EFFECTS OF NEUROMUSCULAR STIMULATION AND CYCLE ERGOMETRY ON FUNCTIONAL ABILITIES OF STROKE SURVIVORS IN KANO STATE, NIGERIA

BY

Abdullahi Sule DAMBATTA

DEPARTMENT OF PHYSICAL AND HEALTH EDUCATION,
FACULTY OF EDUCATION,
AHMADU BELLO UNIVERSITY,
ZARIA

OCTOBER, 2016

EFFECTS OF NEUROMUSCULAR STIMULATION AND CYCLE ERGOMETRY ON FUNCTIONAL ABILITIES OF STROKE SURVIVORS IN KANO STATE, NIGERIA

BY

Abdullahi Sule DAMBATTA (B. Sc, M.Sc Exercise &Sports Science (BUK)
PhD/8373/EDU/2010-2011/ P15EDPE9001

A THESIS SUBMITTED TO THE SCHOOL OF POSTGRADUATE STUDIES, AHMADU BELLO UNIVERSITY, ZARIA, NIGERIA

IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF DOCTOR OF PHILOSOPHY (PhD) IN EXERCISE AND SPORT SCIENCE

DEPARTMENT OF PHYSICAL AND HEALTH EDUCATION,
FACULTY OF EDUCATION,
AHMADU BELLO UNIVERSITY,
ZARIA

OCTOBER, 2016

DECLARATION

I hereby declare that this thesis, titled **"Effects of Neuromuscular Stimulation and Cycle Ergometry on Functional Abilities of Stroke Survivors in Kano State, Nigeria"** is written by me under the supervision of Prof. E. A. Gunen, Prof. M. A. Chado and Prof. J. O. Ayo; and that it is a record of my own research work. It has not been presented in a previous application for a higher degree. All quotations have been indicated in the text and the sources of information are specifically acknowledged in the reference section.

---------- ----------

Abdullahi Sule DAMBATTA Date

CERTIFICATION

This thesis titled: **"Effects of Neuromuscular Stimulation and Cycle Ergometry on Functional Abilities of Stroke Survivors in Kano, Nigeria"** by Abdullahi Sule DAMBATTA meets the regulations governing the award of the degree of Doctor of Philosophy (PhD) in Exercise and Sport Science, Ahmadu Bello University, Zaria, and is approved for its contribution to knowledge and literary presentation.

---------- ----------
Prof. E. A. Gunen Date
Chairman, Supervisory Committee

---------- ----------
Prof. M. A. Chado Date
Member, Supervisory Committee

---------- ----------
Prof. J. O. Ayo Date
Member, Supervisory Committee

---------- ----------
Prof. (Mrs.) T. N. Ogwu Date
Head of Department

---------- ----------
Prof. K. Bala Date
Dean, School of Postgraduate Studies

DEDICATION

To all stroke survivors in developing countries like Nigeria,

for their hope and belief that life never ends with stroke.

AKNOWLEDGEMENTS

The researcher is indebted to every member of the family, friends and well-wishers for their encouragement, love, prayers and moral support. If the researcher has seen far than near, it is by leaning on the shoulders of the elders. It is with deep gratitude that the researcher appreciates the supervisory roles, mentorship and guidance of Prof. E.A. Gunen, Prof. M.A. Chado, Prof. K. Venkarteswarlu and Prof. J. O. Ayo for the time and patience taken to critique, guide and correct this work.

The researcher is grateful to all staff of the Departments of Physical and Health Education (P.H.E), Human Physiology and Veterinary Physiology, Ahmadu Bello University, Zaria; The Head, Department of Physical and Health Education (Prof. T. N. Ogwu); the Coordinator of Internship Programme (Prof. F.B. Adeyanju); the Post-graduate Coordinator (Prof. M. A. Suleiman) Prof. A. I. Kabido, Prof. E. J. Chom, Prof. V. Dashe, Dr. M. Umaru, Dr. M. Aliyu, Dr. B. W. Zamani, Prof. M. U. Kawu, Dr. M. Aliyu, Dr. R. A. Magaji, Prof. K.V. Olorunshola and Prof. M. G. Yakasai for their advices, motivation and support to the researcher in various ways.

Sincere appreciation is owed to Prof. C. E. Dikki, who has been very receptive to the researcher; a mountain of knowledge, rare humility and a father indeed. The researcher appreciates the input, mentorship and kindness of Prof. J. A. Gwani.

Appreciation is owed to the non-academic staff of the department, Mrs M. Ekeleme; Mr. A. Damboyi, Mal. S. Shuaibu, Mr. M. Thomas, Mal. N. Ibrahim and Mr. M. Tanko whose energy and assistance in various capacities made this study comfortable.

The researcher appreciates the Head, Department of Physiotherapy, Aminu Kano Teaching Hospital, Kano, Dr. P. Kodzo; Dr. Jovita A. Daniel, Dr. Bashir Kaka, Dr. A. Kabir (Spine and Orthopaedic consultant), Dr. F. Kabir , Dr S.B Jibrin, Dr. L. A. Kuki, Dr. G. D. Waziri, Alhaji M. Ahmadu, Hajia F. Maikudi and Major M. L. Rano to mension a few, your roles in various ways from the admission, encouragement and support are most inspiring.

All the Staff of Murtala Mohammed Specialist Hospital, Kano, Aminu Kano Teaching Hospital, Kano, Bayero University, Kano and Excellence Physiomed Services, Kano are highly appreciated. in their supports in various capacities.

The researcher appreciates the support, patience and commitment of the participants, whose cooperation, commitment, punctuality and understanding made this work successful. To all the research assistants: Mr. M. Tasiu, Mr. M. Khalifa, Mr S. Abubakar and Mal S. Mohammed; and the printing limited, Master Daniel (Asheolege) and Ado Café, God bless you.

ABSTRACT

This study investigated effects of neuromuscular (NMS) and cycle ergometry (CE) on functional abilities of stroke survivors in Kano State, Nigeria which lasted for 12 weeks. Among the parameters assessed were strength (grip and pinch strength), joints flexibility (range of motion: ROM); that include shoulder, elbow and wrist joints extension while the lower limb include hip extension, knee flexion and ankle dorsiflexion. The cardio-respiratory indices of the participants investigated were systolic blood pressure, diastolic blood pressure, pulse rate and oxygen saturation. Stroke survivors were exposed to neuromuscular stimulation and cycle ergometry exercises three times per week on alternate days for duration of 12 weeks. Training intensity was maintained at 35-45%, 50-55% and 60-65% of their target heart rate for 20, 25 and 30 minutes respectively. The research design used for this study was 2x3x 4 factorial design. A population of 40 male and female subjects participated in the study. Consecutive sampling technique was used to place the participants into two groups (NMS and the CE) respectively of which only 25 reached the 12^{th} week of the study. The data collected was analyzed using descriptive statistics of mean and standard deviation for the demographic data and to answer the research questions. Repeated measures analysis of variance and scheffe post-hoc were used to analyze the null hypotheses. Based on the results of this analysis, it was concluded that NMS and CE significantly improved grip and pinch strength among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$). NMS and CE had significant effect on range of motion of shoulder extension, elbow extension, wrist extension, hip extension, knee flexion and ankle dorsiflexion among stroke survivors in Kano State, Nigeria ($P < 0.001$). NMS and CE caused significant reduction in both the systolic and diastolic blood pressures among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$). NMS and CE had significant reduction on the pulse rate of stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$). NMS and CE had significantly improved the SPO_2 of stroke survivors in Kano State, Nigeria ($P < 0.001$). NMS and CE had significantly improved the coordination of stroke survivors in Kano State, Nigeria ($P < 0.001$). **Ba**sed on the findings, it was recommended that Cycle ergometry could be adopted as a mode of training for improving the functional abilities of

stroke survivors in Kano State, Nigeria. For better improvement in flexibility, both the CE and NMS are recommended as a reliable training protocol in the rehabilitation of stroke patients in Kano State, Nigeria. Training duration for either of the NMS and CE should be continuous for at least a period of 12 weeks for optimum benefits of stroke patients in Kano State, Nigeria.A combination of both NMS and CE training modes are recommended in the rehabilitation of stroke patients in Kano State, Nigeria.

TABLE OF CONTENTS

CHAPTER TWO

CONTENTS **PAGE**

CHAPTER THREE

CONTENTS **PAGE**

CHAPTER FOUR

CHAPTER FIVE

LIST OF TABLES

TABLE **PAGE**

TABLE **PAGE**

LIST OF PLATES

PLATE **PAGE**

LIST OF APPENDICES

CHAPTER ONE

1.0 INTRODUCTION

1.1 Background of the Study

Stroke is a clinical condition that impacts on the life of it survivors, forcing a change in lifestyle and reconstructing their identity. Stroke survivors are usually deconditioned and predisposed to sedentary lifestyle which limits their performance of physical activities of daily living (ADL) (Ellis, Hill & Horn, 2000). The likelihood of a recurrent stroke is associated with continued unhealthy lifestyle (Hoeing, Nusbaum & Brummel–Smith, 1997). Increased unhealthy lifestyle among stroke survivors worldwide has led to stroke becoming the leading cause of death and disability by 2020 (Khuram, 2011). Stroke is one of the most common causes of disability worldwide and the fourth most common neurological disorder after headache, epilepsy and peripheral neuropathy. The prevalence of stroke is increasing geometrically in Africa (Owolabi, 2011). Hamzat and Olaleye (2002) reported that the prevalence of stroke in the African population ranges from 0.9% to 4.0% accounting for 6.5% to 41% of neurological admissions and about 4% to 9% of hospital deaths among black Africans. Stroke occurs due to cell damage and impaired neurological function resulting from restricted blood supply (ischaemia) or bleeding (haemorrhage) into the brain tissue. The injury affects motor and sensory inputs, language, perception, affective and cognitive dysfunctions (Stewart, 1999). Stroke survivors incur considerable costs in health and social services (Evers; Struijs; Ament; Van Genugten; Jager & Vanden Bros, 2004). The paralysis is usually on the side of the body opposite the side of the brain damaged by stroke, and may affect the face, an arm, a leg, or the entire side of the body. This one-sided paralysis is called *hemiplegia* (one-sided weakness is called *hemiparesis*). Stroke patients with hemiparesis or

hemiplegia may have difficulty with everyday activities such as walking or grasping objects (NINDS, 2014). The commonest presentation of stroke is weakness of one side of the body called hemiplegia. There are other presentations such like monoplegia, diplegia, paraplegia and teteraplegia (Evers *et al.,* 2004). This study used participants with left sided hemiplegia to limit the problem of communication during the training.

Most stroke survivors live with residual physical impairments, which may promote a sedentary lifestyle with the resultant secondary complications (Camels *et al.,* 2011). One of such secondary complications commonly observed following a stroke is poor cardio-respiratory fitness (Chu *et al.,* 2004). Low cardio respiratory fitness is related to poor functional performance and low energy expenditure (Hamzat, 2003), as well as increased risk of stroke and cardiovascular disease (Kurl *et al.,* 2003). Ischaemic stroke is defined as an episode of neurological dysfunction caused by focal cerebral, spinal, or retinal infarction; with central nervous system infarction due to ischaemia, based on pathological, imaging, or other objective evidence of cerebral, spinal cord or retinal focal ischaemic injury based on symptoms that persists not less than 24 hours or until death and other aetiologies excluded (Sacco *et al.,* 2011). Stroke caused by intracerebral haemorrhage as a rapidly developing clinical signs of neurological dysfunction attributable to a focal collection of blood within the brain parenchyma or ventricular system that is not caused by trauma. This definition does not exclude silent cerebral haemorrhage defined as a focal collection of chronic blood products within the brain parenchyma, subarachnoid space, or ventricular system on neuro-imaging or neuropathological examination that is not caused by trauma and no history of acute neurological dysfunction attributable to the lesion (Saccoet al., 2011).

Physical inactivity has significant health and functional consequence for survivors of stroke, including an increased risk of a second stroke and mortality (Hormneset al., 2010). Inactivity exacerbates the normal decline in aerobic fitness, putting the cardiovascular capacity of most post-stroke survivors below the level needed for activities of daily living (Mackoet al., 2005; Michealet al., 2005). This inactivity often leads to reduced endurance and strength (Newham and Hsaio, 2001), which are considered as secondary complications. Reduced endurance and strength do in long run directly affects the levels and regular participation in physical activity by stroke survivors (Fujitanet al., 1999). Participation in safe, easy and less expensive physical activity such as cycling post discharge, at home and in clinics may complement the existing therapies in hospitals if proven effective. Stroke survivors experience exhaustion, which may be due to respiratory insufficiency, resulting from low pulmonary diffusing capacity because of mismatched ventilation-perfusion, reduced lung volumes and poor oxygen saturation (SPO_2) (Bjuro, FuglMeyer & Grimby, 1975). It is believed that disuse and atrophy of the exaggerated respiratory and expiratory muscles may compromise functional quality in stroke survivors.

Training can be defined as the systematic and regular participation in exercise to enhance performance (Billat, 2001). Training Programmes like cycle ergometry (CE) and neuromuscular stimulation (NMS) are designed to improve performance of daily living by developing the appropriate energy sources, increasing muscular strength of the affected muscle groups and improving neuro-muscular skill patterns. Aerobic training develops the aerobic energy system aims to increase cardio-respiratory efficiency (the ability of the body to deliver and utilize oxygen, reduce coronary heart disease and improve general health) and assist to improve neuromuscular functions and weight control (Billat, 2001). Stroke

survivors may benefit from continual motor learning and re-learning of cycling and neuromuscular stimulation, which would help to improve the neurological deficits and quality of life.

1.2. Statement of the Problem

Stroke, also called apoplexy or cerebrovascular accident (CVA) occurs when part of the brain is damaged as a result of lack of blood supply called ischaemia or rupture of blood vessel referred to as haemorrhage (Lawrence & Brass, 1992). Stroke is the second most widespread cause of death and chief cause of disability worldwide and its disability and outcomes depend on the severity of neurological deficit (Donna; Fisher; Macleod & Davic, 2008). Most stroke survivors needhelp in physical activities of daily living, which is usually provided by family members, health system or sometimes social institutions (Agarwal, Kobetic, Nandurkar & Marsolais, 2003). Even with complete stroke rehabilitation, 10% of survivors will have severe disability, 11% may have moderate disability, and 78% only minimal disability, while only 1% will recover completely without any deficit (Kobetic *et al.*, 2003). The incomplete healing rates may be due to the fact that stroke paralyzed muscle fibres are not put into use in physical activities of daily living (ADL) and they are liable to atrophy and become weaker and less fatigue resistant.

Participation in regular physical activity can prevent recurrent stroke as approximately 30% of individuals with stroke are at risk of sustaining a second stroke ((Gordon *et al.,* 2004; Kelly *et al.,* 2011). Physical activity (PA) involvement is of paramount importance because few older adults with stroke do not achieve the recommended PA level of 1,000 kcal per week ((Paterson *et al.,* 2007; Sawatzky, 2007) and they undertake much lower levels of PA compared to healthy individuals possibly due to their motor impairment (Acree *et al.,* 2006).

Stroke survivors are often deconditioned and predisposed to sedentary lifestyles that limit performance of activities of daily living.

Stroke survivors demonstrate some residual loss of function on either the right or left side of the body (Macko, Desouza & Tretter, 1997) and frequently have their vascular system at heightened risk and linked comorbid cardiovascular disease (CVD) (Roth, 1993; Wolf, Clagett & Easton, 1999). This has caused the use of robotic tools for delivering motor improvement among stroke survivors (Krebs *et al.,* 2006; Lonn, Yusuf & Arnold, 2006). The modification of multiple risk factors through a combination of comprehensive lifestyle interventions, prescribed exercises and pharmacological therapy are now acknowledged as the cornerstone of prevention of recurrent strokes and acute cardiac events in stroke survivors (Ivey *et al.*, 2004).

The broad fact on stroke survivors showed that they report decreased quality of life and wellbeing (Clarke, 2002), leading to high episodes of depression (Lee, Im, Kim, Choi, Lee & Han, 2012). More so, most stroke survivors cannot maintain rehabilitation for long in the hospital and even after discharge due to financial constraints. In addition, more attention is often given to investigations and pains management than exercise rehabilitation.

Exercise training, irrespective of the mode adapted, produces responses and adaptation of the body systems. In the field of neurorehabilitation, more emphasis has been placed on the responses and adaptation of macrophysiological variables and physical features for advancement such as ambulation than on micro and fine motor tasks like grip, pinch and cardiopulmonary indices (Letombeet al., 2010; Tolendanoet al., 2011).

There is the need to educate stroke patients on regular training using simple available modalities. What is needed is that the patient can use between clinic visits and after discharge to foster and maintain improved muscle balance. Ideally, such tasks should play multiple characteristics and should use the motor learning principle which improves symmetry of movement and with velocity like that of normal walking.

There is a decline in muscle strength, loss of bone mass and decrease flexibility in joints of stroke survivors (Van Furth, 1982; Stary, 1983; Assoian, Grotendorst & Miller et al., 1984). It has been observed that planned and supervized exercise programmes involving joint flexibility for the aged improved joint range of motion (Chesebro, Zoldelyi & Fuster, 1991), which improves the functional abilities of stroke survivors.

InNigeria, few studies have used neuromuscular stimulation and cycle ergometry to improve the neurological deficits associated with stroke survivors. Most treatment methods are not harmonized and focused. Stroke management should be highly effectivein proprioceptive facilitation and utilize equipment that are relatively low in cost and can be used with minimal dependence. Pedaling the limb in CE and NMS in seating and lying positions respectively may satisfy these assumptions.

In most rehabilitation centres, a large percentage of stroke care-givers are surgeons and physicians who focused more on modalities and investigations to manage pains and other complications than on early exercise rehabilitation. Most therapeutic trainings are single limb based, but studies now indicate that sensorimotor activity in one limb may affect the motor output of the opposite limb. Bilateral training yields better increase in mobility than conventional rehabilitation protocols (Westlake & Nagarajan, 2011). To the knowledge of

the researcher, NMS and CE may have been used but not reported to improve grip and pinch strength, flexibility (Joint ROM), coordination (block-box test) and cardio-respiratory indices (BP, PR and SPO_2), However, paucity of information and data on stroke survivors still exists in Nigeria. This study was therefore conducted to find out the effects of NMS and CEprogrammes on the strength, flexibility, coordination and cardio-respiratory performance of stroke survivors in Kano State, Nigeria.

1.3. Research Questions

The study was conducted to answer the following research questions:

i. Will NMS and CE improve the gripstrength of stroke survivors in Kano State, Nigeria?
ii. Will NMS and CE improve the pinch strength of stroke survivors in Kano State, Nigeria?
iii. Will NMS and CE improve flexibility (range of joints movement) of upper and lower limbs of stroke survivors in Kano State, Nigeria?
iv. Will NMS and CE improve systolic and diastolic blood pressures of stroke survivors in Kano State, Nigeria?
v. Will NMS and CE improve the pulse rate of stroke survivors in Kano State, Nigeria?
vi. Will NMS and CE improve the SPO_2 of stroke survivors in Kano State, Nigeria?
vii. Will NMS and CE improve coordination of stroke survivors in Kano State, Nigeria?

1.4. Basic Assumptions

The following assumptions were made for the purpose of this study:

1. NMS improves excitation and increases circulation in paralyzed muscles.

2. Cycle ergometry training improves the muscular strength and mobility in the joints of the lower and upper limbs as well as endurance of stroke survivors.

3. Motor learning and relearning stimulates neuroplasticity and neurodynamism.

4. Exercises prevent cardiovascular diseases in general population.

5. Task-oriented activity such as box and block activity improves coordination.

1.5 Hypotheses

The following hypotheses were raised to guide the conduct of this study.

1.5.1 Major Hypothesis

There are no significant effects of NMS and CE on the strength, flexibility (range of movement: ROM), coordination and cardio-respiratory indices (systolic and diastolic blood pressures, pulse rate and SPO_2) of stroke survivors in Kano State, Nigeria.

1.5.2. Sub-Hypotheses

i. There is no significant effect of NMS and CE exercises on the grip strength of stroke survivors in Kano State, Nigeria.

ii. There is no significant effect of NMS and CE exercises on the pinch strength of stroke survivors in Kano State, Nigeria.

iii. There is no significant effect of NMS and CE exercises on the joint ROM of stroke survivors in Kano State, Nigeria.

iv. There is no significant effect of NMS and CE exercises on systolic and diastolic blood pressure of stroke survivors in Kano State, Nigeria.

v. There is no significant effect of NMS and CE exercises on the pulse rate of stroke survivors in Kano State, Nigeria.

vi. There is no significant effect of NMS and CE exercises on the SPO_2 of stroke survivors in Kano State, Nigeria.

vii. There is no significant effect of NMS and CE exercises on the coordination of stroke survivors in Kano State, Nigeria.

1.6. Significance of the Study

Many studies have been conducted on stroke survivors with emphasis on neuro-developmental theories, pharmacological agents and NMS. Neural changes in the brain occur with NMS due to increased activity in the limbs. It is also believed that repeated stimulation of muscles may cause habituation of reciprocal inhibition in the antagonist muscles leading to a reduction in plasticity, thus increasing strength and preventing disuse. However, this training modality may be expensive and may not be easy to use at home post discharge. There is the need to compare the effects of NMS with that of cycle ergometry exercise on grip and pinch strength, flexibility of the joints of upper and lower limbs, coordination and selected cardio-respiratory indices such as blood pressure, pulse rate and oxygen saturation of stroke survivors. The results of this study will therefore show whether NMS and CE will give similar improvements on the variables.

It is also hoped that the findings will provide stroke survivors with better knowledge on the management of their conditions and thus enable them assist themselves. The results will improve multidisciplinary intervention in the management of stroke survivors in Kano State, Nigeria. This will create early discharge from the hospital, cost effectiveness, reduce psychological burden on patients, family and well wishers. It may help patients to get

integrated into the society as faster as possible. The results of this study will add to the existing body of literature on the management of stroke survivors using NMS and CE modalities in Kano State, Nigeria.

1.7. Delimitation of the Study

This study was delimited to 30 male and female volunteered stroke survivors between the ages of 40 and 60 years, who had left-sided hemiplegia. The clients were patients referred to Murtala Mohammed Specialist Hospital (MMSH), Kano, Nigeria for stroke rehabilitation.

Training modalities were delimited to the use of NMS and CE at intensity levels of 35-45%, 50-55% and 60-65% of maximal heart rate of each participant. Trainingduration was maintained for 20 minutes between the first and fourth week; 25minutes between the 5^{th} and 8^{th} week and 30 minutesbetween the 9^{th} and 12^{th} week. Training was conducted three times per week on alternate days for 12 weeks. The measurements were delimited to grip and pinch strength, flexibility (ROM), coordination (Block box test) and cardio-respiratory indices (systolic and diastolic blood pressures, pulse rate and peripheral oxygen saturation (SPO_2).

1.8. Limitations of the Study

Participants might have been influenced by their initial levels of fitness, experiences on different training mode and occupations. However, the researcher introduced them to the new training modes and emphasized that the participants train on specified days and modes within the delimited area of study.

CHAPTER TWO

2.0 Review of related literature

2.1 Introduction

This study was aimed at determining the effect of neuromuscular stimulation and cycle ergometry on the functional abilities of stroke survivors in Kano State, Nigeria. The functional abilities of the stroke survivors involved measurement of blood pressure, pulse rate and oxygen saturation as the cardio-respiratory assessment; grip strength, pinch strength

as functional strengths measure; joint range of motions as flexibilities of the joints and block and box tests for coordination among individuals leaving with stroke. Stroke-related changes in the brain that regulate the autonomic function can have significant implications for blood pressure control and cardiac function during the acute phase of stroke recovery (Gresham, Duncan & Stason, 1995; Roth & Harvey, 2000). Not all patients have pulmonary disease after stroke, however, respiration may be compromised as a direct result of stroke (particularly brain stem stroke), connected with weakness of respiratory muscles. The effects of these include impaired breathing mechanics, chronic obstructive pulmonary disease or co-morbidities and lifestyle factors like physical inactivity (Olney; Griffin & Monga, 1991). This chapter reviewed related literature under the following subheadings:

2.2 Epidemiology and Rehabilitation of stroke survivors

- 2.2.1 Pathophysiology of stroke
- 2.2.2 Cerebral Infarction of stroke
- 2.2.3 Intracranial and intracerebral Haemorrhage in stroke
- 2.2.4 Transient ischaemic and clinical manifestation of stroke
- 2.2.5 Neuroplasticity in Stroke
- 2.2.6 Post Stroke recovery and management of stroke

2.3 Physical activity participation for stroke survivors

2.4 Neuromuscular Stimulation training for stroke survivors

- 2.4.1. Effects neuromuscular Stimulation and Strength in Stroke Survivors.
- 2.4:2. Effects of Neuromuscular Stimulation and Flexibility in Stroke Survivors

2.5 Effects of Cycle Ergometry on stroke survivors

2.6. Cardio-respiratory evaluation for stroke survivors

2.7. Training guidelines for stroke survivors

2.7:1. Intensity of exercise for stroke survivors

2.7:2. Target pulse rate for stroke survivors

2.7:3.Rate Pressure Product for stroke survivors

2.7:4. Oxygen saturation for stroke survivors

2.7:5. Rate of perceived exertion for stroke survivors

2.2. Epidemiology and Rehabilitation in Stroke Survivors

Stroke is the second leading cause of death globally, and one of the most common causes of long term disability world-wide. It is estimated that 5.45 million people world-wide die every year from stroke and there are over 9 million stroke survivors. Stroke accounts for nearly 5 million disability-adjusted life years lost all over the world (Bonita, 1997). The pattern of this impact varies with the highest rates in parts of Europe, South-East Asia and the Western Pacific. One in four men and nearly one in five women, aged 45 years can be expected to have a stroke. The risk of dying within three months of stroke is about 80%, but varies according to the sub-type of stroke and initial severity. Two-thirds of deaths due to stroke occur in people living in developing countries and 40% of the subjects are aged less than 70 years. Thus, while many of these countries are struggling with the consequences and problems of communicable diseases, non-communicable diseases are on the rise. In addition to the deaths, many surviving stroke survivors are disabled and need help in the activities of daily living, which must be provided by family members, the health system, or other social institutions (Word Health Organization, 1976).

The incidence of first ever stroke is significantly greater among Blacks (232 per 100,000 population per year for men, and 260 per 100,000 for women) compared with Whites (167 per 100,000 population per year for men, and 138 per 100,000 for women). There are also

important racial differences in mortality rates for ischaemic stroke. The mortality rate for Black men is 87.1 per 100,000 and 78.1 per 100,000 for Black women (Ogun, 2000), whereas the mortality for White men is 58.6 per 100,000 and 57.8 per 100,000 for White women. The precise reasons for these differences are unclear, but genetic, geographic, dietary, and cultural factors have been speculated. The incidence of risk factors for stroke such as hypertension, diabetes, and hypercholesterolaemia differ between racial groups (Howard, 2001). Case fatality rate in Africa averages about 35%, but could be as low as 14.9% or as high as 77% due to cerebral haemorrhage (Odusote, 1996). In Nigeria, stroke is a major cause of neurological admissions and its incidence may be on the increase. The extended hospitalization required in survivors with subsequent inability of many to return to work is a great burden on their families and the community. At the Lagos University Teaching Hospital (LUTH) Lagos, stroke was the commonest cause of neurological admissions, whereas at the Ogun State University Teaching Hospital (OSUTH), Sagamu, it was the third commonest cause of medical admissions. At the University College Hospital (UCH), Ibadan, LUTH, and OSUTH, stroke accounted respectively for 5%, 8%, and 17% respectively of medical deaths (Owolabi & Nagoda, 2012; Ojini & Danesi, 2003). A study by Eze *et al.* (2013) showed that stroke was highly prevalent, constituting 11.6% of medical admissions in Abakaliki south-eastern Nigeria. Case fatality for stroke is generally accepted to be 12% within the first 7 days and 19% at 1 month for first-ever stroke, falling drastically to about 9% per annum after the first 30 days (Vantone, 2002). Stroke therefore appears to be a huge problem in Nigeria and places a major financial burden on the inadequate health services in the country. In Nigeria, cerebral ischaemia (CI) accounted for 64%, intracerebral haemorrhage (ICH) for 19%, and subarachnoid haemorrhage for 6% of all strokes, whilea

changing pattern of increasing frequency of haemorrhagic stroke has been suspected (Ogun *et al.,* 2005).

Prevention of complications are important perspective of stroke management due to high risk of arrays of complications like pneumonia, deep vein thrombosis or phlebothrombosis and pressure sores (Indredavik *et al.,* 2008; European Stroke Organization, 2008). Langhone *et al.* (2000) conducted a study among 311 stroke subjects, to determine the frequency of symptomatic complications up to 30 months post stroke. The complications include neurological- recurrent stroke, epileptic seizures; urinary tract infection, chest infection; mobility related-falls, falls with serious injury, pressure sores, deep vein thrombosis, pulmonary embolism,; pain- shoulder and other pain types ; psychological depression, anxiety and confusion. Stroke is a universal epidemic and not only limited to affluent or high income country (Mathers *et al.,* 2006). About 85% of all stroke deaths are recorded in low and middle income countries and also accounts for 87% of entire losses due to stroke in terms of disability-adjusted life years (DALYs) (Mathers *et al.,* 2006). Mortality is expected to be faster in low and middle-income countries (LMIC) like in Africa of which Nigeria is among; due to increased prevalence of risk factors and availability of primary prevention and acute care programs (Strong *et al.,* 2007; Owolabi *et al.,* 2012).

The World Bank redefined economies based on Gross National Income (GNI) per capital, thus, categorizing Nigeria as LMIC with GNI range from $1,046 to $4,125 (World Bank, 2015). This burden of stroke could likely boost over the next few decades in LMIC due to epidemiological transition from the current dominance by infectious conditions and poverty related diseases (Yusuf *et al.,* 2001). The economic burden of stroke is defined as the direct cost of providing medical care to stroke survivors and the indirect costs related to lost

productivity (Taylor, 1996). The impact of stroke on the local economy and financial burden in Nigeria is yet to be estimated (Ogunbo *et al.*, 2005), although it was stated that each year, about 90.000 new cases of stroke will be recorded in Nigeria. It was also stated that it will cost $55million to look after stroke survivors, if they are managed in the public hospital; and it costs a half a million dollars per patient and if managed in a private hospital for a year (The Guardian Newspaper, 25th July, 2015). The Nigerian economy may be more challenged as more resources will be channeled to stroke management in a situation of limited economic stability, hence prevention is a major issue and cheap affordable rehabilitation tools are necessary (Strauss *et al.*, 2002).

