EFFECTS OF A 12-WEEK AEROBIC EXERCISE PROGRAMME ON CARDIORESPIRATORY AND HAEMATOLOGICAL VARIABLES AND QUALITY OF LIFE OF INDIVIDUALS WITH SICKLE CELL ANAEMIA.

BY

AKINOLA, THERESA ODUNAYO

MATRIC NUMBER 112406

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CERTIFICATION

We certify that this research work was carried out by Mrs. Akinola Theresa Odunayo, in the Department of Physiotherapy, College of Medicine, University of Ibadan, Ibadan, Nigeria under our supervision.

(Main Supervisor)

Dr. B. O. A. Adegoke Ph.D (Physiotherapy)
Senior Lecturer/Consultant Physiotherapist,
Department of Physiotherapy, College of
Medicine, University of Ibadan.

(Co-Supervisor)

Dr. O. A. Dosunmu (FWACP)
Consultant Heamatologist/Senior
Lecturer, Department of
Haematology, Lagos State
University Teaching Hospital,
Ikeja, Lagos.

DEDICATION

This work is dedicated to all the people with Sickle Cell disorder and members of my beautiful family, who have given me their full support during the course of this study and above all, to my everlasting father, the Alpha and the Omega.

ABSTRACT

Sickle Cell Anaemia (SCA) is a genetic disorder characterized by predominance of sickle cell haemoglobin. Its management is multidisciplinary but does not routinely include physiotherapy except when certain complications occur. Aerobic exercise improves cardiovascular endurance and function in sickle cell anaemia patients but its effects on their Quality of Life (QoL), Frequency of Crisis (FC), Frequency of Hospitalization (FH) and Length of Hospitalization (LH) have not been well documented.This study was designed to investigate the effects of a 12-week aerobic exercise programme on QoL, cardiorespiratory and haematological variables, FC, FH and LH of individuals with SCA.

The study was a two-group randomised control trial involving 129 consecutively recruited individuals with SCA assigned to exercise (65) and control (64) groups. However, 104 participants (exercise group= 54, control group= 50) completed the study. Data on FC, FH and LH in the 6-months before the study were retrieved from participants' hospital files. The exercise group had 12 weeks of aerobic dance exercises, comprising dancing thrice weekly; the intensity being progressed by increasing the duration by five minutes fortnightly. Data on QoL (using Short Form-36 questionnaire), cardiorespiratory variables [Heart Rate (HR), Cardiorespiratory Fitness Score (CRFS) and Vital Capacity (VC)] and haematological variables [Packed Cell Volume (PCV), Platelet Count (PC) and Mean Corpuscular Haemoglobin Concentration (MCHC)] were collected at baseline and at the ends of the 6^{th} and 12^{th} week. Information on FC, FH and LH during the 6-month follow-up phase was also retrieved from participants' hospital files. Data were analysed using ANOVA, independent and paired t-tests at $p \leq 0.05$.

The mean ages of the exercise (26.1±6.7 years) and control (25.7±5.6 years) groups were not significantly different. At baseline, the QoL (65.1±8.2, 65.4±10.1), HR (75.2±4.5 b/min, 76.8±5.1 b/min), CRFS (52.6±4.1, 54.3±4.5) VC (1714.5±400.4 cc, 1773.9±446.5 cc), PCV (24.7±3.2 %, 24.4±3.4 %), PC (364.6±127.5×10^9/L, 343.6±128.8×10^9 /L) and MCHC (33.7±1.0 g/dl, 33.9±1.1 g/dl) for exercise and control groups were not significantly different. The exercise group compared with the control had better CRFS (50.8±5.5, 54.2±6.4 respectively), VC (2109.4±441.9cc, 1769.9±389.6cc respectively) and PCV (26.1±3.4%, 23.84±3.8% respectively). The exercise group compared with the control had better HR (71.9±3.5 b/min, 76.5±4.9 b/min), CRFS (46.5±6.2, 55.3±6.7), VC (2293.9±374.1 cc, 1755.8±377.6 cc) and PCV (26.8±3.3 %, 23.32±3.5 %) at the end of the 12th week. Although the pre-intervention variables for exercise and control groups were not significantly different for their FC (1.4±1.5, 1.4±1.7 respectively), FH (0.4±0.6, 0.4±0.6 respectively) and LH (0.6±0.9 days, 0.7±1.0 days respectively). However, the exercise group had significantly less FC (0.5±0.6), FH (0.1±0.3) and shorter LH (0.1±0.3 days) in the follow-up phase of the study.

The 12-week aerobic exercise programme improved the quality of life, vital capacity and cardiorespiratory fitness score and reduced their HR, frequencies of crisis and hospitalization and length of hospitalization. Aerobic exercise should be routinely included in the total management of patients with sickle cell anaemia.

Keywords: Sickle cell anaemia, Aerobic exercise, Quality of life, Cardiorespiratory fitness.

Word count: 472 words.

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CHAPTER ONE

INTRODUCTION

1.1 INTRODUCTION

Sickle-cell anaemia (a form of sickle-cell disorder or sickle-cell disease) is a common genetic condition due to a haemoglobin disorder – inheritance of mutant haemoglobin genes (HbS) from both parents. (WHO, 2006; Koffi et al 2010). It was first reported in literature in 1910 by a Chicago Cardiologist and Professor of Medicine, James B Herrick after he observed peculiar elongated sickle shaped red blood cells (RBC) in an anaemic black medical student (Desai and Dhanani, 2004). The disease comprises the homozygous form HbSS and the double heterozygous forms in which the HbS combines with another abnormal hemoglobin mutation e.g. sickle cell HbC (β^c) disease, HbSC, sickle cell HbD-Los Angeles (or Punjab) and sickle cell HbE-beta thalassemia (β^+-results in decreased beta globulin production while β^o-completely eliminates beta globulin production) (Smeltzer and Bare 2003).

The main problems in sickle cell anaemia arise from the tendency of the red blood cells to become sickle-shaped and block capillaries at low oxygen tension (WHO, 2006). This vaso-occlusive crisis can lead to impaired circulation, chronic leg ulcers and infarction, haematuria, priapism, blindness, cholelithiasis, osteomyelitis, jaundice, peptic ulcer and vascular necrosis (Scutellari et al, 2003).

The life expectancy of individuals with sickle cell disease has improved considerably since 1960 when Sir John Dacie described it as essentially a 'disease of childhood'). According to World Health Organization (2006), in the United States of America, median survival was estimated in 1994 to be 42 years for men and 48 years for

women, whereas comparable figures for Jamaica published in 2001 suggested 53 years for men and 58.5 years for women. In Jamaica, the greatest mortality occurs between 6 and 12 months old when 10% of patients die despite considerable experience in the diagnosis and therapy of the condition and absence of malaria. There are, however, no concrete data on the survival of patients with sickle-cell anaemia on the African continent (WHO, 2006).

Patients' quality of life is an increasingly important outcome measure in medicine and healthcare. It is now widely used in clinical trials and in patient management for assessing morbidity and the impact of treatment (Jonathan et al, 2001). Quality of life (Qol) is the descrepancy between actual and desired function (Rumsfeld, 2002). The Committee on Quality of Health Care in America of the Institute of Medicine (2001) reported that quality of life measurement directly, promotes patient-centered care. There is increasing recognition that the impact of chronic illnesses and their treatment should be assessed in terms of Qol in addition to more traditional measures of medical outcomes i.e. morbidity and mortality (Abdel-Gawad, 2002).

McClish et al (2005), using the SF-36 questionnaire of the Medical Outcome Study compared the health-related quality of life of sickle cell patients to that of those without sickle cell on all the sub-scales (physical functioning, role limitation due to physical health, role limitation due to emotional problems, energy/fatigue, emotional well-being, social functioning, pain and general health) and observed that the former group scored significantly worse than the later group on all the sub scales of the instrument except mental health. However, compared to dialysis patients, sickle cell disease patients scored similarly on physical role and emotional role function, social

functioning and mental health, worse on bodily pain, general health and vitality and better on physical functioning. They concluded that interventions in sickle cell disease should consider improvements in health-related quality of life as important outcomes.

Exercise, is a specific type of physical activity, consisting of structured, repetitive body movements executed to improve or maintain physical fitness (Caspersen et al, 1985). Numerous studies have looked at both the physiological and psychological health benefits of exercise (Williams and Lord, 1997; Turnbull and Wolfson, 2002; Eileen, 2009). People who are physically active substantially lower their risks for coronary heart disease, cerebrovascular disease, hypertension, type II diabetes, overweight and obesity, osteoporosis and deterioration of their functional capacity (Vuori, 2001). The specific goals of an exercise program, in terms of desired health benefits, determines the exercise prescription because different types of exercise programs result in different outcomes. For example, aerobic exercise promotes cardiorespiratory fitness by using the large muscle groups in a continuous, rhythmic fashion (American Institute of Sports Medicine, 1998)

Aerobic exercise or endurance exercise is a subdivision of physical exercise that improves cardiovascular and respiratory health (Eileen, 2009). Moreover, it is generally assumed to increase well-being and reduce negative mood states such as anxiety and depression (Byrne and Byrne, 1993). Generally, it is a cheaper and non toxic treatment modality that could be designed to meet the specific needs of the patient (Vuori, 2001).

There are various kinds of aerobic exercise training. One of these is aerobic dance (Moffet et al, 2000). Verghese et al (2003) reported that dance reduced the risk of dementia. A lot of studies have been conducted on the effects of aerobic exercise on

disease conditions such as (coronary heart disease, rheumatoid arthritis and cancer), but there are few reports of investigations on the response of patients with sickle cell anaemia to aerobic exercise training. Alawale (1998) studied patients attending the Haematology Clinic of the University College Hospital, Ibadan, and reported that the patients benefited from an 8-week aerobic exercise programme, with improved cardiovascular fitness and haematological (Packed Cell Volume and Platelet Count) variables . Two other studies have also surveyed the quality of life (Odusanya, 2005) and health status (Ayeni, 2006) of patients with sickle cell disease in Ibadan and Lagos respectively. However, none of these previous studies investigated the effect of aerobic dance on health status of individuals with sickle cell aneamia. Moreover, none of the previous studies has monitored participants for a long period of time following aerobic exercise training in order to investigate the effect of aerobic exercise on the frequency of crises,frequency of hospitalization and length of hospitalization. This study was therefore designed to investigate the effects of a 12-week aerobic dance exercise on selected cardiorespiratory and heamatological variables, quality of life, frequency of crisis, frequency of hospitalization and length of hospitalization of individuals with sickle cell anaemia.

1.2 STATEMENT OF THE PROBLEM

A comprehensive multidisciplinary approach to the management of patients with sickle cell anaemia produces the best results (Stevenson, 2000). Aerobic exercise has been used in patients with severe rheumatoid arthritis to improve their locomotor ability (Moffet et al, 2000), prevent disability resulting from coronary disease in cardiac patients (Ades, 2001), and improve cardiovascular endurance and function in sickle cell patients (Alawale, 1998). However, none of the studies reported the effects of aerobic exercise

on quality of life, frequency of crisis, frequency of hospitalization and length of hospitalization in patients with sickle cell anaemia. This study was designed to bridge this gap in knowledge by investigating the effects of a 12-week aerobic dance programme on selected cardiovascular and haematological variables in individuals with sickle cell anaemia.The study was designed to answer the following questions:

1) What would be the effects of a 12 week aerobic dance on some cardiorespiratory variables (heart rate, cardio-respiratory fitness score and vital capacity) of individuals with sickle cell anaemia?
2) What would be the effects of a 12 week aerobic dance on some haematological variables (packed cell volume, platelet count and mean corpuscular haemoglobin concentration) of individuals with sickle cell anaemia?
3) What would be the effects of a 12-week aerobic dance on the quality of life of individuals with sickle cell anaemia?
4) What would be the effects of a 12-week aerobic dance on the frequency of crisis, frequency of hospitalization and length of hospitalization in individuals with sickle cell anaemia?

