EFFECTIVENESS OF SQUATTING AND TREADMILL EXERCISE ON CARDIOPULMONARY PARAMETERS IN POST STROKE PATIENTS ATTENDING FEDERAL MEDICAL CENTRE BIRNIN KUDU

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(B.Sc. PHYSIOTHERAPY)

A DISSERTATION SUBMITTED TO THE DEPARTMENT OF HUMAN PHYSIOLOGY, FACULTY OF BASIC MEDICAL SCIENCES, COLLEGE OF HEALTH SCIENCES, BAYERO UNIVERSITY, KANO

IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE AWARD OF THE DEGREE OF MASTER OF SCIENCE IN HUMAN PHYSIOLOGY

DECEMBER, 2015

DECLARATION

I hereby declare that this work titled “Effectiveness of squatting and treadmill exercise on cardiopulmonary response in post-stroke patients” is the product of my own research efforts undertaken under the supervision of Dr I. U. Yarube and has not been presented anywhere for the award of a degree or certificate. All sources of information have been duly acknowledged in the text.

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CERTIFICATION

This is to certify that the research work of this dissertation titled: “Effectiveness of squatting and treadmill exercise on cardiopulmonary response in post-stroke patients” and the subsequent preparations of this dissertation by Saude Adamu Usman (SPS/11/MHP/00001) were carried out under my supervision.

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APPROVAL

This is to certify that this dissertation by Saude Adamu Usman (SPS/11/MHP/00001) has been examined and approved for the award of the degree of Master of Science (Human Physiology) by Bayero University, Kano, for its contribution to knowledge and literary presentation.

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ACKNOWLEDGEMENTS

All praise is to ALLAH, the most merciful, beneficent, gracious and forgiving. My profound gratitude goes to my supervisor Dr. Isyaku Umar Yarube, for his guidance and constructive criticism throughout the course of my research and academic pursuit. The teachings on data analysis have been particularly useful. My profound gratitude goes to my internal examiner Prof. M. M. Khashab, and Dr. Salisu Ahmed.

My appreciation goes to my parents, brothers and sisters for giving me the opportunity to plant and nature seed of success in academia. Additional and special thanks to my mother, Habiba Sani. She is the secret behind my success in this life and the positive hope for hereafter. May Allah the Almighty continue to bestow his mercies on my dear mother, ameen. To my husband whose support during the course of this research has been immeasurable, may you remain blessed and loved.

My profound gratitude goes to the management and ethical committee of Federal Medical Centre, Birnin Kudu and my H.O.D Abdullahi Sani Danbatta. My special appreciation goes to Zubairu Usman the Head of Physiotherapy Department, Rasheed Shekoni Specialist Hospital Dutse, for their support during the course of my research. My profound gratitude goes to Salim Abubakar and Yunusa Abdullahi (of blessed memory) who also taught me how to analyse my data and made some useful suggestions toward a successful completion of this study. I will not rest my pen without appreciating those who assisted me during the course of data collection, Isah Abdullahi and Musa Dan'azumi. My gratitude goes to colleagues, classmates and friends such as Usman Zakari, Kani Adamu, Naimat Mustapha, Dr Umma Idris, Hindatu Muhammed, Dr Fatima Abbas, Dr Rukkaya Baballe, Zainab Umar, and others who are too many to mention, thank you all for your relentless assistance and

support. I will like to use this medium to appreciate the support and contributions given to me by the Head of the Department of Human Physiology, M. A. Salim and all the postgraduate lecturers. I also appreciate the role played by all the academic and non academic staff of the Department of Human Physiology, Bayero University, Kano. Lastly, thanks to each and every one who contributed to the development of this research work whose names or portfolios were not mentioned. Thank you all.

DEDICATION

I dedicate this work to all my loved ones.

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LIST OF ABBREVIATIONS

A = spastic recovery stage

A1 = spastic group receiving treadmill exercise

A2 = spastic group receiving squatting exercise

A3 = spastic group receiving combine treadmill and squatting exercise

B = relative recovery stage

B1 = relative recovery group receiving treadmill exercise

B2 = relative recovery group receiving squatting exercise

B3 = relative recovery group receiving combine squatting and treadmill exercise

BMI = body mass index

CP = cardiopulmonary

CV = cardiovascular

DBP = diastolic blood pressure

Exc. = exercise

FEV_1 = forced expiratory volume in one second

$FEV_1\%$ = (FEV_1/FVC) percentage of FVC expelled in one second

FVC = forced vital capacity

HR = heart rate

HR_{MAX} = heart rate maximum

HRR = heart rate recovery

SBP = systolic blood pressure

SES = socioeconomic status

SQ = squatting

TM = treadmill

ABSTRACT

Exercise capacity and CP fitness is seriously reduced after stroke, therefore functional recovery after stroke is a high priority for health care. This study aimed to determine the CP parameters in post stroke patients at the spastic and relative recovery stages, before and after 8 weeks of squatting and/or treadmill exercise regiments. Sixty (60) post stroke patients (34 males (56.7%) and 26 females (43.3%)) matched for age (30 - 70yrs), sex and BMI with mean age of 52.80, were randomly selected in to group A and B comprising of subjects in the spastic and relative recovery stages respectively. Each group was further divided in to subgroups 1, 2 and 3. Participants in the sub-group 1 (A1/B1) received treadmill exc, those in sub-group 2 (A2/B2) received squatting exc. and those in sub-group 3 (A3/B3) received combined treadmill and squatting exercise. All participants received 15 minutes exercise 3 times per week over a period of 8 weeks. Patients were assessed before exercise, at 4^{th} week and at 8^{th} week of exercise. Statistical analysis was performed using descriptive, and paired t–test. There was statistically significant improvement ($p<0.05$) in SBP, DBP, FVC, and FEV_1 in all the subjects, the 2 groups and subgroups, at week 4 and week 8 when compared to week 1 and FEV_1% remain within the range of 74-87%. HR increased slightly at the end of exercise in all the groups, but no significant increase seen in AT and BT. All the groups also showed improvement in HR recovery at week 8 when compared to week 1. Treadmill exercise at the doses provided places less CV stress on the patient and can improve CP response parameters of stroke patients in the spastic and relative recovery stages.

CHAPTER ONE

INTRODUCTION

1.1 BACKGROUND

Stroke is defined as a sudden focal or global neurological deficit lasting more than 24 hours or leading to death with no apparent cause other than a pathological process of blood vessel (Hatano, 1976; WHO, 1988). It is also defined as a condition in which the brain cells suddenly die because of a lack of oxygen (Stuart, 2008). This can be caused by an obstruction in the blood flow, or the rupture of an artery that feeds the brain. The two main types of stroke include ischemic and hemorrhagic stroke, ischemic stroke occurs due to occlusion in the blood vessel caused by either thrombus or embolus while hemorrhagic stroke occur due to ruptured blood vessel caused by hypertension, aneurysm, over treatment with anticoagulant. Both types of stroke result in a lack of blood flow to the brain and a build-up of blood that put too much pressure on the brain. The third type of stroke is called Transient Ischemic Attack (TIA) or mini stroke. Transient Ischemic Attack is known to be a temporary disruption of blood flow to the brain caused by a temporary occlusion as in ischemic stroke with focal or global neurological deficit lasting not more than 24hrs (Davies, 1985; Weiss, 2010). Depending on the severity of the stroke, the patient may suddenly lose the ability to speak (aphasia) or swallow (dysphasia), there may be memory problem or confusion, visual impairment in one or both eyes, weakness of one side of the face, weakness in an arm or leg or paralysis of the limbs on one side of the body, which can cause difficulty with several daily activities like walking, dressing, eating, etc (Davies, 1985; Weiss, 2010). Other complications include incontinence and bladder problem,

loss of balance and coordination, sensory impairments, coma and even death may occur in severe cases (Stuart, 2008).

1.2 STATEMENT OF THE PROBLEM

The prevalence of stroke in Nigeria is 1.14 per 1000 population (Wahab, 2008) and in the middle belt region of Nigeria is 1.31 per 1000 population (Sanya *et al.,* 2015). The use of treadmill and squatting exercise in rehabilitation of post-stroke patients in this part of the environment (Birnin kudu) is common, but the cardiopulmonary responses to these exercise regimens in both men and women has not been investigated or no documented findings has been seen. Majority of studies have only described the cardiopulmonary response of acute stroke and chronic stroke to separate aerobic exercises, but the combined effect of aerobic exercise (treadmill) and resistance exercise (squatting) training on the cardiopulmonary function in post stroke survivors at the spastic and relative recovery stage has received little attention. In addition, the effect of a prescribed 8-week regimen of the treadmill exercise and that of the squatting exercise on cardiopulmonary function in post stroke patients has not been evaluated in this environment.

1.3 AIM OF THE STUDY

The main aim of the study was to determine the cardiopulmonary responses in post-stroke subjects at the spastic and relative recovery stages, before and after an 8-week exercise intervention.

1.4 SPECIFIC OBJECTIVES OF THE STUDY

The specific objectives of the study are to determine:

1. HR, SBP, DBP, FVC and FEV_1 in post-stroke subjects before and after an 8-week exercise regimen.
2. HR, SBP, DBP, FVC and FEV_1 in post-stroke subjects before and after treadmill exercise.
3. HR, SBP, DBP, FVC and FEV_1 in post-stroke subjects before and after squatting exercise.
4. HR, SBP, DBP, FVC and FEV_1 in post-stroke subjects before and after combined squatting and treadmill exercise.

1.5 SIGNIFICANCE OF THE STUDY

The research will provide some baseline data on cardiopulmonary responses to treadmill and squatting exercise in post-stroke patients in our environment. The result of this study might guide clinicians in prescribing more effective exercise rehabilitation protocol for post-stroke patients resident in this environment.

1.6 RESEARCH HYPOTHESES

1. There is no difference in the CP parameters of post-stroke subjects after 8-week exercise regimens.
2. There is no difference in the CP parameters of post-stroke subjects after 8-week of treadmill exercise.

3. There is no difference in the CP parameters of post-stroke subjects after 8-week of squatting exercise.

4. There is no difference in the CP parameters of post-stroke subjects after 8-week of combined treadmill and squatting exercise.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

Stroke is one of the most common causes of long-term disability worldwide, and stroke patients need assistance with activities of daily living, therefore functional recovery after stroke is a high priority for healthcare (McArthur, Quinn, and Higgins, 2011). Recovery from stroke is challenging due to impaired neuromuscular control, decreased functional mobility, balance deficits and reduced cardiopulmonary (CP) fitness (Mackey-Lyons and Hewlett, 2005). Generally, respiratory muscle weakness is attributed to the impairment of the muscles involved in respiration, induced by central nervous system (CNS) lesions (Jandt, Caballero, and Junior, 2011). Sezer, Kutayordu, Sutbeyaz, and Koseoglu (2004) performed a study to evaluate the CP and metabolic responses to maximum exercise and aerobic capacity in 25 hemiplegic patients using upper limb cycle ergometer. The patients showed a significant respiratory dysfunction and reduced exercise tolerance when compared with age marched healthy control group. The result further suggests that a program of ventilatory and aerobic training should be considered early after stroke to avoid CP problems and deconditioning. However, aerobic exercise was found to be effective on aerobic capacity (VO_2 peak) regardless of the stage of recovery in post-stroke patients (Pang, Eng, Dawson, and Gylfadottir, 2006).

Emerging evidence suggests that exercise training in the post stroke population can facilitate improvement in the cardiovascular, respiratory and neuromuscular system (Macko, Ivey, and Forrester, 2005). For a variety of reasons, stroke survivors are not

routinely prescribed adequate aerobic and resistance exercises during stroke rehabilitation which likely exacerbates their decline in CP fitness (Mackey-Lyons and Markrides, 2002). Research studies have demonstrated not only improvement in maximum oxygen consumption (VO_2 peak) after exercise interventions (Macko *et al.,* 2005), but also physical function and psychological well-being or quality of life (Mead, Jones, and Jennings, 2007; Meek, Pollock, Langhorne, and Potter, 2002). Yet, many clinicians do not employ exercise interventions appropriately, perhaps because of the limited amount of research that has identified appropriate screening protocols and optimal dosing of aerobic and resistance exercises (Gordon, Gulanick, Costa, Fletcher, Franklin, and Roth, 2004). Cardiopulmonary fitness is important for performing daily activities and mobility. It has been reported that VO_2 peak values of 20 ml/kg/min is associated with limited physical function for instrumental activities of daily living (Letombe, Cornille, and Delahaye, 2010). Portempa Braun, Tinknell, and Popovich. (1996) observed that documented reduction in cardiovascular fitness may contribute to the increase risk of further stroke or myocardial infarction that stroke patients have in comparison with normal subjects. During exercise, there is a progressive increase in the systolic pressure with an increase in VO_2. Diastolic pressure shows little change with increasing levels of exercise (Franklin, Gordon, and Timmis, 1989; Weisman and Zeballos, 1994). If blood pressure falls as exercise intensity increases, a cardiac limitation or abnormality of sympathetic control of blood pressure such as heart failure, ischemia, aortic stenosis, pulmonary vascular disease or central venous obstruction should be suggested (ATS/ACCP, 2003). Graded treadmill exercise testing with proper safety precautions can be used to assess cardiopulmonary function in paretic stroke patients, also a simple floor walking test can be used to predict adequate neurological function to perform the treadmill exercise test (Macko,

Katzel, Yataco, Tretter, and DeSouza, 1997). It is important therefore, to provide a comprehensive description of cardiopulmonary responses during a squatting and treadmill incremental exercise test for 8 weeks in post stroke survivors ($\geq$ 1 month post stroke and less than 6 months post-stroke) at the spastic and relative recovery stages.

2.1.1 Increased Energy Expenditure and Limitation of Physical Exercise in Post- Stroke Patients

After stroke, people with weakness or paralysis enter a vicious cycle of limited activity and deconditioning that limits functional recovery and exacerbates cardiovascular risk factors (Boyne, Danning, Carl, and Gerson, 2013). Post stroke rehabilitation helps stroke survivors relearn skills that are lost when part of the brain is damaged. These skills include coordinating leg movements in order to walk or carry out the steps involved in any complex activity. The major rehabilitation goals for the stroke patient are preventing complication of prolonged in activity, decreasing the risk of recurrent stroke and cardiovascular events and increasing aerobic fitness. According to Monga, Deforge, and Williams *(*1988), stroke patients have been shown to achieve significantly lower maximal workload than control subjects during progressive exercise testing to volitional fatigue. Other studies which used various adapted ergometry devices or exercise protocols with smaller sample sizes yielded similar findings (Gordon *et al*., 2004; Hoskins, 1975). Oxygen uptake at a given submaximal workload in stroke patients is greater than in normal healthy subjects, possibly because of reduced mechanical efficiency, the effects of spasticity or both. In contrast, peak O_2 uptake is reduced in these stroke patients (Gordon *et al*., 2004). In a

randomized controlled trial that involved 42 hemi paretic stroke survivors, vigorous aerobic exercise training 3 times per week for 10 weeks evoked significant improvements in peak oxygen consumption and work load, submaximal exercise blood pressure response, exercise time, and sensorimotor function (Potempa, Lopez, and Braun, 1995). The findings demonstrated that stroke patients can increase their cardiovascular fitness by a magnitude that is similar to that of healthy older adults who engage in endurance training programs.

Regular aerobic exercise helps to prevent and treat cardiovascular disease (Lazarevic, Antic, vetkovic, and Vlahovic, 2006) and to prevent and reverse arterial stiffening (Tanaka, Dinenno, Monahan, and Clevenge, 2000). Resistance training is also an important physical activity that can prevent or treat lifestyle related disease (Eves and Plotnikoff, 2006) However, high intensity resistance training reduces arterial compliance and increases arterial stiffness (Ebenbichler, Strum, Ganzer, Bodner, Magweth, and Ritsch, 2001). Kawano, Tanaka, and Miyachi (2006) reported that aerobic exercise for 30 minute after resistance training (RT) prevents carotid artery stiffening caused by resistance training in young healthy men. Green, Walsh, Maiorana, Burke, and Taylor (2004), also reported that short term aerobic and resistance training improves endothelium dependent nitric oxide (NO) mediated vascular function in both conduit and resistance vessels, therefore, combined training can improve vascular function. On the other hand, high intensity resistance exercise promotes acute increases in blood pressure to a much larger degree than aerobic exercise (MacDougall, Tuxen, Sale, Moraz, and Sutton, 1985).

According to Miller, Raps, Howard, and Williams (1999), activity intolerance is common among stroke survivors especially in the elderly, such intolerance is likely due to several factors including bed rest induced deconditioning, concomitant left ventricular dysfunction, severity of neurological involvement (e.g flaccidity, spasticity of the extremity, impairment of sensory function of the involved side, impaired trunk balance, weakness of the upper or lower limb, aphasia and mental confusion) and the increased aerobic requirements of walking (Miller *et al*., 1999). According to Water and Yakura (1989), energy expenditure during gait in hemiplegic patients varies with the degree of weakness, spasticity, training and bracing but in general, the oxygen cost of walking (in ml/kg/min) is elevated in hemiplegic patients compared with that of able-bodied subjects of comparable body weight (Roth and Harvey, 2000). In some cases the debilitating motor effects of a stroke can markedly decrease mechanical efficiency and increase the energy cost of walking up to 2 times that of able-bodied person (Gordon *et al*., 2004). The effect of 6 months of treadmill moderate aerobic exercise with a graded treadmill for ambulation on the energy expenditure and cardiovascular demands was studied in stroke patients with chronic hemiparetic gait (Marko, 2001), the training elicited significant reductions in submaximal energy expenditure, these reductions were progressive with continued exercise training. The data suggest that aerobic exercise training and improved cardiovascular fitness might enable activities of daily living to be performed at a lower percentage of the aerobic capacity.