A stroke is certainly not an accident and it is often the result of decades of wear and furring up of the blood vessels supplying the brain. Stroke can occur at any age, but is more common in the elderly, and people with particular lifestyles (Bonita & Beaglehole, 2007).

A stroke risk factor is something that increases ones chances of developing a stroke. There a two categories of risk factors of stroke; the modifiable risks factors and non-modifiable risk factors. Examples of modifiable risks factors include; hypertension, diabetes and obesity, while that of non- modifiable factors includes; age, family history and ethnicity. Certain types of stroke have slight differences in associated risk factors. For example, obesity is associated with cardiovascular risk leading to ischemic strokes, while hypertension is linked to both ischemic and hemorrhagic stroke (Goldstein *et al.,* 2001). Non-modifiable risk factors include all risk factors for stroke that cannot be affected by individual behaviour. These factors include age, gender, family history, ethnicity, and disorders for which there is no known direct cause (Sacco *et al.,* 1997).

Age is an important risk factor that cannot be modified. Most individuals who have a stroke are over 65years, although the average age of stroke victims continues to decrease (Public Health Agency of Canada (PHAC, 2012). The chance of having a stroke approximately doubles for each decade of life after age 55, while stroke is more common among the elderly, an increasing number of people under 65 are also having strokes (Asplund *et al.,* 2009). Telman *et al.* (2010) conducted a study of 656 stroke and transient ischaemicmic attack (TIA) cases in Israel from 2001 to 2008 that were under the age of 65. They found that the mean age of TIA and stroke cases were 50.6 ± 7.5 years and 51.3 ± 7.6 years, respectively. No age is exempt from stroke and when combined with more controllable risk factors such as hypertension, the combination is more important than age alone (Goldstein *et al.,* 2001; O'Donnell *et al.,* 2010).

Family history has been found to be a factor in ischemic as well as hemorrhagic strokes. Hereditary factors may be genetic, but environmental and learned life-styles may also play a

part (Meschia, 2006; Dichgans, 2007). Taking learned behaviour into account makes this factor more complicated than genetics alone. Eating, drinking and exercise habits could explain part of why strokes may appear to have a familial connection. Information on the connection between parental strokes and strokes experienced by their offspring has been riddled with conflict regarding the relative contribution of shared genetic and environmental factors (Kiely, 1993). It has been suggested that there is an increased risks for men, whose mother died of stroke; and women who had a family history of stroke. In 1993, the Framingham study searched for a familial link and their results were inconclusive regarding the role of environmental and genetic influences on familial tendencies for stroke (Kiely *et al.,* 1993).

Arrhythmias of the heart, valvular disease, previous myocardial infarction, and congenital heart defects are noted as significantly increasing the risk of stroke or transient ischemic attack (Goldstein *et al.,* 2001; Dean *et al.,* 2004). Atrial fibrillation alone accounts for up to 15% of the strokes seen in Canada, and this number increases up to one third over the age of 60 (Heart and Stroke Foundation of Canada, 2010).

Stroke incidence and mortality rates vary widely between racial groups; blacks are more than twice likely to die of stroke than whites (Go *et al.,* 2010). In the National Centre for Health (NCH, 2010) statistical update, the report on prevalence of stroke by ethnicity shows 1.8% for Asians, 2.7% for Caucasians, 3.6% for Afro-Americans, and 3.9% for American Indians and residents of Alaska (Go *et al.,* 2010). No prevalence of stroke for Hispanics was reported in that study. From a public health perspective, the National Health and Nutrition Examination Survey (NHANES, 2014) identified that the control (management) of high blood pressure is not as common in Afro-Americans as it is in Caucasian populations even

with the detection and treatment available for hypertension for both groups (Howard *et al.*, 2006).

Modifiable risk factors can be controlled or eliminated through lifestyle changes, exercise and/or the use of pharmaceutical intervention.

Hypertension is defined as systolic blood pressure > 140 mm Hg & diastolic > 90 mm Hg by the Joint National Committee on Prevention, Detection, Evaluation and Treatment of High Blood Pressure (JNC IV, 1997). Uncontrolled hypertension, defined as blood pressure greater than l40/90 mm Hg for an extended period, is an important modifiable risk factor for stroke. Hypertension increases the risk of stroke by two to more than four times, independent of other risk factors (Dobesh, 2006; Powers *et al.*, 2008; Haseqawa, 2010; Nguyen *et al.*, 2010). This increased risk is a result of adaptive structural changes in the blood vessels increasing peripheral vascular resistance that may compromise circulation and enhance the risk for ischemic events (Woo *et al.,* 2004). Hypertension also increases the risk of intracerebral hemorrhage resulting from damage to the fragile walls of the small arteries in the deeper areas of the brain (Woo *et al.*, 2004). Long term ccontrol of hypertension long term can result in a 33% decrease in the risk of stroke (Goldstein *et al.*, 2001). Control of hypertension may require lifestyle changes such as increased physical activity, weight loss, alcohol moderation, smoking cessation, lower fat and salt in diet as well as the use of blood pressure lowering medications (Canadian Hypertension Education Programme (CHEP, 2013). Adhering to such a regimen is difficult for many patients and statistics suggests that only about half of those who have prescribed medications actually take them consistently (Besthor *et al.*, 2008; Haseqawa, 2010).

The microvascular and macrovascular complications of diabetes create one of the most significant risk factors for ischemic stroke (CSN, 2011). The Honolulu Heart Program was fundamental in establishing the risk of stroke related to diabetes in a study conducted from 1965 to 1968 involving over 7000 participants and a 12-year follow up period (Abbott *et al.,* 1987). They showed a 2.5-time greater risk of stroke in cases with diabetes, but at that time, it was only a suggestion that the reason for the increased risk was due to atherosclerosis. The confirmation of this suggestion came in the years to follow with further research (Burchfiel *et al.,* 1998). Chronic high blood glucose was shown to lead to the development of atherosclerosis and subsequently stroke risk. Type II diabetes is preventable and can be controlled in many cases through diet, exercise, medication, and weight control (James *et al.,* 2004; Lakka and Laaksonen, 2007).

Obesity has been recognized for some time as a contributing factor of hypertension. In recent times, it is being examined at least in part as an independent risk factor for stroke. Strazzullo (2010) conducted literature review of prospective studies for their evaluation of obesity in relation to stroke events, their findings showed a positive relationship between overweight, obesity and risk of ischaemic stroke independent from age, life-style, and other cardiovascular risk factors. The relationship between obesity and stroke was further defined with the results of another study that indicated a much stronger association of stroke risk with waist-to-hip ratio than with body-mass index (O'Donnell, 2010).

The relationship between cardiovascular disease, stroke and smoking has been well established in the literature (Shiton and Beevers, 1989). In a study by Aboyans *et al.,* (2006), it was found that smoking was the strongest predictor of the progression of arterial disease.

Smoking is a leading cause of heart disease and is a known risk factor for stroke. Smoking is known to raise triglycerides in blood, damage endothelial cells that line blood vessel walls, cause thickening and narrowing of blood vessels and result in the formation of clots. Furthermore, it has been found that inflammatory reactions to smoking are involved in the pathogenesis of atherosclerosis and reported 30% increase in risk (Raopach *et al.,* 2006; Asplund *et al.,* 2009).

The consumption of alcohol in moderate amounts of 1-2 drinks per day has been associated with a lower risk of ischemic stroke by reducing athero-thrombotic events (Goldstein *et al.,* 2001). However, recent and current heavy (not previous) drinking increases the risk for both hemorrhagic and ischaemic strokes (Hillbom, 1998). No cause and effect relationship has been established between alcohol use and stroke, but studies such as carried out by Elkind *et al.* (2006) have shown correlation between moderate use of alcohol and lower levels of low density lipoproteins (LDL) and lower blood pressures in individuals consuming small regular amounts of alcohol (Goldstein *et al*., 2001).

Dyslipidaemia is a term used to describe excessive levels of blood cholesterol that create plaque deposits on blood vessel walls (O'Rourke *et al.,* 2004; PHAC, 2012). These fatty deposits can occur within the brachiocephalic or internal carotid arteries and cause narrowing of the artery which decreases blood flow to the brain and increases the risk of blockage (O'Rourke *et al.,* 2004; Go *et al.,* 2010; PHAC, 2012). The presence of this buildup is generally not treated unless the individual also has physical symptoms associated with it (O'Rourke *et al.,* 2004). There has however been controversy regarding the effect of fat intake as it relates to levels of cholesterol in the body and the ensuing relationship to cardiovascular disease and stroke (Sauvaget *et al.,* 2004). Hypertension is the most common

and most important stroke risk factor, including isolated systolic hypertension. It is one of the most powerful and prevalent factors for first stroke. It is also an independent risk factor for recurrent stroke and stroke after TIA (Woo *et al.*, 2004).

2.2.1 Pathophysiology of stroke

Strokes are non-progressive in nature and are caused by ischaemia or haemorrhage. A small number of strokes are caused by congenital abnormalities of blood vessels and can result in spontaneous intracranial haemorrhage. These defects of the blood vessels are known as arteriovenous malformations. They are liable to subsequent bleeding and surgical intervention is the only treatment. The major categories of vascular disease in stroke are; infarctive (thrombotic, embolic) and haemorrhagic (intracerebral and subarachnoid). Cerebral infarction accounts for 69% of strokes, primary cerebral haemorrhage for 13%, subarachnoid haemorrhage for 6%, and 12% are of uncertain type (Larry *et al.*, 2011).

The brain requires a constant supply of glucose and oxygen, which are delivered via blood circulation, and accounts for 15% of the resting cardiac output, and 20% of the total body oxygen consumption. Cerebral blood flow remains constant over a wide range of blood pressure and intracranial pressure because of auto-regulation of vascular resistance. Ischaemic stroke occurs when a migrating clot (embolus) or a fixed clot (thrombus) lodges itself in a blood vessel, obstructing the blood flow to the area distal to the blockage. This causes an abrupt interruption to blood flow and leads rapidly to cell death and focal neurological deficit. This thrombus is usually due to atherosclerosis and often associated with hypertension, diabetes mellitus, and coronary or peripheral vascular disease (Jassin and Jouria, 2013). Since there is no reserve of oxygen in the brain, normal cerebral function can continue only for 8 to 10 minutes after cerebral ischaemia; irreversible damage follows after

6 to 8 minutes. The major arteries that are affected are the carotid, vertebra-basilar, and cerebral arteries (Yadav, 2011).

2.2.2 Cerebral Infarction of stroke

Infarction is the death of a section of the brain tissue due to impairment of blood supply. Interruption of blood flow to the brain depletes the adenosine triphosphate (ATP) and since the brain energy reserves of ATP-dependent processes (like sodium-potassium pump) halts. This leads to the accumulation of glutamates which lyses the neurons and other cells of the brain causing irreversible damage and even death (Chodobski *et al.,* 2011). The anatomical location of the lesion determines whether the patient develops a hemiplegia, a sensory deficit, blindness, aphasia, or some other symptoms. The size, location and shape of the infarct and the extent of the tissue damage that results from total cerebral ischaemia brought about by occlusion of a blood vessel are determined by the size of the vascular bed and the adequacy of blood flow through collateral circulation, with the major source of collateral flow being the circle of Willis. In addition to the injurious effects on brain cells, ischaemia and infarction can result in loss of structural integrity of brain tissue and blood vessels, partly through the release of matrix and metalloproteases, which are zinc and calcium-dependent enzymes that break down collagen, hyaluronic acid and other elements of connective tissue. The loss of vascular integrity results in a breakdown of the protective blood-brain barrier that contributes to cerebral oedema, which can cause secondary progression of the brain injury (Chodobski *et al.,* 2011).

2.2.3 Intracranial and Intracerebral Haemorrhage of Stroke

Haemorrhagic stroke can be caused by intracerebral haemorrhage (ICH), which involves bleeding directly into brain tissue, and subarachnoid haemorrhage (SAH), which involves

bleeding into the cerebrospinal fluid that surrounds the brain and spinal cord. This can occur as a result of hypertension that causes lipohyalinosis in the small arteries of the brain causing micro aneurysms to form. The onset is dramatic in nature with severe headache, vomiting, and sometimes loss of consciousness (Allan *et al.*, 2005). Subarachonid haemorrhage (SAH) is bleeding into the surface of the brain, mainly caused by rupture of arterial aneurysms. Aneurysm rupture releases blood directly into the cerebro-spinal fluid (CSF) under arterial pressure. The blood spreads quickly within the CSF, rapidly increasing intracranial pressure. Death or deep coma ensues if the bleeding continues (Pollay, 2012). Symptoms of SAH begin abruptly, occurring at night in 30 percent of cases. There are usually no important focal neurological signs, unless bleeding occurs into the brain and CSF at the same time (Neurological emergencies, 2015). The primary symptom is sudden, severe headache (97 percent of cases) classically described as the "worst headache of my life". The headache is lateralized in 30 percent of patients, predominantly to the side of the aneurysm. The onset of the headache may or may not be associated with a brief loss of consciousness, seizure, nausea, vomiting, focal neurological deficit, or stiff neck (Allan *et al.,* 2005).

This is bleeding into the tissue of the brain commonly caused by hypertension. Intracerebral haemorrhage accounts for 8-13% of all strokes. An accompanying oedema may disrupt or compress adjacent brain tissue. Displacement of brain parenchyma may cause elevation of intracranial pressure (ICP) and potentially fatal herniation syndromes that may result in death or major disability worse than with ischaemic stroke. Intracerebral haemorrhage does not improve during the early period: it progresses gradually in minutes or a few hours (Liebeskind & Lietsep, 2015).

2.2.4 Transient Ischaemic Attack and Clinical Manifestation of Stroke

Transient ischaemic attack (TIA) is defined in the same way as stroke, but with signs and symptoms lasting less than 24 hours. It has also been defined as sudden, focal neurological deficit lacking neuroimaging and clinical evidence of acute infarction, and lasting less than 24 hours. A number of people suffer from a brief focal loss of function with complete resolution within 60 minutes. This sometimes serves as a warning event, which may lead to a major stroke, if not checked. Approximately 60 percent of patients with a completed stroke have had premonitory TIAs. Adams and Graham (1998), recorded that approximately 5% to 10% of people who have TIA will go on to have major stroke. The risk of developing a stroke after a hemispheric TIA can be as high as 20% within the first month, with the greatest risk being within the first 72 hours (AHA, 2005).

Stroke manifests with an abrupt onset of any or combination of the following signs: hemiparesis, hemisensory deficits, monocular or binocular visual loss, visual field deficits, diplopia, dysathria, facial droop, ataxia, vertigo, aphasia, sudden decrease in level of consciousness. Deficits after a stroke consist, amongst other things, of motor problems, sensory problems and spasticity, and can range from complete paralysis of the one side of the body towards relatively minor coordination deficits. Such impairments affect an individual's ability to complete everyday activities (disability) and affect participation in everyday life situations (Landhorne *et al.,* 2009). Based on artery affectation, stroke can present with different clinical manifestation. Middle cerebral artery and internal carotid artery strokes cause contralateral motor and eye dysfunction with speech and sensory deficits, while vertebral/basilar artery strokes cause balance, vertigo, and cranial nerve dysfunction (Jassin and Jouria, 2013).

2.2.5 Neuroplasticity of Stroke

Plasticity is an intrinsic property of the human brain and represents evolutions innovation to

enable the nervous system break away from the restrictions of its own genome and thus acclimatize to environmental pressures, physiologic changes and experiences (Pascual-Leone *et al.,* 2005). The cortex with its numerous synaptic connections is the ideal site for plasticity to take place (Donogue, 1995). Motor recovery after stroke is related to neural plasticity, which involves developing new neural interconnections, acquiring new functions, and compensating for impairment (Takeuchi & Izum, 2013). Cortical reorganization is an important rationale for rehabilitation and a major neurophysiological underpinning of neurological recovery post-stroke (McClure & Teasell, 2014) and it is an on-going state throughout life (Chan, 2014). The brain is highly plastic through development as new connections are formed and removed during use dependent processes. Cortical reorganization after stroke has been found to be comparable with types that occur during normal development, such as motor recovery after stroke and the acquisition of skilled movement patterns in human infants (Cramer & Chopp, 2000; Murphy and Corbett, 2009). Chan (2014) showed that acquisition of expertise is accompanied by structural and functional changes in the brain. Structural plasticity is evident through microscopic output by increased volume and grey matter density of the brain areas involved in control of practiced task. Structural plasticity is also the structural changes that can disappear when practice stops, indicating the diversity of structural plasticity and the need to practice for long in other to establish adaptation (Chan, 2014). In humans, motor activity in the affected limb results in recruitment of cortical areas along the infarct rim, secondary motor area of the contralateral hemisphere and ipsilateral hemisphere that is manifested as increased activation of the cortical regions of the affected hemisphere (McClure and Teasell, 2014).

Neuroplasticity is based on the principle of cortical representation of the body parts in the motor cortex which states that the more a part is used the larger its representation in the motor

cortex and body parts are known to compete for representation in the brain. This can bring about what is known as “use dependent” plasticity or “experience dependent” plasticity or “learned nonuse” of the affected part (Lipert *et al.,* 2000). This is because when stroke occur, it leads to substantial depression in motor control functions like poor strengths and coordination. However, when appropriate technique is applied, learned non-use can be overcome. Training procedures can be used as incentive for the stroke patient if done for consecutive weeks (Lipert *et al.,* 2000).

2.2.6 Post Stroke Recovery and Management

Recovery of stroke survivors is paramount to the caregivers after sustaining the insult (Tilling *et al.,* 2001). Stroke patients recover differently depending on the severity of the initial deficit which is related to the prognosis of the condition (McClure and Teasell, 2014). Recovery from stroke takes two different but related forms which are neurological and functional recovery. The neurological recovery also known as spontaneous recovery involves the brain recovery or reorganization which influences rehabilitation (McClure and Teasell, 2014). A reduction in impairment and disabilities over the first 3-6months after stroke is often called spontaneous recovery which manifests as improvements in motor control, dysphasia and primary neurological functions (Dobkin, 2011; Bruno-Petrina, 2014). Functional recovery refers to improvement of independence in areas such as self-care, strengths and mobility; which is dependent on patient motivations, ability, family support and type of training interventions. This type of recovery may also be influenced by rehabilitation, but may occur independently of neurological recovery (McClure and Teasell, 2014).

Many studies have determined the pattern and time of recovery following stroke (Ahmed *et al.*, 2004; Hamzat and Peters 2009; Horgers *et al.,* 2009). It was reported by Julkunen *et al.* (2005), that better recovery from stroke happens at the first four weeks and there is proof that recovery changes occur in some people up to 6 months. It was reported by Horgan *et al.*, (2009) that recovery may last beyond six months. Even though, there may be spontaneous recovery, there is good evidence that early organized stroke care is related to better outcome (Karla and Langhone, 2007). The superlative predictors of stroke recovery at 3 months are initial neurological insufficiency and age (Gbiri *et al.,* 2014), depression (Pohjasvara *et al.,* 2001), prestroke disability and presence of motor impairment related to poor motor outcome (Appelros *et al.,* 2003). It was also reported by Gbiri *et al.* (2014), that functional recovery at six months is faster in the young than the aged and in hemorrhagic than ischemic stroke survivors.

Motor recovery to the upper limb is more affected than that of the lower limb and recovery in the upper limb is less than the lower limb. The timing of the arrival of movement in the hand is key predictors of motor recovery in the upper limb and to the affected patient generally (Bruno-Petrina, 2014). Couper *et al.* (2012) confirmed this in a systematic review of 58 studies, which revealed that the predictive factor for upper limb recovery following stroke is the initial severity of motor impairment or function. The return of the hand strengths in stroke survivors such as grasp is a good prognostic feature in stroke patients. About 9% of patients with upper limb weakness may gain good recovery of upper limb strengths and flexibility. Many stroke survivors may show some level of motor recovery in the hand by a month and make full functional recovery, by three months of rehabilitation (Bruno-Petrina, 2014). Likewise, about 188 stroke patients were studied and it was revealed that assessment of finger

extension and shoulder abduction within 72 hours after stroke can help predict stroke recovery (Nijland *et al.,* 2010). There was almost certainty that if strengths in the fingers and shoulder abduction returned by the second day post-stroke, then there is 0.98 probability of regaining dexterity by 6 months and evidence shows it can occur in 60% of patients with stroke (Bruno-Petrina, 2014). More than several years ago, it was postulated by Hebb in 1949 that increments in synaptic efficacy occur during learning when firing of a neuron repeatedly produces firing in another neuron of which it is connected, leading to concept of plasticity as a behavioural adaptation or learning that is related to change in function at the level of the synapse (Murphy and Corbett, 2009). The first recovery machinery is resolution of destructive local factors, which generally accounts for early spontaneous improvement after stroke (between the first 3-6 months). These include resolution of local oedema, resorption of local toxins, reperfusion of infarcted penumbra and recovery of incompletely damaged ischaemic neurons. The second recovery mechanism is the reorganization of the central nervous system known as neuroplasticity. This process is known to play an active role in the restoration of function post stroke (Bruno-Petrina, 2014; McClure and Teasell, 2014).

In the management of stroke, the rehabilitation team is usually interdisciplinary as it involves professionals with different skills working together to help the patient. They include: a physiotherapist, an occupational therapist, nursing staff, a speech and language therapist, a physician etc. Some teams may also include a psychologist, physiologist, social workers and a pharmacist, since at least one third of patients manifest with post stroke depression and decreased participation (NINDS, 2014). The treatment given may include the test for the establishment of the type of stroke, which includes brain scan, etc, and treatment to support basic life functions such as breathing and to reduce pain and stress. Management of stroke

survivors can be classified into medical, surgical, and rehabilitative treatment. Once stroke has occurred, management will usually be conservative. However, in a small number of cases, surgery may be indicated. For example; in some patients with subarachnoid haemorrhage, it may be possible to clip an aneurysm to prevent further damage, or drain a haematoma before it causes irreparable damage. Surgery may also be performed to improve cerebral circulation in patients with thrombotic or embolic cerebro-vascular accident (CVA); this includes an endarterectomy (removal of atherosclerotic plaque from the inner arterial wall) or a micro-vascular bypass (surgical anastomosis of an extracranial vessel to an intracranial vessel). The indications for surgery in patients with ICH vary with the site of bleeding. Sub-occipital craniectomy with cerebella decompression can be performed for all cerebella haemorrhages greater than 3cm in diameter (NINDS, 2014).Medications given include clot-dissolving drugs that prevent further clotting. To prevent the progression of stroke symptoms, heparin is often used, and aspirin in ischaemic stroke. Medications useful in treating CVA include:

i. Alteplase (recombinant tissue plasminogen activator, TPA), effective in emergency treatment of embolic CVA. Patients with emboli or thombolytic CVA, who are not candidates for TPA (3 to 6 hours post-CVA), should receive aspirin or heparin.
ii. Long-term use of aspirin or ticlopidine used as antiplatelet agents to prevent recurrent CVA.
iii. Anticoagulants (heparin and warfarin), which may be required to treat crescendo TIAs not responsive to antiplatelet agents.
iv. Antihypertensives, antiarrhythmics, and antidiabetic agents, which may be used to treat risk factors associated with recurrent CVA.
v. Corticosteroids such as dexamethasone to minimize associated cerebral oedema.

vi. Analgesics such as codeine to relieve the headache that typically follows haemorrhagic CVA (Hacke, Donnan, Fieschi, Kaste, von Kummer & Broderick (2004).

vii. During acute exacerbations, supportive measures include bed rest in an intensive care unit so that the patient can be carefully monitored and receive the necessary respiratory care, otherwise the patient is admitted to a stroke unit or a general medical ward. If the stroke is mild and there is adequate domiciliary back up, the patient may be treated at home (Hacke, Donnan, Fieschi, Kaste, von Kummer & Broderick, 2004).

The aim of rehabilitation is to improve quality of life by reducing emotional, functional, physical, cognitive and communication disorders. Physical therapy intervention for patients with stroke includes interventions to remediate impairments and to compensate for functional limitations. These involve the frequent use of motor learning and control approaches to facilitate all activities in stroke rehabilitation. Physical therapy management follows a problem-solving approach and involves the following conditions:

i. Watching for signs of pulmonary emboli, such as chest pain, shortness of breath, dusky colour, tachycardia, fever, and changed sensorium.

ii. Watching for signs of other complications such as infection, cerebral oedema, hydrocephalus, seizures, aspiration pneumonia, deep-vein thrombosis, pressure ulcers, urinary tract infections, contractures, and joint subluxation.

iii. Preventing aspiration pneumonia, by placing the patient in an upright and lateral position to allow secretions to drain and turning the patient frequently.

iv. Assisting in minimizing long-term disability. Deficits can include motor weakness,

coordination and balance problems, diminished corneal reflex, visual field deficits, dysarthria, dysphasia, impaired memory and concentration, and pain.

v. Maintaining joint Range of Motion (ROM) and prevent deformity

vi. Promoting awareness, active movement and use of the hemiplegic side (Langhammer *et al.,* 2000).

One of the aims of managing stroke survivors is the prevention of secondary complications which includes:

i. Improving trunk control symmetry and balance.

ii. Initiating self-care activities and developing independent activities of daily living.

iii. Improving functional mobility skills and encouraging socialization.

iv. Monitoring changes associated with recovery.

v. Approaches to respiratory and vasomotor function and developing cardio-respiratory endurance (Potempa, Lopez, & Braum, 1995).

The idea is to have a coordinated expert interdisciplinary team, including a physiotherapist, occupational therapist, physicians, surgeons and nursing staff who have regular team meetings to help in the outcome of these patients (Clarke & Foster, 2015). For this to be achieved, the main goals should include:

Therapeutic positioning:This is carried out in order to improve awareness of the affected side and also to prevent other complications like bed sores. Positions to avoid are:

i. Lateral side flexion of the head and trunk towards the affected side, and rotation of the head to the unaffected side.

ii. Depression and retraction of the scapula, internal rotation and adduction of the

arm, elbow flexion, forearm pronation, and flexion of the wrist and fingers.

iii. Retraction and elevation of the hip, with hip and knee extensions, prevent adduction of the hip and plantar flexion of the ankle.

Positioning the patient involves correct alignment of the extremities. Rolls can be used to prevent external rotation. High-topped sneakers can be used to prevent food drop when the patient is sitting up and his feet are on the floor. Positioning the patient in a side-lying position on the affected side can help decrease hypertonicity and increase awareness on the hemiparetic side (Jones, Carr, Newham & Barnett-Wilson, 1998).

Range of motion exercise*:* Flexibility refers to the ability to mobilize a part of the body through a broad range of motion without undue strain to the articulations and muscle attachments. This can measure the flexion and extension movements. But there is no general measure for flexibility. The most acceptable and accurate test for flexibility is by the use of goniometer. Goniometer is a protractor type of instrument that measures the joint range of motion. The instrument has two arms attach to the centre of the joint. It should be placed at the joint of investigation and measurement is on degrees. It can be time consuming but it is an objective measure of flexibility during physical fitness. This can be used to assess range of motion in shoulder, trunk, long limb movements including the trunk (ACSM, 2000). This can be active or passive. Active range of motion exercises can be subjective (normal anatomical movements like flexion extension) or objective (task-oriented movements like touching the nose). Range-of-motion (ROM) exercises can be provided throughout the day [National Stroke Association (NSA, 2010).

Functional mobility activities*:* Patients are encouraged to use both sides of the body. Treatment progression and intensity will depend on the patient's response. Such activities

involve rolling (back and forth), bridging, sitting-up from supine position (Jones *et al.*, 1998).

Passive exercises and electrical stimulation:Passive and neuromuscular electrical stimulation (NMS) exercises are common clinical approaches, but NMS is limited in stroke rehabilitation. Both animal and clinical studies have shown functional improvements after these interventions —improve shoulder stiffness after passive stretches, prevent muscle loss (atrophy) of specific denervated muscles, prevent shoulder subluxation in chronic stroke survivors when used in conjunction with conventional upper limb therapy, and help to reduce spasticity effectively in stroke survivors. Even though the therapeutic effect of NMS on spasticity is still controversial and its effects on spasticity are not clearly established, some reported results are variable (Conforto *et al.*, 2010).

Passive movements in hemiplegic stroke survivors before clinical recovery elicit activation of the affected hemispheric patterns that may be critical for the restoration of motor function. Passive exercises were also found to maintain muscle properties and prevent adaptive shortening of muscles, increase joint range of motion by stimulating receptors of kinetic sensation, increase venous and lymphatic drainage, break adhesion formation in the joints, and prevent deep vein thrombosis. Passive exercises have a soothing effect and induce relaxation and can be used to break accessory movements which cannot be localized actively (Jones *et al.*, 1998).

Establishing and maintaining communication with the patient. If the patient is aphasic, a simple method of communicating basic needs is set up. Questions are praised so the patient will be able to answer using the system. Repetitions are done quietly and calmly with gestures, if necessary, to help the patient to understand. Spending time with the patient, and

simplifying language, asking yes – or – no questions, whenever possible (NINDS, 2014).

Provision of psychological support: Setting realistic short-term goals, involving the patient's family in his/her care when possible and explaining his/her deficits and strengths (Duncan, 2005)

Orthopedic Problemsthat can occur should be prevented, such as:

a. Subluxation of the shoulder: occurs when any biomechanical factors contributing to glenohumeral joint stability are interrupted.
b. Pain: due to muscle imbalance, loss of join ROM, improper movement patterns, and improper weight-bearing patterns (Jones et *al.,* 1998).

Prognosis

i. 22% of men and 25% of women who have an initial stroke die within a year (percentage is higher for those greater than 65 years).
ii. 50% of men and women less than 65 years of age who have a stroke die within 8 years.
iii. 14% of those who survived a first stroke, or TIA, have another within 1 year (Lee, Shafe & Cowie, 2011).

Stroke Outcomes

10% Recover almost completely

25% Recover with minor impairments

40% Experience moderate to severe impairments requiring special care

10% require care in a nursing home or other long-term care facility

15% Die shortly after the stroke.

30% Develop pneumonia within first month

10% Risk of recurrent stroke per year

10% Death post-stroke from pulmonary embolisms (NINDS, 2014).

Other morbidity from stroke:

23% Develop multi-infarct dementia

70% Develop depression

27% Major depression

40% depression common among caregivers (NINDS, 2014)

2.3 Physical Activity Participation for Stroke Survivors

Physical activity is a complex multi-dimensional behavior that is often categorized and measured using variables such as frequency, intensity, duration and mode (Valanon *et al.,* 2006). Inactivity exacerbates the normal decline in aerobic fitness and compromised cardiovascular capacity of most stroke survivors below the expected level during activity of daily living (Macko *et al.,* 2007; Michael, *et al.,* 2005). Inactivity could lead to low endurance and strength (Newham and Hsaio 2001), which affects the level of regular participation in most daily routines for survival (Fujitan *et al.,* 1999). Stroke survivors can benefit from counselling on enhanced participation in physical activity (Vander Ploeg *et al.,* 2007). It has been documented that motivation to exercise improves functional participation (Brophy *et al.,* 2013).

