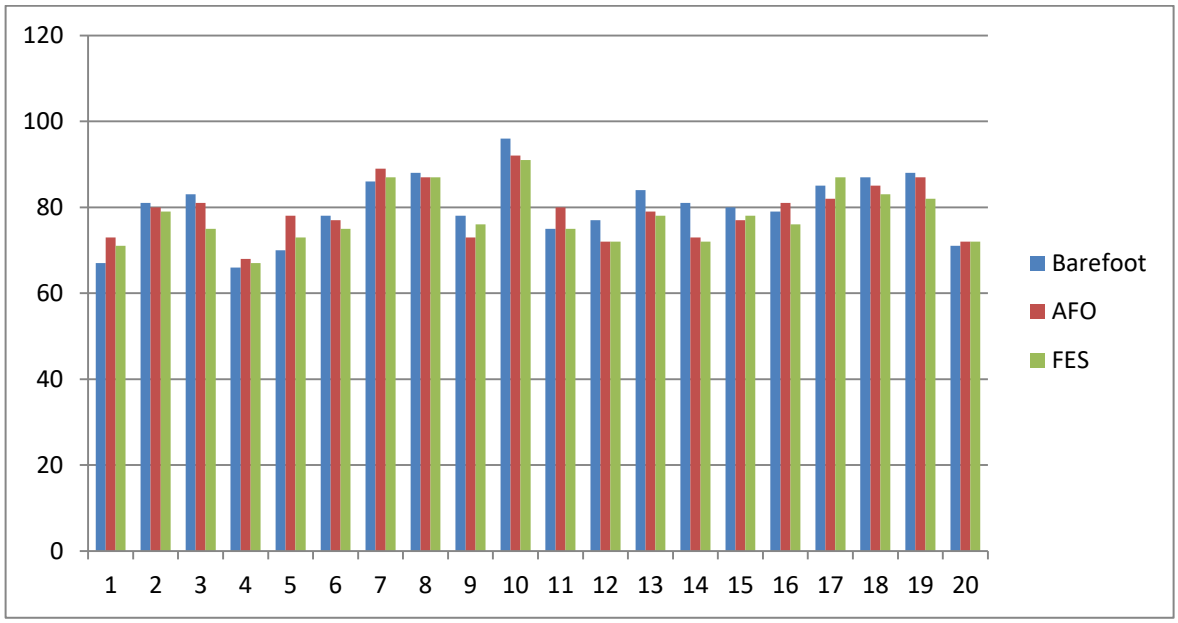
Figure 24: Comparison of swing phase on paretic side in subjects walking barefeet, AFO and FES



The mean duration of swing phase was 29.25 % (SD 28.45) barefoot, 28.45 % (SD 8.93) with AFO and 31.7 % (SD 8.47) with FES. There was statistical difference in swing phase on paretic side among barefoot, AFO and FES (p value 0.04). FES showed statistically significant difference compared to AFO. (p value 0.0078).

<u>Stance-Swing ratio (Non-Paretic side)</u>: <u>Stance phase</u>:

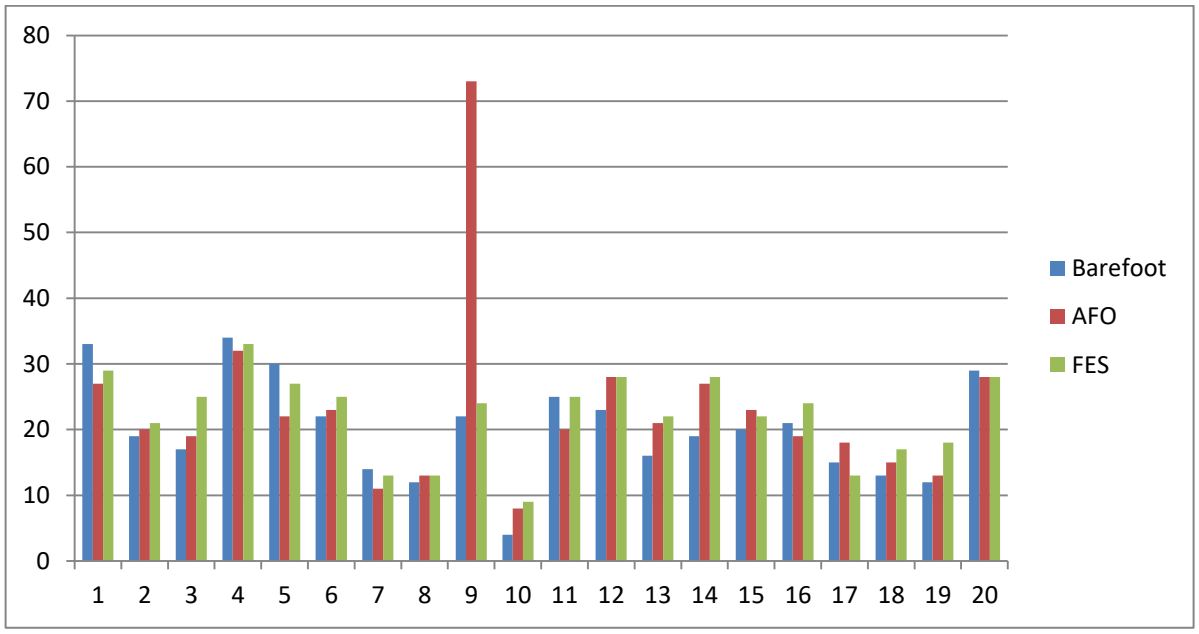
Figure 25: Comparison of stance phase on non-paretic side in subjects walking barefeet, AFO and FES



The mean duration of stance phase was 80 % (SD 7.64) barefoot, 79.3 % (SD 6.43) with AFO and 77.8 % (SD 6.45) with FES. There is statistical difference in stance phase on paretic limb among barefoot, AFO and FES (p value 0.033). FES showed statistically significant difference compared to AFO. (p value 0.0152).