1.3 AIMS OF STUDY

i. To investigate the effects of a 12-week aerobic dance on some cardiorespiratory variables (heart rate, cardio-respiratory fitness score and vital capacity) of individuals with sickle cell anaemia.
ii. To investigate the effects of a 12-week aerobic dance on some haematological variables (packed cell volume, platelet count and mean corpuscular haemoglobin concentration) of individuals with sickle cell anaemia.

iii. To investigate the effects of a 12-week aerobic dance on the quality of life of individuals with sickle cell anaemia.

iv. To investigate the effects of a 12-week aerobic dance on the frequency of crisis, frequency of hospitalization and length of hospitalization of individuals with sickle cell anaemia.

1.4 HYPOTHESES

1.4.1 MAJOR HYPOTHESES

1. There would be no significant differences in cardiorespiratory variables, haematological variables, quality of life, frequency of crises, frequency and length of hospitalization of patients with sickle cell anaemia in the aerobic exercise and control groups at week 0, end of week 6 and end of week 12 of the study.
2. There would be no significant differences in the frequency of crisis, frequency of hospitalization and length of hospitalization of patients in the aerobic dance and control groups at 6 months before and 6 months after the 12-week aerobic dance programme.

1.4.2 SUB HYPOTHESES

1. There would be no significant differences between the heart rate of the experimental and control groups at the three time frames (week 0, 6^{th} week and 12^{th} week) of the study.
2. There would be no significant difference between the cardiorespiratory fitness score of the experimental and control groups at the three time frames of the study.

3. There would be no significant differences between the vital capacity of the experimental and control groups at the three time frames of the study.
4. There would be no significant differences between the packed cell volume of the experimental and control groups at the three time frames of the study.
5. There would be no significant differences between the platelet count of the experimental and control groups at the three time frames of the study.
6. There would be no significant differences between the mean corpuscular haemoglobin concentration of the experimental and control groups at the three time frames of the study.
7. There would be no significant difference between the frequency of crisis of the experimental and control groups at 6 months before the intervention.
8. There would be no significant difference between the frequency of crisis of the experimental and control groups at 6 months after the intervention.
9. There would be no significant difference between the frequency of hospitalization of the experimental and control groups at 6 months before the intervention.
10. There would be no significant difference between the frequency of hospitalization of the experimental and control groups at 6 months after the intervention.
11. There would be no significant difference between the length of hospitalization of the experimental and control groups at 6 months before the intervention.
12. There would be no significant difference between the length of hospitalization of the experimental and control groups at 6 months after the intervention.
13. There would be no significant difference in the heart rate of the experimental group across the three time frames of the study.

14. There would be no significant difference in the cardiorespiratory fitness score of the experimental group across the three time frames of the study.
15. There would be no significant difference in the vital capacity of the experimental group across the three time frames of the study.
16. There would be no significant difference in the heart rate of the control group across the three time frames of the study.
17. There would be no significant difference in the cardiorespiratory fitness score of the control group across the three time frames of the study.
18. There would be no significant difference in the vital capacity of the control group across the three time frames of the study.
19. There would be no significant difference in the packed cell volume (PCV) of the experimental group across the three time frames of the study.
20. There would be no significant difference in the Platelet count of the experimental group across the three time frames of the study.
21. There would be no significant difference in the mean corpuscular haemoglobin concentration of the experimental group across the three time frames of the study.
22. There would be no significant difference in the packed cell volume (PCV) of the control group across the three time frames of the study.
23. There would be no significant difference in the platelet count of the control group across the three time frames of the study.
24. There would be no significant difference in the mean corpuscular haemoglobin concentration of the control group across the three time frames of the study.

25. There would be no significant differences between the quality of life of experimental and control groups at the three time frames of the study.
26. There would be no significant difference in the quality of life of the experimental group across the three time frames of the study.
27. There would be no significant difference in the quality of life of the control group at the three time frames of the study.

1.5 DELIMITATIONS

This study was delimited as follows:

1) Participants: Adolescent and adult sickle cell anaemic patients attending the specialty (Haematology clinic) of Lagos State University Teaching Hospital, Ikeja, Lagos.
2) Cardiorespiratory Variables: Cardiorespiratory fitness score, heart rate and Vital capacity.
3) Heamatological variables: Packed Cell Volume (PCV), Platelet Count (PC) and Mean Corpuscular Haemoglobin Concentration (MCHC).
4) Quality of life: This was measured using the Short-Form-36 Health survey (SF-36) Version 1.0 of the RAND Medical Outcome Study (MOS).

1.6 INCLUSION CRITERIA

The study involved Sickle cell anaemia patients who were screened by the referring physician, certified fit (absence of any symptoms of dyspnea, fainting and syncope after performing the step test for 3 minutes) by the researcher and were English literate.

1.7 EXCLUSION CRITERIA

The following categories of sickle cell disease patients were excluded from the study:

1. Sickle cell anaemia patients who were in sickle cell crisis at the time of recruitment into the study.
2. Sickle cell anaemia patients who had medical complications associated with sickle cell disease such as joint problems, priapism, kidney disease, stroke, retinopathy, acute chest syndrome, chronic anemia.
3. Sickle cell anaemia patients who were pregnant.
4. Sickle cell anaemia patients who were not English literate.

1.8 LIMITATIONS

In the absence of a disease-specific quality of life instrument, a generic questionnaire (MOS SF-36 version 1.0) was used. This might have affected the finding of this study on quality of life. However, the generic instrument used in this study have been found to be valid for use in a previous study on individuals with sickle cell anaemia by McClish et al (2005).

1.9 SIGNIFICANCE OF STUDY

1. The outcome of this study provided an insight into aerobic exercise as an intervention for patients with sickle cell anaemia.
2. The outcome of this study showed that patients with sickle cell anaemia could participate in carefully prescribed and closely monitored aerobic exercise programmes without adverse clinical effects.

3. The outcome of this study should encourage parents of patients with sickle cell anaemia to change their orientation towards their wards participating in monitored aerobic exercise programme, without fear that exercise could trigger a crisis in patients with sickle cell anaemia.
4. The outcome of this study should encourage Physicians to refer their sickle cell anaemia patients to the Physiotherapist not only when certain complications such as bone problems occur, but as part of the wholistic treatment and improving the quality of life of sickle cell anaemia patients.
5. The outcome of this study should also encourage schools to allow patients with sickle cell anaemia to take part in monitored aerobic exercise in order to prevent the sickle cell patients from developing psychosocial problems and stigmatization by peers.
6. The outcome of this study showed that aerobic dance could be prescribed as an enjoyable home programme for suitable patients with sickle cell anaemia. since music and dance have health enhancing effects on social and mental wellbeing of human beings generally, therefore, aerobic dance could prevent sickle cell patients from falling into depressed mood.

1.10 OPERATIONAL DEFINITION OF TERMS

1. **Frequency of Crisis**: This was the number of times the participants suffers from crises 6 months before and 6 months after this study.
2. **Frequency of hospitalization:** This was the number of times the participants were hospitalized 6 months before and 6 months after this study.

3. **Length of hospitalization:** This was the duration of time in days, during which the participant was on admission in the hospital, 6 months before and 6 months after this study. It included daycare admission.

CHAPTER TWO

REVIEW OF THE LITERATURE

2.1 DEFINITION OF SICKLE CELL ANAEMIA

Sickle cell anaemia (SCA) is an inherited disorder of haemoglobin which occurs in any person who has inherited sickle haemoglobin (HbS) from each parent. (Fleming and Lehmann, 1982; Akinyanju and Olujohungbe, 2007). Sickle cell anaemia is a pathological condition characterized by the presence of HbS (Scutellari et al, 2003). Sickle cell haemoglobin (Hb S) is the commonest and most important haemoglobin variant known in humans (Akinyanju and Olujohungbe, 2007). It is caused by a point mutation in the β-globin chain of haemoglobin, replacing the glutamic acid with the less polar amino acid valine at the sixth position of the β chain (Swarup et al, 2009). This mutation causes the sickle cell haemoglobin to form elongated rigid rods which can block small blood vessels, impairing blood flow. This condition leads to shortened red blood cell survival, and subsequent anaemia, often called sickle cell anaemia (WHO, 2006).

2.2 HISTORY OF SICKLE CELL ANAEMIA

Sickle Cell Anaemia (SCA) is one of the commonest genetic disorders world-wide and the most common inherited haematological disease affecting humans (WHO, 2006). There is some evidence that sickle cell disease had been recognized in Africa by black Africans long before the earliest descriptions in the medical literature at the beginning of the twentieth century (Fleming, 1982; Desai and Dhanani, 2004). Also, the history of the condition tracked reports back to 1670 in one Ghanaian family of the Krobo tribe by Dr Konotey-Ahulu (Konotey-Ahulu, 1974; Desai and Dhanani, 2004).

The various ethnic groups in Ghana, including the Twi, Ewe and Ga peoples, identified sickle cell disease as an entity to the extent of pinpointing some cardinal features, such as the recurrent attacks of pain in the bones and joints, the variable severity of the disease and its familial tendency, but with the parents of affected children appearing normal (Fleming, 1982). In Nigeria, sickle cell anaemia apparently remained unrecognised as a distinct disorder by the various ethnic groups until recent times. The Nigerian languages abound with words and phases which describe various symptoms commonly found in patients with sickle cell disease. In the Hausa language for example, expressions such as "rashin jinni" (lack of blood), "ciwon ga' bo bi sai sai" or "amosanin kasha" (recurrent pain in the bones and joints) and "rashin kuzari (lack of energy) are frequently used in relation to sickle cell disease by patients, their relatives and by traditional healers. Age-old concepts of "Ogbanje" (Ibo) and "Abiku" (Yoruba) provide a traditional explanation for recurrent deaths of children in some families (Fleming, 1982). The unequivocal and incontrovertible evidence that sickle cell disease was identified as a distinct abnormality in the traditional societies of Nigeria before it's recognition in 'Western' medicine is lacking (Fleming, 1982). However, the earliest descriptions in the Western medical press were inevitably based on features of the disorder as observed in the descendants of Africans (predominantlyWest Africans) living in the new world (Fleming, 1982). In 1904, Dr James B. H (1861-1954) a physician in Chicago, U.S.A, first noted the presence of cells which were shaped like a sickle in the blood of an anaemic West Indian medical student. This historic observation linked the 'peculiar and elongated sickle shaped red blood cells' with severe anaemia (Desai and Dhanani, 2004). The disease was named "sickle cell anaemia" by Vernon Mason in 1922 (Akinyanju and Olujohungbe, 2007).

The genetic basis for sickle cell anaemia was established by Linus Carl Paulings and coworkers in 1949. They reported that sickle cell anaemia was associated with an alteration of haemoglobin. This was the first time a genetic disease was linked to a mutation of a specific protein, which was a milestone in the history of molecular biology (Akinyanju and Olujohungbe, 2007).

The origin of the mutation that led to the sickle cell gene was initially thought to be in the Arabian Peninsula, spreading to Asia and Africa. It is now known, from evaluation of chromosome structures, that there have been at least four independent mutational events, three in Africa and a fourth in either Saudi Arabia or Central India. These independent events occurred between 3,000 and 6,000 generations ago, approximately 70,000-150,000 years (Desai and Dhanani, 2004).

2.3 EPIDEMIOLOGY

Sickle cell anaemia is the most commonly known hereditary blood disorder (Midence and Elander, 1994). It is estimated that each year over 300,000 babies are born with sickle cell anaemia worldwide, and that approximately 5 percent of the world's populations are carriers of a trait gene for sickle cell anaemia. Sickle cell anaemia is most prevalent in tropical regions; though population migration has spread the disease to most countries (WHO, 2006). Ohaeri and Shokunbi (2002) also reported that, the disease afflicts up to 100million people world wide, predominantly black people in Africa, Europe, America, and those of Asian ancestry. About 0.15 percent of black children in the United States have sickle cell anaemia. The prevalence is lower among adults because patients with SCA have a lower life expectancy (Desai and Dhanani, 2004).