Training is simply defined as the systematic and regular involvement of muscular movement to improve performance (Billat, 2001). It aims at the aerobic energy system, increase cardio-respiratory perfusion, assists in weight control and relaxation and helps to reduce coronary heart disease. There are four methods of enhancing aerobic capabilities, which include fartlek, interval, continuous, and circuit trainings. Training is a series of exercises that a

patient with a peculiar diagnosis performs at a station, finishing each exercise before moving on to the next stage with virtually no rest in between (Prentice, 1994). The principle of overload which is specific to an activity is necessary to improve physiological situations bringing about training effect or response (Kraemar *et al.,* 2002). The specific type of exercise leads to adaptations and the training has to be planned to meet the patient's capabilities (Hecke *et al.,* 2004). Aerobic training should follow the principle of frequency, intensity, time and type (Coyle *et al.,* 1984).

The physical therapist should be knowledgeable about the principles and processes of training to combat stress, illness and disease. Cardio-respiratory fitness is defined as the ability to carry out moderate to high intensity muscular work with large muscle groups for endurance (American College of Sports Medicine, 2000). It also depends on the functional integrity of the pulmonary, cardiovascular, and large skeletal muscle systems to enhance physical activity (ACSM, 2000).

Aerobic training like cycle ergometry is known to have an important effect on cardiovascular function (Globas *et al.,* 2012). In addition to improvement in endurance of people living with stroke, aerobic training helps to prevent many challenges of stroke, especially in Nigeria (Hamzat, 2014). A study on effect of aerobic exercise on total cholesterol level of stroke survivors found no significant effect (Lenon *et al.,* 2008). Aerobic exercise training is an organized regular exercise programme targeted to improve the ability of the system to increase cardio-respiratory efficiency and enhance performance (Billiat, 2001). There is still need to assess physiological variables such as pulse rate, blood pressure, motor function, VO_2 max in stroke neurological rehabilitation (Tolendano *et al.,* 2011).

Sedentary lifestyle is high among stroke survivors and reduces performance of activities of daily living, increases the risk of falls and recurrent cardiovascular diseases (Billinger *et al.*, 2014). Physical activity and exercise training have the capacity to influence physical, psychological and social domains post stroke (Bellinger *et al.*, 2014). Walking ability and upper limb and extremities, and cardio-respiratory fitness are improved following exercise (Harris & Eng, 2010; Veerbeck *et al.*, 2011). More than 30% of stroke survivors report activity restrictions which include societal roles, anatomy and engagements (Gadidi *et al.*, 2011). Reduced VO_2, orthostatic intolerance, increased joint contractures and deep vein thrombosis are peculiar detrimental effects of bed rest. It is associated with decreased volume of blood plasma cardiac output and increased resting pulse rate (Fortney *et al.*, 2011). Early intervention post stroke within 24 hours at regular intervals has been shown to result in earlier walking and improved functional recovery (Cumming *et al.*, 2011). The recommendation of aerobic training modes may include leg, arm or combined arm-leg.

2.4 Neuromuscular Stimulation

Functional electrical stimulation (FES) is a neuromuscular stimulation technique used for stimulating task-specific and functional activities. Some studies report that functional electrical stimulation is efficient for improving upper extremity functions, such as holding, grasping, moving and releasing objects (Alon; Lewitt & McCarty, 2008). The use of neural pathways in the exaggerated areas helps the re-organization and healing process not only in the acute period, but also in the subacute and chronic periods (Duffau, 2006; Lindstrom, 2011). The likelihood of a second stroke is increased due to the persistent unhealthy lifestyle (Hoeing, Nusbaum & Brummel-Smith, 1997). Many stroke victims require extensive care (Abrams; Beers & Berkow, 1995). Scores of co-morbidities are also common to stroke

survivors ranging from coronary heart disease, obesity, and hypertension, type 2 diabetic mellitus and hyperlipidaemia (Black-Schafer; Kirsteins & Harvey, 1999). Stroke either ischaemic or haemorrhagic, presents with symptoms which include weakness (hemiparesis, quadriparesis) or paralysis (hemiplegia, tetraplegia) and impaired balance (ACSM, 2002).

Peak pulse rate during acute exercise will diverge for person's with stroke depending on their age, level of disability (extent of muscle atrophy on affected side), number and severity of comorbidities, degree of spasticity, cognitive impairment and medication use (ACSM, 2002). Evaluating patients outcome measure, and advance knowledge of neuro recovery is paramount in improved management of stroke survivors (Roprer *et al.*, 2002). Research findings reveal that it is more effective than task oriented training activity alone in the rehabilitation of subjects with mild and moderate hemiparesis (Alon, Levitt & McCarthy, 2007). Post-stroke dysfunction is among the mainly investigated neurological disorders, and is one of the most important goals in post stroke rehabilitation. The complex interactions of the neuromuscular system ought to be considered when selecting and developing management methods upsetting the human system and causing the disturbances (Lindquist, Prado, Barnos, Mattoli, Dacosta & Salvini, 2007).

Studies have also shown superior gait improvements, when robotic-assisted locomotor devices like leg propelled wheelchairs are used for stroke survivors (Lindquist *et al.*, 2007). There are limited studies conducted using both propelled upper limbs and neuromuscular stimulation. These changes result in increased morbidity and mortality and reduced quality of life (Roth, 1993; Gillum & Sempos, 1997; Wolf, Clagett & Easton, 1999; American Heart Association, 2002). The cardio-respiratory fitness varies according to age, gender, physical activity levels, body composition and presence of chronic disease or disability (Smith; Blair

& Bonow 2001; Pearson, Blair & Daniels, 2002). The changes in muscle physiology and inflammation, impaired haemodynamic response, altered metabolic health and respiratory dysfunctions can negatively affectdaily activities and exercise performance (Brinkman & Hoskins, 1979; Duncan, Richards & Wallace, 1998). Hypoxia is common post stroke and it makes stroke survivors more prone to respiratory problems (Nachmann *et al.*, 1995). Stroke is one of the principal causes of morbidity and mortality of adults in the developed world and the leading cause of disability in all industrialized countries. Stroke incidence is approximately one million per year in the European Union and survivors can experience several neurological deficits or impairments, such as hemiparesis, communication disorders, cognitive deficits or disorders in visuo-spatial perception (Kwakkel, Kollen & Wagenar, 1999; Schmidt *et al.*, 2007). About 795,000 Americans experience a new or recurrent stroke yearly; with resulting direct and indirect health costs totaling $34 billion and $21.8 billion, respectively. There are currently over 1 million people in the United States who have survived stroke and are existing with minor to severe functional limitation (Kwakkel, Kollen & Wagenar 1999; CDC, 2011).

In chronic stroke, blood flow in the paretic limb is significantly lower at rest and during exercise, when compared to the non-paretic limb (AHA, 2002; Brown; Whisnaut & Sicks, 1989; Gillum & Sempos, 1997). Motor recovery after stroke is related to neural plasticity, which involves developing new neuronal interconnections, acquiring new functions, and compensating for impairment. However, neural plasticity is impaired in the stroke-affected hemisphere. It is important that motor recovery therapies facilitate neural plasticity to compensate for functional loss. A study reported that individuals with left insular cortex experienced an increase in cardiac events such as heart failure within one year after stroke

(Roth & Harvey, 2000). Rehabilitation techniques have been more successful in restoring function in the lower limbs than in the upper limbs. Further recovery of upper limb function should not be expected if functional recovery has not occurred by the 11th week (Roth & Harvey, 2000). Therapeutic interventions such as neurofacilitation techniques, progressive strengthening, bio feed-back and NMS have been used to promote functional recovery, but the outcome of these have yielded inconsistent results (Duncan, 2005). The rehabilitation of the upper extremity has been a great challenge to the rehabilitation circle (Hara & Muraoka, 2006). The understanding of the mechanisms that promote or prevent recovery is crucial to the design of optimized therapies. Voluntary upper limb activity was said to have become possible by using it as a feedback mechanism. In a study using proprioceptive neuromuscular facilitation (PNF), NMS and control group, it was observed that significant improvement was more in patients receiving ES with no improvement to control. The changes reported suggest that neural-plastic changes in the brain may have occurred, apparently due to increased activity in the hand, while performing the exercise. It is also possible that repeated stimulation of muscles may cause habituation of reciprocal inhibition in the antagonist muscles leading to a reduction in plasticity. This may lead to improved control of the limbs due to reduction in neglect syndrome (Chae & Yu, 2000).

Neuromuscular stimulation has been observed to increase muscle strength in spinal cord injured individuals. Large strength gain was noticed, with the rate of increase being substantially greater on the resisted side (Laurer, Smith, Mulcahey, Betz & Johnson, 2011). A low cost clinical exercise system was developed for the spinal cord injury, using NMS. The exercise tolerance was said to be as a result of increase in local muscle strength and

endurance. Muscle bulk of muscle and joint range of motion increases were reported. Many traditional methods, such as neurodevelopment techniques, have been used for rehabilitation of upper extremity function after stroke, but these have not been shown to be efficacious in controlled studies. However, the approach that involves repetitive training of the paretic upper extremity or task-oriented activities leading to functional achievement to stroke survivors has shown promise. Functionally-oriented task practice of a limb has shown substantial evidence of efficacy for individuals with long-term stroke disabilities greater than one year, with encouragement in the use of the affected limb in daily life (Brewer, Hogan, Hickey & Williams, 2012). In controlled design to compare neuro-developmental treatment, the Brunnstrom techniques with conventional treatment, failed to detect any difference in general outcome and upper limb function (Lord & Hall, 1996). The extremity constraint induced therapy evaluation (EXITE) is the first national randomized, single blind study to systematically test a neurorehabilitation therapy among patients that can use the limb having experienced a first stroke within 3 to 9 months of enrolment (Monye, 2012). Functional electrical stimulation (FES) maintains standing posture achieved by simultaneous activation of both sets of quadriceps and glutei muscles for knee and hip extension which enables paraplegics to stand from a seated position and transfer to another surface (Szecsi *et al.*, 2008). Beldalois *et al.* (2011) found that multichannel ES given 10 to 60 minutes, three times per week for a month improved gait performance in paraplegics.

Studies have highlighted the contribution of afferent synaptic activity to central motor control and indicate that reorganization of the sensory and motor systems occurred early following stroke. Stimulation causes tingling – "pins and needles" sensation – on the skin. Most people do not find it uncomfortable, but a few do, and for this reason do not use it.

However, a short period of stimulation at low intensity, frequently overcomes this problem. Even though people are carefully assessed, treatment with ES may not benefit them or they find it complicated to use the stimulator effectively (Miller *et al.*, 2013). People find that the stimulation or the electrodes cause irritation of the skin. This has been addressed by using hypoallergic electrodes or changing the type of stimulation used. In rare cases, it was found that stimulation increases muscle tightness (spasticity) (Miller *et al.*, 2013). Motor and mobility problems are very common after stroke. For the purposes of treatment planning and outcome assessment, is important to reliably and accurately assess motor function in patients with stroke. Hence, this study measures strength and the ROM to assess the response of the subjects to the treatments. While, ES has other problems such as electrocution, electric power failure, cost of purchase, maintenance and affordability; passive exercise is also faced with problems like minimal patient compliance, time consumption, risk of musculo-skeletal disorder during the therapy and as a means of treatment by some patients.

The International Classification of Functioning, Disability and Health (ICF), developed by the World Health Organization (2008), describes functioning from three perspectives: body; person and society classified as "Functioning and Disability"; and contextual factors. Activity limitation or disability are manifested by reduced ability to perform daily functions such as dressing, bathing, reaching or walking, which may make the patient completely dependent or not, depending on the level of impairment or severity of stroke (Roth, Heinemann & Lovell, 1998). Studies have also recognized that certain factors can influence the level of activity limitation such as intrinsic motivation, mood, adaptability, coping skill, cognition, learning ability, severity, type of acquired medical comorbidities, medical stability, physical endurance levels, effects of acute treatments and the amount and type of

rehabilitation training (Roth, 1994; Gresham, Duncan & Stason, 1995). The debilitating motor effects of stroke can markedly reduce mechanical efficiency and increase level of energy cost, resulting in decreased activity and greater exercise intolerance, leading to secondary complications such as reduced cardio-respiratory fitness, muscle atrophy, osteoporosis and impaired circulation to the extremities in stroke survivors (Bjuro, Fugl-Meyer & Grimby, 1975). Rehabilitation programmes designed to optimize functional motor performance, like use of the treadmill in normal ambulant individuals, aerobic exercise training to improve strength with fitness may be incorporated to activity-limited patients (Lopez & Braun, 1995; Visintin, Barbeau & Korner-Bitensky, 1998; Shepherd, 2001).

Despite advances in acute management, stroke remains a major cause of disability worldwide. A number of neurological functions are impaired by stroke, the most common of which is motor disability contralateral to the stroke lesion side. Restoration of walking is considered the main goal of post stroke lower limb rehabilitation with gait speed regarded as a reliable marker of deficit severity (Janssen *et al.,* 2008). Prescribing exercise for stroke survivors is comparable in many ways to prescribing medication based on individual needs and limitations. Aerobic training modes include leg, arm separately or combined arm-leg activity at 40-70% of peak oxygen consumption or pulse rate reserve and perceived exertion with frequency of 3 to 7 days a week and duration of 20-60 minutes, depending on the level of fitness (Palmer-McLean & Harbst, 2003).

Neuromuscular stimulation (NMS) is frequently used to strengthen normal muscles. In pathology, several investigations have shown the merit of NMS for the treatment of amyotrophic related to immobilization. The efficacy of NMS of the quadriceps during rehabilitation after anterior cruciate ligament surgery has been well documented (Monaghan,

Caulfield & Mathuna, 2010). Percutaneous electrodes facing the muscle motor points are used to stimulate muscle stimulation cycles. Stimulation frequencies are chosen to induce contractions, and sensory discomfort related to current intensity varies considerably between subjects, but remains the major limitation to strength. In the context of rehabilitation, addition of ES to voluntary exercise was superior to isometric voluntary contraction alone (Nobbs & Rhodes, 1986; Delitto, Rose & Mckowen, 1988).

2.4.1 Effects of Neuromuscular Stimulation and Strength in Stroke Survivors

The motor unit

An individual muscle is made up of many motor units of different types, giving the muscle its particular characteristics. The motor unit consists of an anterior horn cell, the alpha motor neuron emanating from it and all the individual muscle fibres it supplies. The number of muscles fibres supplied varies from very few in which precise control of movement is required to one to two thousand for large postural muscles. All the muscle fibres of a particular motor unit are the same (Knierim, 1997).

Type I**:** are slow twitch, red fibres because they are highly vascular and predominate in postural muscles. They are slow to contract and relax because the motor neuron supplying them is of small diameter and low conduction velocity with low frequency. They have many oxidative enzymes and fatigue slowly.

Type II: are fast twitches, white, glycolytic fibres. They are less vascular and the motor neuron supplying them is of larger diameter and has a higher conduction rate. They are divided into two subgroups.

Type IIA: Have fewer oxidative enzymes than type I, but are relatively fatigue resistant. They predominate in ordinary low force movement.

Type IIB**:** has the least oxidation enzymes and fatigue rapidly. They produce a large force for short periods and are brought into play during strenuous movement.

During voluntary contraction of a muscle, there is an asynchronous firing of motor neurons resulting in a smooth contraction. The force of a contraction is graded, in general by the increase in the number of motor units recruited (spatial summation) and the frequency of nerve impulses (temporal summation). Itoccurs in the early stages and in low force muscle contraction and later at greater muscle force. In other words, as more motor units become involved, further increase of muscle force is largely achieved by increased rate of nerve impulse firing.

Type I muscle fibres are recruited first and later type II. Prolonged muscle contraction leads to fatigue, rapidly in Type IIB and most slowly in type I. The order of recruitment is largely fixed but can be influenced by cutaneous stimulation. Neuromuscular stimulation (NMS) is different from voluntary contraction in several ways. First, there is synchronous firing of all motor neurons stimulated. Secondly, ES will not stimulate motor units in the same recruitment order as voluntary contraction. In fact, it is largely reversed because:

a. Larger diameter motor neurons (type II) are more easily stimulated
b. Sensory nerves are inevitably stimulated (Knerim, 1997).

Additionally, the frequency of firing is fixed, unlike voluntary contraction. Therefore in order to cause stronger muscular contractions, the current intensity has to be increased to stimulate more motor units (Knierim, 1997). The excitation caused by ES via its motor nerve has both immediate and long-term effects. Muscular contraction and vascular changes are examples of the immediate effect, while muscle strengthening and structural changes in muscle fibres may ultimately result from long-term. The structure of living muscles is not fixed. There is,

for example, a balance between the synthesis and breakdown of the constituent protein. The rate of this can be as much 10% of skeletal muscle protein per day; occurring at a higher rate in type 1 slow twitch fibres than in type II. More anabolism than catabolism will lead to muscle hypertrophy with more muscle and collagenous tissue produced, with atrophy as the reverse case. Where voluntary active exercise is restricted, NMS may be substituted. It is usually applied by surging (or ramping) a series of short pulses at frequencies of 30 and 100 hertz (Chae & Yu, 2000).

Therapeutic purpose of electrical stimulation

Muscle strengthening

A. Normal muscles: The question of efficacy of electrical stimulation (ES) to normal muscles with regard to increase in muscle strength is not entirely resolved in spite of much research. The fact is that ES do increase muscle strength although not quite to the same extent as voluntary exercise. Neuromuscular stimulation (NMS) is not a satisfactory substitute for voluntary activity. However, a number of studies show that NMS combined with voluntary activity led to similar, or in a few cases even greater strength gains than voluntary exercise alone (Mohr, Carlson, Sulentic & Landry, 1985; Stevens-lapsley, Balter, Wolfe, Eckoff & Kohrt, 2012). In a well-controlled study, it was shown that NMS of muscles over a three-week period produce significant gains in muscle strength, being greater in the group treated with high-intensity ES than in the group treated with lower intensities. The force of isometric contraction showed greater gains than that of concentric contraction (Stevens-Lapsley *et al.*,

2012).

The force of voluntary muscle contraction is greater in most but not all subjects than the force that can be produced by NMS of the same musculature. This difference does not seem to be reliable since different stimulators do not produce significantly consistent difference in contraction force (Aureliovas & Aragao, 2012).

B. Weakened muscles: In weakened or weakening muscles, the value of NMS is much clearer and significant gains have been reported with improvement of muscle function. Neuromuscular stimulation at 30 Hz applied to quadriceps of immobilized knees and given 2 seconds on and 9 seconds off cycle for each day for 6 weeks has been shown to reduce muscle atrophy. Quite a number of studies have confirmed the efficacy of NMS in the strengthening of weakened muscles. In a study across sectional area of the quadriceps, diminished muscle girth was observed by 17% in the untreated group, but there was no significant loss in patients that were treated. The effect was considered to be due to the maintenance of protein synthesis in muscles rather than preventing protein break down. Similarly, Increase in quadriceps muscles girth has been observed after using a surged faradic pulse train of 3 seconds at maximum titanic contraction level and within limit of tolerance. It was stated that after the motor threshold is exceeded, very small increases in stimulation amplitude produce relatively large increases in the force of muscle contraction as the strength increases (Synder-Mackler & Robinson (1989). The strengthening effect has been used to promote greater achievement in athletics but any advantage this might have over voluntary effort has not been unequivocally demonstrated. However, its use in the prevention of disuse atrophy appears to be justified (Hansen, 2009).

C. Facilitation of muscle control: Fall and Lindstrom (1991) described the effects of using different electrical frequencies to produce effects in fast and slow muscle fiber types. They suggest that slow fibres would best respond to frequencies of 10-20 Hz, whereas fast fibres would respond to 30-60 Hz. It should be noted that when high frequencies are used, for example above 40 Hz, the fast fibres will not relax between impulses and will produce titanic contraction. If a high frequency is maintained for several seconds, it is capable of fatiguing the fast fibres. Most muscle stimulation programmes are based on using a duty cycle with a rest phase to allow recovery of the motor units, and it is suggested that frequencies not exceeding 40-50 Hz should be used (Fall & Lindstrom, 1991).

Functional electrical stimulation (FES) consists of delivering an electric current through electrodes to the muscles. The current elicits action potentials in the peripheral nerves of axonal branches and thus generates muscle contractions. It has been applied to manage drop foot and has been found that multichannel FES, given 10 to 60 minutes, 3 times per week for 1 month improved gait performance in patients (Belda-Lois; Del Horno; Bermejo-Bosch; Moreno; Pons; Farina *et al*., 2011). However, no follow up was carried out to find out whether these improvements in walking speed were maintained after the FES was stopped (Belda-Lois *et al*; 2011). Stimulation exercise is extensively used therapeutically to initiate and facilitate voluntary contraction of muscles, although it is never possible to distinguish this effect from the strengthening effect already considered (Nobbs & Rhodes, 1986). This idea may be applied in several circumstances which include:

I. Where voluntary muscle contraction is inhibited by pain or injury, like stimulating the quadriceps after knee injury or surgery.

II. In situations where muscle action is not under voluntary control without Practice.

Stimulation of the pelvic floor muscles in the control of incontinence and in stimulation of the *abductor hallucis* in the management of *hallux valgus*.

III. In circumstances in which a new muscle action has to be learned as in after a muscle or motor nerve has been transplanted.

IV. In the later stage of a recovering peripheral nerve lesion to encourage voluntary muscle contraction, where re-innervation has only recently occurred.

V. In situations in which it is necessary to demonstrate to the patient that a particular muscle action of movement can occur normally, where hysterical paralysis is present.

VI. In children with cerebral palsy where ES may enhance muscle contraction and provide sensation so that a child can adda weak response with effective result (Nobbs & Rhodes, 1986).

2.4.2 Effects of Neuromuscular Stimulation and Flexibility in Stroke Survivors

Range of motion of a joint may be limited by different tissues and from different causes. Electrical stimulation of muscles to stretch the shortened tissues has been used for various reasons. Joint limitation of motion due to shortening of soft tissues on one side of the joint has been treated by cyclical electrostimulation of the muscles that stretch the contracture (Jones *et al.,* 1998).

i. The loss of motion due to spasticity of muscles in neurological conditions. Regular passive movements are recommended, often carried out at home by the patient or in the clinic by the specialist. Electrical stimulation (ES) has been applied as an alternative to manual passive movement to help prevent the loss of motion due to plasticity of the antagonist muscles.

ii. Scoliosis: In the treatment of this interesting deformity, the lateral trunk muscle on convexity of the curve are stimulated or exercised electrically. Surface electrodes are attached to the patient's back and muscle contraction is provoked in short cycles at a level which allows the patient to sleep during the treatment. Moderate scoliosis ($20\text{-}45^0$) has shown improvement in over 80% of stroke survivors (Jones *et al.,* 1998).

This is the use of faradic type or similar electrical pulses is long used and applied to the skin to cause muscle contraction. It encompasses both control and rehabilitation of the neuromuscular complex in that strengthening occurs when the muscle is regularly and repeatedly stimulated. This may also have a beneficial effect on muscle spasm. Electrical stimulation has signified an improvement in walking speed and physiological cost index and no significant carry-over effect, unlike in splinting. Similarly ES of the deltoid has been used to prevent glenohumeral subluxation in patients (Carmick, 1994).

The effect of ES on spasticity is not clearly established, partly due to the difficulty in measuring and defining spasticity.

The use of ES in spasticity is generalized via:

i. Stimulation of antagonists to utilize the effect of reciprocal inhibition.
ii. Stimulation of the spastic muscles themselves.
iii. Alternately, stimulating agonist and antagonist muscles.

Neuromuscular stimulation will have the same effect as normal voluntary muscle contraction in causing an increase in muscle metabolism. There will be associated consequences of increased oxygen uptake and carbon dioxide, lactic acid and other metabolite production, as well as raised local temperature and greater local blood flow. There was 20% blood flow

increase after 10% and 30% of maximum voluntary contraction which occurred one minute after NMS had started and continued for some 5 minutes after it had finished (Beelen; Seargent., James & Ruiter, 1995).Not only is the intramuscular blood flow increased but as a consequence of regular muscle contraction and relaxation the flow in adjacent soft-walled veins will be increased, due tothe muscle pumping action. This effect is used therapeutically to help control limb oedema by raising the rate of flow in venous and lymphatic vessels. Blood flow in the femoral artery increased by 18.5% after ES of 0.4 ms pulses at 50Hz in 4 s off cycles. The study also used sufficient current to stimulate the muscle to 15% of its maximum voluntary contraction and the blood flow measured in the femoral artery with an ultrasonic Doppler device. The increased blood flow was noted within 5minutes of the start of ES with graduation to normal level within 1minute of ES cessation. The effect of ES on triceps surae of limb blood flow and microvascular filtration capacity was studied and it was noticed that there was significant increase in blood flow and walking distance with decreased intermittent claudication (Anderson; Whatling; Hudlicka; Gosling; Simms & Brown, 2004).

Muscle fatigue as a consequence of voluntary contraction is well known but it is a complex and not fully understood phenomenon. Initially fatigue is due to depletion of muscle glycogen and available blood glucose with other biochemical limitations. Ultimately, the rate of oxygen utilization is important. Muscular fatigue at a submaximal contraction can be controlled by varying the amount of motor units involved. Prolonged contraction shows increased recruitment of motor units to maintain the same muscle force as fatigue occurs (Low, Reed & Dyson, 2000). It would therefore be expected that electrical stimulation of muscle via the motor nerve would lead to relatively rapid muscle fatigue, since a fixed set of motor units are stimulated with the fibre type preferentially selected. It was shown that

muscle fatigue due to ES was greater than that due to isometric, voluntary contraction of equal force. The degree and duration of fatigue was said to be directly related to the extent of the ES. A study found the evidence of fatigue persisting after long periods after a therapeutic protocol of ES applied to healthy subjects. The use of NMS for paralyzed muscles with intact lower motor neurons, as in spinal cord injuries, has led to interest in the way muscle fatigue responds to NMS with rapid fatigue seen in paraplegic patients (Low, Reed & Dyson, 1987).

Fatigue after exercise, including electrically-induced exercise, may be a necessary stimulus for muscle strengthening, but stimulation of already fatigued muscles is still subject of investigation. The possible risks due to ES have been much considered, but no evidence of any structural or functional damage due to ES has been recorded. Gordon and Mao (1994)conclude that low level of NMS for up to 2 hours a day appears to maintain fatigue resistance in muscles without reducing force.

A denervated muscle is different in many respects from an innervated muscle. This includes its response to electrical stimuli. Without a functional nerve supply, a muscle can only be caused to contract by direct stimulation of the muscle fibre. There are distinguishing items between muscles stimulated via the nerve and direct denervated muscle stimulation (Pieber *et al.,* 2015).

i. Muscle tissue is less excitable than nerve so a greater electric charge is needed.
ii. Slow worm-like contraction results because of slow speed of contraction through the muscle and diminished rate of contraction compared with innervated muscle.
iii. Slow-rising electrical pulses can stimulate muscle because it has less ability to accommodate than nerve.

There has been confusion and controversy over the therapeutic use of NMS on denervated

muscle for many years. The rationale has been to maintain the muscle in as healthy a state as possible by electrically-induced artificial exercise, while awaiting reinnervation. It seems reasonable that making muscles contract with electrical stimuli would substitute for the beneficial effect of normal muscle contraction. Newham points out that skeletal muscle has a great capacity for regeneration. When a muscle is denervated, many structural and functional changes occur (Pieber *et al.,* 2015).

i. Loss of voluntary and reflex activity

ii. Atrophy, degeneration and fibrosis

iii. Fibrillation – spontaneous contraction of muscle fibres.

There is considerable literature concerning the effect of electrical stimulation on denervated muscle, including evidence that it will retard muscle atrophy and degeneration but not completely prevent it (Kern, Salmons, Mayr, Rossini & Carraro, 2005).

The type and amount of stimulation used to achieve this is variable. The best results were achieved with vigorous isometric muscle contraction (Low & Reed, 2000).A review concluded that all the denervated muscle fibres must be activated, and isometric contractions are more effective than isotonic contractions. Regular stimulation should commence as soon as possible after denervation. Neuromuscular stimulation of muscle fibre contraction is a very vital concept that needs to be well demonstrated and recorded for the future. Neuromuscular stimulation for the purpose of helping persons with paralysis of the limbs mainly focuses on neuromuscular transmission peripherally. The choice depends on the conditions and goals of stimulation. Some of these currents are the faradic, direct, sinusoidal and Russian currents (Aureliovas & Anragao, 2012).

2.5 Effects of Cycle Ergometry in stroke Survivors

Cycle ergometry does not require much postural control, compared to the treadmill, and it is a better alternative for individuals with poor balance (Khuram, 2011). The rehabilitation is important to make the patient more independent and enable them regain lost activities. It usually starts from acute care and continues as a rehabilitation programme (AACPR, 1995). It has been shown in systematic reviews that rehabilitation after stroke increases patient survival and reduces the length of inpatient stay (Langhorne & Duncan, 2001). The main purpose of rehabilitation after stroke is to regain motor control in gait and related activities, to improve upper limb function and cope with activities of daily living to increase participation (Khuram, 2011). Approximately 100,000 first ever strokes occur each year. Half of all acute stroke survivors starting rehabilitation will have a marked impairment of function of one arm, and only about 14% of these will regain useful function. Several studies have been carried out, particularly in Africa, pertaining to hemiparetic stroke survivors. Among this, is a study on the effect of treadmill walking and over-ground walking exercise training on recovery of walking function in an African group of stroke survivors. The outcome proves that treadmill and over-ground walking combined with conventional rehabilitation improves walking (Brinkman & Hoskins, 2008).

Cycle ergometry can be used to achieve many functional, physiological and psychological benefits (Pang, Eng, Dawson & Gylfadottir, 2010). Postural imbalance or asymmetrical limb movement between affected and unaffected limbs are commonly observed in post stroke subjects (Szecsi *et al.,* 2008). The software computerized FES assisted cycling used on acute stroke survivors improves quantifiable biomechanical parameters such as smoothness and symmetry of cycling. Janssen *et al* (2008) reported that except for muscle strength, VO_2 peak and MET, no additional effect was seen in assistive ergometry. The cycling effect could be

explained by repetitive voluntary contraction of leg muscles as an adaptation which promotes circulatory adaptations, hypertrophy and histochemical changes in muscles all of which are helpful in relearning how to execute movements. Muscle strength is usually reduced after stroke (Patten; Lexell & Brown, 2004) thus; one of the aims of rehabilitation is to improve muscle strength, walking ability and increase possibilities to manage ADL (Richards; Malouin & Dean, 1999).