Stance-Swing ratio (Non-Paretic side): **Swing phase**:

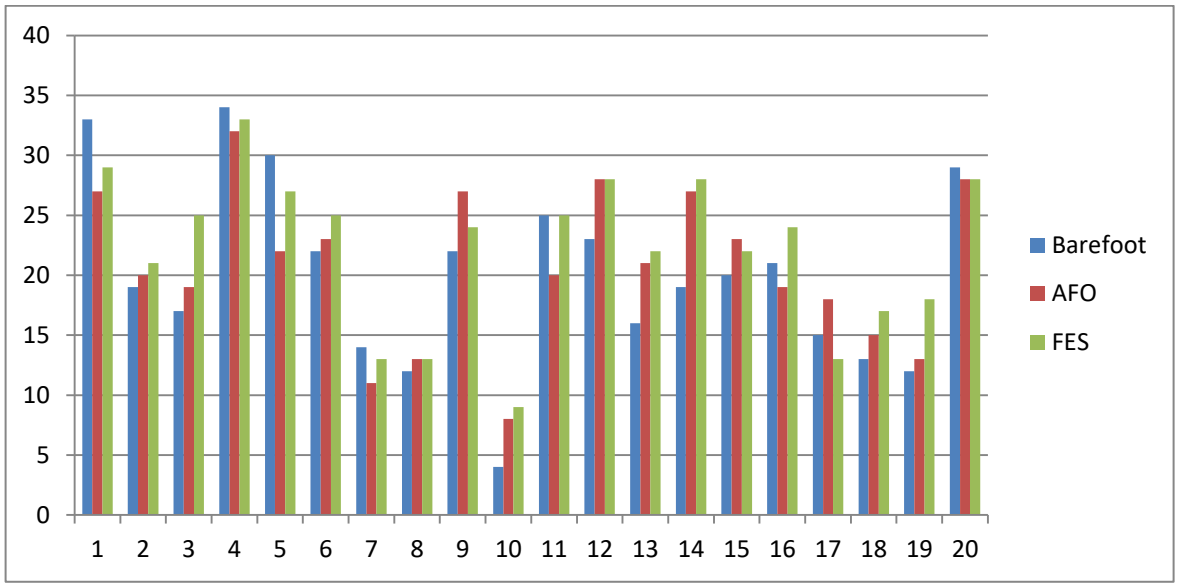
Figure 26: Comparison of swing phase on non-paretic side in subjects walking barefeet, AFO and FES



The mean duration of swing phase was 20 % (SD 7.64) barefoot, 20.7 % (SD 13.22) with AFO and 22.2 % (SD 6.45) with FES. There was statistical difference in swing phase on paretic side among barefoot, AFO and FES (p value 0.033). FES showed statistically significant difference compared to AFO. (p value 0.0251).

Single limb support (Paretic side):

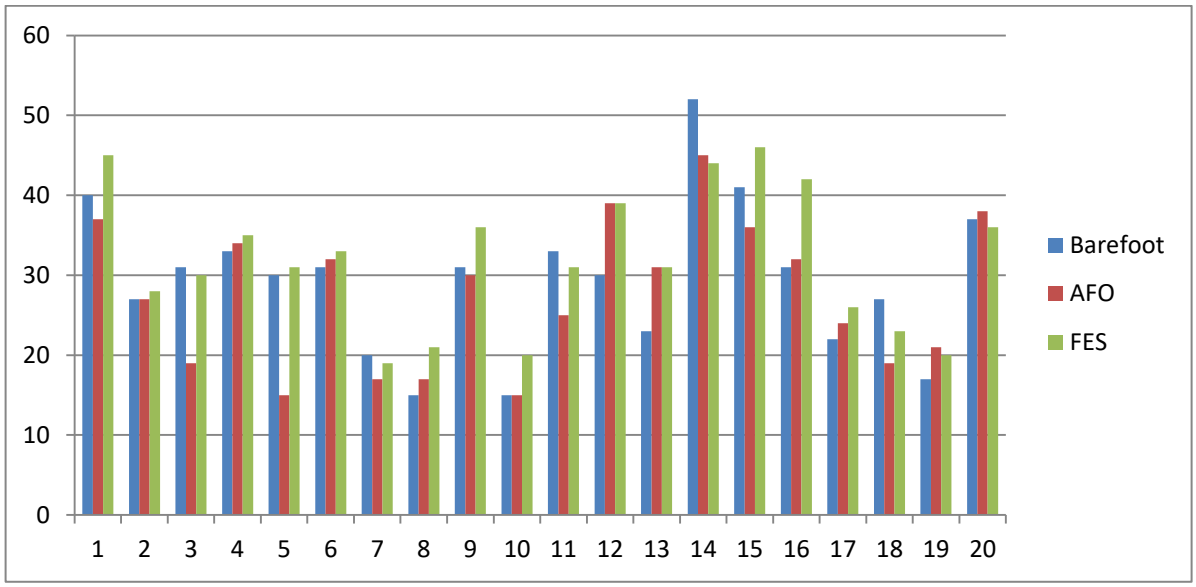
Figure 27: Comparison of single limb support on paretic side in subjects walking barefeet, AFO and FES



The mean duration of single limb support on paretic limb was 20 % (SD 7.64) barefoot, 20.7 % (SD 6.43) with AFO and 22.2 % (SD 6.45) with FES. There was statistical difference in stance phase on paretic limb among barefoot, AFO and FES (p value 0.033). FES showed statistically significant difference compared to AFO. (p value 0.015).

Single limb support (Non-Paretic side):

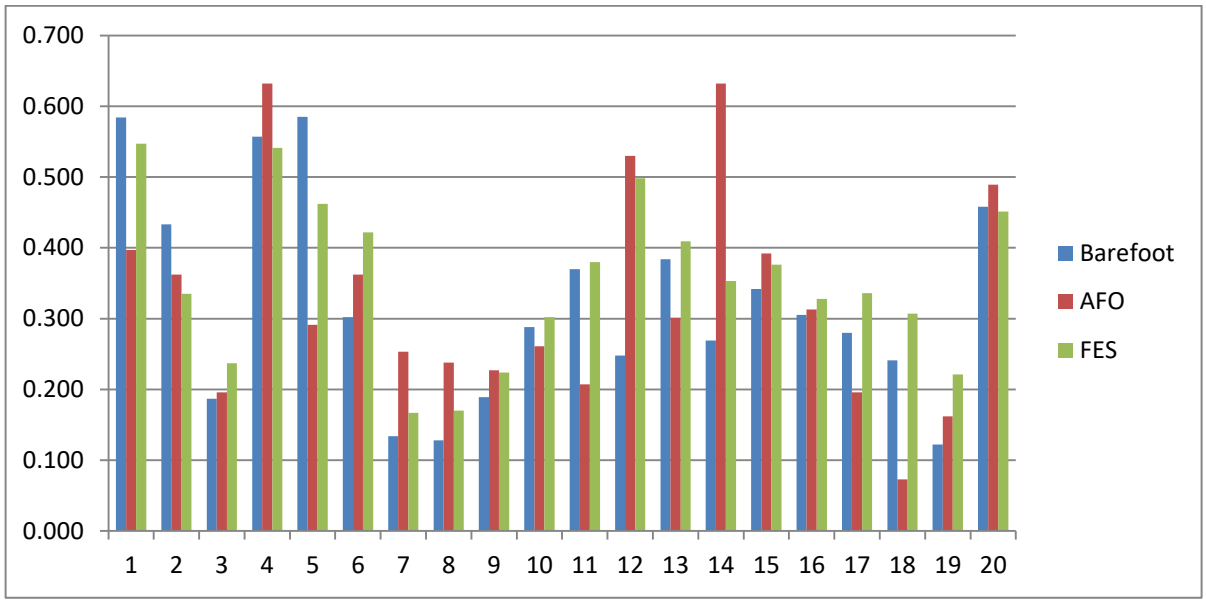
Figure 28: Comparison of single limb support on non-paretic side in subjects walking barefeet, AFO and FES



The mean duration of single limb support on non-paretic limb was 29.3 % (SD 9.22) barefoot, 27.65 % (SD 9.03) with AFO and 31.8 % (SD 8.63) with FES. There was statistical difference in stance phase on paretic limb among barefoot, AFO and FES (p value 0.018). FES showed statistically significant difference compared to AFO. (p value 0.0001).