In Nigeria, it was discovered that, young children between the ages of 2 and 4 years have the highest incidence of SCA with a gradual but progressive decline in the incidence of SCA in the age range 8-16 years (Jawal et al, 2003). WHO (2006) reported that 24% of the Nigerian population are carriers of the mutant gene and the prevalence of sickle-cell anaemia is about 20 per 1000 births. This means that in Nigeria alone, about 150 000 children are born annually with sickle-cell anaemia. Ohaeri and Shokunbi (2002) reported that the incidence of HbSS was about 2% and that of HbSC was approximately 0.7% in Nigeria. The gene has persisted because heterozygous gain a slight protection against the falciparum malaria (Akinyanju and Olugbohungbe, 2007).

It was estimated that the population of people with the sickle cell trait was 8% among black people in the United States of America and between 10% and 40% across equatorial Africa and decreases to between 1% and 2% on the north African coast and <1% in South Africa. This distribution reflects the fact that sickle-cell trait confers a survival advantage against malaria and that selection pressure due to malaria has resulted in high frequencies of the mutant gene especially in areas of high malarial transmission (WHO, 2006). In West African countries such as Ghana and Nigeria, the frequency of the trait is 15% to 30% whereas in Uganda it shows marked tribal variations, reaching 45% among the Baamba tribe in the west of the country (WHO, 2006).

2.4 PATHOGENESIS AND PATHOPHYSIOLOGY OF SICKLE CELL ANAEMIA.

Haemoglobinopathies are characterized by the production of structurally defective haemoglobin due to abnormalities in the formation of the globin parts of haemoglobin (Shah, 2004). Sickle haemoglobinopathies are the genetically determined conditions characterized by structurally abnormal haemoglobin variants; giving rise to haemolytic

disease by virtue of their property to polymerize and assume crescent shapes (Desai and Dhanani, 2004). If two parents (HbAS) who are carriers have a child, there is a 1-in-4 chance of their child developing the disease (HbSS) and a 1-in-2 chance of their child just being a "carrier" (HbAS) (Desai and Dhanani, 2004).

Heterozygotes have a higher resistance to malaria; this is known as "heterozygote advantage" (Desai and Dhanani, 2004). Under conditions of low oxygen tension, HbS molecules tend to polymerize, or "stack" which converts the HbS from a fluid state to a viscous or gel state. Stiff rods of HbS are formed; leading to the deformation of red cells into an elongated or sickle shape. Polymerization will only take place if a cell contains predominantly HbS molecules. Dilution of HbS with normal HbA makes the red blood cells fairly resistant to sickling, because polymerization is a result of reactions between HbS molecules. An HbS and an HbA molecule do not "bond" in the same way as two HbS molecules. The red blood cell of a SCA person generally contains between 80% and 95%HbS, while that of an individual with sickle cell trait (AS) has between 40% and 45% (Desai and Dhanani, 2004).

However, sickling can occur under certain extreme conditions in people with SCA trait. These changes are more likely to supervene when the haemoglobin is deoxygenated particularly with homozygous S haemoglobin. Relatively slight reduction in oxygen tension causes HbS to crystallize, so contouring the erythrocyte into the characteristic sickle shape. The sickling leads to intravascular stasis and occlusion (Ayeni, 2006).

2.5 TYPES OF HAEMOGLOBINOPATHIES.

Disorders of synthesis of haemoglobin are invariably inherited and resulted in either structurally abnormal haemoglobins or quantitative defects of normal haemoglobins (WHO, 2006).

1. SICKLE CELL HAEMOGLOBIN (HbS) β6 GLUTAMIC ACID->VALINE

Sickle cell haemoglobin (HbS), the commonest abnormal haemoglobin, is formed by the substitution of valine for glutamic acid in the sixth position of the β chain (Fleming, 1982). It is generally the most common and the most severe form of the disease (Smeltzer and Bare, 2003; WHO, 2006). Its prevalence in relation to other forms of sickle cell anaemia falls with age and occurs mostly in black people, but has also been frequently found around the Mediterranean Sea and in India (Jawal et al, 2003). The mutation leads to prompt polymerization of the variant upon deoxygenation through the extensive formation of strong hydrophobic bonds, mainly between the newly inserted valine and the β chains of the adjacent haemoglobin molecules. This leads todeformation of the red blood cells, which acquire the sickle cell shape and hinder the microcirculation of blood (Loukopoulos, 2002).

2. HAEMOGLOBIN C β6 GLUTAMIC ACID->LYSINE

This is the next most common abnormal haemoglobin, lysine is substituted for glutamic acid in the sixth position of the β chain (Fleming, 1982; Loukopoulos, 2002). The homozygosity for this variant is associated with decreased solubility, target-shaped red blood cells and haemolytic anaemia

(Loukopoulos, 2002). This condition is confined to the people of West African ancestry (WHO, 2006).

3. HAEMOGLOBIN D β121 GLUTAMIC ACID->GLUTAMINE.

The D-haemoglobins include (D-Ibadan, D-Los Angeles or Punjab and D-Iran) This variant is frequent among the Punjabs in India. It is not usually associated with any clinical manifestations (Fleming, 1982).

4. HAEMOGLOBIN E β26 GLUTAMIC ACID->LYSINE

This is the third most prevalent abnormal haemoglobin in the world. It is found mainly in Asia, though it has been described occasionally in blacks (Fleming, 1982). Lysine is substituted for glutamic acid in the twenty sixth position of the β chain. The variant chain is associated with mild instability and a slightly reduced rate of synthesis. As a result, homozygosity for the variant causes mild haemolytic anaemia, which for reasons not yet completely understood, becomes very severe in compound β^E / β-thalassaemia heterozygotes (Loukopoulos, 2002).

5. HAEMOGLOBIN M

In this condition, the carriers are clearly cyanotic because half of their haemoglobin chains are oxidized to met-haemoglobin, which has a bluish colour. The transport of oxygen is not grossly impaired and the carriers have no major problems. The variants include HbM Boston: α 58 (E7) histidine->tyrosine, Hb

Hyde Park: β92 (F8) histidine-> tyrosine, Hb Saskatoon: β63 (E7) histidine->tyrosine and Hb Milwaukee: β67 (E11) valine->glutamic acid. In some instances the variants are also unstable (Loukopoulos, 2002).

6. HAEMOGLOBIN O Arab β121 GLUTAMIC ACID->LYSINE.

This is frequent among the Pomacs, probably representing the ancient Thracian tribe in Greece and Bulgaria. Its homozygous state is associated with mild haemolytic anaemia, which is aggravated in compound β^0/β-thalassaemia heterozygotes (Loukopoulos, 2002).

7. THALASSAEMIAS

Inherited quantitative defects in the synthesis of globin chains give rise to the thalassaemia syndromes. There are two major types of Thalassaemias, alpha and beta, which are named after the two protein chains that make up normal haemoglobinrms (WHO, 2006). The α-thalassaemia syndromes arise when there is a genetically-determined impairment of the rate of synthesis of α chains; as a result there is an excess of γ chains in the prenatal period and of β chains in the post natal period.. The excess γ chains form a tetramer called Hb Barts (γ_4) and excess β chains form Hb-H (β_4). In thalassaemia disorders, the rate of β chain synthesis is impaired with consequent excess formation of α chains, which do not, however, form any haemoglobin tetramer; but there is ineffective erythropoiesis and impaired production of Hb-A (Fleming, 1982). As a rule, carriers of these mutations display a low mean corpuscular haemoglobin concentration (MCH)

(the red cell haemoglobin concentration is lower than normal) but no other findings or symptoms (Loukopoulos, 2002).

2.6 CLINICAL FEATURES AND COMPLICATIONS OF SICKLE CELL ANAEMIA.

Though the condition is inherited; clinical manifestations begin only after several months. This is because HbF that is present at birth, protects against sickling (Shah, 2004). The clinical manifestations and complications are anaemia, vaso-occlusive phenomenon, chronic organ damage, pain crisis (bone, abdominal and chest pain**),** hand and foot syndrome, chronic leg ulcers (usually in adolescents and adults), pulmonary disease, neurological disease, gall stone, frequent urination, fatigue, increased susceptibility to infections, fever, jaundice (paleness, yellow eyes and/ or skin), poor eye sight or blindness, priapism, rapid heart rate, delayed growth and puberty, excessive thirst, sepsis e.t.c. (Shah, 2004).

1. **Anaemia or Aplastic Crisis:** Sickle cell anaemia patients have severe haemolytic anaemia with Haematocrit values between 18 and 30%. The destruction of red blood cells is independent of cell age (Baker and Silverton, 2001), with the mean red blood cell survival being 10-15 days. Those cells having relatively low level of fetal haemoglobin (HbF) have shorter life span; partly due to a greater chance of becoming irreversibly sickled. The anaemia becomes increasingly severe if erythropoiesis is suppressed. There are two main causes of this condition namely: infection and folic acid deficiency (Smeltzer and Bare, 2003).

2. **Vaso-occlusive Phenomenon:** Polymerization of sickling haemoglobin (HbS) molecules within the red cells raises the internal viscosity and reduces compliance of the cell membrane so that the HbS containing cells are less able to negotiate capillary beds and thus leading to premature red blood cells destruction (haemolysis) and obstruction of blood flow (Vaso-occlusion) (Ayeni, 2006).
3. **Chronic Organ Damage:** By the time SCA patients reach adulthood, there is often objective evidence of anatomic or functional damage to various tissues, due to cumulative effect of recurrent vaso-occlusive episodes. Almost any organ may be involved, but most commonly the lungs, kidney, liver; skeletal system and the skin are so affected (Akinyanju and Olujohungbe, 2007).
4. **Hand and Foot Syndrome:** These problems occur during infancy. It presents as swelling of the dorsum of the hand and foot. Cortical thinning and destruction of the metacarpal and metatarsal bones appear on radiographs 3-5 weeks after the swelling begins. The swelling is not accompanied with leukocytosis or erythyma ((Akinyanju and Olujohungbe, 2007).
5. **Chronic Leg Ulcers:** One of the symptoms of sickle cell disease is the presence of indolent leg ulcer which is characteristically over the malleoli. The ulcers are usually on the lower one third of the leg, above the ankle and on the medial side. The ulcers start as infarcts in the skin, causing small

blisters like lesions; these later develop into necrotic sloughs (Adedoyin et al, 2001).

6. **Musculoskeletal Symptoms:** Skeletal maturity is also delayed but they do have growth spurt and indeed attain average or above average heights-tall thin young men or girls with tall lower limbs. Also, the blood supply to the connective tissues, especially in the hip and shoulder joints can be blocked by the sickle cells resulting in bone damage and poor healing (Balogun et al, 2010).
7. **Cardiopulmonary disease:** The heart and the lungs are affected in several ways by the pathological processes of sickle cell anaemia. Most patients with sickle cell anemia develop abnormal pulmonary function characterized by airway obstruction, restrictive lung disease, abnormal diffusing capacity, and hypoxemia and pulmonary hypertension (Soyanwo et al, 1999). Pulmonary complications account for a large proportion of deaths among adults with sickle cell anaemia (Platt et al, 1994).
8. **Neurological disease:** Sickle cell anaemia is a blood disorder; however, the central nervous system (CNS) is one of the organs frequently affected by the disease (Schatz and McClellan, 2006). Brain disease can begin early in life and often leads to neurocognitive dysfunction. Schatz and McClellan, (2006), in their study on the effect of SCA on neurocognition stated that approximately one-fourth to one-third of children with SCA have some form of CNS sequelae from the disease, which typically manifest as deficits in specific cognitive domains and academic difficulties.