Clinical studies show that acute and prolonged cycling and running exercises enhance the cognitive act (Khuram, 2011). Studies compared 10 weeks of adapted cycle ergometry exercise with passive range of motion to control. Results showed significant differences in VO_2 peak, mobility and motor learning compared to the controls (Potempa, Lopez & Braun, 1995). Another study used matched control design to determine the feasibility of aerobic cycle training to conventional rehabilitation early after stroke. Results showed significant differences in 6 minutes walk test, spatio-temporal gait and stroke impact scale compared to the conventional group (Tang, Sibley, Thomas & Bayley, 2008).

2.6 Cardio-respiratory Evaluations in Stroke Survivors

Most of the clinical assessment tools are based on either self-report or observer-rated measures. Self-report measures, although simple to acquire, can be vague or inaccurate due to poor patient memory, unsound perceptions of performance, or misjudgments of actual capability (Ruben, 1995; Lieberman *et al.,* 1996). Observer-rated surveys by caregivers are often time consuming, involve limited opportunity for repeated observations, and rarely capture changes in functional status that may fluctuate throughout the day (Ruben, 1995). Instrumented devices that automatically and continuously monitor physical activity and

functioning provide an alternative to subjective assessment tools. The objectivity and comprehensiveness of a patient's physical performance record could be improved by a system that remote locations such as the patient's home or community (Kiani *et al.,* 1997).

It has been documented that motivation to exercise improves function (Brophy *et al.,* 2013). Even though both motivation and physical activity have an effect in increasing function, motivation does this by indirectly influencing physical activity levels and thereby increasing function (Brophy *et al.,* 2013). Providing advice on health behaviours without objective feedback of compliance or outcomes may lead the patient to develop "cognitive fantasies"; i.e. belief that they are adhering to the advice when they are not (Oettingen & Mayer, 2002; Naik *et al.,* 2007). Feed-back of progress towards goals can increase motivation, improve self (Locke & Lathem, 2002).

Pulse rate

Regular physical exercise is an important factor to reduce the indexes of cardiovascular and all causes morbimortality. However, there is, apparently, additional and independent benefits of the regular practice of physical exercise and the improvement of the level of aerobic condition. Pulse rate (PR) is mediated primarily by the direct activity of the autonomic nervous system (ANS), specifically through the sympathetic and parasympathetic branches activities over the sinus node autorhythmicity, with predominance of the vagal activity (parasympathetic) at rest, that is progressively inhibited since the onset of the exercise. The HR behavior has been widely studied during different conditions and protocols associated to the exercise. A reduction of the cardiac vagal tone (parasympathetic function) and consequently a diminished PR variability in rest, independently of the protocol of measurement used, is related to an autonomic dysfunction, chronic-degenerative diseases and

increased mortality risk. Individuals with high levels of aerobic condition have a lower resting PR, along with a larger parasympathetic activity or smaller sympathetic activity, but it is not necessarily a direct consequence of the exercise training, as long as other inherent adaptations to the aerobic conditioning can influence the resting HR. The PR response in the onset of the exercise represents the integrity of the vagus nerve, and the PR recovery on the post-exercise transient also denotes important prognostic information; by the way, individuals that have a slow PR recovery in the first minute post-exercise have increased mortality risk (Araujo, 2016). The pulse rate is simply the number of heart beats per minute. An average person's pulse rate is 70 beats per minute (Doohan, 2000). According to the American Heart Association (2010), the maximum safe pulse rate can be calculated by subtracting the persons age from 220. Maximal PR reduces with age, and at the same relative workload, women have a higher PR response than men, which compensates for the lower stroke volumes women have compared to men (Roberts, 2002).

Stroke volumes

The stroke volume, the amount of blood in milliliters (ml) pumped out of the heart with each beat is 70 ml/beat in an average resting person (Ganong, 1999). The stronger the contraction of the heart, the larger the stroke volume. Under conditions of rest, the heart does not fill to its maximum capacity and the ventricles empty only about 50% of their volume during systole, and this leads to low stroke volume. However, during periods of exercise, the stroke volume increases because of both of these mechanisms; the heart fills up with more blood and the heart contracts more strongly (Doohan, 2000).

Cardiac output

Cardiac output is the volume of blood pumped by the heart per minute (ml/min) (Doohan, 2000). For young, healthy men, resting cardiac output averages about 5.6L/min and for women, this value is about 4.9L/min. When the factor of age is considered as well, cardiac output declines to about 2.4 L/min by 80 years (Vincent, 2000). Cardiac output is a function of pulse rate and stroke volume, that is cardiac output in mL/min = pulse rate (beats/min) x stroke volume (ml/beat); thus, increasing either pulse rate or stroke volume increases cardiac output (Doohan, 2000).

The total volume of blood in the circulatory system of an average person is about 5 litres (5000 ml). Therefore, the entire volume of blood within the circulatory system is pumped by the heart each minute (at rest). During vigorous exercise, the cardiac output can increase up to 7 fold (35 L/minute) (Doohan, 2000).

Blood Pressure

Several studies reported that blood pressure (BP) at the utmost workload was elevated in cycle ergometry (CE) than in many exercise protocols (Maeder, Wolber, Atefy, Gadza, Amman, Myers & Rickli, 2005). These results point out that the degree of increase in BP on the CE is likely to be higher than the degree of increase in SBP on the TM. As the increased BP in CE could increase the myocardial burden of patients with BP-related diseases such as stroke, hypertension, and aneurysm, which may have an adverse, effect (Mundal, Kjelden, Sandrick, Erikssen, Thaulow & Erikssen, 1994). It should carefully be considered in the exercise test and exercise prescription. Blood pressure has not been reliable in the discovery of exertional hypotension. Blood pressure should be measured using a standard stethoscope

and sphygmomanometer (Froelicher & Umann, 1995). Systolic and diastolic blood pressure (SBP, DBP) should be recorded during the 5 minutes rest period before the beginning of the graded exercise test (GXT). Rimmer, Riley & Creviston (2000) modified exercise prescription to stroke survivors with abnormal blood pressure during exercise test (systolic $\geq$ 220 mm Hg, diastolic $\geq$ 110 mm Hg). The patients were instructed not to exceed a rate pressure product of 200 (ACSM, 2000). The resting diastolic blood pressure (DBP) should not be less than 100 mm Hg to begin exercising. If the resting DBP is greater than 100 mm Hg, range of motion exercises should be performed until DBP drops below 100. Exercise should be terminated, if blood pressure is elevated to 220/110 mm Hg or higher and should only be resumed, when blood pressure drops below that value (ACSM, 2002).

Blood pressure is to be recorded in the supine, sitting, and standing positions. During the GXT, SBP and DBP should be recorded during the last minute of each stage (usually every 3 minutes) and more frequently if warranted. During the recovery phase (at least 6 minutes) blood pressure is measured immediately after the termination of the GXT (patient in upright position) and every 1 to 2 minutes during the 6-minute recovery period or longer or after the cool down period if involved.During the exercise test, the normal response of the systolic blood pressure is to rise with increasing workload. A decrease in systolic blood pressure of 20 mm Hg or failure of the systolic blood pressure to rise with increasing workload is reason for test termination. Excessive rise in the systolic blood pressure (250 mm Hg) or diastolic blood pressure (120 mm Hg) is also cause for test termination (AACPR, 1995; ACSM, 1995).

2.7. **Training Guidelines and Responses for Stroke Survivors**

Responses and adaptations caused by training is highly dependent on multiple factors related to training, such as frequency, length of the training session and the type of training frequency, such as velocity of muscle action, duration, repetition of the activity and rest intervals. There are several basic principles related to these responses and adaptation. First, the overload principle means that an exercise overload specific to the activity must be applied to enhance physiological improvement and bring about the training response (Kraemer *et al.,* 2002). Second, the specificity principle states that specific exercise elicits specific adaptations (McArdle *et al.,* 1996). Third, training benefits are optimized when training programmes are planned to meet the patient's individual needs and capacities (Heck *et al.,* 2004). The fourth is the reversibility principle, which implies that the beneficial effects of exercise are transient and reversible (Coyle *et al.,* 1984). The exercise session should be supervised by a clinical exercise physiologist along with several assistants. Assistants should be trained to monitor blood Pressure, pulse rate and other vital signs. Participants should wear a pulse rate monitor for the entire session to ensure that they are exercising in the appropriate target pulse rate zone. Participants should be taught how to measure their own rate of perceived exertion (RPE), use the equipment safely even if assisted and understand the warning signs for when to stop exercising. Blood Pressure (BP), PR and evaluating vital signs for the study should be recorded prior to exercise and several times during the exercise session. At the end of each session, BP and PR should be recorded to ensure that they return to the resting values before departure from the study area. Participants should be encouraged to drink plenty of water to avoid dehydration (ACSM, 2002). The instructors should be skepticalto avoid fatigue and delayed onset muscle soreness, though these are common side effects in new resistance trainers. Stroke survivors may aspire to make rapid gains in strength

and can train at a moderate to high intensity level, the instructor should be cautious not to over work the muscle groups (Eng & Tang, 2007).

Many individuals with stroke have hand dysfunction on the hemiparetic side. This may make it difficult to grasp barbells or railings or handles on aerobic training equipment like cycle ergometry. There are several versions of specially-designed gloves that allow contact with the equipment. This will prevent injury while performing resistance training routines. Participants who have poor grip or grasp can use wrist cuffs or leather mitts with Velcro or bandage to secure their hands during rehabilitation programmes (Mota & Silva, 2014).

2.7.1. Intensity of Exercise

There is an intensity or level of aerobic exercise that is necessary to improve cardio-respiratory endurance. Although intensity and duration are separate entities and will be discussed separately, it is difficult to discuss intensity without mentioning duration because of the interaction between the two. Prescribing exercise intensity for all adults who have cardiac or pulmonary problems or those who are apparently healthy, is the most challenging task in designing the exercise programme because individualization and appropriate monitoring are required to ensure that the maximum prescribed intensity is not exceeded. The intensity of exercise is usually expressed in relative terms as a percent of functional capacity or a percentage of age-adjusted maximum heart rate (AAMHR) (ACSM, 1995). By using the data derived from the laboratory and clinical assessments, the clinician can formulate the exercise intensity portion of the rehabilitative exercise programme. Three techniques are commonly used to prescribe and monitor exercise intensity: pulse rate, metabolic energy expenditure (VO_2 or metabolic equivalents (METs), and rating of

perceived exertion (RPE) (Kavanagh, 1994; Temes, 1994, Pollock, Welsch & Graves, 1995; ACSM, 1995; Winslow, 1995).

2.7.2 Target Heart Rate for stroke survivors

During dynamic exercise involving large muscle groups, a relatively linear relationship exists between pulse rate and oxygen uptake. There are several widely used methods for establishing target heart rate (THR) for, apparently, healthy adults, as well as for patients with cardiac and pulmonary disease (ACSM, 1995). There are three commonly used methods of determining THR and target heart rate range (THRR) for the exercise prescription:

i. The heart rate reserve method or Karvonen formula;

ii. Percentage of maximal heart rate achieved on the GXT; and

iii. Resting heart rate plus 20 to 30 bpm. (Pollock, Welsch & Graves, 1995; Kavanagh, 1994; Temes, 1994 & Winslow, 1995; ACSM, 1995).

A fourth method for establishing THRR and THR that will be briefly presented is to plot the relationship between heart rate (PR) and VO_2. The heart rate reserve, Karvonen formula (Karvonen, Kentala & Mustala, 1957) is calculated by taking a percentage (usually 40 to 85 percent) of the difference between the resting heart rate (in the seated position) and the maximal achieved heart rate on a GXT and adding that value to the resting heart rate. The THRR is used to define safety guidelines for exercise intensity during the exercise session. Individuals with low levels of functional capacity should probably begin their exercise regimes at 40 to 50 percent of pulse rate reserve (ACSM, 1995). To ensure a cardio-respiratory endurance training effect, the THR for a specific patient defines the most

appropriate heart rate (HR) within the prescribed THRR. For most individuals, the training intensity would be approximately 60 percent of heart rate reserve (Swain, 1994). For a cardio-respiratory patient with mild to moderate impairment, the recommended THR should be calculated by using a minimum of 50 to 60 of the maximum functional capacity (Hodgkins, 1990). Patients with moderate to severe pulmonary impairment reach their maximum voluntary ventilations (MVVs), equal to maximal voluntary expiration before the cardiovascular maximums are approached. For these patients, exercise intensities that approach their maximum ventilator limits or the upper end of the THRR can be used. Fluctuations of approximately 10 percent normally occur in patient's heart rate during a single exercise session (Brannon *et al*., 1983; ACSM, 1995).

A second method to calculate THRR is to use a fixed percentage of the maximum heart rate (MHR) attained on the GXT. Generally, the percentage used to calculate the THR is between 55 to 90 percent (Pollock; Welsch & Graves, 1995; ACSM, 1995). This method is easier to calculate than the Karvonen method and a number of clinicians prefer it. It has been reported that a THR of 75 percent MHR correlate with the heart rate at ventilatory (anaerobic) threshold (VT) and, in absence of direct measurement of VT, can be used as a guide to determine THR in patients with cardiac disease. Cardiac rehabilitation programmes using the percentage of HRmax attained on the GXT for determining exercise intensity have reported an average of 65 percent (Winslow, 1995). In a case where patients enter rehabilitation with a symptom-limited GXT (SL-GXT), the HR at which the SL-GXT was terminated becomes the maximum HR for that subject. The maximum HR attained on the SL-GXT is then used in formulas for calculation of THR and THRR. If no SL-GXT or GXT has been given, the age-adjusted maximum heart rate (AAMHR) may be used in the formulas for calculating THR

and THRR. The AAMHR may be estimated by simply subtracting the patient's age from 220. The major problems with estimating HRmax arise from individual differences and medications that may alter expected resting and maximum heart rate (Brannon *et al.*, 1993).

The third method commonly used to establish the exercise training THR is to simply take the patient's resting pulse rate and add 20 to 30 beat per minutes to that value. This is the simplest of the three methods that have been presented, and is often used when no GXT or SL-GXT has been performed and the patient has been referred for cardiac rehabilitation (Winslow, 1995).

A fourth method used to establish a patient's THR and THRR is to plot the rate against measured VO_2 max. However, because this method requires an accurate measurement of VO_2 max, it is not practical to use in situations where direct measurement of VO_2 max is not obtained. Estimation of VO_2 max can be obtained from maximal MET levels, achieved on a GXT or an SL-GXT. Calculating THRR by this method is usually between 50 to 85 percent of VO_2 max achieved or estimated (ACSM, 1995).

Metabolic Energy Expenditure

Some clinicians prefer to prescribe exercise intensity by using activities that require a percent of the maximal Metabolic Energy Expenditure (METs) achieved during the evaluation process. Usually, the activity chosen for prescription by this method corresponds to 40 to 85 percent of maximal METs achieved during evaluation (Fox; Naughton & Gorman, 1972). As with any method of exercise prescription, in the beginning phases of rehabilitation, the exercise intensity should be prescribed at the lower end of the target range, in this case, near 40 percent of maximal METs, to monitor HRs by telemetry (in early rehabilitation) and/or

palpation (in later stages of rehabilitation). Adding HR monitoring to prescription by METs ensures that maximal safe exercise will not be exceeded, especially in situations that naturally cause increase in heart rate (for example, hot humid environment) and in the Rate-pressure product (RPP).

The rate pressure product is an excellent indicator of aerobic conditioning because the RPP decreases for a given workload as the patient becomes more conditioned. Cardiac and deconditioned subjects generally have higher RPPs for a given workload than physically-trained individuals. The RPP relates well to measured myocardial oxygen consumption, and it is possible to precipitate a patient's angina repeatedly at the same RPP when a standardized workload or exercise test is performed. The RPP illustrates the importance of both pulse rate and blood pressure responses when writing an appropriate exercise prescription (Kavanagh, 1994; Froehlicher & Umann, 1995).

2.7.4. Pulse Oximetry (SPO_2)

The amount of oxygen carried depending on hemoglobin in the blood is named as SpO2 and this forms the main mechanism for the transportation of oxygen to the cells (SpO2 was used to indicate that a non-invasive measurement was realized using pulse oximeter in this study). Measurement of oxygen saturation gives information about hypoxia (Giuliano *et al*., 2005; Hakemi *et al*., 2005). There is far less information available regarding the capabilities of pulse oximeters during exercise and most of this work is limited to studies of normal subjects and elite athletes (Gaskin & Thomas, 1995).

The use of pulse oximetry in the evaluation of oxygen saturation provides the individual continuous non-invasive measurement of both of the saturation and pulse without an invasive

attempt (Woodrow, 1991). Patients with stroke are prone to respiratory problems for reasons which include: alterations in the central regulation of respiration (Nachmann *et al.,* 1995), sleep apnoea (Bassetti & Aldrich, 1999; Harbison & Gibson, 2000), weakness of the respiratory muscles on the hemiplegic side (Fluck, 1966; Haas & Rusk, 1967; De Troyer, De Beyl & Thirion, 1981), aspiration (Smith *et al.,* 2000), chest infections (Davenport *et al.,* 1996; Langhorne, *et al.,* 2000), left ventricular failure (Davenport *et al.,* 1996), and pulmonary emboli (Davenport *et al.,* 1996; Langhorne *et al.,* 2000). There is significant loss of muscle function resulting from stroke, and survivors have reduced arterial oxygen saturation and peak oxygen uptake (Potempa, Lopez & Braun, 1995; Rimmer, Riley & Crviston, 2000). Barcroft indicated that there was diminish in oxygen saturation of arterial blood during exercise (Moazami, *et al*., 2013; Barcroft, 1975). Penaloza and company similarly reported that they obtained a decline in the oxygen saturation with acute exercise (Penaloza, *et al*; 1962). The pre and post test differences between the groups were compared, any significance was not found (Bijeh and Farahati, 2013; Vogel *et al*., 1962). Short-term training lowers the oxygen saturation of soccer players and this decrease arised due to the sudden need of oxygen in skeletal muscle. A graded exercise test to achieve improved oxygen performance ($VO_{2)}$ and SPO_2 may be performed on a stationary bike or treadmill or in persons with severe hemiplegia, with an arm ergometry. Goodman (1996) noted that arm cranking yields lesser VO_2 than the treadmill. The preferred exercise mode is the stationary cycle since most stroke survivors have challenges with gait and balance. The stationary cycle makes it easier to quantify external work load, because the energy requirements of treadmill walking will vary as individuals change stride length, shift centre of gravity, swing arms, or

hold onto rail (Potempa *et al.,* 1996). Additionally, it is easier to record blood pressure measurements on the cycle ergometry (ACSM, 2002).

The European Ad-Hoc Consensus Group advocated that in all stroke survivors' oxygen saturation should be monitored continuously or at regular intervals (Kelleher, 1989*).* Pulse oximetry allows oxygenation to be monitored continuously and non-invasively (Lindberg, Lennmarken & Vegfors, 1995). Motion artifact can affect oximeter readings in stroke survivors, such problems may be reduced by placing the oximeter on the affected side (Lindberg *et al*., 1995; Cowen, 1997; Woodraw, 1999).

2.7.5. Rate of Perceived Exertion

The patients become familiar with the feeling associated with exercise at the appropriate intensity. There is need for an objective measurement. At that point of perceived exertion, the scale provides the means of monitoring exercise intensity (Borg & Lindaholm, 1967). Borg' rating of perceived exertion scale (Borg, 1973), is used by patient with cardiac disease to rate the intensity of an exercise activity; a rating of 12 to 13 (somewhat hard) on the 20-point scale correspond to approximately 60 percent of heart rate (HR) range, whereas a rating of 16 (hard) corresponds to 85 percent. If the 10-point scale were used, the rating would be between 4 and 6 (ACSM, 1991). Adding a zero on the 20-point scale indicates at what level of HR, the patient is performing the exercise.

Borg's scale of perceived exertion and the revised 10-grade scale

The 10-point ratings scale (Borg, 1973) is as follow

- **i.** 0 = nothing at all
- **ii.** 0.5 = Very, very weak (just noticeable)

iii. 1 = Very weak

iv. 2 = Weak (light)

v. 3 = Moderate

vi. 4 = Somewhat strong

vii. 5 = Strong (heavy)

viii. 7 = Very strong……

ix. 10 = Very very strong (almost maximum) maximal

Table 2.1: Classification of intensity of exercise based on 30 to 60 minutes of endurance training

HRmax	VO_2 max or HRmax (beat/min)	RPE (20-Point scale) %	Intensity
35	30	10	very light
35-59	30-49	10-11	light
60-79	50-74	12-13	moderate
80-89	75-84	14-16	heavy
90	85	16	very heavy

HRmax − maximum pulse rate; VO_2 max− maximum oxygen uptake; RPE− rate of perceived exertion. Source: from Pollock and Wilmore, 1995.

The exercise programme for the stroke survivors shall follow the standard protocol. The stroke survivor shall engage in an hour of physical activity, a minimum of three days per week in a hospital wellness programme or community-based setting like a fitness centre or university-based programme. Self-initiated exercises should be done on days when the subject does not attend the Programme. Exercise programmes should consist of cardiovascular endurance, muscle strengthening and endurance flexibility (ACSM, 2000). Rimmer *et al.,* (2000) initiated a strength training protocol for stroke survivors for one set of 15-20 repetitions. The rate of perceived exertion should be recorded at the training. Participants should stretch at the beginning of flexibility training and at the end of the

exercise session. Stretches should be held for 15-30 seconds for muscles on the hemiparetic side (ACSM, 2002). Participants should begin with intermittent exercise during the first 4 weeks of the programme, after which most of them should be able to complete 30 minutes of continuous exercise in their THRR.

CHAPTER THREE

3.0. RESEARCH METHODOLOGY

3.1. Introduction

This study investigated the effects of neuromuscular stimulation (NMS) and cycle ergometry (CE) training to determine the level of functional abilities of stroke survivors in Kano State, Nigeria. To achieve this purpose, the research design, population, sample size and sampling techniques, instrumentation, training protocol, Procedures for data collection and statistical analysis are described in this chapter.

3.2. Research Design

A 2 x 3 x 4 factorial research design was used for this study. Participants were assigned to two experimental groups of neuromuscular stimulation (NMS) and cycle ergometry (CE). Three assessments were made at baseline, immediately after 6^{th} and 12^{th} week of training on four (4) dependent variables of strength, flexibility, coordination and cardiorespiratory indices of stroke survivors in Kano State, Nigeria.

3.3. Population

The subjects for this study comprised of forty (40) newly referred male and female stroke survivors from all Local Government Areas of Kano State to the physiotherapy Unit of the Murtala Mohammed Specialist Hospital (MMSH), Kano and from the Out-patient Unit of the Neurology Department. Information from the Records Unit showed an average of 5 newly-referred stroke survivors per week during the period of this study (Record officer, Department of Physiotherapy, MMSH, Kano, 2015).

3.4. Sample and Sampling Techniques

The minimum sample size was calculated using the expression of medium effect:

$n = N (Z_1 + Z_2)^2/ES^2$.

n = number of sample size

N = number of groups

$Z_1 = 1.96$

$Z_2 = 0.84$

ES = medium effect; 0.9 adopted (Stroke)

Therefore,

$n = 2(1.96 + 0.84)^2 / 0.9^2$

= 19.35

For expected attrition of 10%, n = 19. + 1.9 = 20.9, approximately = 21 minimum number of stroke survivors participated in this study. Forty (40) were given informed consent but only 30 participants consented to participate in the study.Consecutive sampling technique was used, in which the first two subjects were randomly selected using a coin into the two training groups and other participants were alternately distributed between the two groups (NMS and CE) for the training.

3.4.1 Inclusion Criteria

The inclusion criteria for this study include:

1. History of single unilateral left hemiplegia or hemiparesis not more than one year.
2. 5^0 active wrist extension and 1^0 finger extension (Taube, Uswaite & Elbert, 2002).
3. Male and female adult between the ages of 40 and 60 years.
4. No aphasia- dominantly left sided.
5. Ability to understand and sign the informed consent form.

3.4.2 Exclusion Criteria

The exclusion criteria for this study are:

6. Implanted or pace maker device
7. Unstable/fatal cardiac arrhythmias
8. Clinical evidence of shoulder subluxation from one finger width (Monye, 2012).
9. More than two episodes of stroke and duration of more than a year.

Neuromuscular Stimulation

Training Protocol

This group of participants had the training three times per week for 12 weeks. The subjects rested for 2 minutes as soon as they entered the cubicle of the physiotherapy Unit. The participants were taking medications and were medically certified to be free from major cardiovascular and/or respiratory risk at the time of the study. Each participant sat on the chair and the area of stimulation (left upper limb and left lower limb) was cleaned with soap and water to remove sebum and dirt to reduce skin resistance to improve conductivity. Baseline measurements of the grip strength, pinch strength, joints range of motion of the left upper limb and lower limb; systolic and diastolic blood pressure, pulse rate and oxygen saturation; and block and box activity and the target heart rate was calculated at 35% - 45%

and each participant underwent active upper limb and lower limb possible movement as warm-up for 5 minutes without the electrical stimulator (ES) attached on couch. This was followed by the NMS exercise for 20 minutes along with active possible movements from the participants, at the low intensity (35% - 45%) of their target heart rate. The active electrode was placed on the muscle trunk, while the passive electrode was placed at the most bulging belly of the muscle at a faradic current enough to initiate comfortable visible contraction (Clayton, 1978).

Adaptation was expected at the end of the first 3 weeks. The NMS was progressed to 25 minutes with intensity of 50 – 55% of their target heart rate and also maintained at 3 times per week for 5 weeks and decreased faradic stimulation for muscular contraction. In the last stage, that is the last 4 weeks, patients underwent training with progression to 30 minutes at intensity of 60-65% of their target heart rate and lower intensity of faradism. Measurements of the variables were taken at the end of 6^{th} and 12^{th} week of the training.

All patients in each session were allowed 5 minutes of rest after training. Meals were provided after the training and transport fare (to and fro) was given to all patients for the 12 weeks. Rate of perceived exertion (RPE) of 5-6 were considered for termination of the training. The decreasing stimulatory current will actively allow for active exercises by the participants.

Cycle Ergometry Training Protocols

The participants in this group sat for 2 minutes before the commencement of training in the physiotherapy Unit, where the cycle ergometers were mounted. Participants were assisted to

climb the cycle ergometers and supportive Velcro and bandages were used for stabilizing the affected limbs.

Due to the maximal exertion required for the cycle ergometry training, a warm up was carried out based on the Ethics Committee requirements and ACSM (2010) recommendations for training stroke survivors. Prior to reporting to the Physiotherapy Unit at Murtala Mohammed Specialist Hospital, Kano for the testing, subjects were given the following instructions: no eating, drinking other than water within three to four hours and no heavy exercise within 8 hours prior to testing. Work intensity was adjusted by variations in resistance and cycling rate.

Baseline measurements were taken after 2 minutes rest as the participants came into the cycle ergometry shelves. The participants underwent 5 minutes warm-up and passive stretches then 20 minutes cycling with the left limbs stabilized or aided with Velcro bandages, depending on the patient's ability at 35% - 45% of maximal heart rate (HRmax) for duration of four 3 weeks. During the 4^{th} – 8^{th} week of CE, training session lasted for 25 minutes without resistance while the intensity was increased from 50-55% of the HRmax.

The last session, had the participants rode the cycle ergometer three times per week for 30 minutes at 60-65% of HRmax for 4 weeks (9^{th} – 12^{th} week). All post-training measurements were taken at baseline, immediately after 6^{th} and 12^{th} week.

3.5 Experimental Management

3.5.1 Research Assistants

This study used four research assistants. One Exercise and Sports Scientist and three Physiotherapists were used during the period of this study. The Physiotherapist measured the cardio-respiratory parameters and electrode placement for the participants in the NMS group, and measures the range of joints motions in both groups. The Exercise Scientist measured the grip and pinch strength for both group of participants and man the cycle ergometry group. All the research assistants were trained on the training protocols and measurements of blood pressure, pulse rate and intensity calculation and monitoring, SPO_2, grip strength, pinch strength, joints range of motion and block and box protocols. The training for the cycle ergometry (CE) was conducted on Tuesday, Thursdays and Saturdays while that of neuromuscular stimulation (NMS) was conducted on Monday, Wednesdays and Fridays.

3.5.2. Ethical Approval

The researcher received an introductory letter by the Department of Physical and Health Education, Ahmadu Bello University, Zaria (Appendix C), introducing the researcher to sought permission to carry out the study in Kano State hospital., and the ethical approval was granted by the Hospital Management Board, Kano State (Appendix B) to carry out the at the physiotherapy Department of Murtala Muhammad Specialist Hospital, Kano State, Nigeria.

3.6. Instruments for Data Collection

The following instruments were used for data collection:

i. Hand-grip dynamometer (Camry Digital, EH101, M2C mart manufacturers, China, 2008)

ii. Pinch gauge (SH5001, England, 1992)

iii. Goniometre (Hand made with protractor, Orthopaedic, Kano, Nigeria)

iv. Stop watch (Swiss, Swatch, 1.53volt, Swiss)

v. Wooden box and block (Hand-made, Kano, Nigeria)

vi. Electro stimulator (AMI Surgical, 13ko1126c, Mumbai, India, 2003)

vii. Pulse oximeter (Easy care, 002012, CE 0123, Bangalore, India, 2005)

viii. Cycle ergometer (Hit-up, American Fitness, China)

x. Wrist Electronic Sphygmomanometer (Wrist electronic, CK102S, Gwanzhou, China, 2009).

3.7. Procedures for Data Collection

The extensors of the affected wrist were stimulated, selecting lateral epicondyle for the active electrodes, while the belly of the muscle had the passive electrode. The nature of modality and tactile sensation were explained to the patient, which were tingling sensation and contraction of the muscles under stimulation with the device turned ON. The hamstring and dorsiflexors of the affected limb were mobilized by electrostimulation and active exercise in lying position. The active electrode was placed at the muscle trunk of the back at lumbar plexus, while the passive electrode was placed at the muscle belly. The cycle ergometer was rode without resistance for the stroke participants for 20, 25 and 30 minutes at 35% - 45%, 50 – 55%, 60 – 65% of maximal heart rate (HRmax) and the assessments were taken at baseline, after the 6^{th} and 12^{th} week of training.