Stride Length: (paretic side):

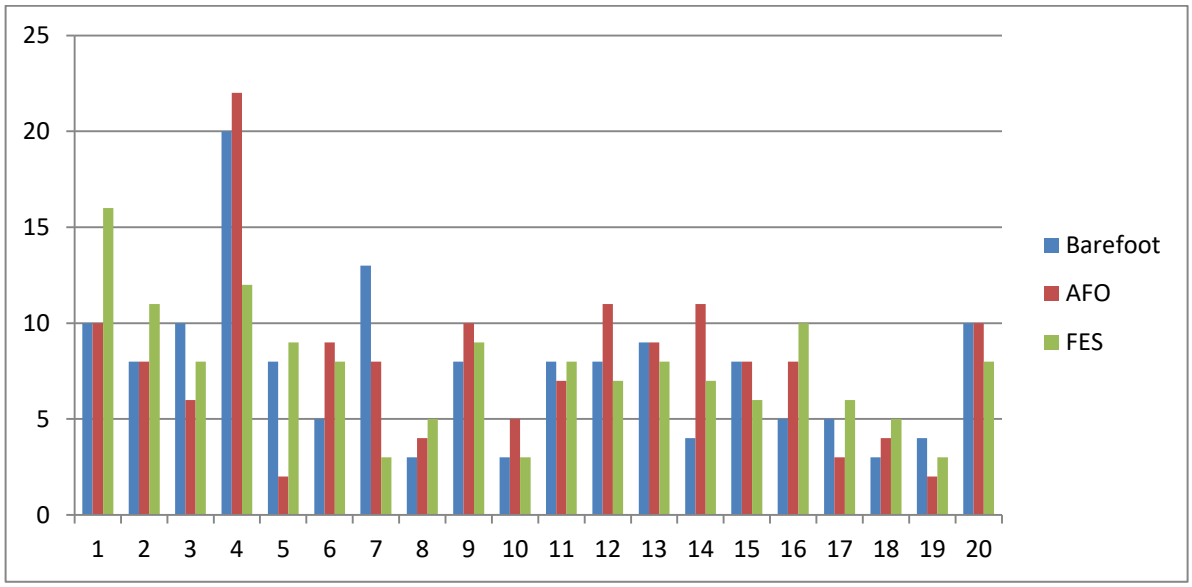
Figure 29: Comparison of stride length on paretic side in subjects walking barefeet, with AFO and FES



The mean stride length was 53.65 cm (SD 23.61) barefoot, 54.3 cm (SD 25.26) with AFO and 59.65 cm with FES (SD 19.29). There was no statistical difference in stride length test among barefoot, AFO and FES (p value 0.08).

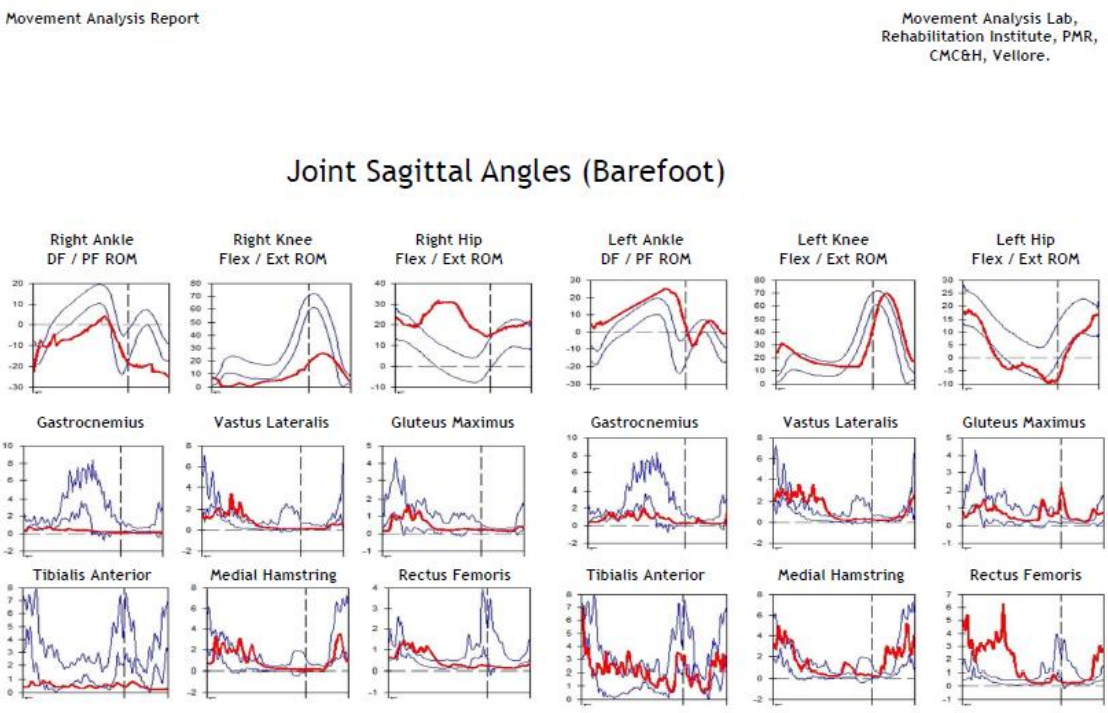
Step Width: (paretic limb)

Figure 30: Comparison of step width on paretic side in subjects walking barefeet, with AFO and FES



The mean step width was 7.6 cm (SD 4.05) on barefoot, 7.85 cm (SD 4.41) with AFO and 7.6 cm with FES (SD 3.2). There was no statistical difference in stride length test among barefoot, AFO and FES (p value 0.77).

Figure 31: Graphical representation of kinematics data (Barefoot)



This is a sample kinematic (sagittal plane) report of a patient with right hemiparesis.

<u>Kinematics and EMG:</u>

Ankle:

- Right side- Initial contact was made with 20 degree of plantar flexion. Ankle remained in plantar flexion throughout the gait cycle and maximal plantar flexion was 25 degree at terminal swing phase. Forefoot rocker was absent.

  <u>EMG</u>: Gastrocnemius contraction was absent during pre-swing phase. Tibialis anterior was silent throughout the gait cycle.

- Left side- Ankle joint kinematics follows normal pattern except there was reduced plantar flexion in pre-swing phase.