Even common household tasks such as bed making, is associated with considerably greater energy requirements among post stroke women than among their healthy counterpart (Gordon *et al*., 2004). The above mentioned variables can create a vicious

circle of further decreased activity and greater exercise intolerance, leading to secondary complications such as reduced cardiopulmonary fitness, muscle atrophy, osteoporosis, and impaired circulation to the lower extremities in stroke survivors (Billinger, Jordan, and Barbara, 2012). Physical activity places greater energy demands on the cardiovascular system of hemiplegic patients than on that of able-bodied subjects, stroke survivors with pre-existing cardiac disease may be at an increased risk for exertion–related adverse cardiac complications (Roth and Hervey, 2000). Pre-existing or post stroke cardiovascular conditions can delay or inhibit participation in a therapeutic exercise program, complicate the rehabilitation and long term post stroke course of care and limit the ability of the patient to perform functional activities independently (Roth, Mueller, and Green, 1988). Stroke patients have a known heightened risk of secondary cardiac complication and recurrent stroke which makes the post stroke period a particularly important time to implement stroke secondary prevention interventions (Roth and Noll, 1994; Wolf, Clagett, and Easton, 1999). An aerobic conditioning program can enhance glucose regulation and promote decrease in body weight and fat stores, also promote decrease in blood pressure (in hypertensive patient), level of total blood cholesterol, serum triglycerides, and low density lipoprotein cholesterol (Franklin and Sanders, 2000). Exercise also increases high-density lipoprotein cholesterol and improves blood rheology, hemostatic variable and coronary artery endothelial function (Hambrecht, Wolf, and Gielen, 2000).

2.1.2 Related Studies

According to Marko *et al*. (1997), the effect of 8-weeks aerobic exercise intervention in sub-acute stroke patients shows that moderate-high intensity aerobic exercise

appear to be beneficial for improving cardiovascular outcomes during submaximal performance of an exercise test. In another work by WilBorn, Greenwood, Wyatt, Bowden, and Grose (2004) conducted on subjects undergoing alternate inverted leg press and upright hack squat using 1RM (one repetition maximum) to grade the exercise intensity in both groups, the findings suggest cardiovascular measures are influenced by intensity of the exercise, rather than apparatus or change in body position during resistance exercise, suggesting that low intensity long duration resistance training elicits a greater systolic blood pressure response than high intensity short duration resistance training.

Kim, Park, and Yim (2014) observed a significant intergroup difference in the FVC, FEV and peak expiratory flow in subjects using an individualized respiratory device combined with other forms of conventional exercise than the conventional physical therapy exercise alone. In a research done by El-Nahas and Sayed (2012) to investigate the influence of early aerobic interval training (using treadmill) on ventilatory functions, walking speed and functional mobility in post-stroke patients, a statistically significant improvement of ventilatory functions and Rivermead mobility index (used for assessing walking speed and functional performance) was revealed. Yoo, Lim, Lee, and Kwon (2014) investigate the cardiovascular response of stroke patients to submaximal under water treadmill exercise and land treadmill exercise. The result shows that the mean maximum increase in BP and HR of underwater treadmill walking were significantly lower than that of land treadmill walking. Also, that the SBP, DBP, mBP decreased significantly after water immersion but HR was unchanged. The researchers believed that the water immersion may have reduced the workload of the cardiovascular system.

While it is known that cardiopulmonary (CP) fitness is reduced after stroke the majority of the studies to date have enrolled a higher proportion of men than women (Marko, 2001). Billinger *et al,* (2012) conducted a retrospective study to investigate the cardiopulmonary response and safety of exercise testing at peak effort in people at the chronic stage of stroke recovery. 62 patients (32 males and 30 female) participated in the exercise testing both males and females had low cardiopulmonary fitness levels. There was no significant difference found between the gender for peak heart rate or VO_2 peak, but males demonstrated higher values for minute ventilation, tidal volume and respiratory exchange ratio. In de-Almeida, Clementino, Rocha, Brandao, Dornelas and de-Andrade, (2011) findings right-sided hemiplegia exhibited greater impairment of the respiratory muscles than left-sided hemiplegia due to the physiologic positioning of the domes of the diaphragm. There is no significant difference seen in SBP, DBP, and HR for dorminant and nondominant side of brain lesion in hemiplegic after exercise (Hamzat and Alabi, 2006).

In a 12 weeks cycle ergometer training study, exercise capacity and self-concept improved in 7 hemiparetic stroke patients. However, there were no direct measurements of cardiopulmonary fitness and motor function improvements were characterized by subjective descriptions rather than standardized functional evaluations. Moreover, these findings support the use of regular aerobic exercise to improve cardiovascular health and fitness after stroke (Thompson, Buchner, and Pina, 2003). The relative cardiovascular stress of physical therapy and occupational therapy sessions in 20 patients undergoing a stroke rehabilitation program was evaluated by

MacKay-Lyons and Makrides (2002). The time per session in which the achieved heart rate was to be within the prescribed target heart rate zone was found to be extremely low, which suggest that these sessions elicited inadequate cardiovascular stress to induce a training effect. Roth, Mueller, and Green (1992) also found that activities that evoked the greatest heart rate increase were performed in the upright position and involved transitional movements.

In another study by Michael and Todd (2008) on exercise treadmill test and estimating cardiovascular prognosis in post-stroke patients, it was shown that abnormal hemodynamic (cardiovascular) responses to exercise treadmill testing may indicate an increased risk of coronary event and death even if evidence of ischemia is absent. In exercise treadmill test exercise duration, blood pressure, heart rate and heart rhythmic responses to exercise have prognostic significance. According to the authors, of the prognostic factors, exercise durations is the one most strongly associated with risk of coronary events and death, independent of age, sex, or known presence and severity of coronary artery disease. A decrease in blood pressure with exercise can reflect severe coronary artery disease or left ventricular systolic dysfunction.

Micheal and Todd (2008) further stated that a heart rate that does not increase adequately during exercise or does not recover rapidly after exercise is associated with an increased risk of death. Also, exercise testing may help to improve the prognosis of patients with an abnormal hemodynamic response to exercise caused by poor general health. In a study involving two 55 year old men without a history of coronary artery disease both undergo an exercise treadmill test to evaluate atypical angina. Both men

had normal results at rest on electrocardiography (ECG), heart rate (70b/m) and blood pressure (130/80mmHg). The duration for treadmill exercise test was 12 mins and 4 mins for patient A and B respectively peak exercise blood pressure was higher in patient A (210/70mmHg) than patient B (120/70mmHg). Both heart rates rise equally to 150 bm. No patient complained of chest pain. Patient 'A' experienced fatigue while 'B' had dyspnea. Drop in heart rate was faster in patient 'A' than patient 'B'. ST-segment depression was equal in both patients (1.5mm), the result shows that patient 'B' with decrease in peak exercise blood pressure is more likely to develop coronary artery disease (Arena, Myers, and Williams, 2007).

A stress test used diagnostically is considered to have a positive result if the patient develops signs and symptoms of ischemia during stress i.e. ST–segment depression and angina (Arena *et al.,* 2007). Treadmill exercise testing can be used both for diagnostic or prognostic purpose. Prognostic variables measured during exercise treadmill testing that predict outcomes are actually indicators of general fitness and function of the autonomic nervous system. They include; exercise duration, exercise hypotension, exercise hypertension, heart rate recovery, chronotropic incompetence (failure of heart rate to rise during exercise), and ventricular ectopy (Marko, Smath, Dobrovolny, Sorkin, Goldberg, and Silver, 2001). ST-segment depression and angina can also be used for prognostic purpose. The exercise duration covered by a patient in an exercise determines the functional capacity of that patient. Another way to measure functional capacity is to measure oxygen uptake during exercise (Arena *et al.,* 2007).

Rehabilitation programs designed to optimize functional motor performance in stroke survivors have incorporated aerobic exercise training with and without partial body weight-supported walking (e.g treadmill exercise) to improve strength and timing of muscle activation and cardiopulmonary fitness (Hesse, Bertelt, and Jahnke, 1995). Kurt, Evelyne, and Julie (1999), studied the effect of body weight supported treadmill training (BWSTT) on cardio respiratory fitness in 2 patients (man and women) recovering from severe traumatic brain injury found out that, after 2 to 3 session of BWSTT per week, both pre and post BWSTT aerobic capacity was measured for the 2 patients and both patients submaximal and peak responses improved. The responses include increase in total treadmill work/exercise duration, peak oxygen uptake, and estimated cardiac stroke volume.

In a study conducted by Takanobu, Mitsuliko, and Komei (2007), on the effect of aerobic exercise before and after resistance training (combined aerobic and resistance training), thirty three young healthy subjects were randomly assigned to groups that ran before resistance training (BRT; 4 males, 7 females), ran after resistance training (ART; 4 males, 7 females), or remained sedentary (SED; 3 males 8 female). The BRT and ART groups performed RT at 80% of one reputation maximum and ran at 60% of the targeted heart rate twice each week for 8 weeks. Both brachial ankle pulse wave velocity (BAPWV) and flow-mediated dilation (FMD) after combined training in the BRT group did not change from baseline. In contrast, BAPWV after combined training in the ART group reduced from baseline, moreover, brachial artery FMD after combined training in the ART group increased from baseline brachial artery diameter, mean blood velocity and blood flow in the BRT and ART group after combined training increased from baseline. These values returned to the baseline during the

detraining period. The values did not change also in the SED group. The result shows that performing aerobic exercise there after (ART) can prevent the deterioration of vascular function.

In a study by Hanson, Slance, Rueckert, and clerk (1995), on the cardiac response to squat the hemodynamic patterns in nine normal men and six heart transplant recipients during 2 minutes of squatting were examined to determine the role of cardiac innervation in the mediation of cardiac responses. Stroke volume was monitored by ensemble averaged thoracic impedance cardiography and blood pressure was determined with a plethysmograph. These techniques provided continuous measurements which were capable of detecting transient and non-steady state changes. Forearm blood flow was measured with venous occlusion plethysmography. Measurements were obtained after 3 minutes of quiet standing, immediately after squatting and at 20, 60 and 120 secs of sustained squatting. At the end both groups exhibited similar increases in stroke volume index (normal subject 10.5 ml/m^2; heart transplant recipients or subjects 10.3 ml/m^2) and mean arterial pressure (normal subjects 8.5 mmHg, heart transplants recipients 5.0 mmHg) which were sustained throughout squatting. Each group also showed an initial decreases in peripheral resistance (normal subject 3.6 unit; heart transplant recipient 7.7 units) followed by a return to baseline values after 20 seconds. Heart rate decreased in normal individuals (10 beats per min) but was unchanged or minimally increased (2 beats per min) in heart transplant recipients. Forearm vascular resistance was conspicuously decreased in normal individual (47.8 units) and not significantly in heart transplant recipients. The researcher concluded that the major haemodynamic responses to squatting (increase cardiac output and increase blood pressure) are similar in normal individuals

and heart transplant recipients. These responses are primarily due to augmented venous return and are not altered by cardiac denervation.

In a study performed by Vallejo, Schroeder, Zheng, Jensky, and Sattler (2006) on the cardiopulmonary responses of older adults to eccentric and concentric resistance exercise (using leg squats) for one week, it was reported that the cardiopulmonary responses were significantly lower during eccentric exercise than during concentric resistance exercise in older subjects. Eccentric resistance exercise was recommended for older persons with lower exercise tolerance and at risk of adverse cardiopulmonary events. Another research was conducted (Kaelin, Swank, Adams, and Barnard, 1999) to determine the safety of one repetition maximum (1 RM) testing using parallel squat and to determine the prevalence of abnormal cardiopulmonary responses, muscle soreness, and muscle injury among patients with chronic obstructive pulmonary disease (COPD). Repetition maximum is the maximal number of times a load can be lifted before fatigue using good technique of lifting, a "1 RM" signifies the maximum resistance a person can move in one repetition of exercise (American College of Sport Medicine, 1998). Blood pressure, heart rate, dyspnea rating and oxygen saturation responses were measured immediately following 1 RM procedure. Results showed no injury, no significant muscle soreness or abnormal cardiopulmonary responses occur as a result of the 1RM parallel squats among the patients. According to the researchers a properly supervised and screened pulmonary rehabilitation population can be 1 RM tested without significant muscle soreness, injury or abnormal cardiopulmonary responses.

2.2 EPIDEMIOLOGY

Cardiovascular disease (CVD) has been a major problem in the developed and developing countries and its burden in these countries is over whelming (Johnston, Mendis, and Mathers, 2009). Stroke is a common neurological disorder and is the second or third leading cause of death and a major cause of long term disability (Johnston *et al.,* 2009). In a study conducted by Mukadas and Misba'u (2009) on the incidence of CVD in the north-western Nigeria, it was observed that hypertension constitute 39.1% of the cardiovascular diseases. According to Adebayo (2013), findings from the experts in Nigeria shows that 160,000 Nigerians suffer stroke yearly and 80% of the cases are preventable circumstances. Also between 2010 and 2012, 272 stroke cases were recorded in National hospital Abuja occuring due to various risk factors like hypertension, diabetes, abdominal obesity and hyperlipidemia (Alkali, Bwala, Akano, Osi-Ogbu, Alabi, and Ayeni, 2013). In a four-year review of stroke admissions in tertiary institution in south western Nigeria by Olufemi, Kolawole, Binbo, Timothy, Olusegun, Adebowale, and Joshua (2011), a total of 101 patients who had stroke were admitted during the review period which accounted for 4.5% of medical admissions and 1.3% of total hospital admissions. According to Adeloye (2014), 19 studies from 10 African countries were selected to determine the prevalence and burden of stroke in Africa between 2009 and 2013, result shows that the burden of stroke in Africa is high and still increasing.

Stroke kills almost 130,000 Americans each year—that is 1 out of every 20 deaths (Centre for Disease Control (CDC), 2015; National Hospital Care Surveys (NHCS, 2015) on average, one American dies from stroke every 4 minutes (Mozaffarin,

Benjamin, Roger, Berry, Blaha and Go, 2015). Every year, more than 795,000 people in the United States have a stroke. About 610,000 of these are first or new strokes. About 185,000 strokes, nearly one of four are people who have had a previous stroke (Mozaffarin *et al.,* 2015). About 87% of all strokes are ischemic strokes, when blood flow to the brain is blocked (Mozaffarian *et al.,* 2015). Stroke costs the United States an estimated $34 billion each year (Mozaffarin *et al.,* 2015; Panel, Ralph, Benjamin, Emelia, Chairy, Joseph, 1997). This total includes the cost of health care services, medications to treat stroke, and missed days of work. Stroke is a leading cause of serious long-term disability (Mozaffarin *et al.,* 2015). Stroke is the fifth leading cause of death for Americans, but the risk of having a stroke varies with race and ethnicity. Risk of having a first stroke is nearly twice as high for blacks than for whites, and blacks are more likely to die following a stroke than are whites. (Howard, Evans, Crouse, Toole, Ryu, and Tegeler, 1994; Mozaffarin *et al.,* 2015). Hispanics risk for stroke falls between that of whites and blacks (Mozaffarin *et al.,* 2015). American Indians, Alaska Natives, and blacks are more likely to have had a stroke than are other groups (CDC, 2012). Although stroke risk increases with age, strokes do occur at any age. In 2009, 34% of people hospitalized for stroke were younger than 65 years (Hall, Levant, and DeFrances, 2012). Age-adjusted prevalence was higher among adults with a lower level of education compared with those with a higher level of education. From 2006 to 2010, no statistically significant change in stroke prevalence was observed among women or among any particular age group, race/ethnicity, or level of education. The country's highest death rates from stroke are in the south-eastern United States (Lloyd-Jones, Adams, and Brown, 2010).

2.2.1 Predisposing Factors of Stroke

The risk factors for stroke include modifiable and non-modifiable risk factors. Martins (2010), compiled 10 modifiable risk factors for stroke such as hypertension, current smoking, abdominal obesity, diabetes, lack of physical activity, poor diet, heavy alcohol drinking, heart disease, psychosocial stress/depression, and oral contraceptives. Transient ischemic attack has also been identified as a risk factor for stroke (Howard, 1994). The non-modifiable risk factors include age, gender, race, ethnicity and heredity. Of all age is the most important risk factor for stroke. This is because after the age of 55 years, every decade is associated with increase in blood pressure in both men and women (Brown, Whisnant, Sicks, and O'Fallon, 1996).

2.3 PATHOPHYSIOLOGY OF STROKE

2.3.1 Ischaemic Stroke

Ischaemic stroke can be thrombotic or embolic in origin. The common pathway of ischaemic stroke is lack of sufficient blood flow to perfuse cerebral tissue due to narrowed or blocked arteries leading to or within the brain (Dirnagl, Ladecola, and Moskowitz, 1999). Narrowing is commonly the result of atherosclerosis, the occurrence of fatty plaques lining the blood vessels. As the plaques grow in size the blood vessel becomes narrowed and the blood flow to the area beyond is reduced (Dirnagl *et al.,* 1999). Damaged areas of an atherosclerotic plaque can cause a blood clot to form, which blocks the blood vessel resulting in a thrombotic stroke (Doyle, Simon and Stenzel-Poore, 2008). In an embolic stroke, blood clots or debris from elsewhere in the body, typically the heart valves in rheumatic valves and atria in atrial fibrillation travel through the circulatory system and block narrower blood vessels

(Doyle, 2008). In the core area of a stroke, blood flow is so drastically reduced that cells usually cannot recover and subsequently undergo cellular death. The tissue in the region bordering the dead tissue known as the ischaemic penumbra is less severely affected. This region is rendered functionally silent by reduced blood flow but remains metabolically active. The ischaemic penumbra may undergo apoptosis after several hours or days but if blood flow and oxygen delivery is restored shortly after the onset of stroke, they are potentially recoverable (Liu, Levine, and Winn 2010). After seconds to minutes of cerebral ischaemia the ischemic cascade is initiated in the core area. This is a series of biochemical reactions in the brain which usually goes on for two to three hours, but can last for days even after normal blood flow returns (Woodruff, Thundyil, and Tang, 2011). The important steps of the ischaemic cascade are as follows:

- Without adequate blood supply and thus lack of oxygen, brain cells lose their ability to produce energy in the form of Adenosine Triphosphate (ATP).
- Cells in the affected area switch to anaerobic metabolism, which leads to a lesser production of ATP and the release of lactic acid.
- Lactic acid is an irritant which has the potential to destroy cells by disruption of the normal acid-base balance in the brain.
- ATP reliant ion transport pumps fail, causing the membrane to become depolarized due to large influx of ions e.g calcium.
- Intracellular calcium levels become too high and trigger the release of the excitatory amino acid neurotransmitter glutamate.
- Glutamate further increases the permeability of the membrane to influx of calcium.
- Excess calcium entry over excites cells and activates proteases (enzyme which digest cell proteins), lipases (enzyme which digest cell membrane lipids), and free radicals.