9. **Gallstones (Cholelithiasis):** Gallstones are small stones, usually made of cholesterol, that form Gallstones are small stones, usually made of cholesterol, that form in the gallbladder. In sickle cell anaemia, This causes the urine produced to be dark in colour or blood stained, a condition known as haematuria (Akinyanju and Olujohungbe, 2007).
10. **Jaundice:** This is caused as a result of increased rate of haemolysis. It is also known as icterus. It is manifested by yellowing of the skin, conjunctival and mucous membranes as a result of increased level of bilirubin in the human body. Usually the concentration of bilirubin in the blood must exceed 2-3mg/dl for the coloration to be easily visible (Akinyanju andOlujohungbe, 2007).
11. **Priapism:** This is only found in males. It is a condition characterized by a persistent and painful erection. Blood vessels become blocked by sickle cells so that blood is trapped in the tissue of the male's organ. It is extremely painful and can result in damage to this tissue causing impotence (Akinyanju and Olujohungbe, 2007).
12. **Retinopathy:** The blood vessels that support the tissue at the back of the eye may be blocked by sickle cells thereby resulting in retinopathy. Regular ophthalmology evaluations and effective treatment can help a person avoid permanent damage to their vision (Akinyanju and Olujohungbe, 2007).
13. **Stroke:** In Europe and America, about one in twelve children with sickle cell anaemia has a stroke. In Africa the proportion is not known, however in Nigeria some studies reported one in fifty (2%) (Akinyanju and

Olujohungbe, 2007; Kehinde et al, 2008). The average age at onset is 6 years but it could affect a child as young as 2 years (Akinyanju and Olujohungbe, 2007).

14. **Sepsis:** This is an ever present danger in sickle cell anaemia and it is frequently the initiating factor in a crisis. Crisis may be precipitated by malaria, bacterial or viral infections (Akinyanju and Olujohungbe, 2007).

2.7 SICKLE CELL CRISES

2. 7. 1 PAINFUL CRISIS

Acute episodes of musculoskeletal pain are the most prevalent symptomatic manifestations of sickle cell anaemia and accounts for most hospital admissions (Sickle Cell Information Centre, 2008). Painful crisis primarily often affects the lumbar spine, abdomen and femoral shaft, but distal parts of the limbs are affected. Recurrences may or may not involve similar sites on successive occasions (Kehinde et al, 2008). Several factors are known to precipitate painful crisis, but majority of attacks are unpredictable and unexplained. Acidosis, hypoxia, and dehydration are known to trigger a crisis. Painful crises usually occur in association with acute infection. Painful crisis do not cause physical changes, functional impairment or focal infarction (Platt et al, 1994).

2. 7. 2 ABDOMINAL CRISES

Small infarcts in the mesentery and abdominal viscera are caused by vasoocclusion by clumps of sickled cells. Abdominal crises can mimic any of the acute abdominal emergencies and fever, rebound tenderness and leucocytosis may occur. Observant patients can usually distinguish sickle crises from other abdominal symptoms.

However differential diagnosis of sickle cell crises from acute abdominal disorders requiring surgical intervention can be carried out (Akinyanju and Olujohungbe, 2007).

2. 7. 3 HAEMATOLOGIC CRISES

Hematologic crisis happens if the spleen's outflow of blood is obstructed, leading to splenic sequestration of blood and splenic enlargement (splenomegaly). Malfunction of the spleen may also make individuals more susceptible to infections from encapsulated organisms such as Haemophilus influenzae type B (HIB), a highly contagious bacterium that causes meningitis, pneumonia, septic arthritis, and cellulitis. Another hematologic crisis, aplastic anemia, can occur when the bone marrow ceases making red blood cells (Platt et al, 1994).

2.7.4 BONE AND JOINT CRISES

In older children and adults, bone or joint crises occur sporadically throughout life in the limbs, spine, ribs and periarticular structures. The most frequently affected sites are the marrow of the humerus, tibia and femur. Tenderness and swelling develop over the site of infarction and pain is intense. Most patients develop little fever and local warmth. Erythema occurs only if the bony lesion is superficial. There will be no radiological changes until resolution and demineralization of cortical bone have begun (Balogun et al, 2010).

2.8 DIAGNOSIS.

The inheritance of SCA or sickle cell trait cannot be prevented but can be predicted, so screening is recommended (McPherson and Pincus, 2007). In the diagnosis of SCA in any black patient with haemolytic anaemia, history of painful crisis, arthroplasty, ankle ulcers, priapism, retinopathy, stroke, jaundice e.t.c can be very helpful (Sickle Cell Information Center, 2008). The diagnosis is made by the presence of a positive sickle cell solubility test (Sickledex), demonstrating that the patient's red cells sickle at low oxygen tension. However, this test does not tell the difference between sickle cell trait and sickle cell anaemia. Another test called haemoglobin electrophoresis is needed to differentiate between the two disease entities (McPherson and Pincus, 2007). Other tests include Slide Elution test, Automated Dithionite, the Techicon System and Sodium Metabisulphate test (Shah, 2004). Risk factors associated with these tests are excessive bleeding, fainting or feeling light-headed, haematoma, infection and multiple punctures to locate veins (McPherson and Pincus, 2007).

2.9 PRECAUTIONS AND TREATMENT

Sickle cell anaemia patients can stay as healthy as possible by taking the following precautions:

1. Eating a balanced, healthy diet (Fahner, 2007).
2. Taking vitamins, including folic acid supplements, as prescribed (Fahner, 2007).
3. Drinking plenty of fluids to prevent dehydration (Fahner, 2007).
4. Avoiding extreme cold or heat (Fahner, 2007).

5. Exercising regularly, but in moderation. Exercise is important for staying healthy, but overdoing it can trigger a crisis in some people, particularly if they become dehydrated, overheated, or exhausted (Fahner, 2007).
6. Getting plenty of rest (Fahner, 2007).
7. Avoiding alcohol, hard drugs, and smoking, which can aggravate sickle cell anaemia and its symptoms. Some people with sickle cell anaemia are prone to lung problems, so smoking is particularly risky and must be avoided (Fahner, 2007).
8. Avoiding places that are low in oxygen. (For example it is not a good idea to go hiking at high altitudes or spend lots of time swimming under water (Fahner, 2007).
9. Preventing serious infections by contacting the doctor as soon as illness symptoms start and ensuring they get immunizations (such as pneumonia and flu vaccines) that the doctor recommends (Fahner, 2007).
10. Learning as much as they can about the disease and keeping regular appointments with the doctor to help prevent complications (Fahner, 2007).

Although there is no cure for sickle cell anaemia, health professionals can do a great deal to help patients. Basic treatment of painful crises relies heavily on pain-killing drugs and intravenous fluids to reduce pain and prevent

complications. Regular health maintenance is critical for people with sickle cell anaemia. Proper nutrition, good hygiene, bed rest, protection against infections and avoidance of other stresses all are important in maintaining good health and preventing complications. Regular visits to a physician or clinic that provides comprehensive care are necessary to identify early changes in the patient's health and ensure immediate treatment (Sickle Cell Information Centre, 2008). The treatment options include the following:

1. **Blood Transfusions:** Blood transfusions correct anaemia by increasing the number of normal red blood cells in circulation. They can also be used to treat spleen enlargement in children before the condition becomes life-threatening. Regular transfusion therapy can help prevent recurring strokes in children at high risk (National Institute of Health, 2007).
2. **Oral Antibiotics:** Giving oral Penicillin twice a day beginning at 2 months and continuing until the child is at least 5 years old can prevent pneumococcal infection and early death (National Institute of Health, 2007; Akinyanju and Olujohungbe, 2007).
3. **Hydroxyurea:** The first effective drug treatment for adults with severe sickle cell anaemia was reported in early 1995, when a study conducted by the National Heart, Lung and Blood Institute in U.S.A showed that daily doses of the anticancer drug hydroxyurea reduced the frequency of painful crises and acute chest syndrome (National Institute of Health, 2007; Akinyanju and Olujohungbe, 2007).

4. **Stem Cell Transplant:** This procedure uses a mixture of the patient's bone marrow and a donor's bone marrow that combine to form healthy new blood cells. It allows for a patient's bone marrow not to be destroyed. The procedure was first carried out in a 12-year old with sickle cell anaemia named Keone Penn of Snellville, Georgia, U.S.A. who also suffered a stroke when he was 5 years old. (National Institute of Health, 2007; Akinyanju and Olujohungbe, 2007).
5. **Zinc administration:** Zinc is given as it stabilizes cell membrane (National Institute of Health, 2007).
6. **Analgesics administration:** Most people with sickle cell anaemia have intensely painful episodes called vasso-oclussive crises. Painful crises are treated symptomatically with analgesic at regular intervals until the crisis has settled. NSAIDS (such as diclofenac or naproxen) are used for milder crises while intravenous opioids are used for managing more severe crises. (National Institute of Health, 2007; Akinyanju and Olujohungbe, 2007).

2.10 PREVENTION OF SICKLE CELL ANAEMIA (WHO, 2006)

Sickle cell anaemia can be prevented. Couples at risk of having affected children can be identified by inexpensive and reliable blood tests; chorionic villus sampling from nine weeks of pregnancy can be performed for prenatal diagnosis. Adoption of such measures goes hand-in-hand with health education. However, prenatal diagnosis can raise ethical questions which differ from one culture to another. Experience has clearly shown that genetic counseling coupled with the offer of prenatal diagnosis can lead to a large-

scale reduction in births of affected children. The risk of having affected children can be detected before marriage or pregnancy; however, to do so requires a carrier screening programme. There is extensive experience with such programmes in low- and high-income countries. For example, in the case of thalassaemia prevention, unmarried people in Montreal (Canada) and the Maldives are offered screening, premarital screening is national policy in Cyprus and the Islamic Republic of Iran, and pre-reproductive screening is emphasized in Greece and Italy. These approaches should be practiced in conformity with the three core principles of medical genetics:

1. The autonomy of the individual or the couple
2. Their right to adequate and complete information
3. The highest standards of confidentiality.

Neonatal diagnosis allows provision of simple protective measures, including information for the parents, prophylaxis with penicillin and antimalarial treatment, all giving a better quality of life for the affected children (WHO, 2006).

At present, a large proportion of the African population receives no attention or care for this disease. As with all chronic disorders, improved management creates a cumulative demand for more services. Surveillance and education must be delivered at the community level through the primary health-care system so as to increase public awareness of the problem and lengthen the survival of affected individuals (Akinyanju and Olujohungbe, 2007; Balogun et al, 2010).

2.11 THE RED BLOOD CELL INDICES

Red blood cell indices are measurements that describe the size and oxygen-carrying protein (hemoglobin) content of red blood cells. The indices are used to help in the differential diagnosis of anemia. They are also called red cell absolute values or erythrocyte indices (Jacobs, 1996; Baker and Silverton, 2001). The indices include the following: mean corpuscular volume (MCV); mean corpuscular hemoglobin (MCH); mean corpuscular hemoglobin concentration (MCHC); and red cell distribution width (RDW). They are usually calculated by an automated instrument as part of a complete blood count.

(i) Mean corpuscular volume (MCV)

MCV is the index most often used. It measures the average volume of a red blood cell by dividing the hematocrit by the RBC. The MCV categorizes red blood cells by size. Cells of normal size are called normocytic, smaller cells are microcytic, and larger cells are macrocytic. These size categories are used to classify anemias. Normocytic anemias have normal-sized cells and a normal MCV; microcytic anemias have small cells and a decreased MCV; and macrocytic anemias have large cells and an increased MCV. Under a microscope, stained red blood cells with a high MCV appear larger than cells with a normal or low MCV. Normal value is 82-98 fl (femtoliters) (Jacobs, 1996).

(ii) Mean corpuscular hemoglobin concentration (MCHC)

The MCHC measures the average concentration of hemoglobin in a red blood cell. This index is calculated by dividing the hemoglobin concentration by the hematocrit. The

MCHC categorizes red blood cells according to their concentration of hemoglobin. Cells with a normal concentration of hemoglobin are called normochromic; cells with a lower than normal concentration are called hypochromic. Because there is a physical limit to the amount of hemoglobin that can fit in a cell, there is no hyperchromic category. Just as MCV relates to the size of the cells, MCHC relates to the color of the cells. Hemoglobin contains iron, which gives blood its characteristic red color. When examined under a microscope, normal red blood cells that contain a normal amount of hemoglobin stain pinkish red with a paler area in the center. These normochromic cells have a normal MCHC. Cells with too little hemoglobin are lighter in color with a larger pale area in the center. These hypochromic cells have a low MCHC. Anemias are categorized as hypochromic or normochromic according to the MCHC index. Normal value is 32-39 g/dl ((Jacobs, 1996).