  EMG: Gastrocnemius contraction was poor during pre-swing phase.

  Tibialis anterior showed phasic contraction throughout the gait cycle.

Knee:

- Right side- Knee flexion was reduced in both stance and swing phase of gait.

  EMG- Vastus lateralis and medial hamstring EMG activity was phasic but poor.

- Left side- Knee joint followed normal pattern.

  EMG: Vastus lateralis and medial hamstring EMG activity was phasic.

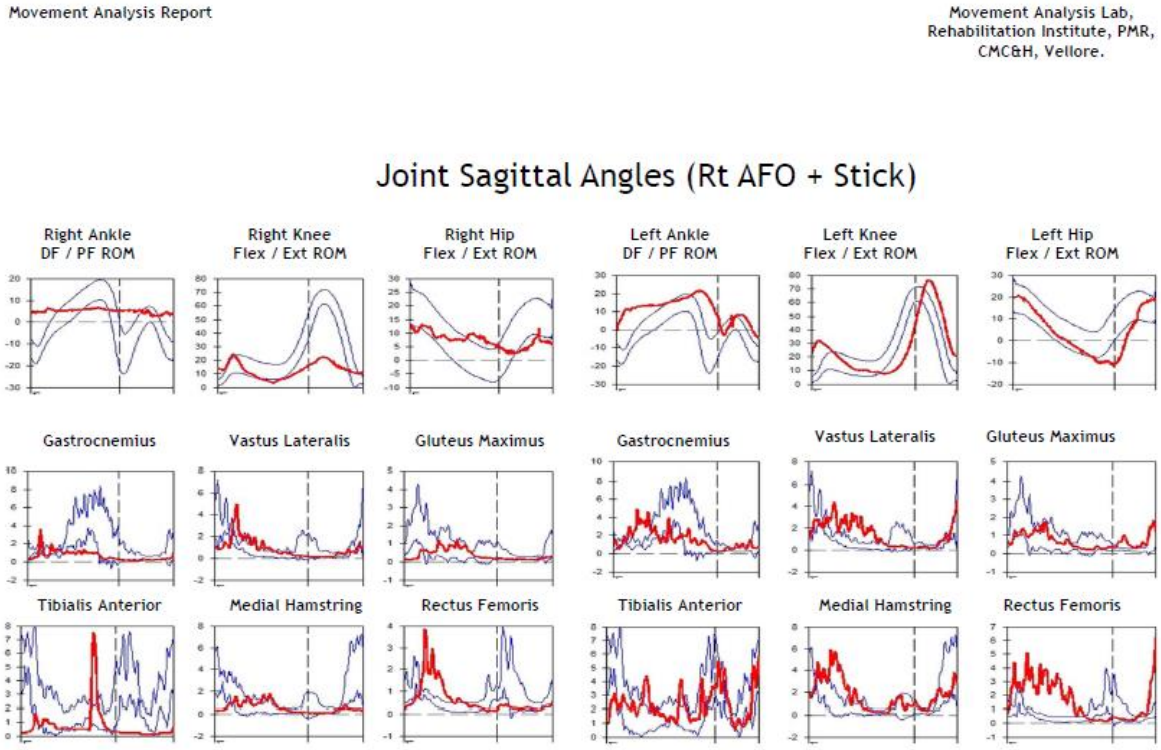
Hip:

- Right side- Hip remained in flexion throughout the gait cycle and maximum flexion was 30 degree.

  EMG- Gluteus maximus activity was diminished in stance phase. Rectus femoris contraction was poor on initial swing phase.

- Left side- Hip joint followed normal kinematic pattern throughout the stride.

Figure 32: Graphical representation of kinematics data (with AFO)

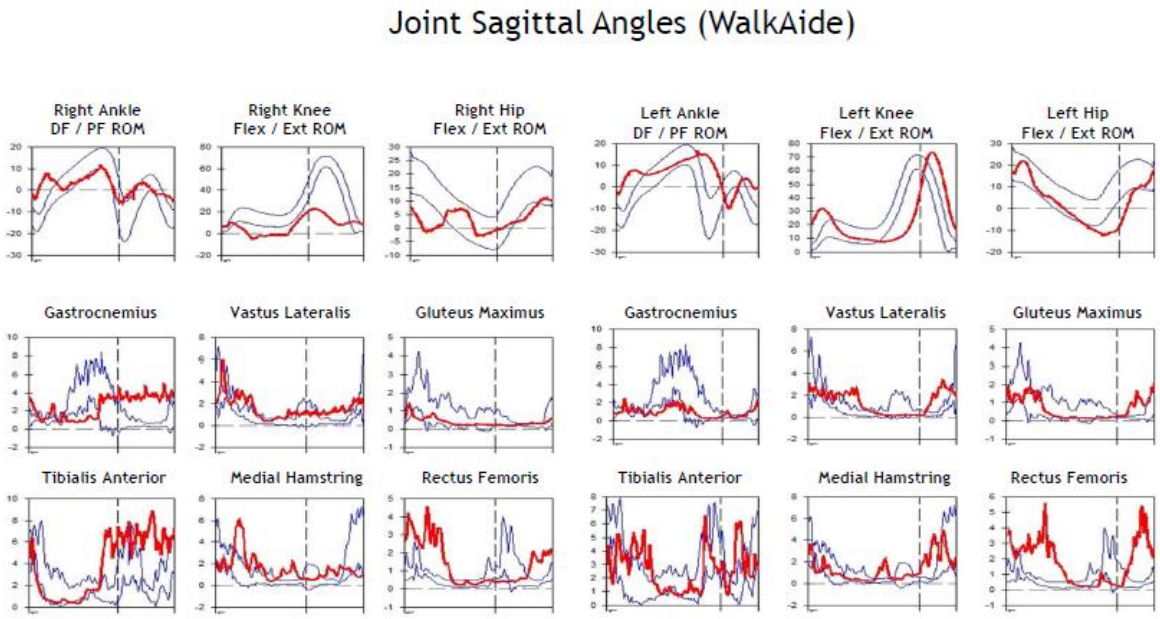


Kinematics (sagittal plane) report of same patient with right hemiparesis walking with right AFO and stick.

- Right Ankle- There was no range of motion noticed. It remained in 5 degree of flexion throughout the gait cycle.
- Right Knee- During stance phase there was flexion at knee and diminished flexion in swing phase.
- Right Hip- Right hip flexion was found to be reduced during pre-swing phase.

All other parameters didn't show any significant changes compared to barefoot.

Figure 33: Graphical representation of kinematics data (with FES)



Kinematic (sagittal plane) report of same patient with right hemiparesis walking with FES(WalkAide).