3.7.1 Validity of Measuring Tools.

All instruments were reliable tools used in stroke rehabilitation and fitness centre. The dynamometers have been used in assessment of adult population having poor hand strength due to peripheral nerve injuries and hand trauma using strength dynamometers (Gill, Reddon, Renny & Stefanyk, 1985), Sphigmomanometers and pulse oximeters are valid cardio-respiratory assessment tools for medical screening of patients with cardiac and pulmonary problems (Howell, 2002; Turner, Speechly and Bignell, 2007). Goniometry is a reliable device to measure limitations, following joint injuries (Norkin and Whites, 1988), while block and box performance test was used in subjects with dexterity problem (Mathiowetz & Ferderman, 1985).

Plate 1: Mini-electrical stimulator for stimulation of muscle (Ami Surgical, 13ko1126c, Mumbai, 2003).

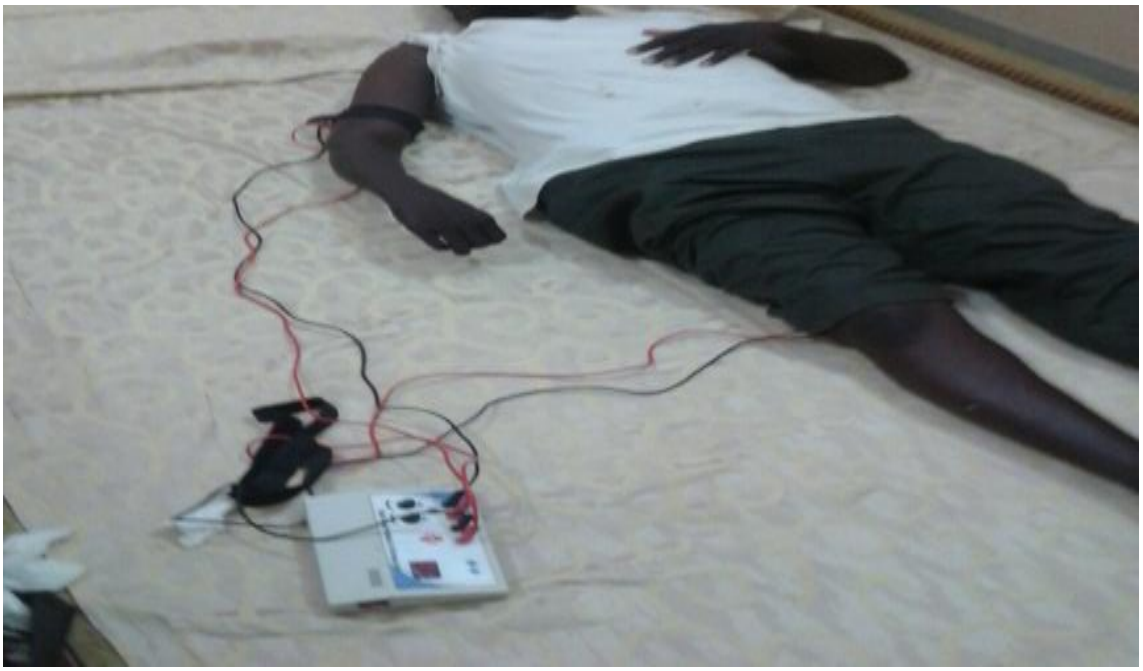

Plate 11: Neuromuscular stimulator for muscular stimulation with active limb movement

Plate 111: A cycle ergometer for movement of limbs

Plate IV: Modified cycle geometry for movement of the limbs of stroke patients

3.7.2 Measurement of Grip Strength

The hydraulic hand dynamometer is a precision instrument for objective measure of poor strength and functional performance in hand trauma and disease (Gill, Reddon, Renny & Stefanyk, 1985). The patient was asked to take a comfortable sitting position, with the upper limb on a table and wrist at the end of the table to avoid use of other synergy muscles. The dynamometer had a scale to measure grip force in kilograms and a red peak hold needle for grip value identification. Grip strength was measured at baseline, immediately after 6^{th} and 12^{th} week of intervention.

The subject held the dynamometer with the affected hand, with the arm at right angles and the elbow by the side of the body. The handle of the dynamometer was adjusted if required, the base should rest on first metacarpal (heel of palm), while the handle should rest on middle of four fingers. When ready the subject squeezes the dynamometer with maximum isometric effort, which was maintained for about 5 seconds. No other body movement was allowed. The subjects strongly encouraged to give a maximum effort (Helen *et al.,* 2011).

The subject was requested to squeeze the dynamometer three times and the average score was recorded for accuracy, at the end of the exercise session.

Plate V: Camry digital hand-grip dynamometer for measuring hand-grip strength

3.7.3 Measurement of Pinch Strength

Pinch strength dynamometer was used to take pinch strength measurement with the subject in a comfortable sitting position. The dynamometer was used to measure pinch strength in kilograms as calibrated on the device. Hydraulic Pinch Gauge – The finger pinch gauge dynamometer can be used to measure pinch strength. It is calibrated in pounds and kilograms of force. Pinch force was applied at the pinch groove between the thumb and the index

finger. The gauge was “zeroed” before each pinch test. The knurled ring of the indicator was grasped and rotated until the zero mark on the indicator is directly beneath the black pointer.

The red maximum pointer was reset before each pinch test. The small knurled knob was rotated to the top of the indicator in a counterclockwise direction until it rests against the black pointer at the zero point. The red maximum pointer remains at the subject's maximum reading until it is reset.

The pinch gauge was used to perform the following three basic pinch tests:

i. Tip Pinch (thumb-index pulp pinch) - thumb tip to index fingertip.

ii. Key Pinch (lateral pinch) - thumb pad to lateral aspect of middle phalanx of index finger.

iii. Palmer Pinch (chuck pinch) - thumb pad to pads of the index and middle fingers. (Baseline Evaluation tools, 2012).

Average of three readings were taken and recorded at baseline, immediately after 6^{th} and 12^{th} week.

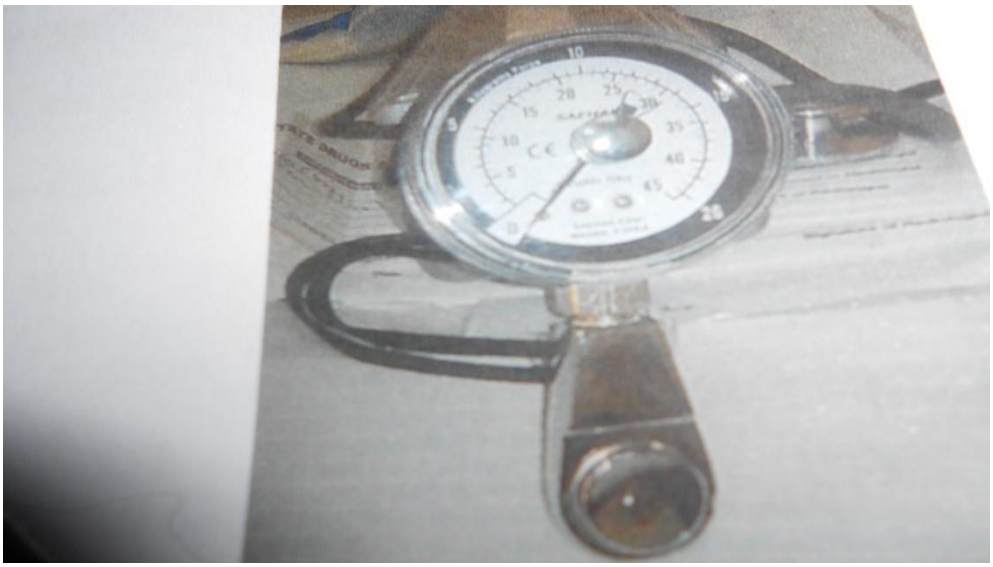

Plate VI: Pinch gauge dynamometer for measuring pinch strength

3.7.4 Measurement of Joint Range of Motion

The participants were asked or assisted to lie comfortably on the couch. The goniometer was placed at the reference points of the shoulder, elbow and wrist joints while the lower limb; hip, knee joints and ankle joints range of motion were also taken to record the possible joint flexibility among stroke participants of the study. These measurements were taken three times and the average score was recorded before, during and after interventions.

Measurements of Goniometer

The researcher palpates the relevant bony landmarks and aligns the goniometer to the part of the body. The degree of measure is based on the level of active movement from the stroke participants. The goniometer was then moved through the available active joint range of motion and the measurement was recorded (Norkin & White, 1988).

The participants were positioned in the lying position, in the cubicle couch. While, the proximal joint was stabilized, the researcher then gently moves the distal arm of the goniometer device through the full joint range of motion carried out by the participant.

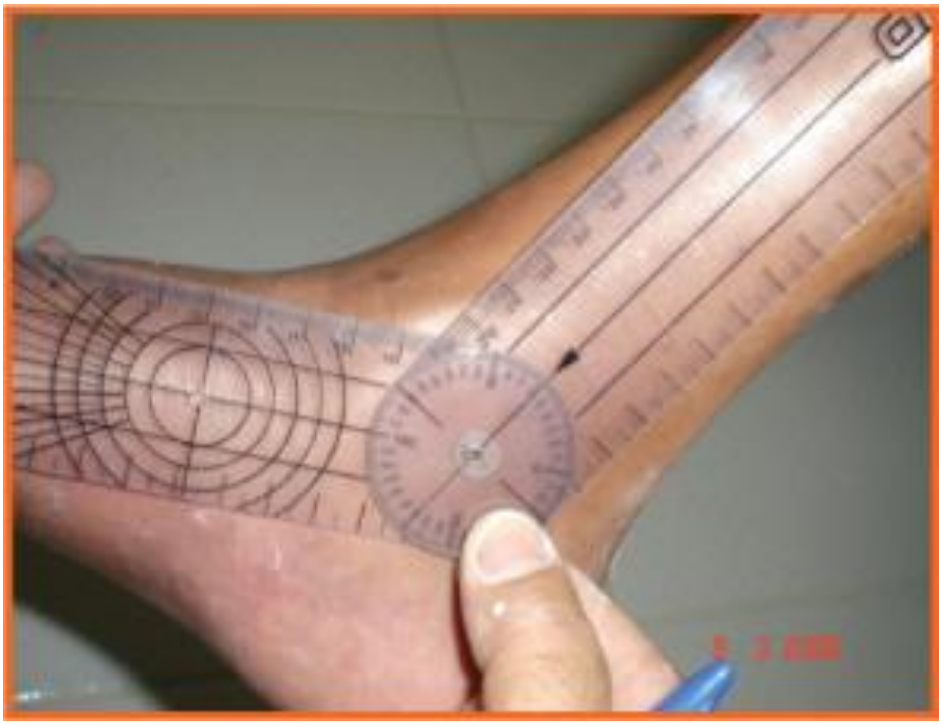

Plate VII: Goniometer for measuring joints range of motion

3.7.5 Measurement of Block and Box Performance (Coordination)

Each subject was comfortably placed on a chair close to the table containing the block and boxes in square shape. The subject was then requested to pick one square block from one section of the box and drop it in the other compartment one after the other in 10 minutes. The block of squares was counted and recorded as the performance.

Individuals are seated at a table, facing a rectangular box that is divided into two square compartments of equal dimension by means of a partition. One hundred and fifty, 2.5 cm, colored, wooden cubes or blocks are placed in one compartment or the other. The individual is instructed to move as many blocks as possible, one at a time, from one compartment to the other for a period of 600 seconds. Standardized dimensions for the test materials and

procedures for test administration and scoring have been provided by Mathiowetz and Ferderman (1985).

To administer the test, the participant is seated opposite the observer in order to observe test performance. The block box Performanceis scored by counting the number of blocks carried over the partition from one compartment to the other during the ten-minute trial period. The Patient's hand must cross over the partition in order for a point to be given, and blocks that drop or bounce out of the second compartment onto the floor is still rewarded with a point. Multiple blocks carried over at the same time count as a single point. Higher scores on the test indicate better gross manual dexterity.

Plate VIII: Block box device for assessing hand dexterity

Plate IX: Block and box test for measuring of coordination

3.7.6 Measurement of Cardio-respiratory Indices

The Cardiorespiratory measures were conducted during the training (NMS and CE). The blood pressure was measured using the digital sphygmomanometer. The pulse rate (PR) was also taken using the hand-stop watch and recorded; SPO_2 was measured using a portable pulse oximeter.

Measurement of Blood Pressure

The blood pressure measurement was carried out using properly sized blood pressure cuff on the participants. The length of the cuff's bladder was placed at the cubital region. The participants were asked to wear loose-fitting clothe that is short sleeved shirt, so that sleeve can be adjusted easily. After the attachment on the arm, the blood pressure is measured by switching on the digital Sphygmomanometer until the values are displayed.

The participant is made to rest for five minutes with the arm resting on a firm surface and feet flat on the floor. The arm was then supported, placing the cuff around the arm at the same level of the participant’s heart to prevent tension and discomfort. The cuff was wrapped

around the wrist with the cuff's lower edge one inch above the proximal radio ulnar joint. The stethoscope's bell was pressed over the brachial artery just below the cuff's edge to measure the diaphragm blood pressure (Turner, Speechly & Bignell, 2007).

Plate X: Digital sphygmomanometer for measuring blood pressure and pulse rate

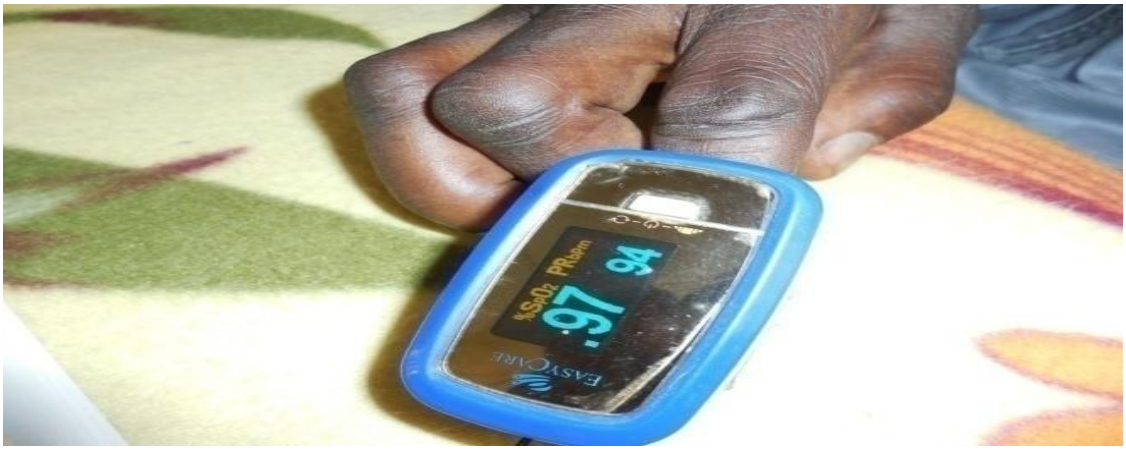

Plate XI: Portable pulse oximeter for measuring oxygen saturation in the blood

Measurement of Oxygen Saturation

Each stroke participant washed his/her hands. This is to reduce transmission of microorganisms and body secretions as standard precaution for prevention of skin infections. The desired sensor site was selected to show the presence of an arterial blood flow to the area. Adequate arterial pulse strength is necessary for obtaining accurate SPO_2 measurements. The sensory finger is placed on the oximeter to allow SPO_2 measure and analysis of waveforms (Howell, 2002). It usually takes about 30 seconds for the procedure. The record for oxygen saturation and waveforms were displayed (Rutherford, 1989). This procedure was repeated three times and the average reading was recorded.

3.8. Procedures for Data Analysis

The descriptive statistics of means and standard deviation were used to compute the demographic characteristics of the participants. Inferential statistics of repeated-measures analysis of variance (ANOVA) were used to evaluate the effects of the training programmes on the selected variables of stroke survivors. Where there are significant effects, the *post-hoc* test was computed to locate the period when the significant change occurred.

All the analyses were computed using the Statistical Package for Social Sciences SPSS version 20.0 (Window Evaluated Version 20, Lead Technology, Illinois, Chicago, USA). An alpha level of 0.01 was used to retain or reject the null hypothesis.

CHAPTER FOUR

4.0 RESULTS AND DISCUSSION

4.1 Introduction

This study investigated the effects of neuromuscular stimulation (NMS) and cycle ergometry (CE) on grip strength, pinch strength; upper and lower limbs joint range of motion, coordination and cardio-respiratory indices of stroke survivors in Kano State, Nigeria. The range of movement assessed were shoulder, elbow, wrist; hip, knee and ankle joints. The cardio-respiratory indices of the participants investigated were systolic blood pressure (SBP), diastolic blood pressure (DBP), pulse rate (PR) and oxygen saturation (SPO_2). Each group of the stroke survivors were exposed to neuromuscular stimulation and cycle ergometry training three times per week on alternate days for duration of 12 weeks. Training intensity was maintained at low to moderate pace for both protocols. The results obtained are presented in line with the research questions, hypotheses and the discussion of the findings in this chapter.

4.2 Results

The NMS and CE groups were exposed to training for a period of 12 weeks with three assessments (at baseline, immediately after 6^{th} and 12^{th} week). Thirty (30) participants started the training but only twenty five met the required attendance (NMS group had 12 and CE group had 13 participants) and whose data have been used for this analysis. A summary of physical characteristics of the participant is presented in table 4.2:1a.

Table 4.2.1a: Physical Characteristics of the Participants

Variables	Group	N	Mean (x)	S.D
Age (Years)	NMS	12	49.00	9.22
	CE	13	48.85	11.90

Duration of Stroke	NMS	12	17.00	2.39
(weeks)	CE	13	14.60	2.00

The mean age of the participants was 49 ± 9.22years and 48.85 ± 11.90 years for the NMS and CE groups respectively. The result also indicated that the NMS group have had stroke for 17±2.39 weeks while the CE group has had it 15±2.00 weeks.

Test of Research questions and Hypotheses

Research question I: Would NMS and CE improve grip strength of stroke survivors in Kano State, Nigeria?

The effects of 12 weeks NMS and CE on grip strength of the participants is presented in table 4.2.2a

Table 4.2.2a: Means and Standard Deviation of the effect of neuromuscular stimulation and cycle ergometry on grip strength of stroke survivors in Kano State, Nigeria

	Neuromuscular stimulation		Cycle ergometry	
Period	Mean (Kg)	S. D.	Mean	S. D.
0 week	2.86	4.369	2.77	2.725
6 weeks	2.80	3.152	3.83	3.792
12 weeks	6.82	5.446	9.31	6.403

Participants who were exposed to the NMS did not show much improvement after the 6th week (2.80 ± 3.15) until at the end of the 12th week of training period where the grip strength improved from the baseline mean score of 2.86 ± 4.369 kg to 6.82 ± 5.44 kg,However, the participants exposed in the CE group improved significantly (3.83kg ± 3.792) after 6thand 12th week (9.31kg ± 6.403) week of the training.

Sub Hypothesis I: There is no significant effect of NMS and CE on grip strength of stroke survivors in Kano State, Nigeria.

The measurement for the grip strength over the three periods (baseline, 6th and 12th week) of the training was computed using repeated-measures analysis of variance. The result of the analysis is presented in table 4.2.2b.

Table 4.2.2b**: Repeated-measures analysis of variance on the effects of NMS and CE on grip strength of the participants**

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	479.353(a)	5	95.871	4.735	0.001
Intercept	1496.045	1	1496.045	73.891	0.000
Type	11.520	1	11.520	.569	0.453
Period	442.053	2	221.026	10.917	0.000*
Type * Period	25.781	2	12.890	.637	0.532
Error	1336.282	66	20.247		
Total	3311.680	72			
Corrected Total	1815.635	71			

F (2, 72) = 4.91 P < 0.000 * significant

Table 4.2.2b shows the results of repeated measures ANOVA of the effects of NMS and CE on grip strength of the participants. Observation of the results revealed that the types training did not differ significantly (P = 0.453) in their effects grip strength of the participants. However, the effects on the grip strength improved by the period of training for the two groups (P = 0.000). Based on these observations, there was sufficient evidence to reject the null hypothesis which states that there is no significant effect of NMS and CE on grip strength of stroke survivors used for this study.

The Scheffe *post-hoc* test procedure on the grip strength measured at the different periods of the training is presented in table 4.2.2c.

Table 4.2.2c: Pair-wise comparison of grip strength measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	.013	.276	1.000
	12 weeks	-5.250(*)	.914	.000*
6 weeks	0 week	-.013	.276	1.000
	12 weeks	-5.263(*)	.877	.000*
12 weeks	0 week	5.250(*)	.914	.000*
	6 weeks	5.263(*)	.877	.000*

* The mean difference is significant (P< 0.001)

The result of the *post-hoc* test revealed that grip strength of the participants in theNMS group improved after the 12^{th} week of training (P = 0.000). In the CE group, significant improvement in grip strength was observed after the 6^{th} and 12^{th} week of training (P = 0.000).

Research question II: Would NMS and CE improve pinch strength of stroke survivors in Kano State, Nigeria?

The effect of 12 weeks NMS and CE on pinch strength of the participants is presented in table 4.2.3a.

Table 4.2.3a: Means and Standard Deviation of the effect of neuromuscular stimulation and

Cycle ergometry on pinch strength of stroke survivors in Kano State, Nigeria

	Neuromuscular stimulation		Cycle ergometry	
Period	Mean(kg)	S. D.	Mean(kg)	S. D.
0 week	0.62	0.671	1.06	0.699
6 weeks	1.08	0.578	1.66	1.096
12 weeks	4.36	2.515	6.50	2.876

Improvement in the pinch strength of the participants in the two training groups had a linear trend with much more effect at the end of 12^{th} week training. The participants in the NMS exercise group showed improvement in the pinch strength after the 6^{th} week (1.08 ± 0.578 kg and 4.36 ± 2.515 kg after the 12^{th} week.

The improvement among the participants in the cycle ergometry improved from the 1.06 ± 0.699 kg at baseline to 1.66 ± 1.096 kg after the 6^{th} week and 6.50kg ± 2.876 after the 12^{th} week. An observation of the data showed that the participants in the CE group improved better than those in the NMS group.

Sub Hypotheses II: There is no significant effect NMS and CE on pinch strength of stroke survivors in Kano State, Nigeria.

The pinch strength of stroke survivors measured during the three periods (baseline, immediately after 6^{th} and 12^{th} week) of training were computed to determine the significance effects of NMS and CE and is presented in table 4.2.3b.

Table 4.2.3b: Repeated-measures analysis of variance on the effects of NMS and CE on pinch strength of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.

Corrected Model	333.378(a)	5	66.676	23.440	.000
Intercept	466.651	1	466.651	164.051	.000
Type	19.950	1	19.950	7.013	.010
Period	302.703	2	151.352	53.207	.000*
Type * Period	10.724	2	5.362	1.885	.160
Error	187.741	66	2.845		
Total	987.770	72			
Corrected Total	521.119	71			

F (2, 72) = 4.91 P < 0.01 * = significant.

Table 4.2.3b shows the results of repeated measures ANOVA on the effects of NMS and CE on pinch strength of the participants. Observation of the results revealed that the training programmes caused statistical significant effects on the pinch strength of the participants by period (P = 0.000). Based on this observation, the null hypothesis which states that there is no significant effect of NMS and CE on pinch strength of stroke survivors in Kano State, Nigeria is rejected.

The Scheffe *post-hoc* test procedure on the pinch strength measured at the different periods of the training is presented in table 4.2.3c.

Table 4.2.3c: Pair-wise comparison of pinch strength of the participants measured at the different periods of the training

(I) Period	(J) Period	Mean Difference	Std. Error	P-value
0week	6 weeks	.533(*)	.163	.023
	12 weeks	4.592(*)	.475	.000*
6weeks	0 week	.533(*)	.163	.023
	12 weeks	4.058(*)	.474	.000*
12weeks	0 week	4.592(*)	.475	.000*

6 weeks	4.058(*)	.474	.000*

* The mean difference is significant at P < 0.01*.

The result of the *post-hoc* test revealed that pinch strength of the participants in the NMS group and CE improved from baseline, immediately after the 6th and 12th week of training in a linear trend.

This means that the two training programmes significantly improved the pinch strength of the stroke participants (P < 0.001) involved in the study.

Research question three: Would NMS and CE improve joints range of motion of upper and lower limbs of stroke survivors in Kano State, Nigeria?

The effect of NMS and CE programmes on joint ROM of the participants is presented in table 4.2.4a.

Table 4.2.4a: Means and Standard Deviation of NMS and CE effects on joints range of motion of stroke survivors in Kano State, Nigeria

		Neuromuscular stimulation		Cycle ergometry	
Parameter	Period	Mean(^{o})	S. D.	Mean(^{0})	S. D.
	0 week	31.17	3.354	40.58	6.256
Shoulder	6 weeks	31.42	2.788	41.83	6.050
extension	12weeks	36.58	4.755	53.50	4.538
	0 week	16.83	8.451	13.42	6.244
Elbow	6 weeks	25.00	8.820	18.58	6.007
extension	12weeks	24.83	7.004	29.75	11.910

	0 week	4.08	3.232	3.25	2.989
Wrist	6 weeks	11.33	7.266	9.83	5.540
extension	12weeks	19.17	9.408	22.33	8.742
	0 week	46.67	8.391	38.42	6.014
	6 weeks	56.67	4.039	58.25	6.503
Knee flexion	12weeks	78.25	4.038	102.83	8.761
	0 week	8.33	3.725	7.92	3.088
Hip	6 weeks	10.00	3.766	11.17	2.887
extension	12weeks	16.50	6.544	7.33	5.087
Ankle					
Dorsiflexion	0 week	5.08	2.503	3.58	1.782
	6 weeks	5.17	2.125	4.00	1.706
	12weeks	7.25	2.598	6.67	1.875

The table above shows the goniometric scores of both group on the joints of both upper limb and lower limb at baseline, immediately after the 6^{th} week and after the 12^{th} week of training.

Sub Hypotheses III: There is no significant effect of NMS and CE on Joints ROM of upper and lower limbs of stroke survivors in Kano State, Nigeria**.**

The data collected at baseline, 6^{th} and 12^{th} week of NMS and CE training was analyzed using repeated measures analysis of variance.

The shoulder extension of the participants showed no much improvement until at the end of the training period where ROM of shoulder improved from a baseline mean score of 31.17 ± 3.354˚ to 36.58˚ ± 4.755˚ after the 12^{th} week for participants in the NMS group while ROM of those in the CE training recorded an increase from 40.58 ± 6.256˚ at baseline to 53.50 ± 4.538˚ after 12^{th} week of training.

The trend in elbow extension in NMS group indicates an increase of 16.83 ± 8.451˚ at baseline to 25.00 ± 8.820˚ after the 6^{th} week but a decrease after the 12weeks (24.83 ±

7.004˚). However, the CE group showed there linear improvement from 13.42 ± 6.244˚ at baseline, 18.58 ± 6.007˚ after 6th week and 29.75 ± 11.910˚ after the 12th week.

The wrist extension showed improvement from the baseline mean score of 4.08 ± 3.232˚ to 11.33 ± 7.266˚ after the 6th week and 19.17 ± 9.408˚ after 12th week for participants in the NMS group. The participants in CE group show improvement of 3.25 ± 2.989˚ at baseline, 9.83 ± 5.540˚ after 6th week and 22.33 ± 8.742˚ after the 12th weeks respectively. Participants in the CE group recorded more increase on the wrist extension than those in the NMS group.

In knee flexion, the NMS group showed increase in joint range of motion from 46.67 ± 8.391˚ at baseline to 56.67 ± 4.039˚ after the 6th week and 78.25 ± 4.038˚ after the 12th week while those in the CE group recorded higher increase of the joint range of motion, of 38.42 ± 6.014˚ at the baseline, 58.25 ± 6.503˚ after the 6th week and 102.83 ± 18.761˚ after the 12th week.

The hip extension in the NMS group showed increase in the joint range of motion from 8.33 ± 3.725˚ at baseline to 10.00 ± 3.766˚ after the 6th week and 16.50˚ ± 6.544 after the 12th week. Those in the CE training had 7.92 ± 3.088˚ at baseline, 11.17 ± 2.887˚ after 6th week and 17.33 ± 5.087˚ after the 12th week respectively. This showed that there was more improvement in the CE group compared to NMS group of training.

The ankle dorsiflexion revealed improvement with NMS and CE programmes on the ankle of stroke survivors. Participants in the NMS group measured 5.08 ± 2.503˚ at baseline, 5.17 ± 2.125˚ after 6th week and 7.25 ± 2.598˚ after the 12th week. In the CE group, had 3.58 ± 1.782˚ at baseline, 4.00 ± 1.706˚ after 6th weeks and 6.67 ± 1.875˚ after 12th week respectively. The data was statistically analyzed using repeated-measures ANOVA and presented in table 4.2.4b.

Sub- hypothesis IV: There is no significant effects of NMS and CE on joint range of motion of upper and lower limbs of stroke patients who undergo training for a period of 12 weeks.

Table 4.2.4b: Repeated-measures analysis of variance on the effects of NMS and CE on shoulder extension of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	4143.569	5	828.714	3.846	.004
Intercept	110528.347	1	110528.347	512.962	.000
Type	2701.125	1	2701.125	12.536	.001*
Period	1243.444	2	621.722	2.885	.063
Type * Period	199.000	2	99.500	.462	.632
Error	14221.083	66	215.471		
Total	128893.000	72			
Corrected Total	18364.653	71			

F (1, 72) = 7.00 P< 0.01 * = significant

The result showed that the training programmes caused statistical significant effects on shoulder extension for the participants by type of training programme (P = 0.001).

The Scheffe *post-hoc* test for the type and three different periods of assessment is presented in tables 4.2:4c and 4.2.4d, respectively.

Table 4.2.4c: Pair-wise comparison of shoulder extension measured at the different period of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	-.750	.442	.353
	12 weeks	-9.167(*)	2.169	.004*
6 weeks	0 week	.750	.442	.353
	12 weeks	-8.417(*)	1.808	.002*
12 weeks	0 week	9.167(*)	2.169	.004*
	6 weeks	8.417(*)	1.808	.002*

* The mean difference is significant at the .05 alpha level.

The result of the *post-hoc* test revealed that shoulder extension of the participants in the NMS and CE shows little increase after the 6th week of training.

Table 4.2.4d: Repeated-measures analysis of variance on the effects of NMS and CE on elbow extension of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	2243.903	5	448.781	3.894	.004
Intercept	32981.681	1	32981.681	286.141	.000
Type	48.347	1	48.347	.419	.519
Period	1781.778	2	890.889	7.729	.001*
Type * Period	413.778	2	206.889	1.795	.174
Error	7607.417	66	115.264		
Total	42833.000	72			
Corrected Total	9851.319	71			

$F(2, 72) = 4.91$ $P < 0.01$ * = significant .