- As the cell membrane is broken down by phospholipases, it becomes more permeable and more ions and harmful chemicals enter the cell.
- Mitochondria break down, releasing toxins and apoptotic factors in to the cell. The cell experience apoptosis.
- If the cell dies through necrosis, it releases glutamate and toxic chemicals in to the environment around it. The toxins poison nearby neurons and glutamate can over excite the neurons.

2.3.2 Haemorrhagic Stroke

Haemorrhagic strokes are due to rupture of a blood vessel leading to compression of brain tissue from an expanding haematoma (Testi and Aiyagari, 2008). This can distort and injure brain tissue in addition the pressure may further lead to loss of blood supply to the affected tissue. Intracerebral haemorrhage is caused by rupture of a blood vessel and accumulation of blood within the brain arising secondary to hypertension, arteriovenous malformations etc. Subarachnoid haemorrhage occurs due to gradual collection of blood in the subarachnoid space of the brain dura secondary to trauma or ruptured aneurysm (Panel, Ralph, Benjamin, Emelia, Chairy, Joseph, Broderick, MarkDyken, Larry, Goldstein, and George 1997).

2.4 EFFECT OF STROKE ON THE HEART AND LUNGS

The major consequences of a stroke are neurological changes, but cardiac problems may also complicate a stroke patient's recovery (Chalela, Julio, Ezzeddine, and Danis, 2004). In the days immediately following a stroke, heart attack, congestive heart failure, abnormal heart rhythms and cardiac arrest are all much more likely to occur

(Rosalyn, 2008). The abnormal heart rhythms include atrial fibrillation and atrial flutter. Both conditions are dangerous, as the upper chambers of the heart are pumping ineffectively, blood pools in the atria sticking on the atrial wall, which may lead to blood clots formation. If clot moves in to the blood stream, they may end up in the coronary arteries (where they could cause heart attack) or the brain (where they could cause another stroke) (Maramattom and Boby, 2006). The chemicals formed in the brain caused by stroke cascade and released in to the bloodstream may be bad for heart functioning (Prosser, Jane, MacGregor, Lees, Diene, and Hacke, 2007). A stroke can directly damage parts of the brain that control the heart. Right hemispheric damage (more than left) makes serious heart rhythm problems and death caused by the heart suddenly stopping much more likely (Myers, Norris, Harchinski, Weingert, and Sole, 1982). Some of the same risks that can lead to a stroke can also make heart problems more likely, including high blood pressure, diabetes, high cholesterol, coronary artery disease and arrhythmias (Panel *et al.,* 1997). Acute stroke may cause cardiac arrhythmias and myocardial cell damage through stroke induced increases in sympathetic tone (Myers *et al.,* 1982). The results of studies by (Roth, 1994, Di Pasquale, Andreoli, and Pinelli, 1986; Moldover, Daum, and Downey, 1984) support the concept that asymptomatic ischemic heart disease is often associated with cerebrovascular disease, and an active investigation of the heart should be considered in cerebrovascular patients in order to plan an optimal, comprehensive rehabilitation program.

It is commonly accepted that the motor cortical representation of the diaphragm and intercostal muscles is bilateral, and thus that these muscles are little influenced by unilateral corticospinal lesions (Sezer *et al.,* 2004). However, it has been shown (Simolowski, Straus, and Attali, 1996; De troyer, De Beyl, and Thirion, 1981;

Przedborski, Brunko, Hubert, Mavroudakis, and De Beyl, 1988; Annoni, Ackermann, and Kesselring, 1990) that hemiplegia due to cerebrovascular disease affects the function of the respiratory muscles on the paralyzed side. Chest roentgenograms of stroke patients showed an elevation of the diaphragm on the affected side (Simolowski *et al*., 1996; Roth, 1994). Furthermore, surface electromyographic recordings of respiratory muscles showed reduced amplitude on the paralyzed side during voluntary hyperventilation (De troyer *et al.,* 1981; Przedborski *et al.*, 1988). Recently, (Simolowski *et al*., 1996; Khedr, El-Shinawy, Khedr, Abdel-aziz, and Awad, 2000) found that stroke patients had abnormal magnetic evoked potentials (MEPs), cortical latency and central conduction time (CCT) of the affected hemisphere, and no bilateral motor representation of each hemidiaphragm. Khedr *et al,* (2000) also demonstrated a significant association between the degree of respiratory dysfunction and MEPs and CCT of the affected hemisphere. In individual case studies, abnormalities of respiration may be associated with small, discrete lesions of the central nervous system, defined by imaging or post mortem, particularly due to stroke (Bianchi, Denavit-Saubie, Champagnat, 1995). Hemispheric ischemic stroke influences respiratory function to a modest degree. Reductions of both chest wall and diaphragm excursion contralateral to the stroke have been reported (Jandt *et al.,* 2011). The latter association correlates well with the localization of the diaphragm cortical representation found by trans-cranial magnetic stimulation and positron emission tomography scanning (Similowski, 1995). Yet there is no clear evidence of cerebral dominance for diaphragm function. Patients with bilateral hemispheric cerebrovascular disease (CVD) show an increased respiratory responsiveness to CO_2 and are liable to develop Cheyne - Stokes respiration suggesting disinhibition of lower

respiratory centre (Koo, Strohl, Gillombardo, and Jacono, 2010). Such a response may persist months to years after the stroke.

Diffuse cortical vascular disease may also lead to a selective inability of voluntary breathing (respiratory apraxia) (Howard, Rudd, Wolfe, and Williams, 2001). Also intermittent upper airway obstruction and apnoea due to periodic fluctuations in the position of the vocal cards is associated with cortical supranuclear palsy due to bilateral lesions of the operculum (Besson, Bogousslavsky, and Regle, 1991). The effect of brainstem dysfunction on respiration, depend on the pathology, localization and speed of onset of the lesion. In patients with bulbar lesions, particularly vascular, the combination of impaired swallowing, abnormalities of the respiratory rhythm reduced vital capacity and reduced or absent triggering of cough reflex all increases the risk of aspiration pneumonia (Miller *et al*., 1999). Nocturnal upper airway occlusion may also contribute to respiration impairment. Unilateral or bilateral tegmental infarcts in the pons (at or below the level of the trigeminal pontine nucleus) may lead to apneustic breathing and impairment of CO_2 responsiveness (Stewart, Howard, and Rudd, 1996). Also similar lesion in medulla may result in acute failure of the automatic respiration (Bogousslavsky, 1990). Infarction of the basal pons or of the pyramids and the adjacent ventromedial portion of the medulla may lead to complete loss of the voluntary system with a highly regular breathing pattern but a complete inability to initiate any spontaneous respiratory movements (Bogousslavsky, 1990; Balami, Chen, and Buchan, 2013). Acute vascular lesions in the lower brainstem compromise respiratory control particularly during sleep leading to irregularities of rate and rhythm of breathing which lead to Cheyne–Stokes respiration, hypopnea and obstructive apnoea, it is likely that size and bilaterality of

the lesions determine the type and severity of abnormalities of the respiratory pattern. In a series of 15 patients with vascular lesions of the brainstem, patients with unilateral lesions in the rostrolateral medulla showed reduced ventilator sensitivity to inhaled CO_2. In these patients there was a minimal effect on breathing while awake, at rest or during exertion however, there was a high incidence of fragmented sleep and obstructive sleep apnoea associated with hypoxemia. The authors concluded that patients with unilateral rostrolateral medullary lesions require monitoring during sleep to diagnose sleep apnoea (Howard *et al*., 2001).

Cardiopulmonary complications and recurrent stroke are leading causes of mortality in the stroke population (Gordon *et al*., 2004). Stroke patients have partial or total weakness of the diaphragm, and intercostal and abdominal muscles on the affected side (Annoni *et al*., 1990; Roth and Noll, 1994; Similowski *et al*., 1996; Lanini, Bianchi, Romagnoli, Coli, Binazzi, and Gigliotti, 2003). The major function of expiratory muscles is to generate a forceful and effective cough (Park, Kang, Lee, Choi, and Kim, 2010). Expiratory muscle weakness results in an ineffective cough, retention of secretions, and inability to maintain a clear airway. This may lead to pneumonia and microatelectasis. Pneumonia is estimated to occur in about one third of stroke patients and is a major cause of morbidity and mortality in these subjects (Harvey, Roth, and Yu, 2007). Weakness of the respiratory muscles results in changes in the lungs and thorax (reduction in lung and thorax compliance) which may lead to a severe restrictive ventilatory impairment (Hart, and Nickol, 2002). Restrictive ventilatory impairment may lead to hypoventilation and hypoxemia (Sezer *et al*., 2004). Both decreased motion and paradoxical motion of the affected half of the rib cage have been described in patients with stroke (Fugl-Meyer and Grimy, 1984).

Furthermore, the efficiency of the unaffected muscles may be decreased due to instability of the chest wall and an inactive lifestyle. A larger hemidiaphragmatic excursion on the unaffected side was observed in these patients (Khedr *et al.,* 2000). Respiratory dysfunction, due to reduced vital capacity (VC), inspiratory capacity (IC), total lung capacity (TLC), forced expiratory volume in 1 sec (FEV1), forced vital capacity (FVC), forced expiratory flow (FEF) 25-75%, maximum voluntary ventilation (MVV), peak expiratory flow (PEF), and expiratory reserve volume (ERV), has been reported after stroke (Roth and Noll, 1994; Annoni *et al*., 1990; Khedr *et al*., 2000; Sezer *et al*., 2004). Peak oxygen consumption VO_2, the criterion measure for exercise capacity, is poor in the stroke population (Potempa, 1996; Arsura, 2005; Pang, Eng, and Dawson, 2005; Rimmer and Wang, 2005; Macko *et al*., 2005; Courban, Calmels, Roche, Ramas, and Fayolle-Minon, 2006). Peak VO_2 in individuals with stroke has been shown to be as low as 50-70% of that age and gender matched in value to sedentary individuals (Mackay-Lyons and Makrides, 2004; Eng, Dawson, and Chu, 2004; Gordon *et al*., 2004). The impairment in cardiopulmonary fitness is related to a combination of physiologic and environmental factors (Kelly, Kilbreath, Davis, Zeman, and Raymond, 2003). Physiologic factors include the loss of strength and coordination, resulting in a reduction in the number of recruitable motor units, and diminished capacity for oxidative metabolism in paretic muscle tissue (Potempa *et al*., 1996). Environmental factors that may contribute to impairment in fitness include bed rest and physical inactivity after the stroke (Mackey, Ada, Heard, and Adams, 1996).

HR is increased at low work rates in stroke patients (Sezer *et al.,* 2004). Decreased cardiovascular fitness, disuse atrophy, weakness, and respiratory dysfunction in stroke

patients may be responsible for this condition (Sezer *et al.,* 2004). Low exercise capacity has been related to an increased risk of various forms of cardiovascular disease in these individuals (Pang *et al.,* 2005). Poor cardiorespiratory fitness has also been linked to a higher risk of stroke and stroke mortality (Arsura, 2005; Pang *et al*., 2005). Stroke is associated with cardiac complications such as cardiac arrhythmias and ischemic heart damage (Katz-Leurer, Shochina, Carmeli, and Friedlander, 2003; Harvey *et al.,* 2007). Cardiac disease has been reported to occur in up to 75% of stroke survivors (Roth, 1993). Both coronary artery disease (CAD) and ischemic stroke, share links to many of the same predisposing, potentially modifiable risk factors such as hypertension, abnormal blood lipids and lipoproteins, cigarette smoking, physical inactivity, obesity, and diabetes mellitus (Roth, 1993; Pearson, Blair, Daniels, Eckel, Fair, and Fortmann, 2002; Goldstein, Adams, Alberts, Appel, Brass, and Bushnell, 2006). Many patients with clinically apparent or silent myocardial ischemia have coexistent cerebrovascular disease. Conversely, many patients with cerebrovascular disease have varying degrees of CAD (Sirna, Biller, Skorton, and Seabold, 1990). Accordingly, recurrent stroke and cardiac disease are the leading causes of mortality in stroke survivors (Gordon *et al.,* 2004).

2.5 EFFECT OF STROKE ON DIFFERENT PARTS OF THE BRAIN

The brain has 3 primary parts, which include the cerebrum, the cerebellum, and the brainstem. Each is responsible for different functions. The brain receives its blood supply from the cycle of Willis formed by 2 internal carotid arteries and 2 vertebral arteries (McMinn, 1997). Blockage or rupture of the vessel at any part of the brain gives a typical presentation due to damage of brain cells in that region (Ledingham

and Warrell, 2000). The cerebrum is the largest; it controls a number of higher functions including speech, memory, learning and the integration of sensory stimuli, and initiation of the final common pathways for movement and fine control of movement. The cerebrum is divided in to a left and a right hemisphere. The left hemisphere controls the majority of functions on the right side of the body and vise versa. Each cerebral hemisphere is composed of the frontal, parietal, temporal and occipital lobes. The frontal lobes consist of prefrontal cortex in front, which is responsible for behaviour and emotions. The back of frontal lobe, consist of the pre-motor and motor areas which produce and modify movement. Damage to this area alters their functions (Moore and Dalley, 1999). The left and right parietal lobes contain the primary sensory cortex which controls sensation (touch and pressure) and a large association area that controls fine sensation (judgement of texture, weight, size, and shape). Damage to right parietal lobe can cause visuo-spacial deficits, making it harder for patient to find his or her way around new or even familiar places. Damage to the left parietal lobe may disrupt a patient's ability to understand spoken and/or written language (Moore and Dalley, 1999). The left and right temporal lobes allow a person to differentiate smells and sounds. They are also responsible for short term memory. The right temporal lobe is primarily involved in visual memory (memory for faces and pictures). The left temporal lobe is primarily involved in verbal memory (memory for word and names). The occipital lobe processes visual information, damage to this lobe can cause visual deficits.

The cerebellum is the second largest area of the brain; it controls reflexes, balance, equilibrium, muscle tone, and coordination of movement. A stroke involving the cerebellum may result in a lack of coordination (ataxia), clumsiness and balance

problems, shaking or other muscular difficulties. This can interfere with a person's ability to walk, talk, and eat (Haslett, Chilvers, John, and Boon, 1999). The brainstem is responsible for a variety of automatic functions that are critical to life, such as breathing, digestion, heartbeat, alertness and arousal. Brainstem stroke is fetal and most devastating and life threatening because they can disrupt the involuntary functions essential to life. People who survive may remain in a vegetative state or be left with severe impairments (Haslett *et al.,* 1999).

2.6 TISSUE-LEVEL ABNORMALITIES AFTER STROKE

There are number of major peripheral changes in skeletal muscle that could propagate disability and contribute to low fitness levels in stroke apart from the brain injury, this include gross muscular atrophy, fibre phenotype shift and associated insulin resistance (Ivey, Hafer-Markeo, and Macko. 2006). The amount of metabolically active tissues accounts in part for how much oxygen a person can utilize. Numerous body composition and hemiparetic muscle tissue abnormalities in chronic stroke contribute to poor cardiovascular health and insulin resistance. These tissue level changes may be primary targets for exercise intervention strategies. Following stroke, gross hemiparetic skeletal muscular atrophy and increased intramuscular area fat are present (Ryan, Dobrovolny, Smith, Silver, and Macko, 2002). Elevated intramuscular fat is associated with insulin resistance and its complications (Ryan *et al.,* 2002). Cellular changes in tissues of the paretic side may also negatively influence fitness, function, and cardiovascular disease (CVD) risk. Specifically, a deficit severity-dependent shift toward a fast-twitch muscle molecular phenotype in the paretic leg results in a more fatigable muscle fibre type that is more insulin resistant, which may contribute to the

high incidence of impaired glucose tolerance (IGT) in this population (Deyne, Hafer-Macko, Ivey, Ryan, and Macko, 2004) (Jakobsson, Edström, Grimby, and Thornell, 1991). Fibre phenotype shift was seen in a myosin heavy chain (MHC) gel electrophoresis of paretic leg muscle biopsies in 13 stroke patients showed elevated proportions of fast type ll fibres as compared with the non-paretic leg muscle. These findings contrast with the relatively equal proportions of slow and fast MHC found in the vastus lateralis of individuals without stroke (Landin, Hagenfeldt, Saltin, and Wahren, 1977; Deyne *et al*., 2004). Furthermore, nearly threefold elevated levels of tumor necrosis factor-α (TNF-α) messenger RNA are reported in hemiparetic quadriceps muscle from patients with stroke compared with those from nonparetic legs and nonstroke control subjects (Hafer-Macko, Yu, Ryan, Ivey, and Macko, 2005). Elevated TNF-α level in skeletal muscle tissue are strongly linked to muscular wasting and insulin resistance in T2DM and advancing age and may contribute to the structural and metabolic abnormalities in hemiparetic skeletal muscle after stroke (De Alvaro, Teruel, Hernandez, and Lorenzo, 2004; Saghizadeh, Ong, Garvey, Henry, and Kern, 1996). Since exercise training has been shown to reduce skeletal muscle TNF-α expression, which improves muscle strength and metabolic function in selected nonstroke populations (Greiwe, Cheng, Rubin, Yarasheski, and Semenkovich, 2001), also modification of inflammatory pathways through exercise therapy warrants consideration in the high CVD risk stroke population. Increased stiffness in the paretic planter flexors has been reported to occur as early as 2 months (Ivey, Hafer-Marko, and Marko, 2008), although it is likely to occur much earlier, therefore stretching pre exercise is necessary to preserve muscle length.