<u>Kinematics and EMG:</u>

Right Ankle:

- Initial contact was made with 5 degree of plantar flexion. Ankle was in dorsiflexion during mid-stance. During swing phase ankle was neutral. Forefoot rocker was absent.
- EMG: Gastrocnemius contraction was absent during pre-swing phase. Tibialis anterior contraction was noticed in swing phase of gait cycle.

Right Knee:

- Knee flexion was reduced in both stance and swing phase of gait.
- EMG- Vastus lateralis and medial hamstring EMG activity was phasic but poor.

Right Hip:

- Flexion was reduced during pre-swing phase.
- EMG- Gluteus maximus activity was diminished in stance phase. Rectus femoris contraction was poor during the initial swing phase.

All other parameters didn't show any significant changes compare to barefoot.

Feedback Questionnaire (patients' satisfaction):

The following table summarizes the patients' satisfaction with AFO and FES at the end of the study. The participants were asked about comfort level, ease of walking on normal and rough terrain, stair climbing, donning and doffing of device, walking distance, effort of walking, stability while walking, appearance of device and orthosis usage after discharge.

Table 5: Patient Satisfaction - Median scores

Question	AFO	FES
Comfort	3(2-4)	4(3-4)*
Ease Normal surface	3(2-4)	4(3-4)*
Ease Rough terrain	2(2-3)	3(3-4)*
Stair climbing	3(3-4)	3(3-4)*
Donning and doffing	3(2-3)	4(4)*
Walking distance	3.5(3-4)	4(3-4)*
Effort of walking	4(3-4)	4(3-4)
Stability	3(3-4)	4(3-4)*
Appearance	3(3-4)	4(4)*
Orthosis usage in future	3(3-4)	4(3-4)*

(Satisfaction scale: 1-Very unsatisfied, 2- Not satisfied, 3- Neutral, 4- Satisfied, 5-very satisfied) * p value- <0.05 *Significantly better scores with functional electrical stimulation (FES) than with the ankle-foot orthosis (AFO)

For all these questions, the median score for FES was 4 (Patient was satisfied with FES) except for walking on rough terrain and stair climbing was 3(Patient was neutral about FES). All scores tended to be higher for FES. Patient satisfaction was not statistically significant for effort of walking.

Effect of order of trial:

Table 6: Effect of order of AFO and FES training i.e. BAF vs BFA

Outcome	p value
10 meter walk test	0.1211
6 minute walk test	0.0447
TUG	0.1617
PCI	0.1416
Stride length (paretic)	0.9097
Single limb support (Paretic)	0.1664
Single limb support (Non-Paretic)	0.5693
Stance swing ratio (Paretic) STANCE	0.1971
Stance swing ratio (Non-Paretic) STANCE	0.1664

Order of trial did not show any effect on the outcome measures, (p value >0.05) except 6 minute walk test. (p value 0.04)

DISCUSSION:

In 2013, stroke was the second leading cause of death comprising 11.8% of all deaths worldwide and third most common cause of disability (4.5% of DALYs). (1) Walking impairment is one of the major disabilities which occurred due to weakness, spasticity and incoordination of lower limb muscles.(2,3) Weakness of ankle dorsiflexor is a major factor for decreased walking endurance.(109) Ankle dorsiflexor weakness with spasticity in plantraflexion results in footdrop. The traditional mode of treatment provided for foot drop is ankle foot orthosis (AFO). The newer modality of treatment is Functional Electrical Stimulation (FES) of the peroneal nerve.(13) Both of these treatment options are well established for the management of foot drop and there is no conclusive evidence to suggest that FES is superior to AFO for correction of foot drop.(14)

The current study was done with the objectives to compare spatiotemporal parameters between barefoot, Ankle-foot-orthosis (AFO) and Functional electrical stimulation as well as to evaluate ankle-foot kinematics in patients with stroke. In addition, patients satisfaction was assessed with AFO and FES by using a questionnaire. The study was a comparative crossover trial.

20 patients with history of cerebrovascular accident were recruited for the study. Majority of the patients were male (19 male patients). Their average age was about 45.5 years. 80% patients had ischaemic stroke. Average duration since stroke was 12 months.

Spatiotemporal Data:

Walking Speed:

Gait velocity was measured by 10 meter walk test. There was statistically significant improvement of gait velocity in both AFO and FES compared to barefoot. Robbins et al reported from a meta-analysis FES improves walking speed in post stroke patients.(91)

There was statistically significant difference in walking speed noticed between the AFO and FES (p value-0.036). Kottink et al. suggested from a randomised control trial that FES resulted in significant increase in walking speed compared to AFO. (110) In contrast, other studies found no difference between use of AFO and FES.(14,105,111,112) The discrepancy can probably be explained by the study participants' characteristics. The patients in our current study had a mean walking speed of 0.36 m/s at baseline whereas participants in the studies of Roos van Swigchem walked at a speed of 1.02 m/s at baseline. An already high baseline walking velocity may have yielded a ceiling effect in their study.

The increase in gait velocity might be explained by several features of FES (WalkAide). First, FES does not restrict ankle mobility, permits easier balance reaction and

plantarflexion movement during push-off (pre-swing phase). Peroneal FES may help to reduce spasticity of the paretic leg and trigger flexion reflex. (113,114)

Although the improvement in the walking speed was statistically significant, they did not meet the established minimally clinically important difference (MCID) for clinical significance (0.04 m/s for AFO and 0.06 m/s for FES in the current study). MCID for gait speed in stroke patients have been reported in literature and range from 0.1 m/s to 0.16m/s. (106) Increase in gait velocity above MCID has been reported in previous literature, this probably as a result of prolonged gait training in other studies. (105,111)

As a disadvantage of transcutaneous FES, skin allergy has been reported in the literature. In this study no participants had any skin problems. (115)

FES of the dorsiflexor does not improve gait quality of the patients with insufficient knee and hip control. Springer et al conducted a study using dual channel FES over hamstring and ankle dorsiflexor and showed improvement of gait speed which did not depend upon initial gait velocity.(95) In this study we used single channel FES (WalkAide). Two of our patients with quadriceps weakness (power MRC 3) and hip extensor weakness showed poor improvement with FES.

Walking endurance:

Walking endurance was measured by 6 minute walk test. There was statistically significant improvement of endurance in both AFO and FES compared to barefoot. Although the improvement in the walking endurance reached statistical significance, it did not meet minimally clinically important difference (MCID). In the present study, change in the 6 minute walk test was 14 meters with AFO and 22 meters with FES. MCID for 6 minute walk test in stroke patients has been reported in literature to be about 34.4 meters. (116)

Physiological cost index (PCI):

PCI was calculated to indirectly measure oxygen consumption of walking. Although there was mean reduction of PCI with AFO and FES compare to barefoot, in this study there was no statistically significant difference noticed between AFO and FES. It could be explained by slow speed of walking.

Balance:

In stroke patients timed up and go (TUG) test is reliable to evaluate which patients have tendency to fall due to imbalance so that fall prevention techniques can be advised. (117) There was statistically significant reduction in time to complete TUG in both AFO and FES compare to barefoot.