(iii) Mean corpuscular hemoglobin (MCH)

The average weight of hemoglobin in a red blood cell is measured by the MCH. The formula for this index is the sum of the hemoglobin multiplied by 10 and divided by the RBC. MCH values usually rise or fall as the MCV is increased or decreased. Normal value is 26-34 pg (picograms) (Jacobs, 1996).

(iv) Red cell distribution width (RDW)

The RDW measures the variation in size of the red blood cells. Usually red blood cells are a standard size. Certain disorders, however, cause a significant variation in cell size. Normal value is 11.5-14.5% (Jacobs, 1996; Pagana and Pagana, 1998).

2.12 EXERCISE

Exercise, is a specific type of physical activity, consisting of structured, repetitive body movements executed to improve or maintain physical fitness (Caspersen et al, 1985). The benefits of exercise include weight reduction, stress reduction, reduced risk of heart disease, improved lung function and improved quality of life (McArdle, 2000). Exercise usually involves three phases.

1. **Warm up phase:** Warming up is necessary before any strenuous activity as it stretches and loosens the body muscles. Warming-up prevents muscle cramps, muscle fatigue and injuries due to sudden jerk. The body could be warmed up in several ways such as walking for about three minutes, climbing the stairs or just step-touch backward and forward, moving the arms one by one in circular motion and jumping for about five minutes. The body ready for the next period when it is warm and loosened up (McArdle, 2000).
2. **Work out phase:** This is the period to perform the main exercise routine. It could last for 10-60 minutes depending on the individuals fitness level.
3. **Cool down phase:** This is the last phase of the exercise programme. This usually involves very gentle movements that would relax the muscles. Some of the exercise movements in the warm up phase can also be performed for five minutes (McArdle, 2000).

2.12.1 PRINCIPLES OF EXERCISE TRAINING

Proper exercise training induces biologic adaptations that improve performance in specific tasks. Effective physiologic conditioning requires adherence to carefully planned

and executed activities (McArdle, 2000). Attention focuses on frequency, duration, type of training, speed, intensity, repetition of the activity and appropriate competition. These factors vary depending on the performance goal (McArdle, 2000). The following principles should guide exercise training:

1. **Overload Principle:** To increase physical fitness through exercise, the body must perform more work per unit of time than it is accustomed to performing. This increase work load combined with the resultant improvement in physical fitness is known as the overload principle (Wilson et al, 1987; McArdle, 2000). Appropriate combinations of training frequency,intensity and duration provide individualized and progressive overload for exercise training of the athlete, sedentary person, disabled individual and even cardiac patient (McArdle, 2000).
2. **Specificity:** This refers to adaptations in metabolic and physiologic systems that depend on the type of overload imposed. For instance, exercise stress like strength/power training, induces specific strength/power adaptations. It also encompasses activities with identical metabolic components. For example aerobic fitness for swimming, running or rowing improves most effectively when the exercise trains the specific muscles required for the specific activity (McArdle, 2000).
3. **Individual adaptation:** Many factors contribute to variations in training responses among individuals. One important factor is the person's relative fitness level and state of training at the start of a conditioning programme and thus may respond to the same training stimulus differently (McArdle, 2000).

4. **Reversibility:** Detraining occurs rapidly when a person stops exercising. After a week or two of detraining, measurable reductions occur in physiologic function and exercise capacity with a total loss of training improvements within several months (McArdle, 2000).

2.12.2 COMPONENTS OF EXERCISE TRAINING

Exercise dosage is a combination of intensity, frequency and duration of effort for the purpose of providing a safe and effective exercise programme (Wilson et al, 1987).

Intensity: The most important component of the exercise prescription is training intensity which is expressed as a percentage of aerobic capacity or maximal heart rate (Wilson et al, 1987). McArdle, (2000) recommended that for developing and maintaining cardiorespiratory fitness in healthy adults, training intensity of between 60 and 90% of maximal heart rate reserve (50 and 85% of VO_2 max) at a training frequency of between 3 and 5 days per week, and training session duration of 15 to 60 minutes should be adopted.

Frequency: Exercise frequency is expressed in number of days per week and varies with the component being developed and the individual's fitness goals. For instance for a favourable cardiorespiratory changes to occur and maintained a minimum number of days is required for engagement in an exercise programme. In most studies, a frequency of three to five days per week is recommended for the improvement of cardiorespiratory function and two to three days per week are needed for retention. (McArdle, 2000).

Duration: Most studies indicate that the duration of exercise necessary for training adaptation of the cardiovascular system is between 20 and 30 minutes per session

(McArdle, 2000). Nevertheless, it has been observed that shorter programmes, between 5 to 10 minutes, have also produced favourable results particularly for individuals with a lower level of physical fitness at the outset (Wilson, 1987). The number of weeks of training is also important in assessing effectiveness of the programme. Training changes may appear in 4 to 6 weeks especially with high intensity exercise (McArdle, 2000).

Mode: Evidence indicates that as long as intensity, duration and frequency of training are adequate and follow sound training principles, the mode of exercise can be variable. The mode of exercise is determined in part by the objectives of the programme (McArdle, 2000).

2.12.3 PHYSIOLOGICAL ADAPTATION TO EXERCISE TRAINING

Adaptations are essentially changes which accompany various types of physical movement. As the body progresses through days, weeks and months of training, its structure and function change to accommodate the consistent workload. These persistent changes are the adaptations that enable the body to respond more favourably to subsequent training sessions. (McArdle, 2000). The main physiological adaptations to exercise occur in the heart, lungs and muscles (McArdle, 2000).

Heart and Circulatory System: The major function of the cardiovascular system during exercise is to deliver blood to the active tissues, which includes the delivery of oxygen and nutrients and the removal of the metabolic waste products (Wilmore, 1982). Exercising can affect the cardiovascular system in different ways depending on the health status of the exerciser and the type of exercise that is being executed (Roberts, 2008). The heart rate has been reported to be the simplest and one of the most informative of the

cardiovascular parameters that can be measured. In sedentary deconditioned individuals, the resting heart rate can exceed 100 beats per minute, while in highly conditioned, endurance individuals, resting heart rates have been reported to be 28-40 beats per minute (Wilmore, 1970). The main heart adaptation is an increase in size as it works to accommodate a greater cardiac output from session to session. As a result, an individual's heart is slower at rest. The area of the heart which undergoes the greatest change is the left ventricle which is responsible for pumping blood to the body (McArdle, 2000). The exercise-lowering effect of blood pressure is as a result of reduced sympathetic nervous system hormones (catecholamines) with training. This response decreases peripheral vascular resistance to blood flow, causing blood pressure to decrease (Martin, 1997).

Pulmonary Adaptations: The harder the muscles work during a training session, the more oxygen they need. This implies that more air must be taken into the lungs and as the level of fitness increases the lungs become more adept at ventilating larger volumes of air during intense physical exertion. Aerobically fit individuals will transport and use the oxygen they inspire more efficiently, therefore allowing the lungs to work at a lower rate (Wilson, 1987). Increase in pulmonary ventilation is accomplished by an increase in both the tidal volume and the respiratory rate, which consequently increases the vital capacity during exercise (Wilmore, 1970)

Muscular Adaptation: During aerobic exercise training, blood flow to the muscles is considerably large in relation to other tissues. This happens due to vasoconstriction of the parasympathetic system and vasodilation of the sympathetic system. Regular aerobic exercise training has been reported to improve fatty acid oxidation in muscles to generate energy for an athlete to exercise at a higher absolute

level of submaximal exercise before experiencing the fatiguing effects of glycogen depletion compared with an untrained person (McArdle, 2000).

2.13 AEROBIC EXERCISE

Aerobic exercise is any activity that uses large muscle groups, can be maintained continuously and is rhythmic in nature and overloads the heart and lungs and causes them to work harder than at rest (McArdle, 2000). Examples of aerobic exercises are: Aerobic dance, Jumping rope, Running, Fitness walking, Swimming, Bicycling, Stair climbing, Cross country skiing etc (Wilmore, 1982).

2.13.1 THE AEROBIC ENERGY PRODUCTION SYSTEM

The aerobic energy system is used by the body during any physical activity that lasts longer than two minutes. The oxidative system requires oxygen to generate ATP and the energy generated can be used for a long period of time (Guyton, 1987). In the aerobic energy system ATP production takes place in the mitochondria. The mitochondria can use carbohydrates (glucose or glycogen) or fats to produce ATP. The actual fuel used depends on the intensity and duration of the exercise and the fitness status of the individual. Increased carbohydrate catabolism during intense aerobic exercise provides for a significantly faster aerobic energy transfer than from fat breakdown and liberates about 6% more energy than fat per quantity of oxygen consumed (McArdle, 2000). Maximum oxygen uptake (VO_2 max) determines how intensely a person can perform endurance exercise and for how long, and it is considered the best overall measure of cardiorespiratory fitness (McArdle, 2000).

2.13.2 AEROBIC FITNESS

Cardiovascular endurance or aerobic fitness is the ability to exercise continuously for extended periods without tiring, and is an important component of many sporting activities (McArdle, 2000).

A person's aerobic fitness level is dependent upon the amount of oxygen which can be transported by the body to the working muscles and the efficiency of the muscles to use that oxygen. The best test for aerobic fitness is the maximal oxygen uptake ($VO_{2max)}$ test (McArdle, 2000). Since this test is very expensive and time consuming, many other simpler tests have been designed to predict $VO_{2max.}$ The many aerobic fitness tests can be classified into submaximal and maximal. Stressing the body by exercising to exhaustion as in maximal tests, is sometimes not appropriate, therefore the submaximal tests which are less demanding can be used (McArdle, 2000). These submaximal tests are;

a) **1-Mile Walk Test**: This test estimates the level of the individual's cardiorespiratory fitness based on the amount of time it takes an individual to complete 1-mile of brisk walking. The exercise heart rate at the end of the walk, age, gender and body weight are also considered. A fast time and a low heart rate indicate a high level of cardiorespiratory endurance (Wilson et al, 1987).

b) **The Astrand and Rhyming Cycle Ergometer Test:** A bicycle ergometer is used to measure the amount of resistance the cycle exerts against an individual's pedaling. This test estimates the maximum oxygen

consumption from the exercise heart rate reached after pedaling a cycle ergometer for six minutes at a constant rate and resistance. A low heart rate after pedaling at a high resistance indicates high maximal oxygen consumption (Wilson et al, 1987).

c) **The 3-minute Step Test:** The recovery from a 3 minutes stepping exercise represents a measure of cardiorespiratory fitness. The participant steps up and down on a bench of a given height at a rate of 24 steps per minute for 3 minutes. A metronome is set at 96 beats per minute (each beat of metronome equals a step with each leg up, up, down, down). At the end of 3 minutes of stepping, the participant still remains standing and the recovery heart rate is counted thirty seconds after the exercise ended. The number of pulse beats in 30 seconds represents the cardiorespiratory fitness score for that participant. Different workers have used benches of various heights. They include a height of 40cm for men and 33cm for women (Havard step test), Canadian Home Fitness Test (40.6cm), Chester Step Test (0.15-0.30metres) and Tecumseh step test (20.3cm) (McArdle, 2000).

2.14 AEROBIC DANCE

Aerobic dance was created by Jackie Sorensen in America in 1969 and has been in Australia since 1981. It is based on dance instead of repetitive exercise. It is never boring, always fun and motivating. It is suitable for children and adults and helps to strengthen the heart and lungs while working every muscle in the body safely (Sorensen and Bruns, 1979). Aerobic dance is a form of low impact exercise and is a good way to improve the

fitness level and avoid strenuous exercise when just starting a fitness programme. It may incorporate exercises with a variety of dance forms, such as disco, jazz and ballet that are performed to music. Adults should get at least 30 minutes of moderate aerobic exercise most days of the week, preferably daily (Sorenson and Bruns, 1979).