2.7 STAGES OF STROKE RECOVERY

Immediately after the onset of stroke there is a stage of cerebral shock with flaccidity and areflexia. Gradually, this is replaced by development of spasticity, hyper-reflexia and abnormal mass movement pattern which is termed as synergy. The duration of flaccidity may vary from days to weeks to infinite. In some cases signs of mild to severe spasticity may be seen in certain groups of muscles like shoulder depressor, shoulder retractors, shoulder internal rotators and adductors, elbow flexors, wrist flexors, forearm pronators, hip extensor, adductors and internal rotators, knee extensors, ankle and toes planter flexors. The stroke recovery stages have been classified into 5 stages by Bronstrum (Bennett and Karnes, 1998) as follows;

Stage 1: Flaccid and no movements.

Stage 2: Spasticity begins and basic limb synergies may be seen as associated reaction.

Stage 3: Limb synergies are produced voluntarily i.e. spasticity increases.

Stage 4: Some movement combination not belonging to synergy may appear, spasticity start declining.

Stage 5: Spasticity disappears, isolated movements are possible and coordination can become normal.

This classification is further simplified by Bobath into only three stages (Davies, 1992) as follows;

Stage 1: Flaccid

Stage 2: Spastic

Stage 3: Spontaneous/Relative recovery

Thus, it is clear that 95% of hemiplegics show spontaneous recovery although the degree of the recovery may vary depending upon various factors and the progress many become stagnant at any point in time (Davies, 1992). Physiotherapy treatment of stroke patient begins from the acute stage, subacute stage, to the chronic stage through the use of various forms of exercises such as passive mobilization, assisted-active exercises, active and resisted exercises. Treadmill running, cycle ergometry, weight lifting, and squatting are some of the active-resisted exercises used in physiotherapy to rehabilitate a stroke patient (Stuart, 2008).

2.8 SQUATTING

Squat (SQ) exercises are motion that human body uses often in real life. Squat is defined as to sit in a low or crouching position with the legs drawn up closely beneath or in front of the body i.e. to sit on one's heels. Squatting is a way or act of exercising by repeatedly assuming a crouching position with the knees bent and the buttocks near the heels (American Heritage, 2011). There are different variant of squatting, squatting can be deep (full) or shallow (half), Squat exercise can be done in almost any location with or without the use of weights or equipment. Adding weights (like dumbbells, barbell etc.) to the squat routine engages the muscles of the upper body essentially giving someone a full body work out in one exercise (Dadoly, 2011). Wall-bar supported squat allows the person's to hold a static bar while squatting to support the weight of the upper body. Squatting is generally used in strength training and fitness (Dadoly, 2011). The movement begins from a standing position with or without weight by moving the hips back and bending the knees and hips to lower the

buttocks near the heels. Squat exercises are great for a total lower body work out. They effectively work most of the major muscle groups of the buttocks, hips and thighs (Stuart, 2008). During wall-bar supported squatting, hip knee and ankle anti-gravity muscles are trained to work cooperatively, concentrically and eccentrically to raise and lower the body mass. Forces are distributed evenly over the three joint in an upright position while resistance is supplied by the body mass on rising up from the squat position (Bloomquist, Langberg, Karlsen, Madsgaard, and Boesen, 2013). Half-squatting is practiced by reaching down to pick up or touch a target object, with the target object placed on a stool of varying height. Doing squat exercise help to tighten and lift the buttock, it increases joint flexibility since the ankles, knees, hips and lower back are all utilized in squatting motion. Squat exercise also strengthens the abdominal and back muscles which are needed to keep balance during the movement (Dadoly, 2011). In healthy subject squatting produces a prompt increase in cardiac output and arterial blood pressure which is accompanied by an immediate decrease in heart rate and forearm vascular resistance. The rise in cardiac output and blood pressure has been attributed to augmented venous return from compression of leg veins, while the decrease in heart rate and forearm vascular resistance are probably due to activation of cardiopulmonary and arterial baroreflexes (Philips, Marchand, and Scheen, 2011).

Plate I: Photograph of subject performing squatting on wall-bar exercise. (Obtained from www.internationalphysicaltherapycentre.com).

2.8.1 Muscles Involved in Squatting

Squat positions are important for some daily activities like toileting, female or male urination, and labour (Brown, 2000). The primary muscles of squatting include sartorius, vastus intermedius, vastus medialis, vastus lateralis, rectus femoris, biceps femoris, semi-tendinosus, semi-membranosus, gluteus maximus, and erector spinae (Clarkson and Gilewich, 1999). Synergy muscles include transverse abdominus, gluteus medius, adductors, soleus, and gastrocnemius. Squat exercise helps in strengthening majorly the primary muscles (Kellis, Arambatsi, and Papadopoulos, 2005).

2.9 TREADMILL

Treadmill (TM) training is a task-oriented exercise model that assumes multiple physiological systems integral to improving mobility recovery and optimizing cardiovascular health after stroke (Ivey *et al*., 2008). The treadmill is a versatile exercise machine that lets users walk, jog and run on flat or uphill (inclined) surfaces while standing on the same place. Treadmills are widely used as exercise machines for medical and non-medical purposes. The machine provides a moving platform with a wide conveyor belt driven by an electric motor (electric treadmill) or a flywheel (manual treadmill). The belt moves to the rear, requiring the user to walk or run at a speed matching that of the belt. The rate at which the belt moves is the rate of walking or running thus, the speed of running may be controlled and measured. Medical treadmills are used for therapeutic and diagnostic purposes. The electric treadmills deliver mechanical energy to the human body through the moving running belt of the treadmill. The subject is passively moved and forced to actively catch up with the

running belt underneath his feet. The medical treadmill provide a safety harnesses and active measuring devices when connected through an interface electrocardiogram, ergospirometry, blood pressure monitor, heart rate monitor etc. (O'Connor, Fitzgerald, Cooper, Thorman, and Boninger, 2002). Treadmill provide effective cardiovascular workout and it is known for its cardiovascular health benefits (Elizabeth, Chumanov, Christa, Wille, Max, and Michalski, 2012). In addition, it also helps strengthen the muscles of the legs and buttocks.

Plate II: Photograph of subject performing treadmill exercise. (Obtained from www.amazon.com).

2.9.1 Muscles Involved in Treadmill Exercise

The muscles involved during treadmill exercise include quadriceps, hamstrings, iliopsoas, calves, glutes, and abdominal muscles (Elizabeth *et al.,* 2012). People who walk and jog on the treadmill regularly gain endurances and strength in the quadriceps. The quads are responsible for knee extension while walking on a treadmill. Hamstrings lie on the posterior thigh; they are used for bending the knees while walking. Treadmill exercise improves the endurance and strength of the hamstrings. The calf muscles also work hard during treadmill exercises. According to a study from the laboratory of medical physics in the Netherlands, most of the work performed by the calf muscles occurs as one pushes off their toes while walking, running, and jumping. Also, the workload on the calf muscles and hamstrings can be increased by increasing the incline on the machine (Rob, 2004; Matte, 2013). The glutes make up the muscles of the buttocks, they help during walking and running. Inclining the treadmill increase the workload on the glutes significantly which help increase the strength, improve endurance and burn body fatt. While walking, jogging, or running the rectus abdominus which is the main abdominal muscles that run down the front of the trunk, contracts to keep the trunk and pelvis in an upright erect position (Nunley, 2014). The rectus abdominis work together with the erector spinae to keep the trunk erect. The rectus abdominus and the erector spinae contractions during treadmill exercise is not enough to improve the strength of the muscles (Nunley, 2014). According to a 2002 study published in the journal of physiology the rectus abdominus is also involved in helping the respiratory cycle during running (Fuller, Megan, Jason, Ralph, Abraham, and Ferhgold, 2002).

2.10 TYPES OF EXERCISE

Physical exercises are generally grouped in to 3 types depending on the overall effect they have on the human body (NIH and NHLBI, 2006). They include aerobic, anaerobic and flexibility exercise.

Aerobic exercise is any physical activity that uses large muscle groups, and cause the body to use more oxygen than it would while resting (NIH and NHLBI, 2006). The goal of aerobic exercise is to increase the cardiovascular endurance otherwise called endurance training (Wilmore and Knuttgen, 2003). Aerobic exercise requires large amount of oxygen to obtain the energy needed for prolonged exercise. Examples of aerobic exercise are cycling, swimming, brisk walk, skipping rope, rowing, hiking, jogging, soccer, tennis, badminton and dancing.

Anaerobic exercise is also called strength or resistance training and can strengthen and tone muscles as well as improve bone strength, balance, and coordination (NIH and NHLBI, 2006). It uses the muscles at high intensity and a high rate of work for a short period. Body obtains energy by burning glycogen stored in the muscles without oxygen. Anaerobic exercises include pushups, lunges, bicep curls using dumbbells, weight training, functional training, eccentric training, sprinting, etc (DesVos, *2*005).

Flexibility exercises stretch and lengthen muscles (NIH and NHLBI, 2006). The goal of flexibility exercise is to improve the range of motion which can decrease the chance of injury (O'Connor, *2*005).

Base on the type of muscular contraction or joint motion Sembulingam and Semblingam (2010), categorised exercise in to 2 types; isotonic and isometric exercise. Isotonic exercise is also called dynamic or cardiovascular or endurance exercise, it can be eccentric or concentric isotonic exercise. It involves movement of the joints and muscle contractions in the arms and legs. In this type of exercise, the heart rate, force of contraction, cardiac output and systolic blood pressure increase. However, the diastolic blood pressure is unaltered or decrease. The reason is because; the peripheral resistance is unaltered or decrease depending upon the severity of exercise. Patients with heart conditions might not be able to do very high intensity isotonic exercises like basketball, competitive swimming, running and cycling because of its effect on the cardiovascular system. Isotonic exercises are aerobic exercises.

Isometric exercises called static or resistance or strength exercises do not involve joint movement. This type of exercise put strain on the heart and blood vessels in people with heart conditions. The exercise, include weight lifting, rock climbing, boxing, wrestling, pushing heavy object etc. During this exercise, apart from increase in heart rate, force of contraction, cardiac output and systolic blood pressure, the diastolic blood pressure also increases. It is because of increase in peripheral resistance during static exercise. Isometric exercises are anaerobic exercise.

2.11 NEURAL CONTROL OF MUSCULAR ACTIVITY

Muscle contraction is initiated by the nervous system. The nervous system comprises of the central nervous system (CNS) and the peripheral nervous system (PNS). The

PNS contains the sensory nerves which transfer information from the body receptors to the CNS, and motor nerves which transport information from the CNS to the muscle fibres. The nerve cell called a neuron is the basic unit of the nervous system. A neuron is composed of a body and projections. The shorter projections are called dendrites; the long one is the axon. Through the dendrite the neuron is able to obtain information from other neurons. The axon then passes the processed information to other cells by spreading the information along the neuron through changes in the voltage in the cell membrane. Once the action potential has reached the end of an axon, a mediator substance e.g acetylcholine is released to complete the transfer of information from one cell to another (Barrett, 2010). The neural control of muscular activity is divided into somatosensory system and somatomotor system. The somatomotor system controls the activities of skeletal muscles which are concerned with posture and movement. This system is constituted by somatic motor nerve fibres that conduct impulse from higher centre to the muscle fibres to initiate contraction. The impulse from the peripheral motor nerve fibre, meet the skeletal muscle fibre at the neuromuscular junction. The binding of the mediator (acetylcholine) to the receptors on the skeletal muscle fibres bring about another action potential which spreads along the muscle cell membranes leading to excitation contraction coupling. The movements of the body depend upon the different groups of skeletal muscles. The voluntary actions and the postural movements are carried out by not only the simple contraction and relaxation of skeletal muscle but also the adjustments of tone in these muscles. The execution, planning, coordination and adjustments of movements of the body are under the influence of different parts of the nervous system, which are together called the motor system. The motor system includes fibres from the spinal

cord and it nerves, cranial nerves, brainstem, cerebral cortex, cerebellum and basal ganglia (Sambulingam and Sambulingam, 2010).

2.12 CARDIOVASCULAR RESPONSES TO EXERCISE

The heart rate increases during exercise mainly because of vagal withdrawal and increase in sympathetic tone. According to Sembuligam and Sembulingam (2010), four factors are responsible for increase in the heart rate during exercise; impulse from proprioceptors in the muscles act through higher centres and increase the heart rate, hypercapnia acts through medullary centres, rise in body temperature acts on cardiac centres via hypothalamus, rise in body temperature also stimulate the SA node directly, circulating catecholamine secreted in large quantity during exercise cause increase in heart rate. Cardiac output increases up to 20 L/min in moderate exercise and up to 35 L/min during severe exercise. The cardiac output increases because of increase in heart rate and stroke volume. Stroke volume increases due to increase in force of cardiac muscle contraction. The venous return increases remarkably during exercise because of muscle pump, respiratory pump, and splanchnic vasoconstriction. During moderate isotonic exercise, the systolic pressure is increased. It is due to increase in heart rate and stroke volume. Diastolic pressure is not altered because peripheral resistance is not affected during moderate isotonic exercise. In severe exercise involving isotonic muscular contraction, the systolic pressure enormously increases but the diastolic pressure decreases. The decrease in diastolic pressure is because of the decrease in peripheral resistance. Decrease in peripheral resistance is due to vasodilatation caused by metabolites. During exercise involving isometric contraction, the peripheral resistance increases. So also, the diastolic pressure along

with the systolic pressure increases (Sabry, 2011). There is a great increase in the amount of blood flowing to skeletal muscles during exercise. The reasons are increase in sympathetic cholinergic fibres activity in the muscles and vasodilator functions of some metabolites like hypercapnia, hypoxia, potassium ions, lactic acid, rise in temperature, and adrenaline (Sembulingam and Sembulingam, 2010).

2.12.1 Blood Pressure (BP)

Blood pressure sometimes referred to as arterial blood pressure, is the pressure exerted by circulating blood upon the walls of the blood vessels (McGhee, 2002; Giang and Tran, 2014). During each heartbeat, blood pressure varies between a maximum (systolic) and a minimum (diastolic) pressure. During systole, there is contraction of the cardiac muscle and pumping of blood from the heart through arteries. During diastole, there is relaxation of cardiac muscle and filling of blood (Sembulingam and Sembulingam, 2010). The BP in the circulation is principally due to the pumping action of the heart (McGhee, 2002; Giang and Tran, 2014), differences in mean BP are responsible for blood flow from one location to another in the circulation. The rate of mean blood flow depends on both blood pressure and the resistance to flow presented by the blood vessels. Mean blood pressure decreases as the circulating blood moves away from the heart through arteries and capillaries due to viscous losses of energy (Klabunde and Richard, 2005). Gravity affects blood pressure via hydrostatic forces (during standing) and valves in veins. Breathing and pumping from contraction of skeletal muscles and pumping of the lungs also influence blood pressure in veins (Klabunde and Richard, 2005). Blood pressure is under control of the nervous and endocrine systems and it varies in healthy people. BP that is

abnormally low is called hypotension, and that which is abnormally high is called hypertension. Both have many causes and can range from mild to severe (Chobanian, 2003). According to the American Heart Association (Aronow, Fleg, Pepine, Artinian, Bakris, and Brown, 2011), for adults who are 18yrs and older blood pressure has been classified in to;

Hypotension - systolic <90mmHg, diastolic <60mmHg.

Normal - systolic 90 - 119mmHg, diastolic 60 - 79mmHg.

Prehypertension - systolic 120 - 139mmHg, diastolic 80 - 89mmHg.

Mild hypertension - systolic 140 - 159mmHg, diastolic 90 - 99mmHg.

Moderate hypertension - systolic 160 - 179mmHg, diastolic 100 - 109mmHg.

Severe hypertension - systolic >_180mmHg, diastolic >_110mmHg.

Systemic arterial pressure is closely related to the product of cardiac output and systemic vascular resistance (Sobel and Roberts 1988). Hypertension is defined as a chronic elevation of arterial blood pressure. In adults blood pressure over 140/90 mm Hg is generally accepted as hypertension (Whiteson, 2007; Victor, 2008). Chronic hypotension, with systolic blood pressure in the range of 85 to 110mm Hg, and without hemodynamic abnormalities such as syncope, is not pathological (Whiteson, 2007; Sobel and Roberts, 1988). Chronic idiopathic hypotension, vasoactive drugs, disorders of the peripheral, autonomic or central nervous system, cardiovascular deconditioning, hypovolemia, and anemia are the causes of orthostatic hypotension (Sobel and Roberts, 1988). During exercise, there is a progressive increase in the systolic pressure with an increase in VO_2. Diastolic pressure shows little change with increasing levels of exercise (Franklin, Gordon, and Timmis, 1989; Weisman and

Zeballos, 1994; ATS/ACCP 2003). Korotkoff technique of measuring BP with sphygmomanometer and stetoscope remains porpular but the use of automated digital BP monitors have been validated to some extend (Shevchenko, and Tsitlik, 1996; El-Feghali, Topouchian, Pannier, ElAssad, and Asmar, 2007).