Table 4.2.4d shows the results of repeated measures ANOVA on the effects of NMS and CE on elbow extension of the participants. Observation of the results revealed that the training (type) did not differ significantly ($P = 0.519$). There effects on elbow extension of the

participants. However, the effects of NMS and CE on the elbow extension with both training increases the joint range of motion (P = 0.001). Based on these observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE on joint ROM (elbow extension) of stroke survivors, involved in the study.

The Scheffe *post-hoc* test procedure on the elbow extension measured at the different periods of the training is presented in table 4.2.4e.

Table 4.2.4e Pair-wise comparison of mean measurement of elbow extension of the participants at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	-6.667	3.236	.192
	12 weeks	-12.167(*)	1.999	.000*
6 weeks	0 week	6.667	3.236	.192
	12 weeks	-5.500	3.092	.309
12 weeks	0 week	12.167(*)	1.999	.000*
	6 weeks	5.500	3.092	.309

* The mean difference is significant at the .01 alpha level.

The result of the *post-hoc* analysis revealed no effect of NMS participants change in elbow extension of the participants until after the 12th week of training. However, elbow extension was noticed to have improved after 6th week of training. In the CE group, the increase in the joint range of motion was linear, immediately after the 6th and 12th weeks of training.

Table 4.2.4f Repeated-measures analysis of variance on the effects of NMS and CE on wrist extension of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	3622.167	5	724.433	16.231	.000*
Intercept	9800.000	1	9800.000	219.564	.000*
Type	1.389	1	1.389	.031	.861
Period	3544.333	2	1772.167	39.705	.000*
Type * Period	76.444	2	38.222	.856	.429
Error	2945.833	66	44.634		
Total	16368.000	72			
Corrected Total	6568.000	71			

F (2, 72) = 4.91 P < 0.01 * = significant.

Table 4.2.4f shows the results of repeated measures ANOVA on the effects of NMS and CE on wrist extension of the participants. Observation of the results revealed that the effects of both training (type) did not differ significantly (p = 0.861). However, the effects was only noticed to during the periods of training for both the NMS and CE groups (P = 0.000). Therefore, there was sufficient evidence to reject the null hypothesis which states that there is no significant effect of NMS and CE on joint range of motion (wrist extension) among the stroke participants.

The Scheffe *post-hoc* test procedure on the wrist extension measured at the different periods of the training is presented in Table 4.2.4h.

Table 4.2.4g Pair-wise comparison of mean of wrist extension measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value

0 week	6 weeks	-6.917(*)	1.206	.000*
	12 weeks	-17.083(*)	1.651	.000*
6 weeks	0 week	6.917(*)	1.206	.000*
	12 weeks	-10.167(*)	1.363	.000*
12 weeks	0 week	17.083(*)	1.651	.000*
	6 weeks	10.167(*)	1.363	.000*

* The mean difference is significant at the .01 alpha levels.

The result of the *post-hoc* test revealed that joint range of motion at wrist extension of the participants in the NMS and CE groups of training improved even at the 6th week of training.

Table 4.2.4h: Repeated-measure analysis of variance on the effects of NMS and CE on hip extension of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	1000.958(a)	5	200.192	10.479	.000
Intercept	10153.125	1	10153.125	531.444	.000
Type	5.014	1	5.014	.262	.610
Period	987.583	2	493.792	25.846	.000*
Type * Period	8.361	2	4.181	.219	.804
Error	1260.917	66	19.105		
Total	12415.000	72			
Corrected Total	2261.875	71			

F (2, 72) = 4.91 P < 0.01 * = significant

Table 4.2:4h shows the results of repeated measures ANOVA on the effects of NMS and CE on hip extension of the participants. Observation of the results revealed that the training (type) did not differ significantly (P = 0.610). However, the effects on the hip extension differed by the periods of training for the two groups (P = 0.000). Based on these

observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE exercises on joint ROM (hip extension) of stroke survivors, involved in the study.

The Scheffe *post-hoc* test procedure on the hip extension measured at the different periods of the training is presented in Table 4.2.4i.

Table 4.2.4i: Pair-wise comparison of hip extension measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	-2.458(*)	.366	.000*
	12 weeks	-8.792(*)	1.077	.000*
6 weeks	0 week	2.458(*)	.366	.000*
	12 weeks	-6.333(*)	.893	.000*
12 weeks	0 week	8.792(*)	1.077	.000*
	6 weeks	6.333(*)	.893	.000*

* The mean difference is significant at the P < .001* alpha level.

The result of the *post-hoc* test revealed that there was progressive improvement in the hip extension of the participants in both NMS and CE groups. The improvement was however observed to be more in the CE group.

Table 4.2.4j: Repeated-measures analysis of variance on the effects of neuromuscular stimulation and cycle ergometry on Knee flexion of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	33017.569(a)	5	6603.514	11.703	.000

Intercept	290449.014	1	290449.014	514.754	.000
Type	642.014	1	642.014	1.138	.290
Period	28968.111	2	14484.056	25.670	.000*
Type * Period	3407.444	2	1703.722	3.019	.056
Error	37240.417	66	564.249		
Total	360707.000	72			
Corrected Total	70257.986	71			

F (2, 72) = 4.91 P < 0.01 * =Significant

Table 4.2:4i shows the results of repeated measures ANOVA on the table shows that there was no significant difference between the two groups (P = 0.290). However, the effects of joint range of motion (ROM) for the knee flexion increase by the periods of training for the two groups (P = 0.000). Based on the result, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE on joint ROM (knee flexion) of stroke survivors, involved in the study.

The Scheffe *post-hoc* test procedure on the knee flexion measured at the different periods of the training is presented in Table 4.2.4k.

Table 4.2.4k: Pair-wise comparison of knee flexion measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	-14.917(*)	2.618	.000*
	12 weeks	-48.000(*)	3.477	.000*
6 weeks	0 week	14.917(*)	2.618	.000*
	12 weeks	-33.083(*)	2.853	.000*
12 weeks	0 week	48.000(*)	3.477	.000*
	6 weeks	33.083(*)	2.853	.000*

* The mean difference is significant at the .001* alpha level.

The result of the *post-hoc* test revealed that knee flexion of the participants in the NMS group improved throughout the periods of training. In the CE, significant improvement in knee flexion was found to be more significant.

Table 4.2.4l: Repeated-measure analysis of variance on the effects of NMS and CE on ankle dorsiflexion of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	124.458	5	24.892	5.505	.000
Intercept	2016.125	1	2016.125	445.901	.000
Type	21.125	1	21.125	4.672	.034
Period	100.750	2	50.375	11.141	.000*
Type * Period	2.583	2	1.292	.286	.752
Error	298.417	66	4.521		
Total	2439.000	72			
Corrected Total	422.875	71			

F (2, 72) = 4.91 P < 0.01 * =significant

Table 4.2.4l shows the results of repeated-measures ANOVA on the effects of NMS and CE on ankle dorsiflexion of the participants. The results revealed that the effect of the type of training did not differ significantly (P = 0.034), but the effects on the range of motion of ankle dorsiflexion increases through the periods of training at the 12^{th} week (P = 0.000). Based on these observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE exercises on joint ROM (ankle dorsiflexion)of stroke survivors, involved in the study.

Further analysis using the Scheffe *post-hoc* test procedure on the ankle dorsiflexion measured at the different periods of the training is presented in Table 4.2.4m.

A further test on the observed means using the Scheffe *post-hoc* test procedure on the mean ankle dorsiflexion measured at the different period of the training is summarized in Table 4.2.4m.

Table 4.2.4m: Pair-wise comparison of ankle dorsiflexion measured at the different period of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0week	6weeks	-.250	.169	.498ns
	12weeks	-2.625(*)	.289	.000*
6weeks	0week	.250	.169	.498ns
	12weeks	-2.375(*)	.205	.000*
12weeks	0week	2.625(*)	.289	.000*
	6weeks	2.375(*)	.205	.000*

* The mean difference is significant at the P< .001 level.

The result of the *post-hoc* test revealed that between the onset and after 6^{th} week of the training, the measured ankle dorsiflexion means were not significantly different. But the measured ankle dorsiflexion, after the 12^{th} week of the training was significantly different from that at 0 week and 6week respectively.

Research question IV: Would NMS and CE exercises reduce blood pressures of stroke survivors in Kano State, Nigeria?

The effects on systolic and diastolic blood pressures by the participants during the duration of the training are summarized in Table 4.2.5a.

Table 4.2.5a: Means and Standard Deviation of the effect of NMS and CE on blood pressure of stroke survivors in Kano State, Nigeria

Blood pressure	Period	Neuromuscular stimulation		Cycle ergometry	
		Mean(mmHg)	S. D.	Mean	S. D.
Systolic	0 week	145.17	11.614	136.67	13.707
	6 weeks	141.67	18.007	139.17	14.434
	12weeks	127.92	14.994	129.17	10.836
Diastolic	0 week	85.33	9.509	85.00	11.677
	6 weeks	88.08	6.345	86.67	10.386
	12weeks	79.75	7.605	72.25	9.555

There was a marked decrease in the systolic blood pressure of participants exposed to NMS from 145.17 ± 11.614 mm Hg at the baseline to 141.67 ± 18.007 mm Hg at 6^{th} week and 127.92 ±14.994 mm Hg after the 12^{th} week. For those in the CE, there was an increase in systolic blood pressure of 139.17 ± 14.434 mm Hg at the 6^{th} week of the training but later decrease after the 12^{th} week (129.17 ± 10.836 mm Hg). The NMS and CE were effective on the systolic blood pressure of the participants after the 12^{th} week training.

A gradual increase was observed in the diastolic blood pressure of the participants in the NMS training after 6^{th} week from 85.33 ± 9.51 mm Hg at baseline to 88.08 ± 6.35 mm Hg, but decreased after the 12^{th} week (79.75 ± 7.605 mm Hg). For CE training group, the

diastolic blood pressure level increases from 85.00 ± 11.68 mm Hg at the baseline, 86.67 ±10.34 mm Hg after the 6^{th} week and then decreased to 72.25mm Hg ± 9.56 after the 12^{th} week of the training. These results showed that was effects of the training modes on the blood pressure of the participants.

Sub Hypotheses IV: There is no significant effect of NMS and CE on blood pressure of stroke patients who under training for a period of 12weeks.

Table 4.2.5b: Repeated-measure analysis of variance on the effects of NMS and CE on systolic blood pressure of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	2835.625	5	567.125	2.841	.022
Intercept	1343980.125	1	1343980.125	6733.546	.000
Type	190.125	1	190.125	.953	.333
Period	2355.250	2	1177.625	5.900	.004*
Type * Period	290.250	2	145.125	.727	.487
Error	13173.250	66	199.595		
Total	1359989.000	72			
Corrected Total	16008.875	71			

F (2, 72) = 4.91 P < 0.01 * = significant

Table 4.2:5b shows the results of repeated measures ANOVA of NMS and CE on systolic blood pressure of the participants. The results revealed that the training modes did not differ significantly (p = 0.333). However, the period of training showed significant effects (P = 0.004) on systolic blood pressure of the two training groups. Based on these observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE on systolic blood pressureof stroke survivors, involved in the study.

Table 4.2:5c: Repeated-measure analysis of variance on the effects of NMS and CE on diastolic blood pressure of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	2096.569(a)	5	419.314	4.801	.001
Intercept	494183.681	1	494183.681	5657.856	.000
Type	171.125	1	171.125	1.959	.166
Period	1746.361	2	873.181	9.997	.000*
Type * Period	179.083	2	89.542	1.025	.364
Error	5764.750	66	87.345		
Total	502045.000	72			
Corrected Total	7861.319	71			

$F_{(2, 72)} = 4.91$ $P < 0.01$ * = significant

Table 4.2.5c shows the results of repeated measures ANOVA on the effects of NMS and CE on diastolic blood pressureof the participants. Observation of the results revealed that the training (type) did not differ significantly (P = 0.166). The effects on the diastolic blood pressurediffered by the periods of training for the two groups (P = 0.000). Based on these observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE exercises on diastolic blood pressure of stroke survivors, involved in the study.

The Scheffe *post-hoc* test procedure on the systolic blood pressure measured at the different periods of the training is presented in Table 4.2.5d.

Table 4.2.5d: Pair-wise comparison of systolic blood pressure measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	.500	4.150	1.000
	12 weeks	12.375(*)	3.778	.022*
6 weeks	0 week	-.500	4.150	1.000
	12 weeks	11.875(*)	2.595	.002*
12 weeks	0 week	-12.375(*)	3.778	.022*
	6 weeks	-11.875(*)	2.595	.002*

***** The mean difference is significant at the .05 alpha levels

The results of the post-hoc test revealed that systolic blood pressure of the participants in the NMS group improve after the 6^{th} and 12^{th} weeks of training. However, in the CE group, significant improvement in systolic blood pressure was observed after the 12^{th} weeks of training only. This shows that all exercises should start with low intensity and the blood pressure should be carefully monitored during the period of exercise because blood pressure increases at the initial level of training. The work of the heart also increases more with sitting position of exercise at the initial level of training compared to lying during the neuromuscular training.

Table 4.2.5e: Pair-wise comparison of diastolic blood pressure measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	-2.208	1.771	.715
	12 weeks	9.167(*)	1.849	.001*
6 weeks	0 week	2.208	1.771	.715
	12 weeks	11.375(*)	1.764	.000*
12 weeks	0 week	-9.167(*)	1.849	.001*
	6 weeks	-11.375(*)	1.764	.000*

***** The mean difference is significant at the .01 apha level.

The Scheffe *post-hoc* test procedure on the diastolic blood pressure measured at the different periods of the training is presented in Table 4.2.5e. The result of the *post-hoc* test revealed that diastolic blood pressure of the participants in the NMS group did not improve after the 6^{th} week of training. However, diastolic blood pressure of the participants improved after 12^{th} week of training. In the CE group, improvement in diastolic blood pressure was at baseline and after 12^{th} weeks of training. It was observed that the NMS and CE have increased blood pressure at the acute phase of the training.

Research question V: Would NMS and CE reduce the pulse rate of stroke survivors in Kano State, Nigeria?

The effects on pulse rate by the participants during the duration of the training are summarized in Table 4.2.6a.

Table 4.2.6a: Means and Standard Deviation of the effects of NMS and CE on pulse rate of stroke survivors in Kano State, Nigeria

	Neuromuscular stimulation		Cycle ergometry	
Period	Mean(beat/min)	S. D.	Mean	S. D.
0 week	79.92	1.445	88.50	3.042
6 weeks	74.33	8.907	78.67	3.614
12weeks	68.33	8.305	66.42	6.259

All the groups in the two training showed gradual reduction in their pulse rate. NMS had a mean pulse rate of 79.92 ± 1.445bpm at baseline, which decreased to 74.33 ± 8.907bpm after the 6^{th} week and 68.33 ± 8.305bpm after the 12^{th} week respectively. For the CE group, the mean pulse rate was 88.50 ± 3.042bpm at the baseline, 78.67 ± 3.614bpm after the 6^{th} week and 66.42b/min ± 6.259 after the 12^{th} week respectively. Generally,there was gradual reduction in the pulse rate of the participants in the two groups implying positive effect on their pulse rates. However, the CE group improved more than those in the NMS group.

Sub Hypotheses V: There are no significant effects of NMS and CE on pulse rate of stroke patients who undergo 12-week training.

The measurement for the pulse rate was carried three times (baseline, immediately after 6^{th} and after 12^{th} week). The independent variables were computed using repeated-measures analysis of variance. The results of the analysis are presented in Table 4.2.6b.

Table 4.2.6b: Repeated-measure analysis of variance on pulse rate of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	3985.111(a)	5	797.022	7.096	.000
Intercept	416176.056	1	416176.056	3705.414	.000
Type	242.000	1	242.000	2.155	.147
Period	3408.361	2	1704.181	15.173	.000*
Type * Period	334.750	2	167.375	1.490	.233
Error	7412.833	66	112.316		
Total	427574.000	72			
Corrected Total	11397.944	71			

$F_{(2, 72)} = 4.91$ $P < 0.01$ * = significant

Table 4.2.6b shows the results of repeated measures ANOVA on the effects of NMS and CE on pulse rate of the participants. Observation of the results revealed that the type of training mode did not differ significantly (P = 0.147). However, the effects on the pulse rate differed by the periods of training for the two groups (P = 0.000). Based on these observations, there was sufficient evidence to reject the null hypothesis that there is no significant effect of NMS and CE exercises on pulse rate of stroke survivors, involved in the study.

The Scheffe *post-hoc* test analysis on the pulse rate measured at the different periods of the training is presented in table 4.2.6c.

Table 4.2.6c: Pair-wise comparison of pulse rate measured at the different period of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	7.708(*)	1.915	.006*
	12 weeks	16.833(*)	2.276	.000*
6 weeks	0 week	-7.708(*)	1.915	.006*
	12 weeks	9.125(*)	1.606	.000*
12 weeks	0 week	-16.833(*)	2.276	.000*
	6 weeks	-9.125(*)	1.606	.000*

***** The mean difference is significant at the .01 alpha levels**.**

The result of the post-hoc test revealed that pulse rate of the participants in the NMS and CE groups reduced after the 6^{th} and 12^{th} week of training.

Research question VI: Would NMS and CE increase Oxygen Saturation (SPO_2) of stroke survivors in Kano State, Nigeria?

The effects of SPO_2 of the participants during the duration of the training are summarized in table 4.2.7a.

Table 4.2.7a: Means and Standard Deviation of the effect of NMS and CE on Oxygen Saturation (SPO_2) of stroke survivors in Kano State, Nigeria

	Neuromuscular stimulation		Cycle ergometry	
Period	Mean(%)	S. D.	Mean(%)	S. D.
0 week	96.67	1.155	96.25	1.603
6 weeks	96.67	1.155	96.25	1.603
12weeks	98.50	0.522	98.17	0.937

There was a modest increase in SPO_2 at the 12^{th} week of training for both programmes.

Sub Hypotheses VI: There is no significant effect in 12 week of NMS and CE on SPO_2 of stroke patients over 12week duration of training

The measurement for oxygen saturation of the participants over the three periods (baseline, $6^{th}$and 12^{th} week) of the two training protocol was computed using repeated-measures analysis of variance. The result of the analysis is presented in table 4.2.7b.

Table 4.2.7b: Repeated-measure analysis of variance on the effects of NMS and CE on SPO_2 of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	59.000	5	11.800	7.907	.000
Intercept	678612.500	1	678612.500	454704.822	.000
Type	2.722	1	2.722	1.824	.181
Period	56.250	2	28.125	18.845	.000*
Type * Period	.028	2	.014	.009	.991
Error	98.500	66	1.492		
Total	678770.000	72			
Corrected Total	157.500	71			

$F_{(2, 72)} = 4.91$ $P < 0.01$ * = significant

The result revealed no significant effects of both training mode on SPO_2 ($p = 0.181$). However, by duration of training, the results revealed significant effects of both training modes on SPO_2 ($P < 0.000$).

Further analysis on the mean measurement using the Scheffe *post-hoc* analysis was done at the different periods of the training summarized in table 4.2.7c.

Table 4.2.7c: Pair-wise comparison of oxygen saturation of the participants measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	.000	.000	1.000ns
	12 weeks	-1.875(*)	.296	.000*
6 weeks	0 week	.000	.000	1.000ns
	12 weeks	-1.875(*)	.296	.000*
12 weeks	0 week	1.875(*)	.296	.000*
	6 weeks	1.875(*)	.296	.000*

* The mean difference is significant at P < 0.01**

The result of the *post-hoc* test revealed that the significant difference in SPO_2 was due to higher value after the 12^{th} week of training.

Research question VII: Would NMS and CE improve coordination of stroke survivors in Kano State, Nigeria; the effects on coordination by the subject during the duration of the training are summarized in table 4.2.8a.

Table 4.2.8a: Means and Standard Deviation of the effect of NMS and CE training improve coordination of stroke survivors in Kano State, Nigeria

	Neuromuscular stimulation		Cycle ergometry	
Period	Mean(N)	S. D.	Mean	S. D.
0 week	5.67	3.939	5.92	2.937
6 weeks	6.00	4.553	5.50	3.606
12weeks	10.92	4.944	11.58	5.696

The table shows a linear increase in the performance of the block-box activity in the NMS and CE training modes. There is a clear indication that the two training mode had significant effect on the block box activity performance of the participants.

Sub Hypothesis VII: **There is no significant effect in NMS and CE on coordination of stroke patients who under gone 12weeks of training**

The measurement for the block box over the three periods (baseline, after 6th week and after 12th week) of the training from the NMS and CE exercises were computed using repeated measures analysis of variance and presented in table 4.2.8b.

Table 4.2.8b: Repeated-measure analysis of variance on the effects of NMS and CE on Coordination of the participants

Source	Sum of Squares	DF	Mean Square	F	Sig.
Corrected Model	484.903(a)	5	96.981	5.070	.001*
Intercept	4155.681	1	4155.681	217.262	.000*
Type	.347	1	.347	.018	.893
Period	480.361	2	240.181	12.557	.000*
Type * Period	4.194	2	2.097	.110	.896
Error	1262.417	66	19.128		
Total	5903.000	72			
Corrected Total	1747.319	71			

$F_{(2, 72)} = 4.91$ $P < 0.001$ * = significant

Table 4.2.8b shows the results of repeated-measures ANOVA on the effects of NMS and CE on coordination of the participants. The results showed that both type of training did not differ significantly (P = 0.893). However, both training are effective on block-box performance activity at the 12th week of the training (P = 0.000). Based on these observations, there was sufficient evidence to reject the null hypothesis which states that there is no significant effect of NMS and CE on coordination of stroke survivors in Kano State, Nigeria.

The Scheffe *post-hoc* analysis on the coordination measured at the different periods of the training is presented in Table 4.2.8c.

Table 4.2.8c: Pair-wise comparison of coordination among the participants measured at the different periods of the training

(I) Period	(J) Period	Mean Difference (I-J)	Std. Error	P-value
0 week	6 weeks	.042	.494	.934
	12 weeks	-5.458(*)	.490	.000*
6 weeks	0 week	-.042	.494	.934
	12 weeks	-5.500(*)	.645	.000*
12 weeks	0 week	5.458(*)	.490	.000*
	6 weeks	5.500(*)	.645	.000*

***** The mean difference is significant at the P < 0.01 alpha levels

The result of the *post-hoc* test revealed that performance of block- box activity of the participants in the NMS and CE group improved at both 6^{th} and 12^{th} weeks of training.

4.3. Discussion

This study investigated effects of neuromuscular (NMS) and cycle ergometry (CE) on functional abilities of stroke survivors in Kano State, Nigeria which lasted for 12

weeks.Among the parameters assessed were strength (grip and pinch strength), joints flexibility (range of motion: ROM); that include shoulder, elbow and wrist joints extension n while the lower limb include hip extension, knee flexion and ankle dorsiflexion.

The results of this study revealed that grip and pinch strength of stroke survivors improved significantly using NMS and CE training protocols, which were observed after the 6^{th} and 12^{th} week. This improvement might have been as a result of increased recruitment of motor unit within the affected skeletal muscles (spacial summation). This results support the findings of the study of Knerim (1997) who tested the effects of electrical stimulation on immediate and long term effect on muscles' adaptation of stroke survivors. The findings of this study further agreed with the investigation of Steven-Lapsley, Balter, Wolfe, Eckoff and Korth (2012) who used NMS and voluntary activity on stroke survivors. Though booth training protocols improved the strength of the participants, cycle ergometry training had higher effects than NMS on grip and pinch strength well as s and oxygen saturation. However, the results of Mohr, Carlson, Sulenic & Landry (1985) did not support this study. Their investigation used NMS, which showed no improvement in strength among stroke participants. The improvement experience on grip and pinch strength became a source of motivation and incentive for stroke survivors used in this study, which confirmed the position of (Lipert *et al.*, 2000).

Stroke survivors used in this study confirmed the efficacy of both training modes on the upper extremity functions, such as holding, grasping, moving and releasing of objects. This finding is in line with the investigation of Lewitt and McCarty (2008; Thrasher *et al. (*2008) and Bae *et al. (*2015) whose results showed the relationship between grip and pinch strength

and activities of daily living in stroke patients. It has also been proved that addition of NMS to voluntary exercise was superior to isometric voluntary contraction alone (Nobbs & Rhodes, 1986; Delitto, Rose & Mckowen, 1988).

Grip and pinch strength greatly improved using both neuromuscular stimulation and cycle ergometry in this study. Nwuga (1993) defined maximum hand grip strength as the functional maximum force that a subject can exert when grip is exerted on object. Hand grip strength is a specific, reproducible, simple and non-invasive test of sympathetic function (Khurana, 1996).

Grip strength alone can be predictive of functional recovery of stroke survivors (Sunderland; Tunson & Bradley, 1992). Waleed, Hana, Ai & Alaan (1999) stated that grip strength is considered the most reliable clinical measure of human strength. Badejo & Olawale (2000) supported this view that grip strength measurement is of great value in physical therapy as it is used to determine the seriousness of upper extremity injuries and success of rehabilitation programme.

Shoulder extension, elbow extension, wrist extension, hip extension, knee flexion and ankle dorsiflexion were the range of motion (ROM) selected and assessed in this study. The results of this study on parameters revealed that shoulder extension, elbow extension, wrist extension, hip extension, knee flexion and Ankle dorsiflexion improved using NMS and CE training at the 6^{th} and 12^{th} week of training. This is as a result of stretching of the muscles and maintaining full length of the soft tissues. This findings support the study of Jones et al (1998) who used cyclical electrical stimulation in the prevention of spasticity among stroke survivors. Fornusek et al (2012) findings also supported the results of this study because as

they also used electrical stimulation to increase ankle dorsiflexion of spinal cord injured patients. The range of motion at the knee joint was found to increase significantly with cycle ergometry for 10 weeks in the study (Khuram, 2011). Also the study of Tang, Sibley, Thomas & Bayley (2008) used 6-minute walk test which improved the range of motion in the lower limb also confirm the finding of this study as a modified cycling. Each of the selected variables were tested using goniometer. For the shoulder extension, significant difference was observed in the measurements taken over the three periods and the significant effects observed was as a results of the greater effects of the CE training than the impact observed with NMS training. The findings of this study agreed with the result of Khuram (2011), who maintained that the main purpose of rehabilitation after stroke is to regain motor control in gait and related activities to improve upper limb functions and cope with activities of daily living.

Systolic and diastolic blood pressure significantly reduced with NMS and CE training protocols which were observed after the 6^{th} and 12^{th} week of training. The reduction in the BP might be as a result of increased cardiac and autonomic functions among the participants. The result of this study on blood pressure revealed that systolic and the diastolic blood pressure levels decreased, especially at the 12^{th} week of training. The results is in line with the study of Wolf, Clagett & Easton (1999) and Roth (1993) who reported that individuals with stroke, frequently have their vascular system at high risk, or have linked cormobid cardiovascular disease because of sedentary lifestyle post stroke.This could have significant effects on cardiac function during activity and exercise. Stroke-related changes in the brain that regulate the autonomic function can have significant implications for blood pressure control and cardiac function during the acute phase of stroke recovery (Gresham, Duncan &

Stason, 1995; Roth & Harvey, 2000). The decrease in the blood pressure of this study indicated was good cardiovascular adaptation to training by the participants, which had far reaching health implication in their lives. The results however did not support the study of Sampson *et al. (2000)* who observed that neuromuscular stimulation initiates painful stimulus, which causes uncomfortable stretches and discomfort and thus, increased the blood pressure at the initial period of treatment of the patients used.

Pulse rate significantly reduced with NMS and CE, observed after the 6^{th} and 12^{th} week of training. This might be as a result of improved autonomic function and cardiovascular adaptation to the training. The results of this study confirmed the position of Ravikiran et al. (2012) who used cycle ergometer and treadmill to assess the cardiovascular responses of sedentary stroke survivors. The study shows that cycle ergometry reduced the pulse rate better than treadmill group. Fletcher, 2001; Hermanson, 1970 & Bruce, 1974, also supported this study when cycle ergometry, treadmill and walking were the modes of exercise. Their study revealed that pulse rate and other cardiovascular indices improved better with cycle ergometry among stroke survivors**.**

SPO_2 improved using NMS and CE observed after the 6^{th} and 12^{th} week of the training. This might be due to improved respiratory muscles and oxygen concentration in the blood through central regulation of respiration. This finding supports the study of Pearlson, Blair & Daniels (2002) who used aerobic exercise such as walking. Other studies like those Potempa et al. (2000) and Goodman (1966) supported this study because oxygenation was improved using arm cranking and treadmill exercises. Hypoxia is a common post stroke complication and it makes stroke survivors more prone to respiratory problems (Nachmann *et al.*, 1995). Most stroke survivors do not have pulmonary disease post stroke, however, respiration may be

compromised as a direct result of stroke (particularly brain stem stroke), which affects the respiratory muscles of the patients. Patients with stroke are prone to respiratory problems for reasons which include: alterations in the central regulation of respiration (Nachmann *et al.*, 1995), sleep apnoea (Bassetti & Aldrich, 1999; Harbison & Gibson, 2000) and weakness of the respiratory muscles on the hemiplegic side (Fluck, 1966; Haas & Rusk, 1967; De Troyer, De Beyl & Thirion, 1981), This leads to impaired breathing mechanics and other respiratory co-morbidities with initiating physical activity (Olney; Griffin & Monga, 1991).

The results of this study revealed that coordination improved with NMS and CE; which were evidence after the 6^{th} and 12^{th} week of training as assessed using the block-box performance activity. This improvement might be as a result of neuroplasticity, better cognitive function and organized muscle activation of the affected group of muscles. This finding is in conformity wth other studies that used aerobic training like cycling which improved the stability, balance and coordination of the participants (Krebs *et al.,* 2006; Lonn, Yusuf & Arnold, 2006). Research findings revealed that block-box activity is effective as a task-oriented training activity in the rehabilitation of subjects with mild and moderate hemiparesis (Monye, 2011; Alon, Levitt & McCarthy, 2007). Functionally-oriented task practice of upper and lower limbs have shown substantial evidence of efficacy for individuals with long-term stroke disabilities of greater than one year; particularly when encouraged to use of the affected limbs in activity of daily life (Brewer, Hogan, Hickey & Williams, 2012).

The use of robotics as tools for rehabilitative services aimed at improving motor abilities among stroke survivors is becoming common these days (Krebs *et al.,* 2006; Lonn, Yusuf & Arnold, 2006). Stroke survivors may benefit from participation in most aerobic physical activities like cycling (Pearson, Blair & Daniels, 2002); arm cranking and light resistance

training (Smith, Blair & Bonow, 2001). This finding is consistent with the results obtained by Hamzat (2014), who reported that despite improvement in endurance of people living with stroke, aerobic training helps to prevent many challenges faced by stroke survivors, especially in Nigeria.