Secondary Outcome measures:

Stride length was increased with AFO and FES compared to baseline but the difference was not statistically significant. This could be the cause of poor walking speed. Percentage of single limb support increased with AFO and FES. FES had statistically significant difference compared to AFO. Stance-swing ratio in paretic and non-paretic limb progressed towards normal with both AFO and FES. There was statistically significant difference with FES compared to AFO.

Kinematics:

Ankle-foot Kinematics:

Ankle was in plantarflexion during swing phase of gait during barefoot gait analysis. Tibialis anterior contraction was absent. With FES, ankle reached to near neutral during swing phase due to phasic contraction of tibialis anterior and evertors. While walking with AFO there was no motion in ankle.

Knee and hip kinematics:

These parameters remained grossly unchanged with AFO and FES.

Feedback Questionnaire (patients' satisfaction):

Satisfaction with FES orthotic substitute was found to be greater that with AFO, which was reported in earlier studies. (111,118) In this study, patients reported significantly better satisfaction with FES than AFO despite no greater increase in speed and endurance. Van Swigchem et al reported that patients were not more satisfied with donning and doffing the FES, ascending / descending stairs compared to AFO. (119)

Though factors such as comfort, cosmesis, ease of donning were favourable, economic factors have to be considered during prescription of FES in post stroke patients with foot drop. This feedback highlights the need of a low cost FES device for developing countries.

CONCLUSION:

1. Both AFO and FES has significant improvement in gait parameters compared to barefoot walking.
2. FES was statistically significant improvement in walking speed and endurance compared to AFO.
3. There was a trend in reduction of physiological cost index with FES, though not statistically significant.
4. The satisfaction level was higher with FES users.

LIMITATIONS OF THE STUDY:

Present study has a few limitations. The patients were not stratified by gait speed. The duration of the study was limited to training with each device for 7 days. Duration of FES training was for 2 hours a day. The feedback questionnaire which was used was not formally validated.

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ANNEXURES:

1. Institutional review board (IRB) acceptance letter

2. Patient information sheet

3. Informed consent form

4. Patient proforma

5. Patient Data – Excel format

Institutional review board (IRB) acceptance letter

OFFICE OF RESEARCH
INSTITUTIONAL REVIEW BOARD (IRB)
CHRISTIAN MEDICAL COLLEGE, VELLORE, INDIA

Dr. B.J. Prashantham, M.A., M.A., Dr. Min (Clinical)
Director, Christian Counseling Center,
Chairperson, Ethics Committee.

Dr. Anna Benjamin Pulimood, M.B.B.S., MD., Ph.D.,
Chairperson, Research Committee & Principal

Dr. Biju George, M.B.B.S., MD., DM.,
Deputy Chairperson,
Secretary, Ethics Committee, IRB
Additional Vice-Principal (Research)

July 17, 2017

Dr. Gourav Sannyasi,
PG Registrar,
Department of PMR,
Christian Medical College,
Vellore – 632 002.

Sub: **Fluid Research Grant NEW PROPOSAL:**
Comparison of gait with ankle foot orthosis (AFO) and functional electrical stimulation (FES) in patients following stroke.

Dr. Gourav Sannyasi (Emp. No. 21332), PG Registrar, PMR, Dr. George Tharion, PMR, Dr. Navin B. P (Emp. No. 3358), PMR, Dr. Rajdeep Ojha (Emp. No. 31799), Bioengineering.

Ref: **IRB Min. No. 10684 [OBSERVE] dated 01.06.2017**

Dear Dr. Gourav Sannyasi,

The Institutional Review Board (**Blue**, Research and Ethics Committee) of the Christian Medical College, Vellore, reviewed and discussed your project titled "Comparison of gait with ankle foot orthosis (AFO) and functional electrical stimulation (FES) in patients following stroke" on June 01st2017.

The Committee reviewed the following documents:

1. IRB Application format
2. Consent Form and Patient Information Sheets
3. Cvs of Drs. Gaurav, Naveen B, Rajdeep, George Tharion and Ms Gowri.
4. No. of documents 1- 3.

The following Institutional Review Board (Blue, Research & Ethics Committee) members were present at the meeting held on June 01st2017 in the BRTC Conference Hall, Christian Medical College, Bagayam, Vellore 632002.

INFORMATION SHEET FOR INFORMED CONSENT

Study Title: Comparison of gait with Ankle Foot Orthosis (AFO) and Functional

Electrical Stimulation (FES) in patients following stroke.

You are requested to participate in a study which will compare between orthosis [AFO] and FES for the treatment of post stroke foot drop. The final conclusion will be made after completion of the study.

What is foot drop and it's management? Foot drop, also called "drop foot" is the inability to lift the front part of the foot which leads to dragging of the foot along the ground while walking. It can have many different causes which includes stroke, traumatic brain injury, multiple sclerosis, cerebral palsy, peripheral neuropathy. People with foot drop due to stroke usually are provided with an ankle-foot orthosis (AFO), that keeps the ankle in a neutral position while walking. AFO is fabricated from polymers of plastic, it extends from upper $1/3^{rd}$ of the leg to the toes. An AFO is applied over the leg and a shoe can be worn over it. However, the use of functional electrical stimulation (FES) of the nerve supplying muscles of the foot [peroneal nerve] is growing as an alternative treatment option. Ankle is freely mobile with the application of FES, whereas, with an AFO the ankle mobility is passively restricted. FES is a device which electrically stimulates the peripheral nerve so that the foot is moved in the upward direction.

This project will compare between ankle-foot orthosis (AFO) and functional electrical stimulation (FES) for correction of post stroke foot drop.

Does this study have any side effects? You would not face any direct or indirect risks on participating in the study.

If you take part what will you have to do?

If you agree to participate in this study, you will be admitted in our rehabilitation centre for a period of one week.

Initially you will have to:

A. You will have to walk 10 meters and time will be measured for intermediate 6 meters for gait velocity.
B. Endurance will be measured by the distance you walk in a period of 6 minutes on level ground.
C. Your heart rate and the speed at which you walk will be measured, following which the energy required to walk will be calculated this is called the Physiological cost index
D. In the gait analysis lab at the Rehabilitation Institute, other measures like the force generated during the time of push off, speed while walking, length of each step, step clearance ability while you walk will be measured.

You will be trained to walk with an orthosis [AFO] and functional electrical stimulator [FES] for one week each. Following the training, the above measurement will be repeated. If you are already a user of orthosis [AFO] then you will be trained only with FES.

There will be no change in the other treatment and investigations which are advised by your doctor. No blood tests will be required for this study.

Will you have to pay for the study? No

What happens if you choose to withdraw from study participation: Your participation in the study will be voluntary. You are free to withdraw at any point of time from the study. There will be no change in treatment if you choose to withdraw from the study.