2.12.2 Heart Rate (HR)

Heart rate is the number of contractions of the ventricles per unit of time, and is usually expressed as beats per minutes (Anna, 2013). In clinical practice the normal range of the resting heart rate in adults is regarded as between 60 and 100 beats per minutes as measured by palpation of the pulse (Anna, 2013). Heart rate is subjected for variation during normal physiological conditions such as exercise, emotions etc however, under physiological conditions, the altered heart rate is quickly brought back to normal. It is because of the perfectly tuned regulatory mechanism in the body through the nervous system (Sembulingam and Sembulingam, 2010). Heart rate training zone has been categorised in to 60% – 75% easy, 75% - 85% moderate and 85% - 95% hard (Fishpool, 2002). Exercise testing can be terminated when subjects reach an estimated or arbitrary percentage of their age-predicted maximal HR (HR_{max}) (ACSM, 2000). HR_{max} is also used as a criterion for achieving peak exertion in the determination of maximum Aerobic capacity. Maximum HR was estimated at 220 minus the participants age, the difference between HR at rest and HR maximum can be used to calculate 50% or 85% heart rate reserve for subsequent training (ACSM, 2000).

2.13 RESPIRATORY RESPONSES DURING EXERCISE

Pulmonary ventilation is the amount of air that enters and leaves the lungs in one minute. It is the product of tidal volume and respiratory rate. Sembulingam and Sembulingam (2010) reported that during exercise, hyperventilation, which includes increase in rate and force of respiration, occurs. In moderate exercise, respiratory rate increases to about 30/min and tidal volume increases to about 2000 mL. Thus, the pulmonary ventilation increases to about 60 L/min during moderate exercise. In severe muscular exercise, it rises still further up to 100 L/min. The thought or anticipation of exercise (psychic phenomenon) increase the rate and force of respiration due to activation of the higher centres, like sylvian cortex and motor cortex of the brain. Exercise induced hypoxia and hypercapnea stimulate the chemoreceptors to send impulse to the respiratory centres which in turn increase the rate and force of respiration. Proprioceptors activated during exercise send impulse to the medullary respiratory centre to increase the breathing rate. Also rise in body temperature during exercise directly stimulate the respiratory centres (Barrett, 2010). During exercise blood flow through the pulmonary capillaries is increased. As a result of this, the diffusing capacity of alveoli for oxygen is increased from 21 ml/min at rest to 45 ml/min during moderate exercise. During exercise, oxygen consumed by the tissues particularly skeletal muscles is greatly increased because of vasodilatation in the muscles which increase the amount of blood flow and oxygen.

2.13.1 Maximum Oxygen Consumed (VO_2)

VO_2 max is the amount of oxygen consumed under maximal aerobic metabolism. It is the product of maximal cardiac output and maximal amount of oxygen consumed by

the muscle. In a normal active and healthy male, the VO_2 Max is 35 - 40 mL/kg body weight/min. In female, it is 30 - 35 mL/kg/min. There is an increase of VO_2 Max by 50% during exercise (Sembulingam and Sembulingam, 2010). According to Ivey, Hafer-Marko, and Macko (2006) retrospective findings on 131 chronic hemiparetic stroke patients showed a mean peak aerobic capacity (VO_2 peak) of 13.6 ± 4 ml/kg/min during progressive graded treadmill walking. The peak O_2 consumption rate in the chronic hemiparetic stroke group was roughly half that of age-matched healthy control group (range of 25 - 30 (ml/kg)/min). These low VO_2 peak levels compromise functional mobility after stroke.

2.13.2 Respiratory Muscle Functions

The diaphragm, external intercostals, and the parasternal part of the internal intercostals and scalene are essential muscles of respiration because these are active even during quiet breathing in normal individuals. The sternocleidomastoid, pectoralis, trapezius, latissimusdorsi and serratus anterior muscles are accessory inspiratory muscles. They are inactive during quiet breathing in normal subjects but help to elevate the thoracic cage during strenuous circumstances (Celli, 1994; De Troyer, 1993). Expiration is passive during normal quiet breathing. Active expiration occurs during exercise, speech, singing, the expiratory phase of coughing or sneezing, and in pathologic states such as chronic bronchitis. The main muscles of expiration are the muscles of the abdominal wall, including the rectus abdominis, the external and internal oblique muscles, the transversusabdominis; and the internal intercostal muscles (Levitzky, 2007). Maximum respiratory pressures (PI max, PE max) and maximal voluntary ventilation (MVV) are used for respiratory muscles evaluation. PI

max and PE max measure respiratory muscle strength. The lower limits of normal maximal inspiratory pressure are 75 cm H_2O for men and 50 cm H_2O for women. The lower limits for maximal expiratory pressure are 100cm H_2O for men and 50cm H_2O for women (ATS/ACCP 2003). MVV is often used as an index of inspiratory muscle endurance that approximates the maximal ventilation during exercise (VE max). The reduction in MVV and in turn, VE max may lead a ventilatory limitation to maximal oxygen transport (VO_2) and exercise capacity (Ries, 1994).

2.13.3 Exercise Capacity

Exercise capacity is the maximum amount of physical exertion that an individual can sustain before exhaustion. Exercise performance reflects a coordinated response of cardiovascular, pulmonary, neuromuscular and musculoskeletal functions during the activity. Reduced exercise capacity may indicate dysfunction in any portion of the complex exercise response (Walker, Hall, and Hurst, 1990). Maximum oxygen uptake (VO_2) is the best index of exercise capacity and the gold standard for cardiopulmonary function or physical fitness (Franklin, 1989; ATS/ACCP 2003, Weisman and Zeballos, 1994). Genetic factors, age, gender, body size and muscle mass, habitual level of activity, and physical conditioning and training are the main determinants of normal VO_2 (ATS/ACCP 2003; Franklin, 1989). VO_2 can increase from a resting value of about 3.5 ml/ minute to 30-50 ml/min/kg during exercise in normal subjects (ATS/ACCP 2003). A reduced VO_2 is a general indicator of reduced exercise capacity and functional reserve capacity of the cardiovascular and respiratory systems. Exercise capacity is mainly limited by cardiovascular, respiratory and peripheral factors such as neuromuscular or musculoskeletal abnormalities (ATS/ACCP 2003;

Weisman and Zeballos, 1994). Both the timed walk test (6 or 12 min) and cardiopulmonary exercise testing (CPET) are used for evaluation of exercise capacity and functional impairment (Shah, Delisa, and Gans, 2005). Oxygen uptake (VO_2), carbon dioxide exhaled (CO_2), minute ventilation (VE), heart rate (HR), electrocardiography (ECG) ,blood pressure, respiratory rate (RR), respiratory exchange ratio (RER), the ratio of physiologic dead space to tidal volume (VD/VT), oxygen saturation (SaO_2), anaerobic threshold (AT) and power output (PO) can be monitored during the CPET.

2.13.4 Ventilation

Ventilation is defined as the movement of air from outside to inside the body into the gas exchange units of the lungs. Ventilation of the lungs allows exchange of gas between blood and atmospheric air (Murray, Wyngaarden, Smith, and Bennett, 1992). In normal adults, the resting minute ventilation (VE) of 5 to 6 L/min can be increased to as much as 150 L/min during short periods of maximal exercise (Franklin, 1989). Increased ventilation during exercise is associated with an increase in both depth and frequency of breathing. During a low level of exercise, increases in ventilation are accomplished primarily by increases in tidal volume. As exercise progresses, both tidal volume and frequency increase until 70 to 80% of peak exercise. Further increases in ventilation result solely from increase in frequency (ATS/ACCP 2003; Weisman and Zeballos, 1994). Maximal ventilation and resting ventilation do not appear to be affected by physical training, but ventilation at submaximal loads is decreased (Levitzky, 2007).

2.13.5 Vital Capacity (VC)

Vital capacity is the maximum volume of air that one can exchange in a single breath or the maximum volume of air that can be expelled out forcefully after a deep (max.) inspiration (Simon, Michael, Chinchilli, Phillips, Sorkness, and Lemanske, 2010). In normal adults, vital capacity values ranges between 3 to 5 liters increases with height, decreases with advanced age, and increases with pulmonary health and overall fitness (Pellegrino, Viegi, Brusasco, Crapo, Burgos, Casaburi, and Vander, 2005). VC is decreased in restrictive lung disease but normal or slightly decreased in obstructive lung disease, FVC, FEV_1 and FEV_1% are important outcomes measured to distinguish COPD from restrictive lung disease (Al-Ashkar, Mehra, and Mazzone, 2003). Spirometer is used to measure VC and lung volumes.

2.13.6 Forced Vital Capacity (FVC) and Forced Expiratory Volume in one Second (FEV_1)

Forced vital capacity is the volume of air that can forcibly be blown out from the lungs after full inspiration and it ranges from 2800 ml to 5000 ml (Perez, 2013). FEV_1 (timed vital capacity) is the volume of air that can forcibly be blown out in one second after full inspiration, or the volume of air expelled in the first second of a forced expiration (Stanojevic, Wade, and Stocks, 2008). Healthy individuals expel about 75% to 85% of their FVC in the first second of breath (Al-Ashkar *et al*., 2003; Stanojevic *et al*., 2008). A value of 83% is considered normal (Sembulingam and Sembulingam, 2010). Restrictive lung disease is characterized by a decrease total lung capacity (TLC) which is normal or increased in obstructive lung disease due to air trapping (Al-Ashkar *et al*., 2003). In restrictive lung disease **FEV_1** is normal or minimally

decreased and the FVC is decreased, leaving a normal or even increased FEV_1/FVC ratio (Bateman, Hurd, Barnes, Bousquet, O'Byrne, and Pederson, 2000; Al-Ashkar *et al*., 2003). This is because the lungs are restricted from fully expanding making it difficult to take a full breath with reduced airflow in to the lung, the restrictive factor may be intrinsic (e.g pneumonia, tuberculosis), extrinsic (e.g scoliosis, kyphosis, rib fracture) and neurological (e.g paralysis of diaphragm, myasthenia gravis, Gullein Barren Syndrome) (Al-Ashkar *et al*., 2003). In restrictive diseases (such as pulmonary fibrosis) the FEV_1 and FVC are both reduced proportionally and the FEV_1% value may be normal or even increased as a result of decreased lung compliance (Reynolds, Goldman, and Ausiello, 2008; Fauci, Braunwald, Kasper, Hauser, Longo, and Jameson, 2008). FEV_1 in obstructive lung disease (e.g chronic bronchitis, asthma, emphysema) is decreased, **FVC** is usually normal or slightly decreased due to the premature closure of airway in expiration and the FEV_1/FVC ratio is decreased (Al-Ashkar *et al*., 2003; Langhammer, Johnsen, Gulsvik, Holmen, and Bjermer, 2001). The decrease in FEV_1 is as a result of increase in airway resistance causing the air in the lung to be exhaled at a slower rate and in smaller amount compared to normal healthy person (Al-Ashkar *et al*., 2003).

2.13.7 Forced Expiratory Ratio (FEV_1%)

FEV_1% (FEV_1/FVC) it is the percentage of FVC expelled in the first second of a forced expiration (Olofson, Bake, and Tengelin, 2008). In healthy adults this should be approximately 70% to 85% (declining with age) (Simon, 2010). In patients with obstructive lung disease FEV_1/FVC decreases and can be as low as 20-30% in severe obstructive airway disease while restrictive disorders have a near normal FEV_1/FVC

(Nathell, Nathell, Malmberg, and Larsson, 2007). According to the Global Initiative for Obstructive Lung disease FEV_1% value of less than 69% indicate obstructive lung disease is highly likely and a value of 85% or greater is suggestive of restrictive lung disease (Al-Ashkar *et al*., 2003).

2.14 SOCIOECONOMIC STATUS (SES)

Social status means rank or position that one holds in a group or society (American Psychological Association, 2007). SES is an economic and sociological combined total measure of a person's work experience and of an individual's or family's economic and social position in relation to others, based on education, occupation and income (Galobardes, Shaw, Lawlor, Smith, and Lynch, 2006). Education refers to level of literacy; occupation refers to profession or skill; and income refers to wages, salaries, profit, rents, pension etc (American Psychological Association, 2007). These three factors determines where or status to place an individual. The three SES include high, middle and low. Low income families or individual focus on meeting immediate needs and do not accumulate wealth that could be passed on to future generation (Gary, McMillan, Jones, and Ainley, 2000). High income families with higher and expendable income can accumulate wealth and focus on meeting immediate needs while being able to enjoy luxuries. The middle income families focus on meeting immediate needs while trying to accumulate little wealth but not able to enjoy luxuries (American Psychological Association, 2007). Higher levels of education are associated with better economic status example those with degree makes better earning than those with diploma (Gary *et al*., 2000). Professional and skilled occupations are associated with better economic status example doctors, lawyers,

professors, engineers makes better earning than teachers, farmers, cooks, helpers, maids, cleaners etc (American Psychological Association, 2007).

CHAPTER THREE

MATERIALS AND METHODS

3.1 STUDY AREA

Federal Medical Centre, Birnin-kudu was used as the study area. It is located in Birnin-kudu local Government area of Jigawa state. The hospital is a tertiary institution with multiple speciality units including Physiotherapy unit, which served as the centre for the study. The number of new stroke cases seen in the physiotherapy unit per week ranges from 2 to 5 patients. The number of follow-up per day in Physiotherapy Department is about 15 to 20 patients. Most of the patients came from Dutse, Jahun, Gumel, Ringim, Birnin kudu, Gwaram local governments, and neighbouring states like Bauchi and Kano. They predominantly belong to the Hausa and Fulani tribes.

3.2 STUDY POPULATION

The study population included all stroke patients attending Physiotherapy clinic in Federal Medical Centre, Birnin kudu.

3.3 STUDY DESIGN

The study was a randomized clinical trial (RCT), eligible patients were those within the age range of 30 to 70 years, with post-stroke duration of not less than one month and not more than six month, and placed on exercise regimens as part of the routine post-stroke physiotherapy for the first time. In addition, the following inclusion and exclusion criteria were considered.

The inclusion criteria: Single episode of hypertensive stroke at least 1 month post-stroke; Controlled blood pressure; ability to sit and stand; independent with ambulation can cover > 50 yards (45 m) with or without an assistive device (Billinger *et al.,* 2012); patients in the spastic or relative recovery stages.

The exclusion criteria: Patients with cardiac or other conditions that are absolute contraindications for exercise (e.g. acute cardiac failure, renal failure); patients in the flaccid stage of stroke recovery; patients with history of tuberculosis or any chronic pulmonary condition; patients with unstable blood pressure, cognitive impairment.

3.4 SAMPLING TECHNIQUE AND GROUPING

Stratified random sampling using balloting was employed to select patients in to group A, which comprised of patients in the spastic recovery stage and group B comprised of patients in the relative recovery stages. Each group consisted of 30 subjects (MacKay-Lyons and Makrides, 2002; Kim *et al.,* 2014; Billinger *et al.,* 2012). Barthel index scale for assessing functional mobility, modified Asthword scale for grading of spasticity, and Bobath stroke recovery staging were used to select both patients in the spastic and relative recovery stages. Each group consisted of patients with both left-sided and right-sided hemiplegia matched for age, sex, and BMI (BMI was calculated to select patients with BMI less than 30kg/m^2, using the formula: BMI = wt/h^2 in kg/m^2). Each group was further divided in to sub-group 1, 2, and 3 consisting of 10 subjects each. The patients in the sub-group 1 were subjected to treadmill exercise

(AT/BT), subgroup 2 to squatting exercise (AS/BS), and subgroup 3 to combined squatting and treadmill exercise (AC/BC).

3.5 LIST OF EQUIPMENTS

The following instruments were used for the research purpose:

- Motorized Treadmill (American fitness YK-07360) was used for walking.
- Wall bars (Wooden locally made by Alhassan furnitures, zoo road Kano) was used to assist subject in squatting.
- Sphygmomanometer (Accusson W421, UK) and Stetoscope (3M Littmann Cardiology III Stetoscope, US)
- Omron mz arm monitor (HEM 7102-E) was used to measure HR.
- Spirolab DATOSPIR (Sibelmed model120A,) was used to measure the forced vital capacity and forced expiratory volume in 1 sec (FVC, FEV1).
- Others include: Harson weighing scale (Model: H89 RED) used to measure weight of the each subject; Standiometer (seca) used to measure height of each subject; the formula BMI = wt/h^2 (kg/m^2) to measure body mass index of each subject; Stopwatch used to monitor time for the exercise.
- Questionneire

3.6 PROCEDURE FOR EXERCISE AND DATA COLLECTION

1 Patients were screened according to the selection criteria after the pre-intervention clinical examinations that were performed by specialised physicians before referring the patient for physiotherapy. Recorded demographic characteristics

and clinical information include gender, age, BMI, affected body side, type of stroke, socioeconomic status (SES), functional mobility and medications.

2 Prior to commencement of the study, subjects had a full description for the exercise testing protocol. The subject were instructed to abstain from consuming heavy food and caffeine 3 hours prior to the scheduled test, also to avoid vigorous physical activity for 24 hours prior to the exercise testing.

3 At study entry the researcher familiarized each subject with the equipments, procedures of performing the exercise, by performing the squat or walk test for 1 minute in front of the subjects, and explained the Borg-rating of perceived exertion (RPE) to the participants.

4 The temperature was maintained at room temperature. The exercise test was terminated if the subject met absolute test termination criteria. Emergency stop key was attached to the patient's shirt to act as safety switch (for automatic stop if the patient cannot maintain the walking speed) while walking on the treadmill.