This finding agreed with that of Bellinger (2014), who reported that physical activity and exercise training have the capacity to influence physical, psychological and social domains of stroke survivors.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.1 Summary

It is a known fact that training, irrespective of the mode produce significant responses and adaptation to the body systems. This study investigated the effects of neuromuscular stimulation (NMS) and cycle ergometry (CE) on selected body parameters of stroke survivors at the Physiotherapy Unit of Murtala Mohammed Specialist Hospital, Kano. The training was conducted for a duration of 12 weeks with measurement taken at the baseline, 6^{th} and 12^{th} week of training. Among the parameters assessed were strength (grip strength and pinch sstrength), joint flexibility (range of motion (ROM). Parameters investigated in this regard were movements of the affected upper limb which include shoulder, elbow, and wrist joints extension while the lower limb include hip extension, knee flexion and ankle

dorsiflexion. Other assessed parametersof the participants investigated were cardio-respiratory indices of systolic and diastolic blood pressures, pulse rate (PR) oxygen ssaturation (SPO_2) and coordination (block-box test).

The study used the factorial experimental approach to investigate the effects of the two training types on the selected parameters of stroke patients, who attend the Physiotherapy Unit of Murtala Mohammed Specialist Hospital, Kano through a random procedure. The 30 selected participants were divided into two groups and were then exposed to two different training protocols (NMS and CE). Data were collected at the baseline, 6^{th} week and 12^{th} week respectively. The collected data was analyzed using the Statistical Package of Social Sciences (SPSS). Among the procedures selected for the tests were descriptive statistics of mean and standard deviation and repeated-measures analysis of variance.

5.1.1 Summary of findings

This result of this study revealed that:

I. NMS and CE significantly improved grip and pinch strength among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$).

II. NMS and CE had significant effect on range of motion of shoulder extension, elbow extension, wrist extension, hip extension, knee flexion and ankle dorsiflexion among stroke survivors in Kano State, Nigeria ($P < 0.001$).

III. NMS and CEcaused significant reduction in both thesystolic and diastolic blood pressures among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$).

IV. NMS and CE had significant reduction on the pulse rate of stroke survivorsin Kano State, Nigeria ($P < 0.001$).The improvement was better in the CE group than in the NMS group ($p = 0.000$).

V. NMS and CE had significantly improved the SPO_2 of stroke survivors in Kano State, Nigeria ($P < 0.001$).

VI. NMS and CE had significantly improved the coordination of stroke survivors in Kano State, Nigeria ($P < 0.001$).

5.2 Conclusion

Based on the findings of this study, the following conclusions were made:

I. NMS and CE significantly improved grip and pinch strength among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$).

II. NMS and CE had significant effect on range of motion of shoulder extension, elbow extension, wrist extension, hip extension, knee flexion and ankle dorsiflexion among stroke survivors in Kano State, Nigeria ($P < 0.001$).

III. NMS and CE caused significant reduction in both the systolic and diastolic blood pressures among stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$).

IV. NMS and CE had significant reduction on the pulse rate of stroke survivors in Kano State, Nigeria ($P < 0.001$). The improvement was better in the CE group than in the NMS group ($p = 0.000$).

V. NMS and CE had significantly improved the SPO_2 of stroke survivors in Kano State, Nigeria ($P < 0.001$).

5.3 Recommendations

Based on the findingsof this study, the following recommendations were made.

I. Cycle ergometry could be adopted as a mode of training for improving the functional abilities of stroke survivors in Kano State, Nigeria.

II. For better improvement in flexibility, both the CE and NMS are recommended as a reliabletraining protocols in the rehabilitation of stroke patients in Kano State, Nigeria

III. Training duration for either of the NMS and CE should be continuous for at least a period of 12 weeks for optimum benefits of stroke patients in Kano State, Nigeria.

IV. A combination of both NMS and CE training modes are recommended in the rehabilitation of stroke patients in Kano State, Nigeria.

5.4 Recommendation for further study

The research concentrated on stroke participants in physiotherapy unit of Murtala Mohammed Specialist Hospital, Kano. The study could be extendedusing larger sample size, similar age range, duration of stroke, sex matched and initial level of fitness for controlling the cofounding variables and statistical variability in human study of this type.

REFERENCES

Abbott, R.D; Donahue, R.P & MacMahon, S.W. (1987) Diabetes and the risk of *stroke*. The Honolulu Heart Program. *Journal of American Medical Association,* 257: 949-952.

Aboyans, V., Criqui, M., Demenberh, J.O., Knoke, J.D., Ridker, P.M. & Fronek, A. (2006). Risk factors for progression of peripheral artery disease in large and small vessels. *Journal of Circulation*, 113(22): 2623-2629.

Abrams, W.B., Beers, M.H. & Berkow, R. (1995). *The Merck Manual of Geriatrics*. Whitehouse State, NJ: Merck Research Laboratories, 1148.

Acree, L.S., Longfors, J., Fjeldstad, A.S., Fjeldstad, C., Schank, B., Nickel, K. J., Montgomery, P.S & Gardner, A.W. (2006). Physical activity is related to quality of life in older adults. *Journal of Health Quality of Life Outcomes*,4: 1- 6.

Adams, H. P; del Zoppo, G; Alberts, M. J; Bhatt, D. L; Brass, L; Furlan, A; Robert, L; Grubb, Randall, T; Higashida, E. C; Chelsea Kidwell, J; Patrick D Lyden, D; Lewis, B; Morgenstern, A; Qureshi, I; Rosenwasser, R.H; Scott, P.A; Eelco, F.M & Wijdicks., A.E. (2015). Guidelines for the Early Management of Adults With Ischemic Stroke A Guideline From the American Heart Association/American Stroke Association Stroke Council, Clinical Cardiology Council, Cardiovascular Radiology and Intervention Council, and the Atherosclerotic Peripheral Vascular Disease and Quality of Care Outcomes in Research Interdisciplinary Working Groups: The American Academy of Neurology affirms the value of this guideline as an educational tool for neurologist. *Circulation,* 115(20): 478-534.

Agarwal, S., Kobetic, R., Nandurkar, S & Marsolais, E. B. (2003). Functional electrical stimulation for walking in paraplegia: 17-year follow-up of 2 cases. *Journal of Spinal Cord Medicine*,26(1): 86-91.

Agyemang, C., Addo, J., Bhopal, R., Aikins, A. G. & Stronks, K. (2009*).* Cardiovascular disease diabetes and established risk factors among populations of sub – Saharan Africa Descent in Europe; A literature review. *Journal ofGlobal Health,* 11: 5-7.

Ahmed, R., Zuberi, B. F. & Asfar, S. (2004). Stroke scale score and early prediction of outcome after stroke. *Journal of the College of Physicians and Surgeons Pakistan*. 4(5): 267-267.

Allan, S. M; Tyrrell, P. J. & Rothwell, N. J. (2005). Interleukin-1 and neuronal injury.*Journal of National Review*, 5: 629-640.

Alon, G; Lewitt, A.F.& McCarty, P.A. (2008). Functional electrical sitmulation (FES) may modify the poor prognosis of stroke survivors with severe motor loss of the upper extremity: A preliminary study. *American Journal of Physical Medicine and Rehabilitation,* 87: 627-36.

Ambrosini, E., Ferrente, S., Pedrocchi, A., Ferrigno, G. & Molteni, F. (2011). Cycling induced by electrical stimulation improves motor recovery in postacute hemiparetic patients: A randomized controlled trial. *Journal of Cerebral Circulation*, 42: 1068-1073.

American College of Sports Medicine (2002). Resources for Clinical Exercise Physiology: muscular, neuromuscular, neoplastic, immunologist & hematologic conditions. Philadelphia, Lippincott Williams & Wikins, 1: 8.

American College of Sports Medicine. (2000). *ACSM's Guidelines for Exercise Testing and Prescription*, 6th ed. Baltimore: Lippincott Williams & Wikins. 1-368.

American Heart Association (AHA) (2010).Guideline for the management of heart failure: A report of the American College of Cardiology Foundation/American Heart Association task force on practice guideline. *Circulation*, e240-327. doi: 10.1161/CIR.0b013.

American Heart Association (1997). Monounsaturated Fatty Acids and Risk of Cardiovascular diseases. *Circulation*, 100(11): 1253 - 1258.

American Heart Association (2005). *Heart Disease and Stroke Statistics.* Update. Dallas, Texas: *American Heart Association*, 60.

Anderson, S. I., Whatling, P., Hudlicka, O., Gosling, P., Simms, M & Brown, M. D. (2004). Chronic Transcutaneous Electrical Stimulation of Calf Muscles improves Functional Capacity without Inducing Systemic Inflammation in claudicants''. *European Journal of Vascular and Endovascular Surgery*, 27(2): 201-209.

Ashe, M. C., Millir, W. C., Eng J. J. & Noreau, L. (2009). Older adults, Chronic Disease and Leisure Time Physical Activity. *Journal of Gerontology*, 55: 64-72.

Asplund, K., Karvanen, J., Giampaoli, S., Jousilahti, P., Niemela, M., Broda, G. & Kulathinal, S. (2009). Relative Risks for Stroke by Age, Sex, and Population based on follow-up of 18 European populations in the Morgan project. *Journal of Cerebral Circulation*, 40: 2319-2322.

Badejo, O. O., & Olawale, O. A. (2000). Determination of Normative Maximum Hand Grip Strength in male and female participants. *Journal of the Nigerian Medical Rehabilitation Therapist*, 5(2): 24-26.

Bailey, M & Macwhenell, C. (1997). Clinical Mentoring of Dry Vascular Lower limbs Amputees during initial Gait Training. *Journal of Physiotherapy*, 83(6): 278-283.

Baseline Evaluation tools. (2012). *Measurement of Grip Strength using Pinch Gauge*. Retrieved from http://www.baselineproducts.net

Bassetti, C & Aldrich, M. S. (1999*).* Sleep apnoea in acute cerebrovascular diseases: final report on 128 patients. *Sleep*, 22: 217–223.

Barcroft, J. (1975). The Respiratory Function of the Blood: Part I. Lessons from High Altitudes. London, Cambridge University Press, pp: 137.

Beelen, A., Sergeant, A. J., Jones, D. A. & Ruiter, C. J. (1995). Fatigue and Recovery of Voluntary and Electrically elicited Dynamic Force in humans. *Journal of Physiology*, 484(1): 227-235.

Belda-Lois, J. M.,Mena-del Horno, S., Bermejo-Bosch, I., Moreno, J. C., Pons, J. L., Farina, D., Iosa, M., Molinari, M., Tamburella, F., Ramos, A., Caria, A., Solis-Escalante, T., Brunner, C&Rea, M. (2011). Rehabilitation of Gait after Stroke: a review towards a top-down approach, 13(8): 66.

Bell, L.M., Watts, K., Sinfarikas, A., Thompson, A., Ratnam, N., Bulsara, M., Finn, J., O' Doiscoll, G., Green, D.S., Janes, T.W. & Davis E.A. (2007). Exercise alone reduces insulin resistance in obese children independently of changes in body composition. *Journal of Clinical Endocrinology & Metabolism*, 92: 4230-4235.

Bellinger, P. M. (2014). B-Alanine Supplementation for Athletic Performance: An update. *Journal of Strength and Conditioning Research*, 28(6): 1751-1770.

Bijeh, N & Farahati. S. (2013). The Effect of Six Months of Aerobic training on Renal Function Markers in Untrained Middle-Aged Women*; International Journal of Sport Studies*, 3(2): 218-224

Billat, V. I; Slawinski, I; Danel, M & Koralsztein, J. P. (2001). Effect of free versus constant pace on performance and oxygen kinetics in running. *Medicine and Science in Sports and Exercise*, 33: 2082-2088.

Billiant, A. E. (2001). Ischaemic Stroke rate in humans, *Journal of Cerebral Circulation*, 32(10): 231-237.

Bjuro, T., Fugl-Meyer, A. R & Grimby, G. (1975). Ergonomic studies of standardized domestic work in patients with neuromuscular handicap. *Scandinavian Journal of Rehabilitation Medicine*, 7: 106–113.

Black-Schaffer R. M., Kirsteins A. E & Harvey R. L. (1999). Stroke rehabilitation, co-morbidities and complications. *Archives of Physical Medicine and Rehabilitation*, 80: S8-S16.

Blair, S.N., Kohl, H. W., Paffenbarger, R. S., Clark, D. G, Cooper, K. H. & Gibbons, L. W. (1981). Physical fitness and all-cause mortality: a prospective study of healthy men and women. *Journal of American Medical Association*, 262: 2395-2401.

Boden, M., Prince, T.B. & Perseghin, G. (1996). Mechanism of free fatty acid-induced insulin resistance in human. *Journal of Clinical Investigation*, 97: 2859-2865.

Bohannon R & Walsh S. (1992). Nature, reliability and predictive value of muscle performance measures in patients with hemiparesis following stroke. *Archives of Physical Medicine and Rehabilitation*, 73: 721-725.

Bonita, R. (1997). Prevalence of Sstroke and Stroke-related Disability. *Journal of Cerebral Circulation*, 28(8): 1527–1529.

Bonora, E., Mohutte, P., Zankanaro, G., Cigolini, M., Querena, M., & Cacciatore, V. (1989). Estimation of *invivo* insulin action in man: comparison of insulin tolerance test with euglycemic and hyperglycemic glucose clamp studies. *Journal of Clinical Endocrinology & Metabolism*, 68: 374-378.

Brannon, F.J., Foley, M.W., Starr, J.N & Saul, M.L. (1983).*Cardiopulmonary Rehabilitation:* Basic Theory and Application. 3rd Edition., Davies Company, Philadelphia, 1-486.

Brewer, L., Horgan, F., Hickey, A. & Williams, D. (2012). Stroke rehabilitation: Recent advances and future therapies. *International Journal of Medicine*, 106(1): 11-25.

Brinkmann, J. R. & Hoskins, T. A. (1979). Physical Conditioning and Altered Self-Concept in Rehabilitated Hemiplegic Patients. *Journal ofPhysiotherapy*. 59: 859-865.

Bropy, P., Zankanaro, G., Cigolini, M., Querena, M. & Cacciatore, V. (2013). Chronic Transcutaneous Electrical Stimulation of Calf Muscles improves Functional Capacity without Inducing Systemic Inflammation in claudicants''. *European Journal of Vascular and Endovascular Surgery*, 7(2): 20-29.

Brouns, F. & Cargill, C. (2002). *Essentials of Sports Nutrition*; England; John Wiley and Sons Limited, 2nd edition, 1- 227.

Brown,D. A.*&DeBacher*, G. A. (2014). Pedaling exercise in persons with *poststroke*hemiplegia. *Journal of Physiotherapy*, 23(5): 102-110.

Brown, R. D., Whisnant J. P., Sicks, J. D., O'Fallon, W. M. & Wiebers, D. O. (1989). Stroke incidence, prevalence, and survival: secular trends in rochester, Minnesota, *Journal of Cerebral Circulation*, 27: 373–380.

Bruno-Petrina, A. (*2014). Motor Recovery in Stroke*. Retrieved from http://emedicine.medscape.com/article/324386-overview.

Cauraugh, J., Light, K., Kim, S., Thigpen, M. & Behrman, A. (2000).Upper extremity improvements in chronic stroke: Coupled bilateral load training. Restoration Neurology Neuroscience, 27(1): 17–25.

Chodobski, A., Zink, B. J. &Szmydynger-Chodobska, J. (2011). Blood-brain barrier pathophysiology in traumatic brain injury. *Transnational Stroke Research,* 2(4): 492-516.

Ciccone, C. D. (2002). *Pharmacology in Rehabilitation*. Pennsylvania; F.A. Davis,89(5): 1-257

Clarke, P., Marshall, V., Block S. E & Colantonio, A. (2002). Well being after stroke in canadian seniors: Finding from the Canadian study of health and aging. *Journal of Cerebral Circulation,* 33: 1016 -1021.

Clayton, E. B & Forsters, A. (1982). *Clayton's Electrotherapy Theory and Practice.* 8th ed. *British Journal of Sports Medicine,* 16(4): 1-268.

Consumer Union, (1998). Do you know your triglyceride? *Consumer Reports on Health*, 10(11): 7-8.

Cooper, A., Alghamdi, G. A., Alghamdi, M. A., Altowaijri, A, &Richardson, S.(2012). The relationship of lower limb muscle strength and knee joint hyperextension during the stance phase of gait in hemiparetic stroke patients. *Journal for Researchers and Clinicians in Physical Therapy,* 17(3): 150-6.

Coyle, E. F., Hagberg., J. M., Hurley, B. F., Martin, W. H. & Holloszy, J. O. (1996). Carbohydrate feeding during prolonged strenuous exercise can delay fatique. *Journal of Applied Physiology, Respiratory Environment and Exercise Physiology*, 55(1): 230-235.

Coyle, E. F., Martin, W. H., Sinacore, D. R., Joyner, M. J., Hagberg, J. M. & Holloszy, J. O. (1984). Time course of loss of adaptations in stopping prolonged intense endurance training. *Journal of Applied Physiology*, 57: 1857-1864.

Cruz, M. I., Weigensberg, M. J., Huang, T. F, Ball, G., Shaibi, G. O. & Goran M. I. (2004). The metabolic syndrome in overweight Hispanic youth and the role of insulin sensitivity. *Journal of Clinical Endocrinology & Metabolism*, 89: 108-113.

Cumminig, T. B., Thrift, A. G., Collier, J. M., Churilov, L., Dewey, H. M., Donnan, E. A. & Bernhartt, J. (2011). Very early mobilization after stroke fast, tracks return to walking. *Journal of American Heart Association,* 42: 153-158.

Damasceno, A., Gomes, J., Azevedo, A., Carrilho,C., Loso, V., Lopes, H., Madade,T., Pravinrai,P., Silva-Mantos, C., Jalla, S., Stewart,S & Lunet,N. (2010). An epidemiological study of stroke hospitalizations in Maputo, Mozambique. *Journal of cerebral circulation,* 41: 2463-2469.

Davenport, K. L., Julius, S. M. & Davenport, P. W. (2007). Perception of preservatory resistive loads in Asthmatic children with Attention Deficit Disorder. *Journal of Clinical Medicine, Respiratory and Pulmonary,* 1: 17-14.

De Troyer, A., De Beyl, D. & Thirion, M. (1981). Function of the respiratory muscles in acute hemiplegia. *American Review of respiratory Disability*, 123: 631–632.

Deklin, J. T. & Ruderman, N. (2002). Diabetes and exercise: The risk benefits profile revisited. In: Ruderman, N. Devlin, J.T, Schneider, S.H. & Krisra, A. (Eds). Handbook of Exercise in Diabetes. Alexandria, VA; *American Diabetes Association,* 253–268.

Depress, J. P. (1994) Dyslipedemia and Obesity. Bailers. *Journal of Clinical Endorcrinology and Metabolism,* 8: 629-660.

Dobkin, B. H; Xu, X; Batalin, M; Thomas, S&Kaiser, W. (2011). Reliability and validity of bilateral ankle accelerometer algorithms for activity recognition and walking speed after stroke. *Stroke, Journal of Cerebral Circulation*, 42(8): 2246-50

Donnan, G. A., *Fisher,*M., Macleod, M. & Davis, S. M. (2008). Epidemiology of stroke patients. Stroke. *Lancet,* 371(9624): 1612-23.

Doohan, J.(2000). *Cardiac Output.* Retrieved on April 20; 2011,from http://www.biosbcc.net/doohan/sample/htm/COandMAPhtm.htm

Drygas, W., Kostka, T., Jegier, A. & Kunski, H. (2000). Long-term effects of different physical activity levels on coronary disease factor in middle-aged men. *Journal of Sports Medicine,* 21: 235-241.

Duncan, P., Richards, L., Wallace, D., Stoker-Yates, J., Pohl, P., Luchies, C., Ogle, A., &Studenski, S. (1998). A randomized, controlled pilot study of a home-based exercise program for individuals with mild and moderate stroke. *Journal of Cerebral Circulation*, 29(10): 2055-2060.

Duncan, P. W. (1997). Synthesis of intervention trials to improve motor recovery following stroke. *Top Stroke Rehabilitation,* 3: 1-20.

Duncan, P. W., Zonwitz, R. & Baters B. (2005). Make agreement of adult stroke rehabilitation Care. A clinical practice guideline in Stroke, *Journal of Cerebral Circulation*, 36: e100 - e143.

Duncan, P., Zorowitz, R., Barbara, B., Choi. J., Glasberg, J., Lamberty, K. & Reker, D. (2005). Management of adult stroke rehabilitation Care. *Journal of Cerebral Circulation*, 36: 100-143.

Durstine, L., Peter, W. G., Paul, G. D., Michael, A. J., Mathew, L. A. & DuBose, K. D. (2002). Blood lipids and lipoprotein adaptation Co-exercise: A qualitative analysis. *Sports AVU Medicine*, 13: 50-62.

Ellis, Hill, C & Horn, S. (2000). Hanged in identity and self-concept: A new theoretical approach to recovery following a stroke, *Clinical Rehabilitation*, 14(3): 279-288.

Evers, S. M., Struijs, J. N., Ament, A. J., Van Genugten, M. L., Jager, J. C. & Vanden Bros, G. A. (2004). International comparison of stroke cost studies. *Journal of Cerebral Circulation*, 35: 1209-1215.

Fall,M. &*Lindstrom, S.* (1991). Electrical stimulation: A Physiology approach to the treatment of urinary incontinence. *North American Journal of Urology*, 18: 393-7407.

Fletcher GF, Balady GJ, Amsterdam EA, Chaitman B, Eckel R, Fleg J, et al.(2001). Exercise standards for testing and training: a statement for healthcare professionals from the American Heart Association.Journal of Circulation; 104(14):1694-740.

Fluck, D. C. (1966). Chest movements in hemiplegia. *Clinical Sciences, 31: 383–388.*

Folsom, A. R., Ameti D. K., Hutchinson, R. G., Liao, F., Clegg, L. & Cooper L.S. (1997). Physical activity and incidence of coronary heart disease in middle age women and men. *Medicine in Science Sports and Exercise*, 29: 901-909.

Ford, E. S. (2005). Risk for all-cause mortality, cardiovascular disease, and diabetes association with the metabolism syndrome: a summary of the evidence. *Diabetes care*, 28: 1769-1778.

Fox, S. M., Naughton, J. P. & Gurman, P. A. (1972). Physical Activity and Cardiovascular health: The Exercise prescription, frequency and type of activity. *Journal of Concepts and Cardiovascular Diseases*, 41: 25-30.

Ganong , W.F. (1999). Review of medical physiology (19^{th}).Starford Connecticum. Appleton and Lange. 1-181.

Gaskin, L & Thomas, J. (1995). Pulse oximetry and exercise. *Journal of Physiotherapy*.8 (5): 254-261.

Garg, A. Bantte, J. P., Henry, R. R., Coulston A. M. & Griver K. A. (1998). Effects of varying carbohydrate content of diet in patients with insulin dependent diabetes mellitus. *Journal of American Medical Association*, 271: 1421-1428.

Gillum, R. F & Sempos, C. T. (1997). The end of the long-term decline in stroke mortality in the United States? *Journal of Cerebral Circulation*, 28: 1527–1529.

Gladman, J., Lincoln, N & Barer, D. (2003). A randomized controlled trial of domiciliary and hospital based rehabilitation for stroke patients after discharged from hospital. *Journal of Neurology, Neurosurgery and Psychiatry,* 55: 960-966.

Globas, C., Becker, C., Cerny, J., Lam, J. M., Lindemann, U., Forrester, L. W. & Macko, R. F. (2012). Chronic stroke survivors benefit from high-intensity aerobic treadmill exercise: a randomized control trial. *Neurorehabilitation and Neural Repair*, 26(1): 85-95

Goldstein, L. B., Bushnell, C. D., Adams, R. J., Appel, L. J., Braun, L. T., Chaturvedi, S. & Pearson, T. A. (2011). Guidelines for the primary prevention of stroke: A Guideline for Healthcare Professionals from the American Heart Association/American Stroke Association. *Journal of Cerebral Circulation*, *42*(2), 517-584.

Goodman, J.M. (1996). Assessment of Exercise Capacity and Principles of Exercise Prescription. Golbourt, I., Yaari, S. & Medalic, J.H. (1997). Isolated high density lipoprotein- cholesterol as risk factor for coronary heart disease mortality: 21 year follow up of 8000 men. *Journal of Arteriosclerosis, Thrombosis and Vascular Biology*, 17: 107-113.

Gordon, N.F., Gulanick, M; Costa, F., Fletcher, G., Franklin, B.A., Roth, E.J. & Shephard, T. (2004). Physical activity and exercise recommendations for stroke survivors: an American Heart Association scientific statement from the Council on Clinical Cardiology, Subcommittee on Exercise, Cardiac Rehabilitation, and Prevention; the Council on Cardiovascular Nursing; the Council on Nutrition, Physical Activity, and Metabolism; and the Stroke Council. *Circulation,* 109(16): 2031-41.

Gordon, T. & Mao, J. (1994). Muscle atrophy and procedures for training after spinal cord injury, *Journal of Physical Therapy*, 74: 50-60.

Giuliano, K.K & Higgins, T.L. (2005). New generation pulse oximetry in the care of critically ill patients. *American Journal of Critical Care,* 14: 26-39.

Guo, W., Kawano, H., Piao, L., Itoh, N., Node, K. & Sato, T. (2011). Effects of aerobic exercise on lipid profiles and high molecular weight adiponectin in Japanese workers. *Journal of International Medicine,* 50(5): 389-395.

Haas, A., Rusk, H. A., Pelosof, H., & Adam, J. R. (1967). Respiratory function in hemiplegic patients. *Archives of Physical Medicine and Rehabilitation, 48: 174–179.*

Habibzadeh, N. (2010). Effects of aerobic exercise on some selected metabolic syndromes in young obese women. *Achives of Kinesiology*, 4: 24-403
.

Hakemi, A & Bender, J.A. (2005). Understanding pulse oximetry advantages and limitations. Home Health Care Management and Practice, 17: 416-418.

Hamzat, T. K & Olaleye, O. A. (2002). Stroke Rehabilitation; when should ambulation commence (abstract). *Journal of the Nigerian Medical Rehabilitation Therapist*, 7(29): 23-25.

Hamzat, T. K., Olaleye, O. A. &Akinwumi, O. B. (2014). Functional ability, community reintegration and participation restriction among community-dwelling female stroke survivors in Ibadan, Ethiopian Journal of Health Science, 24(1): 43–48.

Hara, Y., Ogana, S, & Muroakam Y, (2006). A home based rehabilitation program for the hemiplegic upper extremity by power assisted FES, *Journal of Cerebral Circulation*, 29.

Harbison, J. & Gibson, G. J. (2000). Snoring, sleep apnoea and stroke: Chicken or scrambled Egg*? Quarterly Journal of Medicine, 93: 647–654.*

Harris, J. E. & Eng, J. J. (2010). Strength Training improves upper limb function in individuals with Stroke. A meta-analysis. *Journal of Cerebral Circulation,* 14(1): 136-140.

Helen, C., Roberts, H. J., Denison, H. J., Martin, H. P., Patel, H. S., Cooper, C. & Avan, A. S. (2011). A review of the measurement of grip strength in clinical and epidemiological studies: towards a standardised approach. *Journal of Age and Ageing*, 40(4): 423-429.

Hermansen L, Ekblom B, Saltin B. (1970). Cardiac output during submaximal and maximal treadmill and bicycle exercise. Journal of Applied Physiology; 29(1):82-6.

Hiraoka, K. (2005). Rehabilitation Effort to Improve Upper Extremity Function on Post Stroke Patients: A Meta Analysis. *Journal of physical Therapy Science*, 13: 5-9.

Hoenig H., Nusbaum N & Brummel-Smith K. (1997). Geriatirc rehabilitation: state of the art. *Journal of American Geriatric Society*, 45: 1371-1381.

Hollard, L. J & Bouffard, E. L. (1992). Rating of perceived Exercise, Heart rate and oxygen consumption in Adults with Multi Sclerosis. *Quarterly Journal of Physical Activity*, (9): 64-73.

Howell, M. (2002). The correct use of pulse oximetry in measuring oxygen status. *Professional Nurse*, 17 (7): 416-18.

Huang, T. T., Ball, G. D. & Frauls, P. W. (2007). Metabolic Syndrome in Youth: Current Issues and Challenges. *Journal of Applied Physiology, Nutrition and Metabolism*, 32: 13-22.

Hussein, N. Y., Christin, M. A., Raji, K. K., Thomas, R. V., Frank, K & John, P. K. (2009). Effect of exercise and caloric restriction on insulin resistance and cardometabolic Risk factor in older obese adults: a randomized clinical trial. *Journal of Gerontology*, 64(1), 90-95.

Hosseini, S.M & Ghanbari Niaki, A. (2013). The effect of progressive short-time aerobic training on OGTT and insulin level in young men; *International Journal of Sport Studies*, 3(3): 313-318.

Ibrahim, S. (1990). *Clinical epidemiology of stroke*. New York: Oxford university press, pp. 183.

Ivey, F. M., Gardner, A. W., Dobrovolny, C. L & Macko, R. F. (2004). Unilateral impairment of leg blood flow in chronic stroke patients, *Journal ofCerebrovascular Diseases*, 18(4): 283–289.

James, R., Gines, D., Menlove, A., Horn, S. D., Gassaway, J., & Smout, R. J. (2004). Nutrition support (tube feeding) as a rehabilitation intervention. *Archieves of Physical Medicine Rehabilitation, 86*(12) S82-S92.

Janssen, T. W., Beltman, J. M., Elich, P., Koppe, P. A., Konijnenbelt, H., DeHaan, A & Gerrits, K. H. (2008). Effects of electric stimulation-assisted cycling training in people with chronic stroke. *Archives of Physical Medicine and Rehabilitation*, 89: 463-469.

Jatau, R. S., Venkateswarlu, K., Dikki, C. E. & Haruna, F. R. (2012). Effects of moderate intensity interval training on running on lipids and lipoproteins of sedentary Nigeria male adults. *Journal of Health, Physical Education, Sports and Leisure Studies*, 3(2): 144-155.

Kang, J., Robertson, R. J., Hagbert J. M. Kelley, D. E., Goss, F. L. Dastiva, S. G., Suminski, R. R. & Utter A. C. (1996). Effect of exercise intensity on glucose and insulin metabolism in obese Individuals and obese NIDDM patients. *Journal of Diabetes Care*, 19(4): 311-319.

Kelley G. A., Kelley, K. S. & Vu Tran, Z. (2005). The effect of arobic exercise on lipid and lipoprotein in overweight and obese adults. *International Journal of Obesity*, 29(8): 881-893.