What happens after the study is over? We plan to recruit 20 participants in the study. At the end of one year, comparison would be done between the various paramaters obtained among the participants. Following which an interpretation of the results will be done.

Will your personal details be kept confidential? You will be assigned a unique ID while filling the proforma and data entry. Further reference will be in relation to this number. All the data collected from you will be stored in a computer which will be protected by password. The results of this study may be published in a medical journal or at a conference, but your identity will not be disclosed in any manner.

If you have any further questions, please ask:

Gourav Sannyasi

Dept. of Physical Medicine and Rehabilitation

C.M.C. Vellore

Ph: 9432086987, 04162283023

Email: gourav91.cmc@gmail.com

Informed Consent Form for Subjects

Informed Consent form to participate in a research study

Study Title: Comparison of gait with Ankle Foot Orthosis (AFO) and Functional

Electrical Stimulation (FES) in patients following stroke.

Study Number: _____

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age: _____

(Subject)

i) I confirm that I have read and understood the information sheet dated _____

(for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my medical care or legal rights being affected. [ ] (iii) I understand that doctors in CMC, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose. [ ]

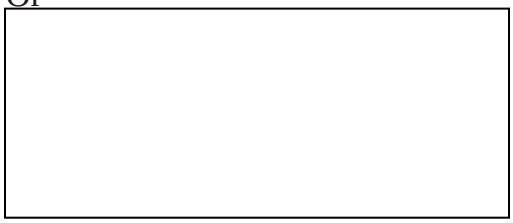
(v) I voluntarily agree to take part in the above study. [ ]

Signature (or Thumb impression) of the Subject/Legally Acceptable

Date: _____/_____/_____

Signatory's Name: _____ Signature:

Or



Representative: _____

Date: _____/_____/_____

Signatory's Name: _____

Signature of the Investigator: _____

Date: _____/_____/_____

Study Investigator's Name: _____

Patient proforma sheet

Study Title: Comparison of gait with Ankle Foot Orthosis (AFO) and Functional Electrical Stimulation (FES) in patients following stroke.

Name-

Patient's hospital no-

Age-

Sex-

Address-

Phone no.-

Duration since stroke-

Type of stroke- Ischaemic/ Haemorrhagic

Anatomical location-

Etiology of stroke-

Handedness- Left/right

Risk factors- Hypertension/Diabetes/Smoking/IHD/Dyslipidaemia/Alcohol/Family history.

ON EXAMINATION:

Pulse:

BP:

Cognitive assessment: Alert, conscious, cooperative.

Cranial nerves:-

Tone- Upper limb-

Lower limb-

Power: **Right** **Left**

Upper limb-

Shoulder

Elbow

Wrist

Lower limb-

Hip- Flexion

Extension

Abduction

Adduction

Knee-

Flexion

Extension

Ankle-

Dorsiflexion

Plantarflexion

Modified Rankin Scale (score)-

Upper limb-

Lower limb-

Additional findings -

Outcome parameters will be measured at baseline, with AFO and with FES.

Patient Data- Excel form

Name	Protocol -	10 m Walk Test (sec)			Speed	m/sec		6 min walk test (meters)			TUG (seconds)		
	BAF-1/BFA-2	B	A	F	B	A	F	B	A	F	B	A	F
1	1	12.05	11.18	10.76	0.8	0.9	0.92	370	400	412	10.8	12.5	11.9
2	1	39.42	37.09	35	0.25	0.26	0.28	97	104	111	37.5	39	34
3	2	35.09	33.1	31.9	0.28	0.3	0.31	108	112	124	38.9	37	36.1
4	2	12	11.1	9.5	0.8	0.9	1.05	290	342	373	11.61	10.8	10
5	2	24.8	23.2	22.1	0.4	0.43	0.45	152	159	165	26.4	25.1	23.8
6	2	35.09	28.7	25.4	0.28	0.34	0.39	110	124	136	27.35	25	22.12
7	1	44.27	41.5	39	0.22	0.24	0.25	88	98	100	37.5	39	34
8	1	27.34	26.04	25	0.36	0.38	0.4	130	138	147	25.87	24.32	23
9	1	29.7	28.9	30	0.33	0.34	0.33	130	134	139	15.88	14.09	13.6
10	1	42.66	40.2	43.98	0.23	0.24	0.22	80	87	77	43.8	44.9	42.5
11	2	26	29.8	24.9	0.38	0.33	0.4	138	139	154	28	27	26
12	2	29.8	24.8	23.1	0.33	0.4	0.43	129	143	150	18.9	17.3	17
13	2	34.09	31	28.2	0.29	0.32	0.35	120	129	140	31.8	28	25.1
14	2	24.1	17.8	19	0.41	0.56	0.52	165	210	204	21	17	17
15	1	25.6	23	20.2	0.39	0.43	0.49	152	160	186	23	21.5	19
16	2	37.8	32.4	28	0.26	0.3	0.35	110	122	143	34.9	32.9	27.8
17	1	36	31	31.4	0.27	0.32	0.31	105	121	117	32.3	31.8	27.6
18	1	34.6	40	45.6	0.28	0.25	0.21	110	88	79	30.2	33	29
19	1	67.3	50	46.8	0.14	0.2	0.21	72	85	90	50	53	47.8
20	2	22	18.4	17	0.45	0.54	0.58	156	187	200	20.2	19	16.5

Name	Protocol -	10 m Walk Test (sec)			Speed	m/sec		6 min walk test (meters)			TUG (seconds)		
	BAF-1/BFA-2	B	A	F	B	A	F	B	A	F	B	A	F
1	1	12.05	11.18	10.76	0.8	0.9	0.92	370	400	412	10.8	12.5	11.9
2	1	39.42	37.09	35	0.25	0.26	0.28	97	104	111	37.5	39	34
3	2	35.09	33.1	31.9	0.28	0.3	0.31	108	112	124	38.9	37	36.1
4	2	12	11.1	9.5	0.8	0.9	1.05	290	342	373	11.61	10.8	10
5	2	24.8	23.2	22.1	0.4	0.43	0.45	152	159	165	26.4	25.1	23.8
6	2	35.09	28.7	25.4	0.28	0.34	0.39	110	124	136	27.35	25	22.12
7	1	44.27	41.5	39	0.22	0.24	0.25	88	98	100	37.5	39	34
8	1	27.34	26.04	25	0.36	0.38	0.4	130	138	147	25.87	24.32	23
9	1	29.7	28.9	30	0.33	0.34	0.33	130	134	139	15.88	14.09	13.6
10	1	42.66	40.2	43.98	0.23	0.24	0.22	80	87	77	43.8	44.9	42.5
11	2	26	29.8	24.9	0.38	0.33	0.4	138	139	154	28	27	26
12	2	29.8	24.8	23.1	0.33	0.4	0.43	129	143	150	18.9	17.3	17
13	2	34.09	31	28.2	0.29	0.32	0.35	120	129	140	31.8	28	25.1
14	2	24.1	17.8	19	0.41	0.56	0.52	165	210	204	21	17	17
15	1	25.6	23	20.2	0.39	0.43	0.49	152	160	186	23	21.5	19
16	2	37.8	32.4	28	0.26	0.3	0.35	110	122	143	34.9	32.9	27.8
17	1	36	31	31.4	0.27	0.32	0.31	105	121	117	32.3	31.8	27.6
18	1	34.6	40	45.6	0.28	0.25	0.21	110	88	79	30.2	33	29
19	1	67.3	50	46.8	0.14	0.2	0.21	72	85	90	50	53	47.8
20	2	22	18.4	17	0.45	0.54	0.58	156	187	200	20.2	19	16.5