2.15 EXERCISE AND SICKLE CELL ANAEMIA

Despite the great efforts made towards understanding the principles of haemoglobin sickling and management; it is disappointing that so little of these findings are reflected in improved medical care for the patient. The management of painful crises has not been improved substantially over the years and there is a dearth of clinical trials directed towards improving clinical management of complications (Koffi et al, 2010).

There are few reports of investigations of exercise responses of patients with sickle cell anaemia. (Alawale, 1998, Charache et al, 2003 and Oyono-Enguelle et al, 2009). In their studies cardiorespiratory and haematological variables were considered. They observed that sickle cell anaemia patients responded to changes as a result of the exercise work out. Sanya and Obakin, (1999) compared the cardiopulmonary response of patients with sickle cell anaemia with the normal controls and they concluded that the response of patients with sickle cell anaemia to exercise featured a relatively high cardiac output manifesting as a higher than expected heart rate for a sub-maximal workload. This shows that the sickle cell anaemia patient could be easily fatigued and should not be subjected to high physical exertion as their healthy peers. Exercise therapy programme in the course of physiotherapy for sickle cell anaemia patients must be carefully prescribed, closely monitored and cautiously progressed (Sanya and Obakin, 1999)

2.16 QUALITY OF LIFE

Quality of Life (Qol), Health Status (HS) and Functional Status (FS) are three concepts often used interchangeably to refer to the same domain of health, since almost all aspects of life can be health-related (McDowell and Newell, 1996).

The health domain ranges from negatively valued aspects of life including death; to the more positively valued aspects such as role function or happiness. The boundaries of definition usually depend on why one is assessing health as well as the particular concerns of patient, clinicians and researchers. The term Qol is used because clinicians focus on quantifying the severity of a disease or disability and carefully assess the treatment plans to efficiently identify the care the patient requires, monitors progress and determine the treatment outcome (Yeomans, 2002).

2.16.1 MEASURES OF QUALITY OF LIFE

Medical interest in the Qol was stimulated by success in prolonging life and by the realization that this may be a mixed blessing as patient wants to live, not to merely survive (McDowell and Newell, 1996). Either self or interviewer administered questionnaires can be used to measure cross-sectional differences in Qol between subjects at a point in time (discriminative instruments) or longitudinal changes in Qol within subjects during a period of time (evaluative instruments). Reliable discriminative instruments are able to reproducibly differentiate between persons. Responsive evaluative measures are able to detect important changes in Qol during a period of time, even if those changes are small McDowell and Newell, 1996).

McClish et al, (2005) using the MOS 36-item Short-Form, compared the Qol scores of patients with sickle cell anaemia with that of national norms and three chronic disease cohorts: asthma, cystic fibrosis and haemodialysis patients. They also assessed whether sickle cell disease specific variables (genotype, pain crisis and utilization) were independently predictive of SF-36 subscales. They found that SCA patients scored significantly worse than national norms on all subscales, except for mental health. Qol scores were similar for physical function, role function and mental health as compared to asthma patients, but worse for bodily pain, vitality, social function and general health subscales. Compared to dialysis patients, sickle cell anaemia patients scored similarly on physical role and emotional role function, social functioning and mental health, worse on bodily pain, general health and vitality and better on physical functioning. Genotype did not influence Qol except for vitality. However, scores significantly decreased as pain levels increased. The MOS SF-36 was administered because the investigator could not find a sickle cell disease specific Qol instrument.

Two basic approaches to quality of life measurements are available, namely: generic instruments that provide a summary of Qol; and specific instruments that focus on problems associated with single diseased state, patient groups or areas of function.

Generic instruments include health profiles and instruments that generate health utilities. Investigations in Qol have lead to instruments suitable for detecting minimally effective effect in clinical trials for measuring the health of populations and for providing information for quality decisions. Health profiles, which describe Qol indices in a set of scores, and health indices that summarize health in a single number are used to measure Qol of individuals. The generic health profiles include the Rand Short Form-36 Health

survey 1.0 (Ware and Sherbourne, 1992), Rand Short Form-36 Health Survey (Ware and Sherbourne , 1992), the Short Form-20 Health survey (Stewart et al, 1988), the Multi Level Assessment Instrument (Lawton et al, 1982), Disability and Distress Scale (Rosser and Kind, 1978), Quality of Well Being Scale (Rosser and Kind, 1978), Quality of Well Being Scale (Kaplan and Bush, 1994) e.t.c.

The disease specific health profiles include Physical and Mental Impairment of Function Evaluation scale (Gurel, 1972), Functional Assessment Index (Crewe et al, 1981), Quality of Life Index (Spitzer et al, 1980), COOP Charts For Primary Care Practice (Nelson et al, 1987), Functional Status Questionnaire (Jette et al, 1986), DUKE Health Profile (Parkerson et al, 1990), Self Evaluation of Life Functional Scale (Linn and Linn, 1984), Functional Living Index- Cancer (Schipper, 1984).

However, some of these instruments can be used as either generic or specific Qol instruments. These include: the Nottingham Health Profile (Hunt et al, 1981), McMaster Health Index Questionnaire (Chambers et al, 1976), Sickness Impact Profile (Bergner, 1976) etc.

2.16.2 THE RAND SHORT FORM-36 HEALTH SURVEY (VERSION 1.0)

The Rand Short Form-36 Health Survey (version 1.0) is so called because the scoring method (a simpler and more straight forward procedure) differs from that of the MOS SF-36. It is a 36-item instrument adapted from the longer instruments completed by patients participating in the Medical Outcome Study (MOS), an observational study of variations in physician practice styles and patient outcomes in different systems of health care delivery (Hays and Shapiro, 1992). The Short Form-36 Health Survey was

developed into a developmental form in 1988; standard form in 1990 and version 1.0 in 1996. It was developed to measure various aspects of an individual's health in a comprehensive yet brief manner and from the individual's point of view. The Short form-36 was designed to meet the requirements of being able to measure changes in health over a prolonged period of time; which was lacking in the MOS Short Form-20 Health Survey. The SF-36 Health Survey is therefore commonly used as a quality of life index measure in population studies (McDowell and Newell, 1996). The RAND 36-Item Health Survey (version 1.0) measures eight health concepts: physical functioning, bodily pain, role limitations due to physical health problems, role limitation due to personal or emotional health problems, emotional well-being, social functioning, energy/fatigue, and general health perceptions. It also includes a single item that provides an indication of perceived change in health.

i. Questions 3, 4, 5, 6, 7, 8, 9, 10, 11, and 12; constitute the physical function scale.
ii. Questions 21 and 22 constitute the bodily pain scale.
iii. Questions 13, 14, 15 and 16 constitute the role limitation due to physical health concepts.
iv. Questions 17, 18 and 19 constitute the role limitation due to personal or emotional health problems.
v. Questions 24, 25, 26, 28 and 30 constitute the emotional well being scale.
vi. Questions 20 and 32 constitute the social functioning scale.
vii. Questions 23, 25, 26, 28 and 31 constitute the energy/fatigue scale.

viii. Questions 1, 33, 34, 35 and 36 constitute the general health scale.

The instrument has been found to be very reliable. The reliability (r) for the various domains of the instrument has been observed to be 0.93 (physical functioning), 0.84 (role functioning/physical), 0.83 (role functioning/emotional), 0.86 (energy/fatigue), 0.90 (emotional well being), 0.85 (social functioning), 0.78 (pain), 0.78 (general health). The instrument has a Cronbach's alpha of 0.76 to 0.90 for the scoring scales (Ware and Sherbourne, 1992).

2.16.3 SCORING OF RAND SF-36 ITEM HEALTH SURVEY (VERSION 1.0).

The instrument can be self administered or administered by interview, either in person or by telephone, in less than 15 minutes. Scoring the RAND 36-Item Health Survey is a two-step process. First, precoded numeric values are recoded per the scoring key given in Appendix F. All Items are scored such that a high score defines a more favourable quality of life. In addition, each Item is scored on a 0 to 100 range so that the lowest and highest possible scores are set at 0 and 100, respectively. Scores represent the percentage of total possible score achieved. In step 2, items in the same scale are averaged together to create the 8 scale scores. Appendix G lists the items averaged together to create each scale. Items that are left blank are not taken into account when calculating the scale scores. Hence, scale scores represent the average for all items in the scale that the respondent answered (Ware and Sherbourne, 1992).

2.17 JUSTIFICATION OF THE STUDY

Review of literature has shown that exercise generally affects the cardiorespiratory and haematological variables in sickle cell disease but that there is a dearth of studies on sickle cell anaemia in this setting. Most of the literatures available for referencing are from other countries and populations. (The summary of previous studies on sickle cell anaemia and exercise is presented in Table A). A number of factors have been found to affect the Quality of life, cardiorespiratory and heamatological variables of individuals with sickle cell disease from previous studies. However, none of these studies have reported the effects of aerobic exercise on Quality of life, frequency of crises, frequency of hospitalization and length of hospitalization in patients with sickle cell anaemia. This study intends to bridge this gap in knowledge by looking at two groups (experimental and control groups) of patients with sickle cell anaemia.

TABLE 1: SUMMARY TABLE FOR PREVIOUS STUDIES ON SICKLE CELL ANAEMIA AND EXERCISE.

S/N	Author and year	Country of study	Sample size and sample characteristics	Age distribution of sample	Gender distribution of sample	Objective
1.	Sanya and Obakin 1999	Nigeria	30 subjects with sickle cell anaemia and 40 healthy controls	18 years and above	Males and females	The response of patients with sickle cell anaemia to exercise test was compared with their age matched counterparts
2.	Covitz et al, 1983	USA	22 Adolescent subjects with sickle cell disease and 12 controls	Not available	Not available	Cardiac performance was studied by radionucleotide angiography at rest and during exercise.
3.	Manno et al, 1983	USA	10 Patients with sickle cell disease	19-53 Years	Not available	Biventricular function was evaluated at rest and during exercise.
4.	McConnell et al, 1990.	Cincinnati, Ohio.	43 Subjects (8 with sickle cell disease and 35 controls).	5-23 years.	Not available	Haemodynamic response to exercise in patients with sickle cell disease and controls were compared.
5.	Pianosi et al, 1991.	Quebec, Canada.	30 Children with sickle cell disease and 16 healthy controls.	Not available	Males and females	Cardiac output and oxygen delivery during exercise in sickle cell disease were studied.

S/N	Author and year	Country of study	Sample size and sample characteristics	Age distribution of sample	Gender distribution of sample	Objective
6.	Pianosi .P, 1991	Quebec, Canada	34 sickle cell disease subjects and 16 controls.	Adults	Not available	Ventilation and gas exchange during exercise in sickle cell disease were studied.
7.	Alawale, 1998	Nigeria	20 sickle cell disease subjects.	16-30 years	9 Males and 11females	Cardio-respiratory and haematological adaptation of patients with sickle cell anaemia to a 12-week endurance exercise training programme was studied.
8.	Braden et al, 1996	USA	18 subjects	Not available	Males and females	Cardiovascular function during rest and exercise in patients with sickle cell disease and coexisting alpha thalassa 2 was studied.
9.	Callahan et al, 2002	America	Not available	Not available	Female	Cardiopulmonary responses to exercise in women with sickle cell anaemia were studied.
10.	Platt, 1982.	USA	5 Subjects	Not available	Not available	To see if SS erythrocytes were abnormally fragile when exposed to shear forces; that could be generated in small vessels of exercising muscles.

CHAPTER THREE

MATERIALS AND METHODS

3.1 MATERIALS

3.1.1 PARTICIPANTS

The participants in this study were 129 sickle cell anaemia patients who were attending the Haematology Clinic of the Lagos State University Teaching Hospital, Ikeja, Lagos. They comprised male and female patients (adolescents and adults); referred by the physician, consented to participate in the study and met the following inclusion criteria.

Inclusion criteria

Sickle cell anaemia patients who were screened by the referring physician and certified fit (absence of any symptoms of dyspnea, fainting and syncope after performing the step test for 3 minutes) by the researcher.