5 The procedure for treadmill exercise sessions began with phase-1 stretching of the quadriceps, hamstring muscles and gluteus to ensure less risk of injury and a better chance of performing exercise. Subject then stood on the treadmill conveyor belt holding the hand rails, as the motor start rolling, subject took a minutes' walk warm-up (phase-2) on the treadmill at a speed of 0.03 km/h after which the exercise intensity was increased gradually to the prescribed speed of 0.1 km/h (minimum speed required to maintain 50 - 85% of maximum heart rate) for 13 minutes (phase-3/active phase), then a minute walk cool-down (phase-4) was employed before terminating the exercise. Adherence to normal gait pattern was emphasized through visual observation to ensure that heel strike, swing phase and stance phase were performed near normal.

6 The procedure for squatting exercise session was also divided into 4 phases, it started with stretching (phase-1) of the quadriceps, hamstring muscles and gluteus to ensure less risk of injury and a better chance of performing the proper technique. Warm-up (Phase 2) began with half squats for a duration of one minute followed by the active phase (phase-3) where subjects were instructed to perform the full squat continuously for 13 minutes with a minimum of 2 to 3 squat per minute, the 4^{th} phase (cool dawn) immediately follows with a half squat which lasted for a minute. The squat technique was instructed as follows:

- Stand with your feet hip width apart with hands holding the wall bars;
- Tighten and pull in your abdominal muscles;
- Lower your body as if you are going to sit in a chair (the motion maybe slow or fast);
- Stop when your legs are parallel to the floor, or when the buttocks come in contact with a reference point device;
- Stay in this position for a few seconds, then press down onto your heels and slowly rise back up to a standing position with the arms supporting the weight of the upper body by grasping the wall bars.

7 Participants in the treadmill, squatting and combined treadmill-squatting groups completed 8-weeks exercise intervention for 15 minutes duration 3 times per week.

8 Cardiopulmonary (CP) response parameters were measured before the commencement of exercise in the first week, then after exercise at the fourth week and eight week. Post exercise vital signs were measured to ensure a return to pre-exercise values. Measurements were taken at rest before the commencement of exercise, at the peak of the exercise and 5 and 20 minute after the exercise.

9 The exercise parameters included systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate, (HR), forced vital capacity (FVC), and forced expiratory volume in one second (FEV_1).

10 Automated digital arm BP monitor was used to measure the heart rate, with the patient seated, relaxed and not speaking, the monitor which was already neatly placed on the exposed left arm and supported at the level of the heart was turned on by selecting the appropriate button, which automatically allows the cuff to inflate and deflate. The monitor could detect the heart rate as the cuff deflates and displays the measurement on a screen.

11 Sphygmomanometer was used to measure the blood pressure (SBPand DBP), immediately after taking the HR, arm cuff connected to the sphyg was neatly placed round the left arm about 2.5 - 5 cm above the cubital fossa with the center of the bladder over the brachial artery. Stetoscope bell was placed over the brachial artery below the anteriocubital space before inflating the cuff. The cuff pressure was gradually released by slowly opening the air release knob. The first sound was recorded as the SBP and DBP was recorded when the final muffled sound disappear.

12 Spirometer "DATOSPIR 120" was used by the researcher to measure the FVC, and FEV_1 in accordance with the recommendations of the Datospir user manual. Patients' demographic and anthropometric characteristic was assessed before applying ventilatory function test. Subjects seated before the measurement, after appropriate placement of nose clip and mouth piece each subject was asked to do a forced quick expiration after maximum inhalation and end the manoeuvre with inhalation. After doing at least three acceptable and repeatable FVC manoeuvres the largest FVC and FEV_1 were recorded (Farrell 1981, Ghai 2007) and calculate the FEV_1/FVC ratio. All measurements were taken with the help of well-trained assistants.

3.7 ETHICAL CONSIDERATIONS

The study was conducted in accordance with the institutional guidelines after the approval from the Federal Medical Centre institutional ethical committee. An informed written consent was obtained from each subject after they were informed about risk and benefits of participating in the research.

3.8 DATA ANALYSIS

Data were collated and analysed using the SPSS version 20.0 statistical software (IMB Inc., Chicago, U.S.A). Student's t-test was used to compare mean values between any two groups. Values are expressed as mean ± S.E.M. P values < 0.05 were considered significant.

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 RESULTS

4.1.1 Socio-demographic Characteristics of Participants

- A total of sixty (60) patients with stroke (34 males (56.7%) and 26 female (43.3%)) participated in the study as shown in figure 4.1.

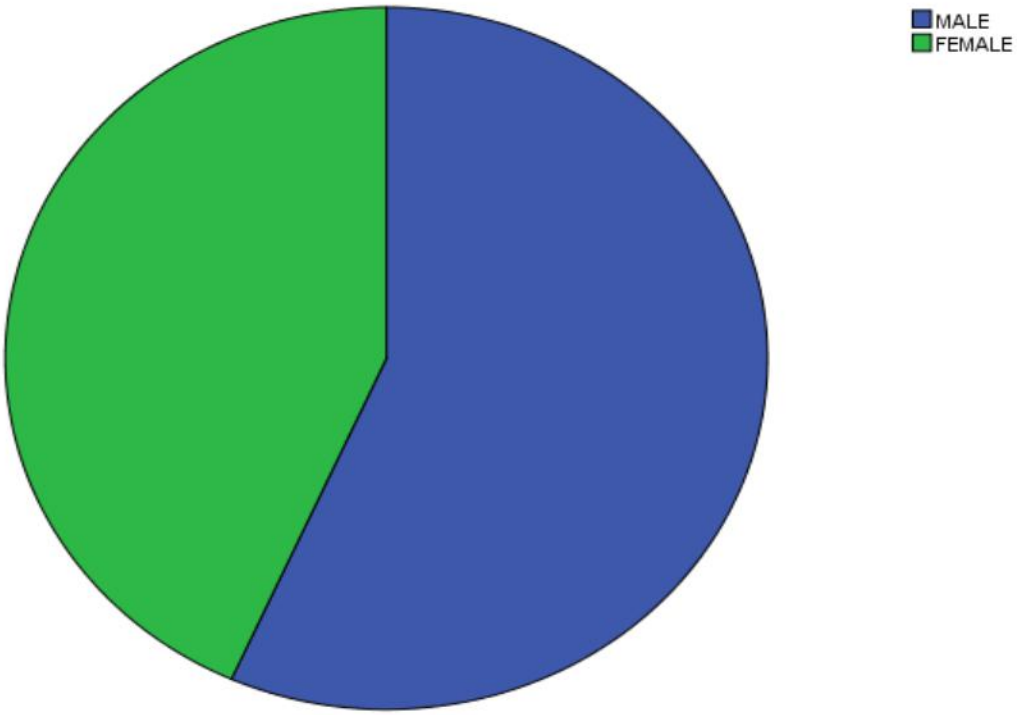

Figure 4.1: Distribution of the subjects by sex.

- The mean age of the participants was 52.80 years, males had 55.62 years and females had 49.12 years, those in the spastic stage and relative recovery stage had mean age of 52.63 and 52.97 years, respectively. Figure 4.2 shows the mean age of subjects.

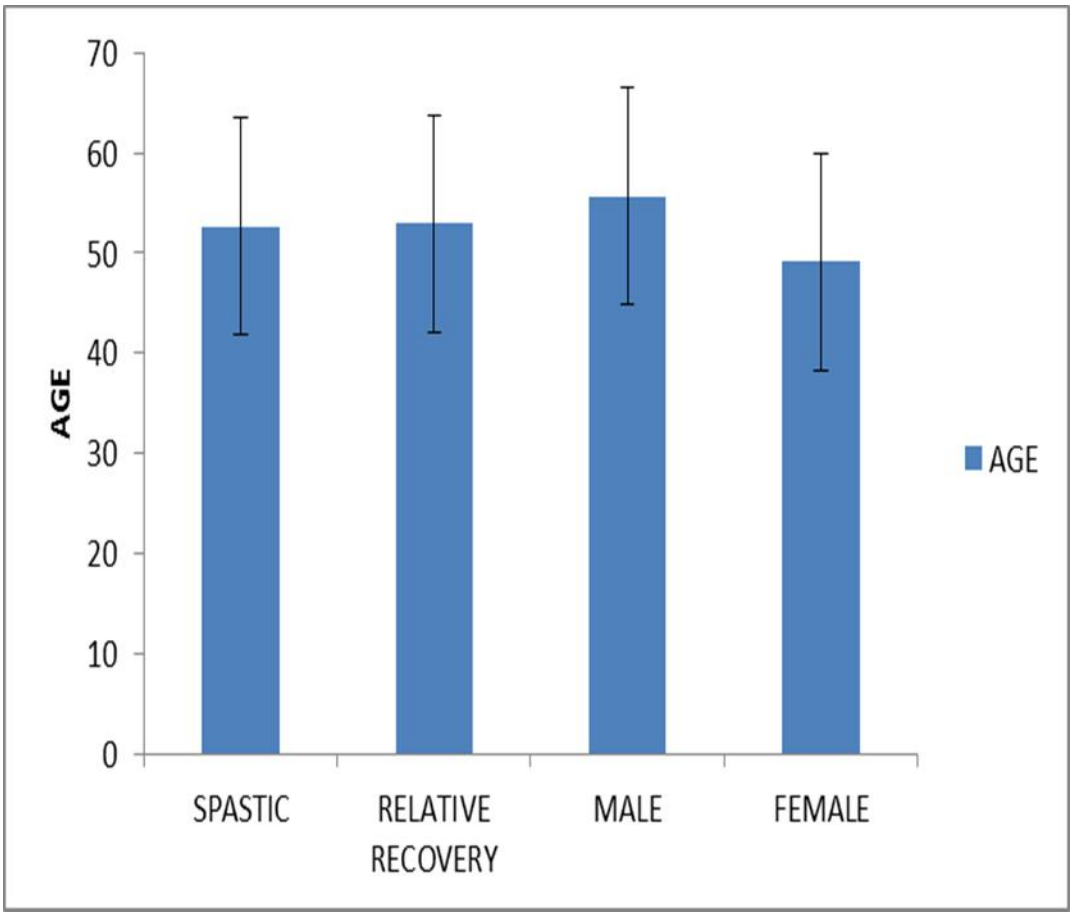

Figure 4.2: Distribution of sex and stage of stroke recovery of the participants by age.

- 85% of the subjects were from the low income socioeconomic status only 15% were in the middle class and none were from the high income class as seen in figure 4.3.

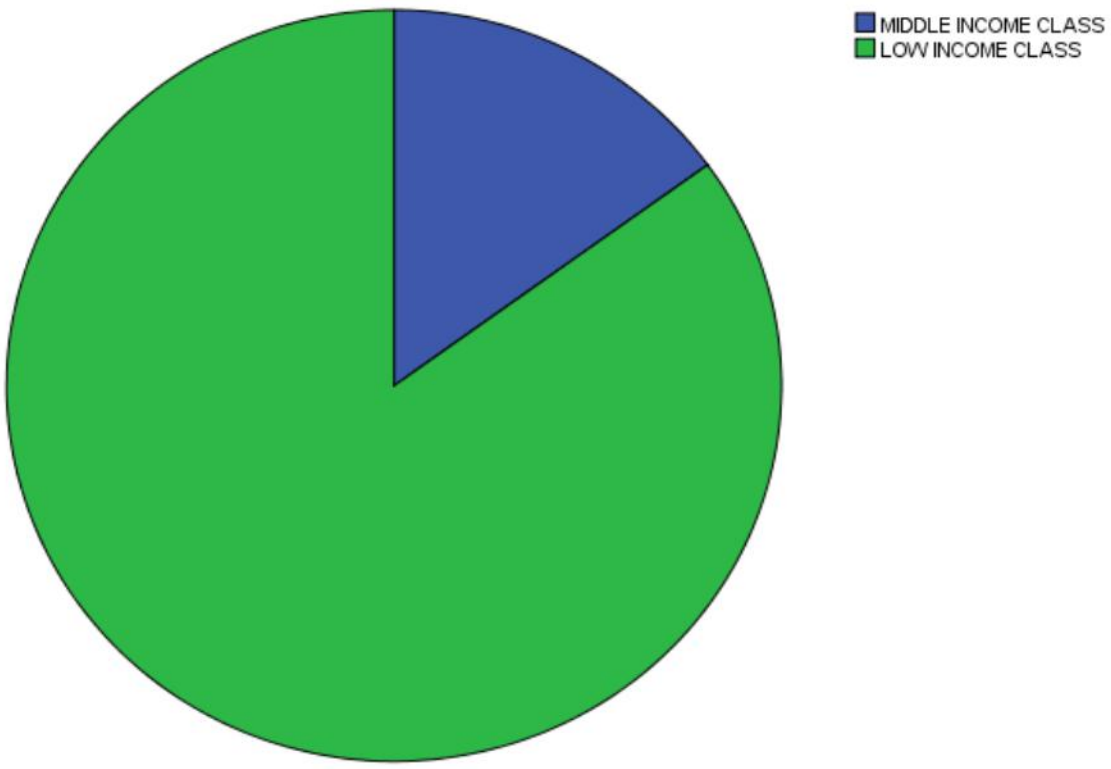

Figure 4.3: Distribution of socio-economic status of the subjects.

4.1.2 Anthropometric characteristics

The mean BMI of participants was 22.25kg/m^2 (male 22.42 and female 22.03), males are a little bit taller also having more weight (1.73m^2 – 65.74kg) than the females (1.67m^2 – 61.12kg). Those in the spastic stage had BMI of 22.68kg/m^2 as compared with 21.82 for participants in the relative recovery stage. Figure 4.4 shows the participants BMI across stroke recovery stages and gender.

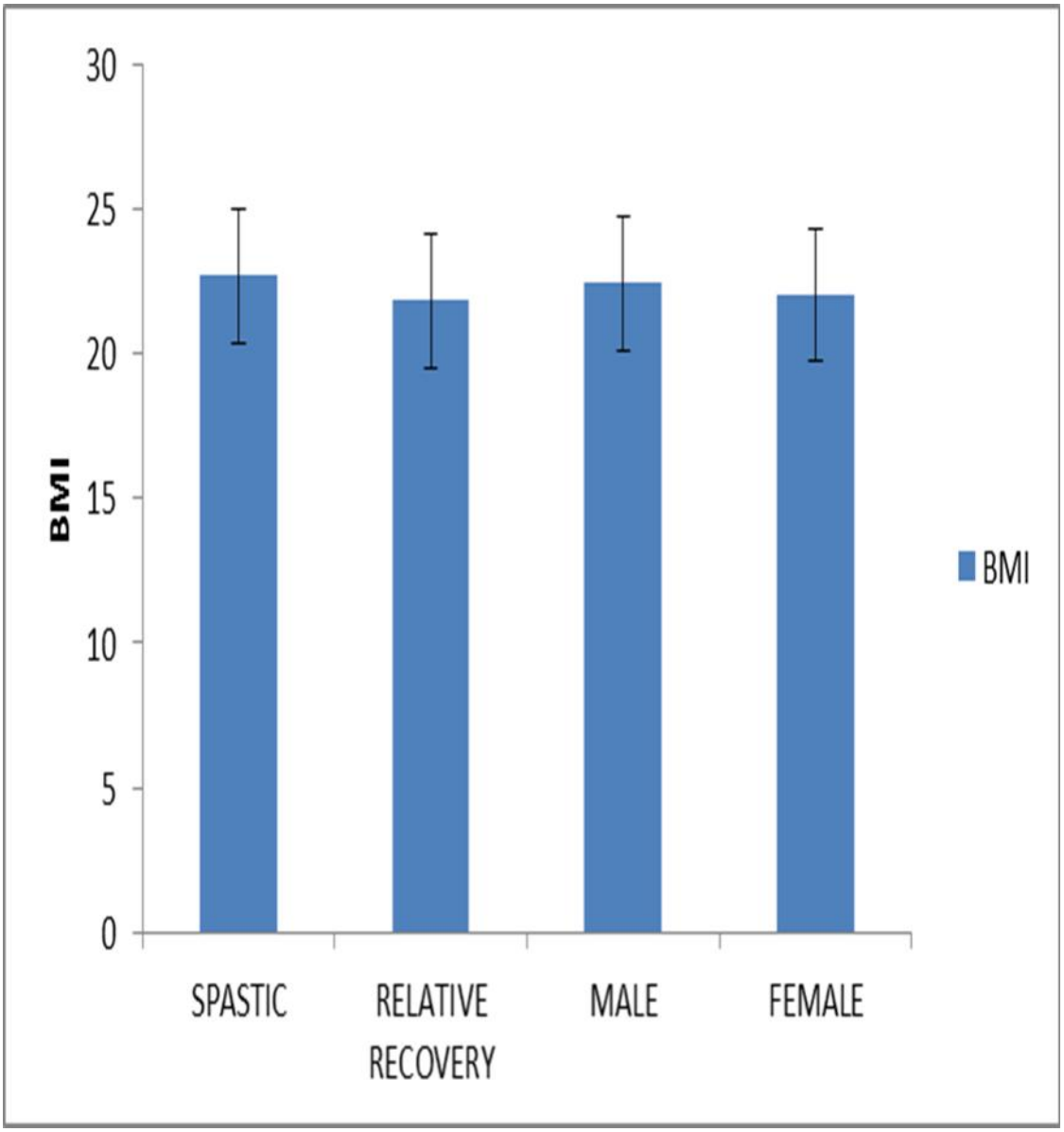

Figure 4.4: Distribution of body mass index (BMI) of the entire subjects according to sex and stage of stroke recovery.

4.1.3 Stroke Characteristics

Table 4.1a represent the participant's stroke characteristic. Barthel index score for functional mobility was used to assess subjects pre and post exercise, significance difference ($p<0.05$) was seen among the two groups at the end of 8-week in table 4.1b.