SUB	Protocol -	PCI			Stride Length (Paretic) cm			Stride Length (Non Paratic) cm		
	BAF-1/BFA-2	Barefoot	AFO	FES	B	A	F	B	A	F
1	1	1.092	0.757	0.609	97	66	91	98	65	89
2	1				75	62	58	76	63	23
3	2	4.173	4.173	4.173	31	33	40	32	49	40
4	2	0.415	1.275	0.632	89	##	87	62	102	52
5	2	0.793	0.749	1.272	98	49	78	101	87	75
6	2	1.729	0.552	0.792	50	60	70	49	69	71
7	1	9.586	3.073	3.6	21	39	26	28	47	25
8	1	9.231	3.225	3.37	21	38	27	36	40	44
9	1	1.818	1.422	1.128	32	39	38	57	60	61
10	1				52	47	55	6	29	22
11	2	1.155	0.686	0.319	60	34	62	61	41	61
12	2	0.59	1.009	0.797	42	85	90	50	52	83
13	2	0.773	0.422	0.938	65	51	70	65	51	67
14	2	0.843	0.851	1.172	48	##	63	81	116	98
15	1	0.502	1.17	0.664	59	67	65	59	62	64
16	2	2.036	0.729	1.191	49	51	53	49	59	54
17	1	3.406	3.693	1.532	47	33	56	47	55	37
18	1	3.033	3.252	2.114	43	13	55	38	53	56
19	1	2.157	2.136	1.983	21	27	37	26	35	37
20	2	1.052	1.137	0.637	73	78	72	59	82	54

Name	Protocol -	Normallized Stride Length (Paretic) cm			Normallized Stride Length (Non Paretic) cm		
	BAF-1/BFA-2	nslp_b	nslp_a	nslp_f	nslnp_b	nslnp_a	nslnp_f
1	1	0.584	0.397	0.547	0.587	0.39	0.533
2	1	0.433	0.362	0.335	0.442	0.364	0.134
3	2	0.187	0.196	0.237	0.191	0.296	0.238
4	2	0.557	0.632	0.541	0.385	0.637	0.327
5	2	0.585	0.291	0.462	0.602	0.519	0.446
6	2	0.302	0.362	0.422	0.297	0.42	0.432
7	1	0.134	0.253	0.167	0.184	0.307	0.16
8	1	0.128	0.238	0.17	0.224	0.251	0.276
9	1	0.189	0.227	0.224	0.332	0.35	0.357
10	1	0.288	0.261	0.302	0.033	0.158	0.123
11	2	0.37	0.207	0.38	0.375	0.25	0.373
12	2	0.248	0.53	0.498	0.294	0.308	0.486
13	2	0.384	0.301	0.409	0.383	0.301	0.396
14	2	0.269	0.632	0.353	0.452	0.647	0.547
15	1	0.342	0.392	0.376	0.344	0.36	0.373
16	2	0.305	0.313	0.328	0.305	0.365	0.332
17	1	0.28	0.196	0.336	0.279	0.329	0.221
18	1	0.241	0.073	0.307	0.215	0.298	0.311
19	1	0.122	0.162	0.221	0.157	0.207	0.216
20	2	0.458	0.489	0.451	0.372	0.513	0.34

Name	Protocol -	Step Width (Paretic) cm			Step Width (Non Paratic) cm		
	BAF-1/BFA-2	swp_b	swp_a	swp_f	swnp_b	swnp_a	swnp_f
1	1	10	10	16	20	11	8
2	1	8	8	11	8	8	10
3	2	10	6	8	7	9	8
4	2	20	22	12	20	18	14
5	2	8	2	9	7	6	8
6	2	5	9	8	6	14	6
7	1	13	8	3	13	7	8
8	1	3	4	5	4	6	6
9	1	8	10	9	6	7	9
10	1	3	5	3	3	4	3
11	2	8	7	8	12	8	7
12	2	8	11	7	8	7	10
13	2	9	9	8	7	6	10
14	2	4	11	7	4	15	12
15	1	8	8	6	4	3	7
16	2	5	8	10	8	9	10
17	1	5	3	6	15	3	7
18	1	3	4	5	6	4	2
19	1	4	2	3	2	3	3
20	2	[redacted]					18

Name	Protocol -	% Single Limb Support (Paretic)			% Single Limb Support (Non Paratic)			Walking speed (Paratic) m/min			Walking speed (Non Paratic) m/min		
	BAF-1/BFA-2	slsp_b	slsp_a	slsp_f	slsnp_b	slsnp_a	slsnp_f	wsp_b	wsp_a	wsp_f	wsnp_b	wsnp_a	wsnp_f
1	1	33	27	29	40	37	45	47	35	41	52	24	40
2	1	19	20	21	27	27	28	15	15	13	16	15	8
3	2	17	19	25	31	19	30	11	18	15	10	18	31
4	2	34	32	33	33	34	35	42	49	40	27	48	37
5	2	30	22	27	30	15	31	27	47	21	28	28	21
6	2	22	23	25	31	32	33	18	24	21	16	22	21
7	1	14	11	13	20	17	19	7	9	7	9	10	7
8	1	12	13	13	15	17	21	5	7	6	6	8	8
9	1	22	27	24	31	30	36	19	23	17	20	21	20
10	1	4	8	9	15	15	20	7	6	8	1	5	19
11	2	25	20	25	33	25	31	28	18	25	26	18	25
12	2	23	28	28	30	39	39	21	32	31	22	30	32
13	2	16	21	22	23	31	31	14	19	18	15	18	18
14	2	19	27	28	52	45	44	21	41	34	23	42	37
15	1	20	23	22	41	36	46	24	25	27	25	25	26
16	2	21	19	24	31	32	42	18	19	21	17	21	22
17	1	15	18	13	22	24	26	10	14	14	10	13	12
18	1	13	15	17	27	19	23	16	4	12	6	8	11
19	1	12	13	18	17	21	20	5	8	8	7	7	21
20	2	29	28	28	37	38	36	24	24	25	43	24	18