3.1.2 INSTRUMENTS

1) **Weighing Scale:** (Secca, Germany) was used to measure the weight of the participants. It was calibrated from 0 to 200kg. Weight was recorded to the nearest 1.0 kg.
2) **Height Meter:** A height meter (Holtain-Crymych, UK) was used to measure height of participants in meters. It was calibrated from 0 to 2 meters; measurement was taken to the nearest 0.01 m
3) **Stethoscope:** (Littman, England) was used to listen to the heart rate of the participants.

4) **Stop watch:** (Liaoning, China) digital stop watch was used to time all activities as necessary.
5) **Sphygmomanometer:** (Accoson, UK) was used to measure the blood pressure of participants. It was calibrated from 0 to 300mm Hg.
6) **A Spirometer:** (Creative Biomedics, U.S.A) was used to measure the vital capacity of the participants in cubic centilitres.
7) **Disposable mouth piece:** This was fixed to the spirometer for each participant to prevent mouth to mouth infection.
8) **Step Bench:** A bench platform (20.3cm high) was used to carry out exercise for the Tecumseh cardiorespiratory fitness test (Plate 6) (McArdle, 2000).
9) **A metronome:** (Wittner, West Germany) was used to dictate a uniform cadence for the Tecumseh cardiorespiratory fitness test (Plate 6).
10) **Television Set:** (Samsung, China) was used to display the aerobics dancing steps for the participants to see. This was centrally placed on a high wall shelf for easy visualization by the subjects.
11) **Video Player:** (Samsung, China) was used to play the hip-hop computer disc.
12) **Aerobics CD:** (Shami Productions, 2006, U.S.A) was inserted into the DVD player so that the participants could perform the dance steps as observed on the television screen.
13) **The Rand 36-Item Health Survey (version 1.0):** This was used to measure the quality of life of the participants. It is a generic instrument with 36 items; which measures eight domains of health (physical functioning, bodily pain, role

limitations due to physical health problems, role limitations due to personal or emotional problems, emotional well-being, social functioning, energy/fatigue and general health perceptions.) It also includes a single item that provides an indication of perceived change in health. It is scored in a two-step process (Appendix E). The lowest and maximum scores obtainable are 0 and 100. The instrument has the following values for the different domains: reliability (r) 0.93 (physical functioning), 0.84 (role functioning), 0.83 (role functioning/emotional), 0.86 (energy/fatigue), 0.90 (emotional well being), 0.85 (social functioning), 0.78 (pain), 0.78 (general health) (RAND Health, 2007).

14) **Data sheet**: This was used to record the data of the participants. A copy of the data sheet (Appendix D) and an informed consent form (Appendix C) were attached to each copy of the questionnaire (Appendix E).

3.3 METHODS.

3.3.1 SAMPLING TECHNIQUE

Consecutive sampling technique was used to recruit participants from the Haematology Clinic of the Lagos State University Teaching Hospital, Ikeja, Lagos (LASUTH).

3.3.2 RESEARCH DESIGN

The research utilized a two-group randomized controlled trial design.

3.3.3 VENUE OF RESEARCH

The venue of this research was the Out-Patient Haematology Clinic of the Lagos State University Teaching Hospital, Ikeja, Lagos (LASUTH). This was because it was closer to the point of recruitment of the patients.

3.3.4 PROCEDURES FOR DATA COLLECTION

This study was in three phases (observational, experimental and follow-up). Ethical approval was sought and obtained for this study from the Research and Ethics Committee of the Lagos State University Teaching Hospital, Ikeja, Lagos (LASUTH) (Appendix A). The rationale and procedure for the study were explained to the participants so as to seek and obtain their informed consent (Appendix B, C). The group to which the first participant was assigned was determined by tossing a coin. Tail was for experimental group while head was for control group and subsequent participants were recruited consecutively and alternately assigned to the experimental and control groups as they become available. The participant's demographic data such as age, sex, weight, height and occupation were sought and recorded on the data sheet (Appendix D).

Observational Phase: Data on frequency of crisis, frequency of hospitalization and length of hospitalization at six months before the study were recorded.

Experimental Phase: Data on cardiorespiratory, haematological variables and quality of life were recorded at baseline (week 0), end of 6^{th} week and end of 12^{th} week of the study.

Follow-up Phase: Data on frequency of crisis, frequency of hospitalization and length of hospitalization at six months after the study were recorded.

Data on weight and height of participants were collected only at week 0, while data on blood pressure, heart rate, vital capacity, cardiorespiratory fitness score, packed Cell volume, platelet count, mean corpuscular haemoglobin concentration and quality of life were collected and recorded at week 0 and at the ends of the 6th and 12th weeks of the study for both experimental and control groups.

1) Blood Pressure: The participant sat on a comfortable chair and the cuff of the sphygmomanometer was wrapped round his/her upper arm at the same vertical height as the heart. The cuff was inflated by the cup as required while the pressure was released the researcher listened to the pulse rate through the stethoscope. The systolic pressure was the pressure at which the first sound was heard while the diastolic pressure was the pressure at which the last sound was heard. The readings were recorded on the data sheet (McArdle, 2000) (Plate 1).

2) Weight: Each participant stood barefooted on the weighing scale, looking straight ahead while the researcher read off the weight to the nearest 1.0kg (McArdle, 2000) (Plate 2).

PLATE 1: Measurement of blood pressure of participants .

PLATE 2: Measurement of participant's weight

3) Height: Each subject stood barefooted against the height meter. While he looked straight ahead, the pointer was slid to the top of his head to read off the equivalent height. The height was recorded to the nearest 0.01m (McArdle, 2000) (Plate 3).

4) Body Mass Index: From the weight and height measured, the Body Mass index was calculated as the ratio of the weight to the square of the height i.e.

$$\text{Body Mass Index} = \frac{\text{weight}}{(\text{Height})^2} \text{ Kg/m}^2 \text{ (McArdlle, 2000)}$$

5) Heart Rate: The participant was allowed to rest for 10 minutes before the heart rate was taken. This was to allow the heart beat to become well regulated for accuracy. The participant sat on a chair and the diaphragm of the stethoscope was placed on the left side of his chest between the fourth and fifth rib (fifth intercostals space). The heart beat was then counted for 60 seconds (McArdle, 2000).

6) Vital Capacity: The participant sat on a chair and was asked to inhale as much air as possible and exhale forcefully into the mouth piece of the spirometer (Plate 4). The procedure was repeated thrice so as to obtain three readings. The average of the three readings was taken as the participant's vital capacity and recorded in cubic centiliters. Disposable mouth pieces were used for each of the participants to prevent mouth to mouth infection among the participants (Gomersall, 1999).

PLATE 3: Measurement of participant's height

PLATE 4: Measurement of participant's vital capacity

7) Cardiorespiratory Fitness Score: The participant's cardiorespiratory fitness was assessed with the Tecumseh step test. This was assessed before the commencement of the aerobic dance programme. Each participant was asked to perform the stepping cycle to a four step cadence of up-up-down-down. Both male and female subjects completed 24 step-ups per minute (up and down twice within 5-seconds span). The metronome was set at 96 beats per minute to give a cadence of one footstep per beat. Each participant performed the test for exactly 3 minutes continuously. On completion of the 3 minutes, while the participant was still standing, the pulse rate was counted by placing the middle fingers over the radial artery. The pulse rate was counted and recorded for a thirty second period exactly 30 seconds after stopping the exercise. It was recorded in beats per minute. The number of pulse beats in 30 seconds recovery period represented the cardiorespiratory fitness score (McArdle, 2000) (Plate 5).

PLATE 5: Measurement of cardiorespiratory fitness using the step test

8) Packed Cell Volume (PCV)/Haematocrit: The tip of the participant's thumb was pricked with a new injection needle and a capillary tube

was about upper three-quarters filled with the discharging blood. The end of the tube was then sealed with a suitable sealant (plasticene) and the capillary tube put inside the Sysmex machine and centrifuged for 5 minutes. The Sysmex blood analyzer automatically displayed the PCV measured on the screen (Plate 6). The normal value for an adult male is 39%-51% while for an adult female it is 34%-46%. (Baker and Silverton, 2001)

9) Platelet Count: Five millilitres of fresh blood was drawn from a vein in the subject's inner elbow region since blood drawn from a vein produces more accurate result than blood drawn from the fingertip (Baker and Silverton, 2001). The blood was then emptied into a specimen bottle to which anti-coagulant has been added, before the test began. The collection of blood took only a few minutes. The platelet count was then digitally estimated by the Sysmex blood analyzer (Plate 6). The normal value for an adult is 150-400×10^9/L (Baker and Silverton, 2001).

10) Mean Corpuscular Haemoglobin Concentration (MCHC): Five millilitres of fresh blood was drawn from a vein in the subject's inner elbow region since blood drawn from a vein produces more accurate result than blood drawn from the fingertip. The blood was then emptied into a specimen bottle to which anti-coagulant has been added, before the test began. The test bottles were carefully arranged horizontally inside the sysmex machine tray and the MCHC was digitally analyzed (Plate 6). The normal value for an adult is 32-37g/dl (Jacobs, 1996; Pagana and Pagana, 1998 and Baker and Silverton, 2001).

PLATE 6: Sysmex blood analyzer (USA): used for PCV, PC, MCHC, MCH and MCV analysis in the study.

11) Quality of life: The quality of life of the participants was evaluated with RAND 36-Item Health Survey (version 1.0), which was self administered by the participants (Appendix E).

The following parameters were assessed in the laboratory by the Haematologist:

The following pieces of information were obtained by interview and/or retrieved from the participants' case notes and was recorded on the data collection sheet:

12) Frequency of crisis at 6 months before the study and 6 months following the end of the study.
13) Frequency of hospitalization at 6 months before the study and 6 months following the end of the study.
14) Length of hospitalization at 6 months before the study and 6 months following the end of the study.

3.3.5 AEROBIC EXERCISE TRAINING PROCEDURE

The procedure for the aerobic exercise training comprised of three phases as listed below:

a. **Warm up:** This was performed for duration of 5 minutes by the participants in the exercise (experimental) group. It involved slow movements that stretched the muscles and improved blood circulation to prevent muscle stiffness, risk of injury and improve performance (McArdle, 2000). The warm up exercise involved the following activities.

Deep breathing exercises: While standing upright, with the arms extended straight out to the side, the participants inhaled deeply as much as possible while raising the arms from shoulder level until the hands came together over the head. That position was held for 2 seconds. Then the arms were lowered to the sides during exhalation. This was repeated 15 times.

Arm rotation: The arms were extended straight out to the sides from the shoulders. The hands are then rotated in circles. The hands were rotated forward 20 times and 20 more while rotating backwards. This was done for 2 minutes.

Body rotation: The participant placed the feet wide apart and bent forward at the waist. Then the body was rotated in slow circles through ten repetitions. This was done for two and half minutes.

b. **Work out:** This comprised of aerobic dance. The work load was increased forthnightly by increasing the duration of the exercise by varying durations of 20, 25, 30, 35, 40 and 45 minutes thrice weekly.

c. **Cool down:** This was done after the work out and lasted for 5 minutes. It comprised of slow dance steps and breathing exercise.

3.3.5.1 THE EXPERIMENTAL GROUP

This group participated in a group aerobic dance for varying intensities of 20, 25, 30, 35, 40 and 45 minutes thrice weekly. The duration of the exercise programme was 12-weeks (Plate 7). The participants wore light clothing and the aerobic exercise was performed in the morning between 8 am -12pm, in order to reduce the effect of temperature on the participants. The participants also continued with the use of their prescribed drugs. Participants were assessed by the referring physician to ensure that the female participants were not pregnant and that none of the participants had complications associated with sickle cell anaemia such as organ damage, priapism and retinopathy. Participants that had some or all of these complications were excluded from the study. The participants also continued with the use of their prescribed drugs. Any participant that did not attend the aerobic dance programme for 3 consecutive sessions within 1 week were considered attrition and were excluded from this study.

Precautions.