Kelley, D. E. Goodpaster, B., Wing, R. R. & Simoneau, J. A. (1999). Skeletal muscle fatty acid metabolism in association with insulin resistance, obesity and weight loss, *American Journal of Physiology*, 277: E1130-E1141.

Kelley, G. S., Kelley, K. S. & Vu Tran, Z. U. (2004). Walking, lipids, and lipoproteins, meta-analysis of randomized controlled trials. *Journal of Preventive Medicine*, 38: S438-S445.

Kelly, P; Westlake,' B. &Nagarajan, S. S.' (2011). Functional connectivity in relation to motor performance and recovery after stroke, Front System, Journal of Neuroscience, 5: 8-12.

Kern, H., Salmons, S., Mayr, W., Rossini, K. & Carraro, U. (2005). Recovery of long term denervated human muscles induced by electrical stimulation, *Journal ofMuscle and Nerve*, 31: 98-101.

Khuram, H. (2011). Effect of aerobic physical training on stroke survivors. *Umea*, 2: 42-48.

Khurana, V. G. (1996). Investigating the relationship between *stroke* and obstructive sleep apnea. *Journal of Cerebral Circulation*, 27: 428-432.

Kiani, K., Snijders, C. J.& Gelsema, E. S. (1997). Computerized analysis of daily life motor activity for ambulatory monitoring. *Journal of Technology Health Care*, 5(4): 307-318.

Kolapo, T. H., Echezona, H., Ekechukwu, E., Feigin, V. L., Lawes, C. M & Bennett, D. A (*2003*). A review of population-based studies. *Journal of Epidemiology and Public Medicine*, 34(2): 494-501.

Kraemer, J. L., Worm, P. V., Faria, M. B. & Maulaz, A. (2002). Brain abscess following ischemic stroke with secondary hemorrhage. *Archieves of Neuropsychiatry*, 66(1): 104-106 retrieved from www.scielo.br/pdf/anp/v66n1/28.pdf.

Kraemer, W. J., Adams, K., Caferelli, E. I. & Dudley, G. A. (2002). American College of Sports Medicine Position Stand. Progressive need in resistance training for healthy adults. *Journals of Medical Science and Sport Science*, 34(2): 364-480.

Krebs, H. I., Aisen M. L., Volpe, B. T & Hogan, N. (2006). Quantization of continuous arm movements in humans with brain injury, *Proc National Academy of Science*, 96: 46445-46491.

Kurl, S., Laukanen, J. A. & Rauramaa, R. (2003). Cardio-respiratory fitness and the risk for stroke in men. *Archives of Internal Medicine*, 163: 1682–1688.

Kwakkel, A., Kollen, B. G., Van der grand, J & Prevu , A. Y. (2003). Probability of regaging dexterity in the flaccid upperlimb impact of severity of paresis and time since onset in acute stroke. *Journal of Cerebral Circulation*, 34(9): 2189- 2186.

Lakka, H. M & Leakman, M. (2007). The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *Journal of American Medicine Association,* **288**: 2709–2716.

LaMonte, M. J., Blair, S. N., & Church, T. S. (2005). Physical activity and diabetes prevention. *Journal of Applied Physiology*, 99: 1205-1213.

Langhorne, P., Stott, D. J., Robertson, L., MacDonald, Jones, L, McAlpine, C., Dick, F; Taylor, G.S. & Murray G. (2000). Medical complications after stroke: a multicenter study. *Journal of Cerebral Circulation*, 31: 1223–1229.

Lavie, C. J., Mailander, L. & Milani, R. V. (1992). Marked benefits with sustained-release niacin therapy in patients with isolated very low levels of high density lipoprotein cholesterol and coronary artery disease. *American Journal of Cardiology*, 69:1083-1085.

Lawer, R. T., Smith, B. T., Mulcahey, M. J.,Betz, R. R. & Johnson, T. E.(2011).Effects of cycling and electrical stimulation on bone mineral density in children with spinal cord injury. *Journal of Spinal Cord*. 49: 917-923.

Lawrence, M. & Brass, M. D. (1992).Stroke. http//www.doc.med.yale.edu/heartbk/18.pdf.

Lee, S. Y., Kim, B. R., Choi, J. H., Lee, S. J. & Han, E. Y. (2012). The effects of neuromuscular electrical stimulation on cardiopulmonary function in healthy adults. *Annual Journal of Rehabilitation Medicine,* 36: 849-856.

Lee, S., Shafe, A. C., & Cowie, M. R. (2011). UK stroke incidence, mortality and cardiovascular risk management 1999–2008: time-trend analysis from the General Practice Research Database. *British Medical Journal*, 1: e000269. doi: 10.1136/bmjopen.

Lennon, O., Carey, A., Gaffney, N., Stephenson, J. & Blake, C. (2008). A pilot randomized controlled trial to evaluate the benefit of the cardiac rehabilitation paradigm for the non-acute ischaemic stroke population. *Clinical Rehabilitation,* 22: 125–133

Leon, A. S. & Sanchez, O. A. (2001). Response of blood lipids to exercise training alone or combined with dietary intervention. *Medicine & Science in Sports & Exercise,* 3(6): S502-S515.

Letembe, D. B., Henry, A. C. & Watt, H. M. (2010). Clinical Guidelines for *Stroke* Management. *Journal of American Medicine Association,* 10(12): 23-35.

Liberman, A. M., Cooper, F. S., Shankweiler, D. P. & Studdert-Kennedy, M. (1997). Perception of speech code. *Psychological Review,* 74: 431–461.

Liebeskind, D. S. & Leitsep, I. R. (2015). Aortic occlusion for cerebral *ischaemia*: from theory to practice. *Journal ofCirculation Cardiology & Respiration,* 10: 31–36.

Liepert, J., Bauder, H., Wolfgang, H.R., Miltner, W.H., Taub, E. &Weiller, C. (2000). Treatment-induced cortical reorganization after stroke in humans. *A Journal of cerebral Circulation,* 31(6): 1210-6.

Lindberg, L.G., Lennmarken, C. & Vegfors, M. (1995). Pulse oximetry: clinical implications and recent technical developments. *Acta Anaesthesiologists in Scand*inavian, 39: 279–287.

Linder, C.W., Durant, R.H. & Mahauney, O.M. (1983). The effect of physical conditioning on serum lipid and lipoprotein in white male adolescents. *Journal Medicine & Science in Sport & Exercise*, 15: 233-236.

Lindquist, A.R., Prado, C.L., Barros, R.M., Mattioli, R., Da Costa, P.H & Salvani, T.F. (2007). Gait training combining partial body weight; effects on post stroke gait. *Journal of Physical Therapy*, 87: 1144-1154.

Lindstrom, M., Lindberg, M. & Sjostrom, S. (2011). Home bittersweet home: the significance of home for occupational transformations. *International Journal of Social Psychiatry, 57*(3): 284-99.

Locke, E. A., & Latham, G. P. (2002). Building a practically useful theory of goal setting and task motivation. *American Psychologist, 57*(9), 705-717.

Lonn, E., Yusuf, S. & Arnold, M. Y. (2006). Homocysteine lowering with folic acid and B vitamins in vascular disease: *New England Journal of Medicine,* 354(15): 1567- 1577.

Lord, J. P. & Hall, K. (1986). Neuromuscular re-education versus traditional programs for stroke rehabilitation. *Archieves of Physical Medicine and Rehabilitation,* 67:88 – 91.

Low, J. & Reed, A. (2000).*Electrotherapy Explained: Principles and Practice*. 3rd edition, Oxford Butterworth-Heinemann, 246.

Luukinen, H., Koshi, K. & Paippala, P. (1995). Risk factors for recurrent falls in the elderly in long-term institutional care. *Public Health,* 109: 57-65.

Macko, R. F., DeSouza, C. A & Tretter, L. D. (1997). Treadmill aerobic exercise training reduces the energy expenditure and cardiovascular demands of hemiparetic gait in chronic stroke Patients. A preliminary report. *Journal of Cerebral Circulation,* 28: 326-330.

Macko, R. F., Ivey, F. M., Forrester, L. W., Hanley, D., Sorkin, J. D., Katzel, L. I., Silver, K. H. & Goldberg, A. P. (2005). Treadmill exercise rehabilitation improves ambulatory function and cardiovascular fitness in patients with chronic stroke: a randomized, controlled trial. *Journal of Cerebral Circulation,* 36: 2206–2211.

Maeder, M., Wolber, T., Atefy, R., Gadza, M., Amman, P., Myers, J & Rickli, H. (2005). Impact of the exercise Mode on exercise capacity; bicycle testing revisited. *Chest*. 128(4): 2804 – 2811.

Manning, C. D. & Palmproy, V. M. (2003). Effectiveness of treadmill retraining on gait retraining of hemiparetic stroke patients, *A systematic review of current evidence physiotherapy*, 89 (c): 337-349.

Manning, C. D. & Pomeroy, V. M. (2013). Recovery of walking function in stroke patients: the Copenhagen Stroke Study. *Journal of Neurologic Physical Therapy*, 94: 839-844.

Mathiowetz, V. & Ferderman, S. (1985). Box and Block Test of Manual Dexterity: Norms for 6-19 Year Olds. *Canadian Journal of Occupational Therapy*, Revue Canadienne d'ergothérapie, 52(5): 241-246.

McClure, A., Meyer, M., Teasell, R.&Hussein, N. (2014).Stroke: More than a 'brain attack'. *International Journal of Stroke*, 9(2): 188-90.

McGill, H. C. & McMahan, G. A. (2010). Starting early to control all risk factors in order to prevent coronary heart disease. *Journal of Clinical Epidemiology*, 5: 87-93.

Moazami, M., Bijeh, N., Gholamian, S. (2013). The Response of Plasma Leptin and Some Selected Hormones to 24-weeks Aerobic Exercise in Inactive Obese Women; *International Journal of Sport Studies*, 3(1): 38-44.

Mohan, K. M., Wolfe, C.D., Rudd, A. G., Heuschmann, P. U., Kolominsky-Rabas, P. L., Grieve, A. P. (2011). The Rebirth of Stroke:Risk and Cummulative Risk of Stroke recurrency: A Systematic Review and Meta-analysis. *Journal of Cerebral Circulation*, 42(5): 1489-94.

Mohr, T., Carlson, B., Sulentic, C. & Landry, R. (1985). Comparison of isometric exercise and high volt galvanic stimulation on quadriceps femoris muscle strength. *Journal ofPhysical Therapy*, 65: 606-609.

Monaghan, B., Caulfield, B. & Mathuna, D. O. (2010). *Surface Neuromuscular Electrical Stimulation for Quadriceps Strengthening Pre and Post Knee Replacement*. John Wiley & Sons. pp, 24.

Monye, A. (2011). *Constraint Induced Movement Therapy and Electrical Stimulation in Stroke Patients.* Unpublished project. Bayero University, Kano, pp, 113.

Muhammad, M. S. (2003). *Effects of Aerobic Exercise Training on Selected Physiological Variables in non-Insulin Dependent Diabetics*. Unpublished PhD Dissertation, Ahmadu Bello University, Zaria.

Mundal, R., Kjelden, S.E., Sandrick, L., Erikkssen, G., Thaulow, E & Erikssen, J. (1994). Blood pressure risk factor in healthy post menopausal women. *Journal of Hypertension.* 24(1): 56-62.

Nachtman, A., Siebler, M., Rose, G., Sitzer, M. & Steinmetz, H. (1995). Cheyne-Stokes respiration in ischaemic stroke. *Journal of Neurology*. 45: 820-821.

Narayani, U. & Sudhan R. R. (2010). Effect of aerobic training on percentages of body total cholesterol and HDL-C among obese women. *World Journal of Sport Sciences*, 3 (1): 33-36.

Norkin, C. C. & White, D. J. (1988). *Techniques and Procedures, in Measurement of Joint Motion*: A guide to goniometry, C. C. Norkin and D. J. White, Editors. FA Davis: Philadelphia, 24.

Nwuga, G. O. (1990). A study of the relationship of the handgrip in industrial workers. *Journal of Industrial Medicine and Surgeons*, 56: 296–300.

O'Donnell, M. J., Xavier, D., Liu, L., Zhang, H., Chin, S. L., Rao-Melacini, P., Rangarajanm, S., Islam, S., Pais, P., McQueen, M. J., Mondo, C., Damasceno, A., Lopez-Jaramillo, P., Hankey, G. J., Dans, A.L., Yusoff, K., Truelsen, T., Diener, H. C., Sacco, R. L., Ryglewicz, D., Czlonkowska, A., Weimar, C., &Yusuf, S. (2010). Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the Interstroke study): a case-control study. *Lancet*, 376(9735): 112-23.

Oettingen, G. & Mayer, D. (2002). The motivating function of thinking about the future: Expectations versus fantasies. *Journal of Personality and Social Psychology*, 83:1198–1212.

Odusote, K. A. (1995). Management of ischaemic stroke – recent advance. *Nigerian Journal of Clinical Practices*, 5(2): 130-138.

Ogun, S. A. (2000). Acute stroke mortality at Lagos University Teaching Hospital- a five year review. *Nigerian Journal of Hospital Medicine*, 10: 8-10.

Ogunbo, B., Ushewokunze, S., Ogun, S. A. & Walker, R. (2005). How can we improve the management of stroke in Nigeria, Africa?. *African Journal of Neurological Sciences*. 5(2): 130-138.

Ojini, F. I. & Danesi, M. A. (2003). A 3 year review of neurologic admissions in admissions in University College Hospital, Ibadan, Nigeria. *West African Journal of Medicine*, 5: 38-41.

Olney, S., Griffin, M. & Monga, T. N. (1991). Work and power in gain of stroke patients. *Archieves of Physical Medicine and Rehabilitation*, 67: 92-98.

Owolabi, L. F. & Nagoda, A. S. (2012). Stroke in developing countries: Experience at Kano, Northwestern Nigeria. *Journal of Clinical*, 7(1): 9-14.

Owolabi, M. O. (2011). Taming the burgeoning stroke epidemic in Africa. *West Indian Medical Journal*, 60(4): 412-21.

Palmer-McLean, K & Harbst K. B. (2003). Stroke and Brain Injury. In: Durstine, J. L, Moore, G .E, eds. ACSM's Exercise Management for Persons with Chronic Diseases and Disabilities. 2nd ed. Champaign, Ill: *Journal of Human Kinetics*, 238–246.

Pang, M. Y., Eng. J. J., Dawson, A. S. & Gylfadottir, S. (2006). The Use of Aerobic Exercise Training in Improving Aerobic Capacity in Individual with Stroke. A meta analysis. *Journal of Clinical. Rehabilitation*, 20: 97-111.

Pascual-Leone, A., Fregni, F., Boggio, P.S., Mansur, C.G., Wagner, T., Ferreira, M.J., Lima, M.C., Rigonatti, S.P., Marcolin, M.A., Freedman, S.D. &Nitsche, M. A. (2005). Transcranial direct current stimulation of the unaffected hemisphere in stroke patients. *Neurological Report*, 28, 16(14): 1551-1555.

Patten, C., Lexell, J. & Brown, H. E. (2004). Weakness and Strength training in persons with Post-stroke hemiplegia: rationale method and efficacy. *Journal of Rehabilitation Research and Development*, 41(3A): 293-312.

Pearson, T. A., Blair, S. N. & Daniels, S. R. (2002). American Heart Association Science Advisory and Coordinating Committee. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update: consensus panel guide to comprehensive risk reduction for adult patients without coronary or other atherosclerotic vascular diseases. *Journal ofCirculation*; 106: 388–391.

Penaloza, D., Sime, F., Banchero, N., and Gamboa, R. (1962). Pulmonary hypertension in healthy man born and living at high altitudes. *Journal of Medicine and Thorax*., 19: 449

Pollock, M. L., Feigenbaum, M. S. & Brecheu, W. F. (1995). Exercise prescription for physical fitness. *American Academy of Kinesiology and Physical Education*, 47: 320-357.

Potempa, K., Braun, L.T., Tinknell, T. & Popovich, J. (1996). Benefits of aerobic exercise after stroke. *Journal of Sports Medicine*, 21: 337-346

Potempa, K., Lopez, M. & Braum, L. T. (1995). Physiological outcomes of aerobic exercise training in hemiparetic stroke patients. *Journal of Cerebral Circulation*, 26: 101-105.

Powell, J., Pandyan A. D., Granat, M., Cameron, M & Stott, D. J. (1999). Electrical stimulation of wrist extensors in poststroke hemiplegia. *Journal of Cerebral Circulation*, 30: 1384-9.

Prentice, R. L., Szatrowski, T. P., Kato, H. & Mason, M. W. (1994). Leukocyte counts and Cerebrovascular Disease, leukocytes in acute ischemic stroke, *Journal of Cerebral Circulation*, 25: 2149-2152.

Richards, C. L & Malouin, F. (2000). Task-related circuit training improves performance of locomotor tasks in chronic stroke: a randomized, controlled pilot trial. *Archives of Physical Medical Rehabilitation*, 81: 409-417.

Richards, J. S., Jackson, W. T. & Novack, T. N. (1997). *Central Nervous System Conditions: Assessing Medical Rehabilitation Practices*. The promise of outcome reasearch. Baltimore. Paul and Brooks, 346.

Rimmer, J. H., Braunschweig, C. & Silverman, K. (2000). Effects of a short- term health promotion intervention for a predominantly African- American group of stroke survivors. *American Journal of Preventive Medicine*, 18: 332-338.

Rimmer, J. H., Riley, B. & Creviston, T. (2000). Exercise training in a predominantly African-American group of stroke survivors. *Journal of Medical Science and Sports Exerise*, 32.1990-1996.

Roberts J. E. (2002). The "push" for evidence: Management of the second stage. *Journal of Midwifery and Women's Health*. 47(1): 2–15.

Rohrer, B., Fasioli S., Krebs, H. I., Hughes, R., Volpe, B. T., Frontera, W. R.., Stein, J. & Hogan N. (2002). Movement smoothness changes during stroke recovery. *Journal of Neuroscience*, 22: 8297-98304.

Roth, E. J. &Harvey, R. L. (2000). Incidence of and risk factors for medical complications during stroke rehabilitation. *Journal of Stroke*, 32(2): 523- 529.

Roth, E. J., Heinemann, A. W., Lovell, L. L,. Harvey, R. L., McGuire, J. R. & Diaz, S. (1999). Impairment and disability: their relation during stroke rehabilitation. *Archives of Physical Medicine and Rehabilitation*, 79(3): 329-35.

Roth, E. J. (1994). Heart disease in patients with stroke. Part II: Impact and implications for rehabilitation. *Archives of Physical Medicine and Rehabilitation*, 75(1): 94-101.

Rutherford, K. A. (1989). Principles and application of pulse oximetry. *American Journal of Clinical Critical Care Nursing*, 1: 649-657.

Sacco, R. L., Adams, R., Albers, G., Alberts, M. J., Benavente, O., Furie, K., Goldstein, L. B., Gorelick, P., Halperin, J., Harbaugh, R., Johnston, S. C., Katzan, I., Kelly-Hayes, M., Kenton, E. J., Marks, M., Schwamm, L. H. & Tomsick, B. (2006). Guidelines for prevention of stroke in patients with ischemic stroke or transient ischemic attack: a statement for healthcare professionals from the American Heart Association/American Stroke Association Council on Stroke: co-sponsored by the Council on Cardiovascular

Radiology and Intervention: the American Academy of Neurology affirms the value of this guideline. *Stroke, Journal of Cerebral Circulation*, 37(2): 577-617.

Sacco, R. L., Kargmann, D. E. & Zamanillo, M. C. (1995). Race-ethnic differences in stroke risk factors among hospitalized patients with cerebral infarction: the Northern Manhattan Study. *Journal ofNeurology,* 45: 659–663.

Schaechter, J. D. (2004). Motor rehabilitation and brain plasticity after hemiparactic. *stroke. Progress in Neurobiology,* 7: 61-72.

Schmidt, H., Werner, C., Bernhardt, R., Hesse, S & Krüger, J. (2007). Gait rehabilitation machines based on programmable footplates. *Journal of Neuroengineering and Rehabilitation*, 4(2): 231-238.

Shepherd, R. B. (2001). Exercise and training to optimize functional motor performance in stroke: driving neural reorganization? *Journal of Neural Plasticity*, 8: 121–129.

Shinton, R. &Beevers, G. (1989). Meta-analysis of relation between cigarette smoking and stroke. *British Medical Journal*, 25: 298-394.

Smith, H A., Lee, S. H., O'Neill, P. A. & Connolly, M. J. (2000). Combination of bedside swallowing assessment and oxygen saturation monitoring of swallowing in acute stroke: a safe and humane screening tool. *Journal of Age and Ageing*, 29: 495–499.

Smith, S. C., Jr., Blair, S. N & Bonow, R. O. (2001). AHA/ACC guidelines for preventing heart attack and death in patients with atherosclerotic cardiovascular disease: 2001 update: a statement for healthcare professionals from the American Heart Association and the American College of Cardiology. *Circulation*, 104: 1577–1579.

Stewart, D.G. (1999). Stroke rehabilitation. epidemiological aspects and acute management. *Archieves of Physical Medicine and Rehabilitation*, 80: S4-S7.

Strazzulla, G. (2010). Cardiovascular effects of brain natriuretic peptide in essential hypertension. *Journal of Hypertension*, 25(5): 1053-1057.

Strong, K., Mathers, C. & Bonita, R. (2007). Preventing *stroke*: saving lives around the world. *Lancet Neurology*, 6(2): 182-187.

Sunderland, A., Tunson, D. J. & Bradley, E. L. (1992). Enhanced physical therapy improves recovery of arm function after stroke. A randomized controlled trial. *Journal of Neurology Neurosurgery and Psychiatry*, 55: 530.

Surgeon General of the Public Health Service. (2004). Surgeon General's Report on Physical Activity and Health (S/N 017-023-00196-5); 1996. Retrieved from http://www.fitness.gov/execsum.htm.

Synder-Mackler, L. & Robinson, A. J. (1989). "*Clinical Electrophysiology*" .Baltimore William and Wilkins,292.

Szecsi, J., Krewer, C., Muller, F & Straube, A. (2008). Functional electrical assisted cycling of patients with subacutestroke. kinetic and kinematic analysis. *Journal of Clinical Biochemistry*, 23: 1086-1094.

Tang, A., Sibley, K.M., Thomas, S.G. & Bayley, M.T. (2008). Effects of an aerobic exercise program on aerobic capacity, spatiotemporal gait parameters, and functional capacity in subacute stroke. *Journal ofNeurorehabilitation and Neural Repair, 23*(4): 398-406.

Tang, A., Sibley, K. M., Thomas, S. G., Bayley, M. T., Richardson, D., Mcllroy, W. S. & Brooks, D. (2009). Effect of an aerobic programme on aerobic capacity spatotemporal gait parameters and funcational capacity in Sumba cute stroke. *Journal of Neurorehabilitation and Neural Repair,* 24(8): 1025-1036.

Taub, E., Uswaite, G. & Elbert, T. (2002). New treatment in neurorehabilitation founded on Basic Research. *Neuroscience*, 3: 228-236.

Teixeira-Salmela, L. F., Olney, S. J. & Nadeau S. (1999). Muscle strengthening and physical conditioning to reduce impairment and disability in chronic stroke survivors. *Archieves Physical Medicine and Rehabilitation*, 80: 1211 – 1218.

The National Health and Nutrition Examination Survey. (2014).Heart Disease and Stroke Statistics2014,Update.Retrievedfromhttps://circ.ahajournals.org/content/129/3/e28.full.pdf

Thomas, J. R. & Nelson, J. K. (1996). Research Methods in Physical Activity. *Human Kinetics*, Champaign I.L. 21.

Tolendano, B. E. (2011). Stroke endemic heart disease, *Journal of Cerebral Circulation*, 81(1): 361-369.

Turner, M.J., Speechly, C. & Bignell, N. (2007). Sphygmomanometer calibration--why, how and how often? *Australian Family Physician,* 36(10):834-838.

Van der Ploeg, H. P., Streppel, K. R., Van Der Beek, A. J., Van Der Woude, L. H., Vollenbroek-hutten, M. M. & Van Harten, V. H. (2007). Successfully improving physical activity behaviour after rehabilitation. *American Journal of Health Promotion*, 21: 153-159.

Vogel, J.H.K., W.F. Weaver, R.L.. Rose, S.G., Jr. Blount and R.F., Grover (1962). Pulmonary hypertension on exertion in normal man living at 10,150 feet (Leadville, Colorado). Med Thorac., 19: 461.

*V*an Hecke M. V., Dekker, M., Nijpels, G., Moll, A. C., Heine, R. J., Bouter L. M., Polak, B. C & Stehouwer C. D.(2005). Inflammationand endothelial dysfunction are associated with retinopathy: the Hoorn study. *Diabetologia,48: 1300–1306.*

Vantone, C. (2002). Unesterified plant steroids and stanol lower LDL-Cholesterol concentrations, equivalent in hypercholesterolemic persons. *American Journal of Clinical Nutrition*, 76: 1272-1276.

Venkateswarlu, K. (2012). Theory of Athlete Training (2nd ed.). Mimeographed Textbook PHE. Department, Ahmadu Bello University, Zaria, Nigeria. 94.

Verbeek, H. H., Alves, M. M., Groot, J. W., Osinga, J., Plukker, J. T., Links, T. P. & Hofstra, R. M. (2011). The effect of four different tyrosine Kinese inhibitors on Medullary and Pupillary Thryroid cancer cells. *Journals of Clinical Endocrinology and Metabolism*, 98 (6): 991-995.

Vincent, J. L. (2000). How to interprete cardiac output and haemodynamic indexes. *Critical Care Cardiology*, 19-27.

Vincent-Onabajo, G. T, Lawan A. K; Oyeyemi, A. Y; & Hamzat T. K. (2014). Functional self-efficacy and its determinants in Nigerian stroke survivors. *Top Stroke Rehabilitation*, 36(6): 504-11.

Visintin, M., Barbeau, H & Korner-Bitensky, N. (1975). New approach to retrain gait in stroke Patients through Body weight Support and treadmill Stimulation, 29: 1122–1128.

Wasserman, D. H., Gree, R. J., Williams, P. E., Lacy, D. B. & Abeemrad, N. N. (1991). Interaction of GUT and liver nitrogen metabolism during exercise. *Metabolism*, 40: 307-314.

West, J.B., Gill, M.B., Lahiri, S., Milledge, J.S., Pugh, I.G.C.E and M.P. (1962). Arterial oxygen saturation during exercise at high altitude. Journal of Appl ied Physiology., 17: 617.

Wildman, R. E. C. & Miller, B. S. (2004). Sport and Fitness Nutrition; Australia; Thomson Wadsworth, pp, 509.

William, M. H. (2007). Nutrition for Health Fitness and Sport; Boston; McGraw-Hill, Higher Education, 478.

Winslow, F. E & Scott, J. (2002). History and Information about Winslow. Arizona. Retrieved from www.Jeff Scott.com, 12 - 26.

Wojtaszewski, J. F. P., Nielsen, J. N., & Richter, E. A. (2002). Exercise effects on muscle insulin signalin and action. Invited review: Effect of acute exercise on insulin signaling and action in humans. *Journal of Applied Physiology*, 93: 384 - 392.

Wolf, P. A., Clagett, G. P. & Easton, J. D. (1999). Preventing ischemic stroke in patients with prior stroke and transient ischemic attack: a statement for healthcare professionals

from the stroke council of the American Heart Association. *Journal of Cerebral Circulation*, 30(9): 1991-1994.

Woo, D., Haverbusch, M., Sekar, P., Kissela, B., Khoury, J., Alexander, S. D., Kleindorfer, D., Szaflarski, J., Pancioli, A., Jauch, E., Moomaw, C., Sauerbeck, L., Gebel, J. & Broderick, J. (2004). Effect of Untreated Hypertension on Hemorrhagic Stroke. *Journal of Cerebral Circulation, 35: 1703-1708.*

Woodrow, P. (1999). Pulse Oximetry. *Nursing Standard*, 13: 42 – 46.

World Bank (2015).Report on Stroke Cases in Africa. World Bank Publications, 1 – 1315.

World Health Organization. (*1976)*; *Experience from a Multicentre Stroke Register*: a preliminary report. 54(5): 541-53.

Yadav, S., Schanz, R., Maheshwari, A., Khan, M.S., Slark, J., De Silva, R., Bentley, P., Froguel, P., Kooner, J., Shrivastav, P., Prasad, K. &Sharma, P. (2011). Bio-Repository of DNA in stroke (BRAINS): a study protocol. *BMC Medical Genetics*, 2: 12:34.

Yan, T., Li, Q., Chen, X. & Ye, T. (2016). Adverse effects of bone marrow stromal cell treatment of stroke in diabetic rats, *International Journal of Stroke,* 42(22): 3551–3558.

Yu, C., Ghen, Y. & Cline, G.W. (2002). Mechanisms by which fatty acid inhibits insulin activation of IRS-1associated phosphatidynositol 3 kinase activity in muscle. *Journal of Biological and Chemistry*, 277: 502390-502396.

Yu, D.T, Chae, J., Walker, M.E; Kirsteins, A., Elovic, E.P., Flanagan, S.R., Harvey, R.L., Zorowitz, R.D., Frost, F.S., Grill, J.H., Feldstein, M&Fang, Z.P. (2004). Intramuscular neuromuscular electric stimulation for poststroke shoulder pain: a multicenter randomized clinical trial. *Archives of Physical Medicine and Rehabilitation.* 85(5): 695-704.

APPENDIX A

INFORMED CONSENT FORM

My name is MrSuleAbdullahi Dambatta, of Department of Physical and Health Education, Ahmadu Bello University, Zaria.

This study entitiled '' Effects of Neuromuscular Stimulation and Cycling Exercises on Functional Abilities of Stroke Patients in Kano, Nigeria'' The study duration will be three times per week for 8 weeks.

The study is expected to find out the most important of the treatment methods to bring improvement to your affected hand and leg.

The risk associated with this study is very little and not beyond feeling of stretch on the muscles or some difficulty climbing to the cycle ergometer.

The benefit of this study if significant on any modality will help your condition have a better focus of treatment method and help hospitals and rehabilitation centres treat other patients of your type with a reference.

In participating for this study, your transportation fares and lunch will be given. And your confidentiality is secured.

It is fully voluntary and you have the right to withdraw at anytime from the study and I will appreciate should you sign below, if you accept to participate in the study.

You can communicate us if interested to know the result of the study or if published through our contacts. Phone: 081297973636 or hannyfortis@gmail.com.

---------- ----------

Abdullahi SuleDambatta Participants

Witness