Table 4.1a: Stroke Characteristics of Subject who Participated in the Study (n=60)

Variables	n	%
Type of Stroke:		
Heamorragic	43	71.70
Ischeamic	17	28.30
Side of Weakness:		
Right	46	76.70
Left	14	23.30
Stroke Recovery Stage:		
Spastic stage	30	50.00
Relative recovery stage	30	50.00
Medications:		
Beta-blockers	11	18.30
ACE inhibitors	12	20.00
Calcium channel blockers	11	18.30
Combined medications	23	38.30

Table 4.1b: Barthel Scale for Functional Mobility among the Participants

	PRE	POST
GROUP	Mean ± S.E.M	Mean ± S.E.M
Spastic	10.50 ± 0.28	12.67 ± 0.46 [a]
Relative Recovery	11.17 ± 0.39	13.00 ± 0.46 [b]

[a] = significant ($p<0.05$) between (before exc) and week 8 in the spastic group, [b] = Significant ($p<0.05$) between (before exc) and week 8 in the relative recovery group.

4.1.4 Effect of Exercise on CP Response of All the Post-Stroke Subjects who participated in the study

As shown in table 4.2, there was statistically significant difference ($p<0.05$) in the SBP, DBP, HR, FVC and FEV_1 at week 4 and 8 when compared to week 1. The FEV_1% shows no statistically significant difference ($p>0.05$).

Table 4.2: Effect of Exercise on CP Parameters of all the Post-Stroke Subjects after an 8-week Exercise Protocol (Mean ± S.E.M.) (n=60), (df = 59)

Parameters	Week1 (before) Mean±S.E.M	Week 4 Mean±S.E.M	Week 8 Mean±S.E.M
SBP	127.27±0.92	124.35±1.14[a]	121.77±0.78[b]
DBP	83.62±0.70	81.98±0.73[a]	79.68±0.53[b]
HR	85.03±0.80	86.83±0.78[a]	87.05±0.70[b]
FVC	2.79±0.08	2.92±0.08[a]	3.17±0.08[b]
FEV1	2.18±0.79	2.30±0.08[a]	2.52±0.08[b]
FEV1%	78.80±1.86	79.38±1.69	80.02±1.52

[a] = significant ($p<0.05$) week 4 compared to week1 (before exc), [b] = significant ($p<0.05$) week 8 compared to week1 (before exc).

4.1.5 Effect of Exercise on CP Response of Post-Stroke Subjects in the Spastic Stage of Recovery (Group-A)

As shown in table 4.3, there was a statistically significant difference ($p<0.05$) in SBP, DBP, HR, FVC and FEV_1 at week 4 and week 8 when compared to week 1. No statistical significant difference ($p>0.05$) was found in FEV_1% at week4 and week 8.

Table 4.3: Effect of Exercise on CP Parameters of Post-Stroke Subjects in the Spastic Stage of Recovery after 8-week Exercise Protocol (Mean ± S.E.M.), (n=30), (df =29)

Parameters	Week1 (before) Mean±S.E.M	Week 4 Mean±S.E.M	Week 8 Mean±S.E.M
SBP	126.70±1.30	125.00±1.11[a]	120.80±1.04[b]
DBP	84.70±0.89	82.80±0.84 [a]	80.07±0.57[b]
HR	85.27±1.17	87.10±1.20 [a]	87.30±1.07 [b]
FVC	2.73±0.10	2.87±0.10 [a]	3.12±0.09 [b]
FEV1	2.24±0.10	2.34 ±0.10[a]	2.57±0.09[b]
FEV1%	81.98±2.33	82.01±2.10	82.33±1.74

[a] = significant ($p<0.05$) week 4 compared to week1 (before exc), [b] = significant ($p<0.05$) week 8 compared to week 1 (before exc).

4.1.6 Effect of Exercise on CP Response of Post-Stroke Subjects in the Relative Recovery Stage (Group-B)

As shown in table 4.4, there was a statistically significant difference ($p<0.05$) in SBP, DBP, HR, FVC, FEV_1 and FEV1% at week 4 and week 8 when compared to week 1.

Table 4.4 Effect of Exercise on CP Parameters of Post-Stroke Subjects in the Relative Recovery Stage after 8-week Exercise Protocol (Mean ± S.E.M.), (n=30), (df =29)

Parameters	Week1 (before) Mean±S.E.M	Week 4 Mean±S.E.M	Week 8 Mean±S.E.M
SBP	127.83±1.31	123.70±1.99[a]	122.73±1.14[b]
DBP	82.53±1.05	81.17±1.18 [a]	79.30±0.90 [b]
HR	84.80±1.11	86.57±1.02 [a]	86.80±0.92 [b]
FVC	2.85±1.12	2.97±0.13 [a]	3.21±0.12 [b]
FEV1	2.13±1.12	2.26±0.12 [a]	2.47±0.12 [b]
FEV1%	75.62±2.81	76.75±2.60 [a]	77.71±2.45 [b]

[a] = significant ($p<0.05$) week 4 compared to week1 (before exc), [b] = significant ($p<0.05$) week 8 compared to week1 (before exc).

4.1.7 Effect of Type of Exercise on CP Response Parameters of the Post-Stroke Subjects in the Spastic Stage of Recovery

Table 4.5 shows that there was no statistically significant difference ($p>0.05$) in HR in the treadmill exercise subgroup as well as the FEV_1% in all the subgroups at the end of the 8-week when compared to week1. There was no statistically significant difference ($p>0.05$) in SBP, DBP, HR, FVC, FEV_1 and FEV_1% within the spastic sub-groups at week 1 and at week 8.

Table 4.5: Effect of Type of Exercise on CP Response Parameters of Post-Stroke Subjects in the spastic stage, at the end of the 8-week exercise protocol (Mean ± S.E.M, df = 9)

	Treadmill (n=10)		Squatting (n=10)		Treadmill + Squatting n=10)	
Parameter	Week1	Week 8	Week1	Week 8	Week1	Week 8
SBP	126.80±1.52	120.00±0.80[a]	125.50±3.37	119.70±2.61[b]	127.80±1.51	122.70±1.54[c]
DBP	82.20±1.93	78.40±0.75[a]	86.50±1.30	80.20±1.22[b]	85.40±1.09	81.60±0.70[c]
HR	84.90±2.21	86.20±1.93	85.00±1.89	87.90±1.68[b]	85.90±2.19	87.80±2.08[c]
FVC	2.77±0.17	3.31±0.11[a]	2.57±0.19	2.95±0.20[b]	2.86±0.15	3.11±0.15[c]
FEV_1	2.07±0.18	2.61±0.16[a]	2.27±0.18	2.56±0.18[b]	2.37±0.18	2.55±0.17[c]
FEV_1%	75.37±5.33	78.70±3.78	88.07±1.46	86.89±1.36	82.50±3.62	81.41±3.02

[a] = significant ($p<0.05$) week 8 compared to week1 (before exc), [b] = significant ($p<0.05$) week 8 compared to week1 (before exc), [c] = significant ($p<0.05$) week 8 compared to week1 (before exc).

4.1.8 Effect of Type of Exercise on CP Response Parameter of the Post-Stroke Subjects in the Relative Recovery Stage

Table 4.6 also shows that there was no significant difference ($p>0.05$) in HR in the treadmill exercise group as well as the FEV_1% in all the sub-groups at the end of the 8-week when compared to week 1. There was no statistically significant difference ($p>0.05$) in SBP, DBP, HR, FVC, FEV_1 and FEV_1% within the relative recovery sub-groups at week 1 and at week 8.

Table 4.6: Effect of Type of Exercise on CP Response Parameters of Post-Stroke Subjects in the Relative Recovery Stage, at the end of the 8-week Exercise Protocol (Mean ± S.E.M, df = 9)

	Treadmill (n=10)		Squatting (n=10)		Treadmill + Squatting (n=10)	
Parameter	Week1	Week 8	Week1	Week 8	Week1	Week 8
SBP	128.00±2.16	119.40±2.07[a]	128.00±2.01	124.10±1.68 [b]	127.50±2.81	124.70±1.90[c]
DBP	81.30±1.88	78.00±1.53 [a]	81.80±1.67	78.60±1.24 [b]	84.50±1.89	81.30±1.78 [c]
HR	85.70±1.92	86.30±1.80	84.40±2.28	87.20±1.65 [b]	84.30±1.67	86.90±1.46 [c]
FVC	2.79±0.23	3.16±0.24 [a]	2.88±0.25	3.22±0.24 [b]	2.89±0.18	3.26±0.16 [c]
FEV_1	1.98±0.21	2.33±0.22 [a]	2.20±0.24	2.56±0.24 [b]	2.23±0.19	2.54±0.19 [c]
FEV_1%	72.05±5.51	74.12±4.55	76.66±4.15	79.02±3.52	78.14±5.17	78.55±4.73

[a] = significant ($p<0.05$) week 8 compared to week1 (before exc), [b] = significant ($p<0.05$) week 8 compared to week1 (before exc), [c] = significant ($p<0.05$) week 8 compared to week1 (before exc).

4.1.9 Effect of Exercise on CP Response of Post-Stroke Subjects According to Sex

Table 4.7 shows that there was statistically significant difference ($p<0.05$) in SBP at week 1 between gender and no significant difference seen at the end of 8 week. There was no statistical significant difference ($p>0.05$) in DBP, HR and FEV_1% between males and females, FVC and FEV_1 shows statistical significant difference ($p<0.05$) between males and females at week 1 and at week 8. Both males and females showed statistically significant difference ($p<0.05$) in SBP, DBP, HR, FVC and FEV_1 at the end of week 8 when compared to week 1. Females showed statistical significant difference ($p < 0.05$) in FEV_1% at week 8 when compared to week 1 while the males did not ($p > 0.05$).

Table 4.7: Effect of 8-week Exercise Protocol on CP Response Parameters of Post-Stroke Subjects Distributed According to Sex (Mean ± S.E.M) Males (n=34), Females (n=26)

	Week 1		Week 8	
Parameters	Males	Females	Males	Females
SBP	128.94±0.82	125.08±1.76 [a]	122.88±0.68[*]	120.31±1.53[#]
DBP	84.47±0.82	82.50±1.18	80.29±0.56[*]	78.88±0.96[#]
HR	84.00±0.96	86.38±1.32	86.47±0.85[*]	87.81±1.17[#]
FVC	3.07±0.08	2.43±0.11[a]	3.45±0.07[*]	2.80±0.11[b#]
FEV1	2.44±0.09	1.86±0.10 [a]	2.76±0.09[*]	2.21±0.10[b#]
FEV1%	79.99±2.61	77.24±2.62	80.22±2.07	79.76±2.27[#]

[a] = significant ($p<0.05$) males compared to females at week1 (before exc), [b] = significant ($p<0.05$) males compared to females at week 8, [*] = significant ($p<0.05$) week1 compared to week 8 males, [#] = significant ($p<0.05$) week1 compared to week 8 females.

4.1.10 Effect of Exercise on CP Response of Post-Stroke Subjects According to the Side of Hemiplegia

Table 4.8 shows that there was statistically significant difference ($p<0.05$) in SBP, DBP, HR, FVC, FEV_1, and FEV_1% between week1 and week 8 in both right-sided and left-sided hemiplegia, except in the left-sided hemiplegia no statistically significant difference seen for FEV_1% ($p > 0.05$). Also, no statistically significant difference ($p>0.05$) seen in SBP, DBP, HR, FVC, FEV_1, and FEV_1% between the two side of affectation at week 1 and at week 8.

Table 4.8: Effect of 8-week Exercise Protocol on CP Response Parameters of Post-Stroke Subjects Distributed According to the Side of Hemiplegia (Mean ± S.E.M.)

	Right-sided (n= 46)		Left-sided (n=14)	
Parameters	Week1	Week 8	Week1	Week 8
SBP	127.61±1.01	121.70±0.93*	126.14±2.13	122.00±1.40#
DBP	84.13±0.76	79.91±0.58*	81.93±1.59	78.93±1.26#
HR	85.20±0.86	87.20±0.76*	84.50±2.02	86.57±1.69#
FVC	2.78±0.09	3.17±0.08*	2.83±0.19	3.16±0.17#
FEV1	2.17±0.09	2.53±0.09*	2.23±0.18	2.49±0.18#
FEV1%	78.89±2.21	80.44±1.82*	78.52±3.42	78.67±2.69

Where * = significance ($p<0.05$) week1 compared to week 8 right-sided, # = significant ($p<0.05$) week1 compared to week 8 left-sided.

4.1.11 Effect of Exercise on Heart Rate Recovery

Table 4.9a and 4.9b describe the heart rate variability and recovery after exercise. A similar HR response was observed across the sub-groups and the stage of recovery. There was an increase in HR to the maximum at the peak of exercise followed by gradual decrease in HR from the peak value to lower values at 5 minutes and 20 minutes post-exercise. At week 1, HR value before exercise when compared to 20 minutes post-exercise in both groups were statistically different ($p<0.05$). At week 8, HR values at 20 minutes post-exercise were statistically same ($p>0.05$) in the relative recovery stage when compared to before exercise and statistically reduced ($p<0.05$) in the spastic stage when compared to before exercise.

Table 4.9a: Exercise-induced HR variability at the begining of exercise protocol (Mean ± S.E.M.)

	Timing of HR measurement			
Groups	before exc.	peak of exc	5 min after exc	20 min after exc.
All (n=60)	85.03±0.80	103.18±1.29[a]	98.68±1.28[b]	87.72±0.94[c]
A (n=30)	85.27±1.17	104.90±1.71[a]	100.17±1.64[b]	88.77±1.45[c]
B (n=30)	84.80±1.11	101.47±1.90[a]	97.20±1.95[b]	86.67±1.19[c]
AT (n=10)	84.90±2.21	110.90±2.39 [a]	109.10 ±2.37 [b]	90.80±2.68[c]
AS (n=10)	85.00±1.89	102.40±2.84 [a]	98.30±2.65 [b]	89.10±2.76[c]
AC (n=10)	85.90±2.19	101.40±2.87 [a]	96.10±2.69 [b]	86.40±2.08 [c]
BT (n=10)	85.70±1.92	105.00±3.51 [a]	101.20±3.48 [b]	88.00±2.23 [c]
BS (n=10)	84.40±2.28	100.10±2.62 [a]	96.00±2.61 [b]	86.50±2.28 [c]
BC (n=10)	84.30±1.67	99.30±3.69 [a]	94.40±3.87 [b]	85.50±1.75 [c]

[a] = significant ($p<0.05$) for at peak of exercise compared to before exercise, [b] = significant ($p<0.05$) for five minute after exercise compared to before exercise, [c] = significant ($p<0.05$) for twenty minute compared to before exercise.

Table 4.9b: Exercise-induced HR variability at 8-week (Mean ± S.E.M.)

	Timing of HR measurement			
Groups	before exc.	peak of exc.	5 min after exc.	20 min after exc.
All (n=60)	87.30±0.67	103.60±0.78[a]	96.72±0.60[b]	87.05±0.70
A (n=30)	88.10±1.00	104.80±1.31[a]	97.50±0.98[b]	87.30±1.07[c]
B (n=30)	86.50±0.89	102.40±0.81[a]	95.93±0.70[b]	86.80±0.92
AT (n=10)	87.10±1.84	104.80±2.33 [a]	97.10±1.49 [b]	86.20±1.93[c]
AS (n=10)	88.50±1.63	104.60±2.12 [a]	97.00±1.56 [b]	87.90±1.68[c]
AC (n=10)	88.70±1.88	105.00±2.57 [a]	98.40±2.10 [b]	87.80±2.08 [c]
BT (n=10)	85.20±1.50	100.90±1.47 [a]	95.60±1.22 [b]	86.30±1.80
BS (n=10)	87.40±1.75	103.60±1.47 [a]	97.00±1.09 [b]	87.20±1.65
BC (n=10)	86.90±1.41	102.70±1.31 [a]	95.20±1.36 [b]	86.90±1.46

[a] = $p<0.05$ for at peak of exercise compared to before exercise, [b] = $p<0.05$ for five minute after exercise compared to before exercise, [c] = $p<0.05$ for twenty minute compared to before exercise.

4.2 DISCUSSIONS

Emerging evidence suggests that exercise training in the post stroke population can facilitate improvement in the cardiovascular, respiratory and neuromuscular system (Macko *et al.,* 2005). This study aimed to determine the cardiopulmonary responses parameters in post stroke patients at the spastic and relative recovery stages before and after 8 weeks of squatting and/or treadmill exercise regiments.

All the participants successfully completed the exercise protocol except 2 subjects who interrupted their exercise for 2 sessions due to elevated blood pressure and return after the blood pressure was stable. The participants in the two groups and sub-groups showed significant decrease in pre-exercise SBP (by 3 mmHg) and DBP (by 2 mmHg), increase in FVC (by 0.13L) and FEV_1 (by 0.12L) at week 4 when compared to those measured at week1. The participants in the two groups and sub-groups also showed significant decrease in pre-exercise SBP (by 6 mmHg) and DBP (by 3 mmHg), increase in FVC (by 0.4L) and FEV_1 (by 0.3L) at week 8 when compared to those measured at week1. These changes are normal-positive physiological responses to prolong exercise showing that the treadmill and squatting exercise has been effective on the cardiac and respiratory muscle performance enhancement of the post-stroke patients in all the groups, leading to an improvement in the cardiovascular conditioning to physical exercise. Treadmills deliver mechanical energy to the human body through the moving running belt of the treadmill. The subject actively walk/run to catch up with the running belt underneath his feet, this process gives the person effective cardiovascular and pulmonary work out through the involvement of all the

antigravity muscles (Fuller *et al*., 2002; Elizabeth *et al*., 2012). Wall-bar supported squat exercise engages the muscles of the upper body and lower body essentially giving someone a full body work out in one exercise (Stuart, 2008), which may have effect on the CP response parameters.

WilBorn *et al*. (2004) conducted study on 16 subjects undergoing alternate inverted leg press and upright hack squat using 1RM (65% to 85% of 1RM) to grade the exercise intensity in both groups, the findings suggest increase in HR, SBP, and DBP, and a return to the resting value after exercise. The cardiovascular measures are influenced by intensity of the exercise, rather than apparatus or change in body position during resistance exercise. This study supports the findings of this work.

Kim *et al.* (2014) investigated the effect of individualized respiratory muscle training device combined with conventional physical therapy exercise on the pulmonary function and exercise capacity of 20 stroke patients (10 exp. 10 controls). Improvement and a significant intergroup difference were observed in the FVC, FEV_1 and peak expiratory flow after exercise. His findings suggest that exercise of the respiratory muscle using an individualized respiratory device combined with other forms of conventional exercise had a positive effect on pulmonary function and exercise capacity than the conventional physical therapy exercise alone.

This findings support (Forjaz *et al*., 2000) study on 16 normotensives performing ergometry exercise at varying intensities to determine the influence of exercise on the blood pressure (BP) and HR. They found out that exercising at lower or higher

intensities lead to decrease in BP after exercise and at lower intensity the HR decreases but at higher intensity the HR increases after exercise.

HR increased slightly at week 4 (by 2 b/m) and week 8 (by 2 b/m) when compared to week 1 in both groups or sub-groups but the increase was statistically insignificant in the treadmill sub-groups (by 1 b/m) as compared to the squatting and combine treadmill-squatting exercise sub-groups (by 2 b/m). During aerobic or resistance exercise the HR increases due to vagal withdrawal and increase sympathetic tone. Heart rate recovery after exercise in people with healthier heart and those who exercise regularly is faster due to higher parasympathetic activity and lower sympathetic activity (Dimkpa, 2009). The relative cardiovascular stress of physical therapy and occupational therapy sessions in 20 patients undergoing a stroke rehabilitation program was evaluated by MacKay-Lyons and Makrides (2002). The time per session in which the achieved heart rate was to be within the prescribed target heart rate zone was found to be extremely low, which suggest that these sessions elicited inadequate cardiovascular stress to induce a training effect but in this study the increase the heart rate may be due to increased cardiovascular stress placed on the subjects by the 3 forms of exercise and poor endurance.

A similar HR response was observed across the sub-groups and the stage of recovery. There was an increase in HR to the maximum at the peak of exercise followed by gradual decrease in HR from the peak value to lower values at 5 minutes and 20 minutes post-exercise. HR recovery (HRR) after exercise improved at week 8 when compared to week 1 across all the groups. Heart rate recovery is mainly thought to be

due to parasympathetic reactivation, studies (Tiukinhoy, Beohar, Hsie, 2003; Kligfield, Cormick, Chai, Jacobson, Feuerstadt, Hao, 2003) have indicated that HRR can be improved or modified in normal or abnormal conditions through exercise training and available evidences significantly reveal the potential importance of post-stroke HRR as an index of cardiovascular health (Dimkpa, 2009).

Changes in FEV_1% show no significant difference in the values at week 1 when compared to week 4 and week 8 however, the values from both groups and sub-groups remain within a range of 74 to 87%. These may be because most of the stroke patients have normal pulmonary function since FEV_1% of 70 to 85% is considered normal (Simon *et al*., 2010), while some present with mild signs of restrictive respiratory disease where the FVC is lower than the total lung capacity but the FEV_1 remain within normal range and FEV_1% may be normal or slightly increased (Bateman *et al*., 2000; Al-Ashkar *et al*., 2003).

Males and females showed improvement in SBP, DBP, HR, FVC and FEV_1 at the end of week 8 when compared to week 1. This shows that the exercise is effective in both males and females but the males showed significant higher values for FVC and FEV_1 than the females at week 1 and at week 8. This result may be by chance due to the fact that males have higher FVC than females. This partially support the findings of Billinger *et al*. (2012) on cardiopulmonary response and safety of treadmill exercise testing at peak effort in 62 patients (32 males and 30 female) during the chronic stage of stroke recovery. Both males and females had low cardio-respiratory fitness levels. No significant difference was found between gender for peak heart rate or VO_2 peak at the end of the intervention. Males demonstrated higher values for minute ventilation,

tidal volume, and respiratory exchange ratio. The results based on side of affectation in all the subject shows that right-sided and left-sided affectation improved and responded well except $FEV_1\%$ ($p>0.05$) in the left sided affectation group which shows no significant difference, although the number of patients with L sided stroke could not be matched with R sided stroke due to the large number of R sided stroke cases seen in this community. According to Hamzat and Alabi, 2006 nil significant difference seen in SBP, DBP and HR for dominant and non-dominant side of brain lesion in hemiplegic after exercise, but in de-Almeida *et al*. (2011) findings right-sided hemiplegia exhibited greater impairment of the respiratory muscles than left-sided hemiplegia due to the physiologic positioning of the domes of the diaphragm on the affected side.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.1 SUMMARY

There was statistically significant difference ($P < 0.05$) in SBP, DBP, HR, FVC, and FEV_1 in the entire subject at week 4 and week 8 when compared to week1, except the $FEV_1\%$ ($p > 0.05$). Effect of exercise at week4 and week8 when compared to week1 in each group (A and B) irrespective of type of exercise shows statistically significant difference ($p < 0.05$) in participants cardiopulmonary response parameters except $FEV_1\%$ in group A which shows no significant difference ($p > 0.05$). Statistically significant difference ($p < 0.05$) for SBP, DBP, FVC, and FEV_1 was observed between week1 and week 8 in the treadmill (AT, BT) squatting (AS, BS) and combine treadmill–squatting (AC, BC) groups. Heart rate shows no statistically significant difference ($p > 0.05$) between week 1 and week 8 post exercise in the AT and BT group but there was significant difference in the AS and BS, AC and BC sub-groups. Also $FEV_1\%$ shows no statistically significant difference ($p > 0.05$) between week1 and week 8 post exercises in all the sub-groups. There was statistically significance difference ($p < 0.05$) in FVC, FEV_1 and $FEV_1\%$ between males and females but no significant difference was observed for other parameters at week 1 and at week 8. Significant improvement was seen at the end of 8 week when compared with week 1. Right sided (R) and left sided (L) affectation shows no statistically significant difference ($p > 0.05$) in cardiopulmonary response parameters at week 1 and at week 8, but there was statistically significant difference ($p < 0.05$) in CP parameters between week 1 and week 8 in both R and L side of affectation except $FEV_1\%$ ($p>0.05$) in the left sided affectation group. There was statistically significant

difference ($p<0.05$) in the HR variability before exercise when compared to at peak of exercise, 5 minute and 20 minute after exercise at week 1 and at week 8 in the 2 groups and subgroups. HR recovery 20 minute after exercise improved at week 8 when compared to week 1 in all the groups.

5.2 CONCLUSION

- There was a decrease in SBP, DBP, and an increase in HR, FVC, and FEV_1 due to the 8-week exercise regimen in all the post-stroke subjects, base on the stages of recovery, and type of exercise.
- FEV_1% in all the groups and sub-groups remain within the range of 74-87% at the end of the 8-week.
- HR increase in the 3 subgroups shows that squatting exercise and combine treadmill-squatting exercise places more cardiovascular stress on the subjects than the treadmill exercise alone.

5.3 RECOMMENDATIONS

Based on the results of this study, the following recommendations are proposed:

- 8 weeks of Treadmill exercise interventions at the doses described in this study may be recommended to post-stroke patients resident in our environment to improve their cardiopulmonary conditioning and exercise tolerance, which is vital for activity of daily living.

- Further study should be conducted on squatting and combined Td and SQ exercise.
- Further study should be conducted to determine the ECG changes in response to the two forms of exercise among post stroke patients in the spastic and relative recovery stages.

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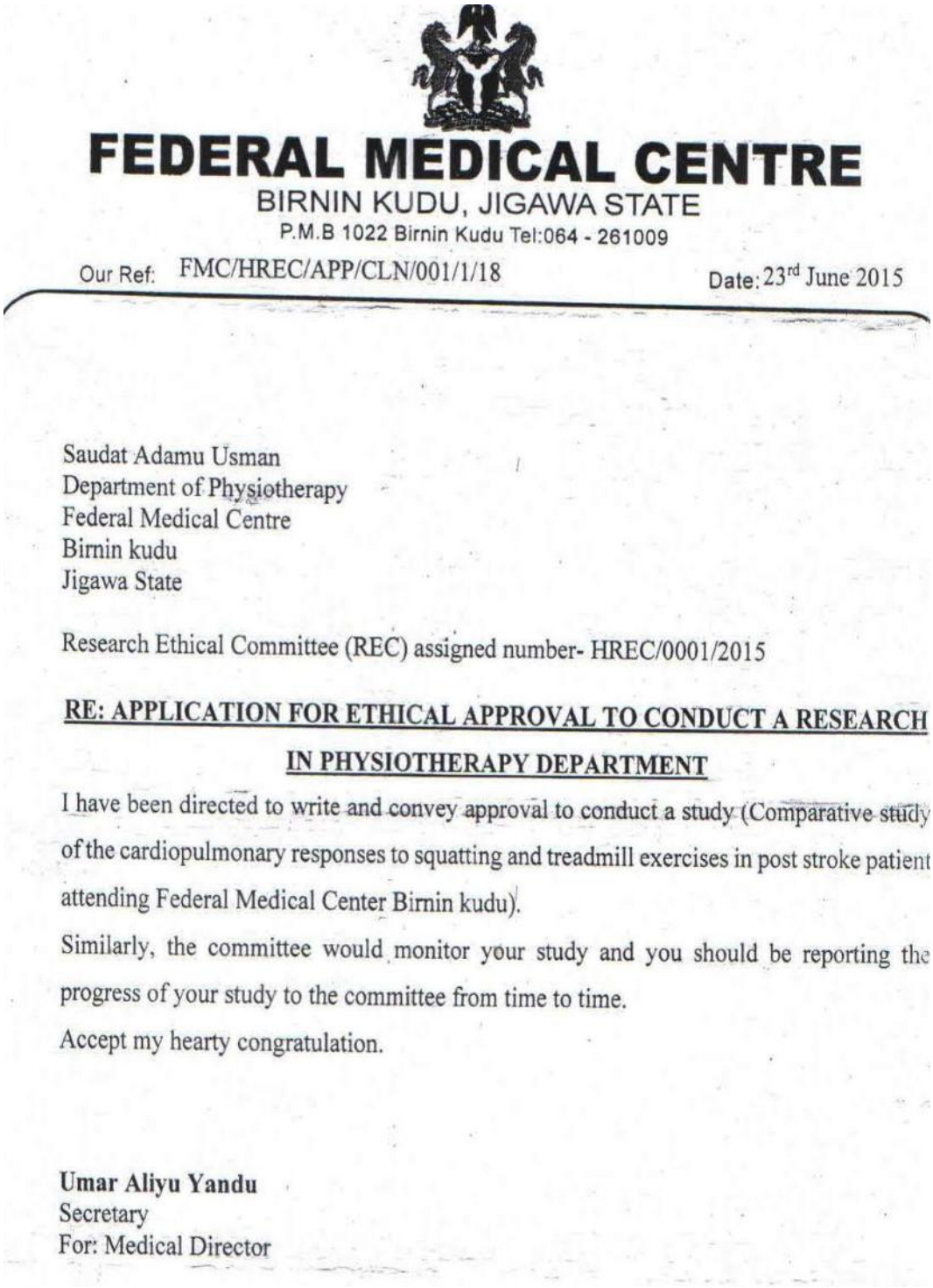
APPENDIX I

ETHICAL APPROVAL



FEDERAL MEDICAL CENTRE
BIRNIN KUDU, JIGAWA STATE
P.M.B 1022 Birnin Kudu Tel:064 - 261009

Our Ref: FMC/HREC/APP/CLN/001/1/18 Date: 23rd June 2015

Saudat Adamu Usman
Department of Physiotherapy
Federal Medical Centre
Birnin kudu
Jigawa State

Research Ethical Committee (REC) assigned number- HREC/0001/2015

RE: APPLICATION FOR ETHICAL APPROVAL TO CONDUCT A RESEARCH IN PHYSIOTHERAPY DEPARTMENT

I have been directed to write and convey approval to conduct a study (Comparative study of the cardiopulmonary responses to squatting and treadmill exercises in post stroke patient attending Federal Medical Center Birnin kudu).

Similarly, the committee would monitor your study and you should be reporting the progress of your study to the committee from time to time.

Accept my hearty congratulation.

Umar Aliyu Yandu
Secretary
For: Medical Director

APPENDIX II

INFORMED CONSENT FORM

DEPARTMENT OF HUMAN PHYSIOLOGY, FACULTY OF MEDICINE

BAYERO UNIVERSITY, KANO

I am Saude Adamu Usman, a final year MSc student of human physiology department, faculty of medicine Bayero University, Kano. I write to seek for your consent to participate in my clinical research on the cardiopulmonary response of stroke patients to squatting and treadmill exercises.

During this research you will be receiving treadmill and/or squatting exercises for period of 8 weeks and your heart rate, blood pressure and respiratory function will be assessed before and after the exercise regiment. Please be informed that all information obtained from you will be treated with utmost confidentiality, and used purely for research purpose. You are free to refuse to participate in this study, you also have the right to opt-out of the study any time. I will be very grateful if you offer your full cooperation and participation for the success of the study, as this will further improve the rehabilitation of stroke patients.

Consent: the study has been well explained to me and I appreciate the procedure involved, I therefore volunteer to be part of the study.

_____

SIGNATURE OF THE PARTICIPANT AND DATE

_____

SIGNATURE OF THE RESEARCHER AND DATE

_____

SIGNATURE OF WITNESS AND DATE

APPENDIX III

BARTHEL INDEX OF ACTIVITIES OF DAILY LIVING

Activity and Score

FEEDING

0 = unable

5 = needs help cutting, spreading butter, etc., or requires modified diet

10 = independent

BATHING

0 = dependent

5 = independent (or in shower)

GROOMING

0 = needs to help with personal care

5 = independent face/hair/teeth/shaving (implements provided)

DRESSING

0 = dependent

5 = needs help but can do about half unaided

10 = independent (including buttons, zips, laces, etc.)

BOWELS

0 = incontinent (or needs to be given enemas)

5 = occasional accident

10 = continent

BLADDER

0 = incontinent, or catheterized and unable to manage alone

5 = occasional accident

10 = continent

TOILET USE

0 = dependent

5 = needs some help, but can do something alone

10 = independent (on and off, dressing, wiping)

TRANSFERS (BED TO CHAIR AND BACK)

0 = unable, no sitting balance

5 = major help (one or two people, physical), can sit

10 = minor help (verbal or physical)

15 = independent

MOBILITY (ON LEVEL SURFACES)

0 = immobile or < 50 yards

5 = wheelchair independent, including corners, > 50 yards

10 = walks with help of one person (verbal or physical) > 50 yards

15 = independent (but may use any aid; for example, stick) > 50 yards

STAIRS

0 = unable

5 = needs help (verbal, physical, carrying aid)

10 = independent

TOTAL

(0–100)

(Mahoney and Barthel, 1965).

APPENDIX III

MODIFIED ASHWORTHSCALE FOR GRADING SPASTICITY

Grade and Description

0 No increase in muscle tone

1. Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of the range of motion ROM when the affected part(s) is moved in flexion or extension.
2. Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.
3. More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.
4. Considerable increase in muscle tone, passive movement difficult.
5. Affected part(s) rigid in flexion or extension. Bohannon and Smith (1987).

APPENDIX IV

QUESTIONNAIRE

Age ☐

Sex ☐

Side of Affectation ☐

Stroke Duration ☐

Height ☐

Weight ☐

Chronic Pulmonary/Cardiac/Renal Condition ☐

Functional Mobility Barthel Index Score

☐ 0 → Immobile or < 50 yards

☐ 5 → Wheelchair independent, including corners, > 50 yards

☐ 10 → Walks with help of one person (verbal or physical) > 50 yards

☐ 15 → Independent (but may use any aid; for example, stick) > 50 yards

Asworth Spasticity Score

☐ G-0 → No increase in muscle tone

☐ G-1 → Slight increase in muscle tone, manifested by a catch and release or by minimal resistance at the end of ROM when the affected part(s) is moved.

☐ G-2 → Slight increase in muscle tone, manifested by a catch, followed by minimal resistance throughout the remainder (less than half) of the ROM.

☐ G-3 → More marked increase in muscle tone through most of the ROM, but affected part(s) easily moved.

☐ G-4 → Considerable increase in muscle tone, passive movement difficult.

☐ G-5 → Affected part(s) rigid in flexion or extension.

Type of Stroke H I

Stroke Recovery Stage Spastic Relative

Medication:

- Beta blockers
- ACE inhibitors
- Calcium channel blockers
- Combination

Socioeconomic Status

Low =A3, B3 and C3 Middle = A2, B2 and C2 High =A1, B1 and C1

A: Level of Education

1- Education up to University Level
2- Secondary but not up to university
3- No Schooling or up to Primary Level only

B: Income per Month

1- Above Minimum Wage
2- National Minimum Wage
3- Below National Minimum Wage

C: Occupation

1- Professionals

2- Technicians, Skilled Workers and Well to do Traders

3- Unskilled Workers

Table 1: Distribution of Subjects Age and BMI by Sex and Stages of Recovery

Variable		Mean ± SD
BMI		22.25 ± 2.20
	Male	22.42 ± 2.31
	Female	22.03 ± 2.09
	Spastic stage	22.68 ± 2.40
	Relative recovery	21.82 ± 1.92
Age		52.80 ± 10.80
	Male	55.62 ± 10.84
	Female	49.12 ± 10.29
	Spastic stage	52.63 ± 11.26
	Relative recovery	52.97 ± 10.23

Table 2: Distribution of Subject by Sex and Socioeconomic Status

Variable	frequency	%
Gender		
Male	34	56.7
Female	26	43.3
SES		
Low	51	85
Middle	9	15
High	0	0