The following precautionary measures were taken in accordance with guidelines by Sorensen and Bruns (1979):

i. There was a doctor on standby during the exercise programme in case of any emergency, to assist with the resuscitation of sickle cell anaemia patients who might collapse during the exercise.

ii. All the participants danced tall to prevent muscle strain and fatigue.

iii. Participants drank water as needed during their workout to prevent dehydration.

PLATE 7: Sickle cell anaemia patients in aerobic dance session.

3.3.5.2 THE CONTROL GROUP

This group did not participate in the aerobic dance exercise programme; but were using their prescribed drugs over the period of 12-weeks. The participants were assessed by the referring physician to ensure that participants did not have complications associated with sickle cell anaemia such as organ damage, priapism and retinopathy. Participants that had some or all of these complications were excluded from the study.

3.4 DATA ANALYSIS

1. Data were summarized using descriptive statistics of mean and standard deviation.
2. Repeated measure Analysis of Variance (ANOVA) was used to compare each of heart rate, vital capacity, cardiorespiratory fitness score, packed cell volume, platelet count, mean corpuscular haemoglobin concentration and health status scores of:
    a) The experimental group across weeks 0, 6 and 12 of the study.
    b) The control group across week 0, 6 and 12 of the study.
3. Independent t- tests were used to compare the following variables between the experimental and control groups at the baseline, 6 weeks and 12 weeks of the study:
    a) Heart rate
    b) Vital capacity
    c) Cardiorespiratory fitness score

d) Packed Cell Volume

e) Platelet Count

f) Mean Corpuscular Heamoglobin Concentration

g) Quality of life.

4. Independent t- tests were used to compare the following variables between the experimental and control groups at 6 months before the study:

   a) Frequency of crisis

   b) Frequency of hospitalization

   c) Length of hospitalization.

5. Paired t-test was used to compare the values of the following variables 6 months before and 6 months after for the experimental and control group:

   a) Frequency of crisis

   b) Frequency of hospitalization

   c) Length of hospitalization.

The alpha level for t-test and ANOVA was set at 0.05. Post-hoc analysis for significant repeated measures ANOVA was done using paired t-test with the alpha level at 0.017 using the Bonferroni adjustment.

CHAPTER FOUR

RESULTS AND DISCUSSIONS

4.1 RESULTS

4.1.1 PARTICIPANTS

One hundred and twenty nine participants diagnosed to have sickle cell anaemia (SCA) by consultant haematologists with the Lagos State University Teaching Hospital, Lagos were recruited into the study. Sixty five participants were recruited into the experimental group while sixty four participants were recruited into the control group. Four (3.10%) of the participants dropped out because they relocated to other states as a result of employment, fourteen (10.85%) because of schooling, one (0.78%) died as a result of sickle cell crisis developed after the study and he was not given a prompt medical intervention as a result of industrial action by medical doctors in Lagos State, and six (4.65%) stopped coming without any reason and they could not be reached on the phone. Eleven (16.92%) participants dropped off from the experimental group while 14 (21.86%) participants dropped from the control group.

One hundred and four (80.62%), of the initial 129 participants (54 experimental and 50 control) completed the study and only their data were analyzed. Fifty-five (52.88%) of the participants were females and 49 (47.12%)

were males. The incentives of free routine drugs, tests, frequent telephone contacts and transport fare were probably responsible for their good compliance.

4.1.2 PHYSICAL CHARACTERISTICS OF PARTICIPANTS

The mean age, weight, height and BMI of participants in the experimental group were 26.06±6.67 years, 52.39±5.66kg, 1.61±0.07m and 20.24±2.02kg/m^2 respectively, while the mean age, weight, height and BMI for the control group were 25.66±5.59 years, while the mean age, weight, height and BMI for the control group were 25.66±5.59 years, 50.82±6.10kg, 1.61±0.06m and 19.64±1.60kg/m^2 respectively. Independent t-test at $\alpha = 0.05$ did not show any significant difference between the mean age ($p= 0.686$), weight ($p= 0.476$), height ($p= 0.656$) and BMI ($p=0.097$) of participants in both groups (Table 2).

4.1.3 COMPARISON OF THE CARDIORESPIRATORY VARIABLES OF PARTICIPANTS IN EXPERIMENTAL AND CONTROL GROUPS AT WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

4.1.3.1 HEART RATE

The mean heart rates of the experimental group were 75.20±4.50 beats/min, 75.81±4.54 beats/min and 71.93±3.50 beats/min at week 0, end of the 6th week and end of the 12th week of the study respectively, while that of the control group were 76.76±5.13 beats/min, 76.34±5.02 beats/min and 76.48±4.89 beats/min respectively at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at $\alpha = 0.05$ showed that

the groups' heart rate were not significantly different at week 0 but the control group had significantly greater (p= 0.000) heart rate than the experimental group at the end of 12th week of the study (Table 3). The trends of the heart rate of the experimental and control groups are presented in Figure 1, while the heart rate of the control group remained unchanged, that of the experimental group increased between week 0 and week 6 but decreased substantially between week 6 and week 12 of the study.

TABLE 2

PHYSICAL CHARACTERISTICS OF PARTICIPANTS

	Group			
	Experimental	Control		
	n=54	n=50		
Variables	Mean±SD	Mean±SD	95% CI	p
Age (years)	26.06± 6.67	25.86±5.59	-2.97 to1.96	0.686
Height (m)	1.61±0.07	1.61±0.06	-0.02 to 0.03	0.656
Weight(kg)	52.39±5.66	50.82±6.10	-1.45 to3.08	0.476
BMI (kg/m^2)	20.24±2.02	19.64±1.60	-0.11 to 1.31	0.097

CI= Confidence Interval.

SD= Standard Deviation

P= 0.05

TABLE 3

COMPARISON OF CARDIORESPIRATORY PARAMETERS OF PARTICIPANTS AT WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Variables	Time Frame	Group Experimental (n=54) Mean±SD	Control (n=50) Mean±SD	95%CI	p
HR (b/min)	Week 0	75.20±4.50	76.76± 5.13	-3.43 to 0.32	0.103
	Week 6	75.81±4.54	76.34± 5.02	-2.38 to 1.33	0.576
	Week 12	71.93±3.50	76.48± 4.89	-6.20 to -2.91	0.000*
CRFS (b)	Week 0	52.63±4.14	54.26± 4.53	-3.32 to 0.05	0.058

	Week 6	50.80±5.48	54.22± 6.39	-5.73 to -1.11	0.004*
	Week 12	46.52±6.17	55.30± 6.69	-11.28 to-6.28	0.000*
VC (cc)	Week 0	1714.50±400.42	1773.94±446.50	-224.18 to 105.30	0.476
	Week 6	2109.35±441.87	1769.92±389.62	176.87 to 501.99	0.000*
	Week 12	2293.89±374.12	1755.80±377.55	391.81 to 684.37	0.000*

*Significant difference between experimental and control groups at p=0.05

Key: HR=Heart rate, CRFS=Cardio-respiratory fitness score, VC= Vital capacity

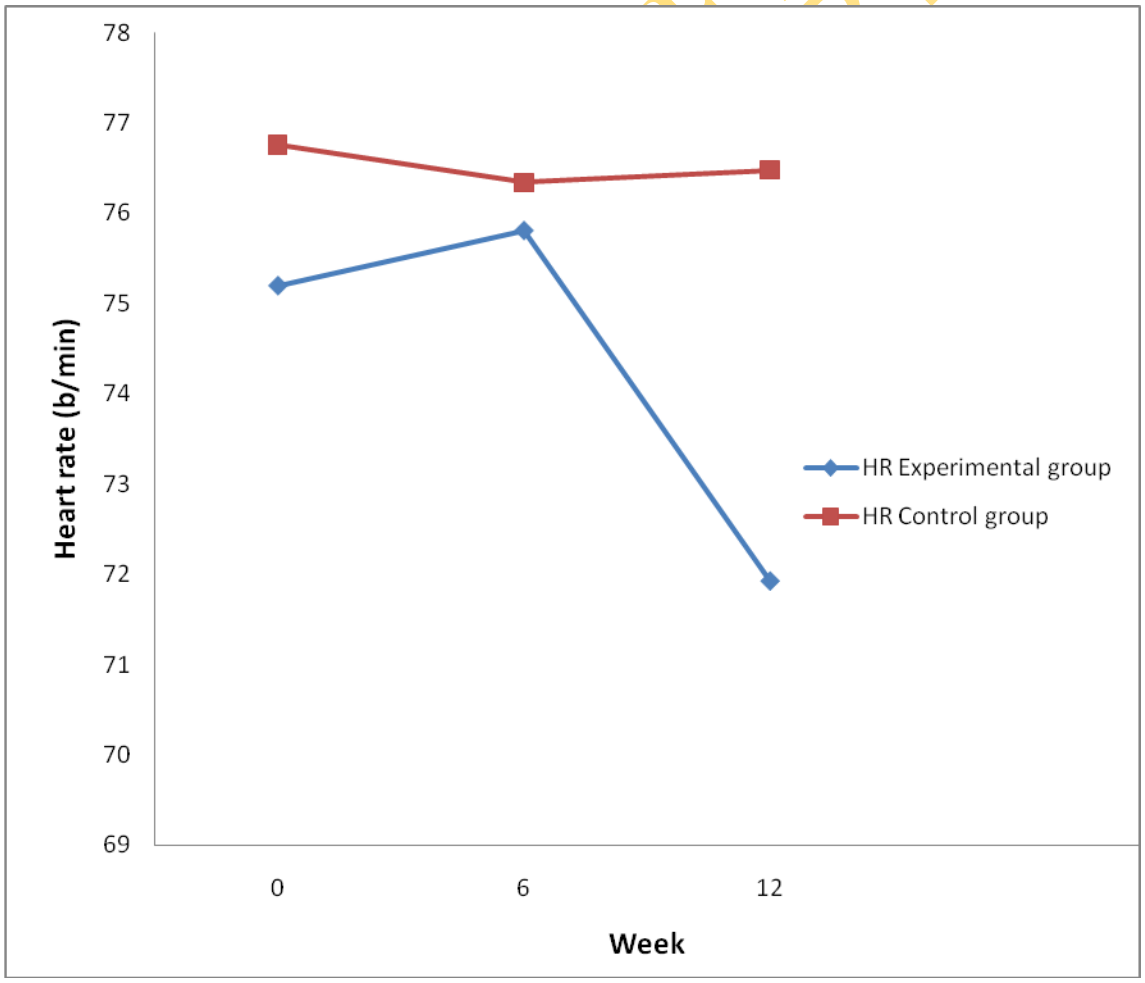

FIGURE 1: changes in the heart rate across the three time frames.

4.1.3.2 CARDIORESPIRATORY FITNESS SCORE

The mean cardiorespiratory fitness scores (CRFS) of the experimental group at the beginning of week 0, end of the 6th week and end of the 12th week of the study were 52.63±4.14, 50.80±5.48 and 46.52±6.17 respectively, while the scores for the control group were 54.26±4.53, 54.22±6.39 and 55.30±6.69 at the beginning of week 0, end of the 6^{th} week and end of the 12^{th} week of the study respectively. Independent t-test at $\alpha = 0.05$ showed that the control group had significantly greater CRFS (poorer fitness) than the experimental group at the end of the 6^{th} week ($p= 0.004$) and the 12^{th} week ($p=0.000$) of the study (Table 3). The trends of the participants cardiorespiratory fitness score across the three time frames are presented in Figure 2, while the cardiorespiratory fitness scores or the control group remained unchanged, that of the experimental group increased between week 0 and week 6 but decreased substantially between week 6 and week 12 of the study.

4.1.3.3 VITAL CAPACITY

The mean vital capacity (VC) of the experimental group were 1714.50±400.42cc, 2109.35±441.87cc and 2293.89±374.12cc at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively, while the mean VC for the control group were 1773.94±446.50cc, 1769.92±389.62cc and 1755.80±377.55cc at week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at $\alpha = 0.05$ showed that the control group had significantly poorer vital capacity than the experimental group at end of the 6th week ($p= 0.000$) and 12th week ($p= 0.000$) of the

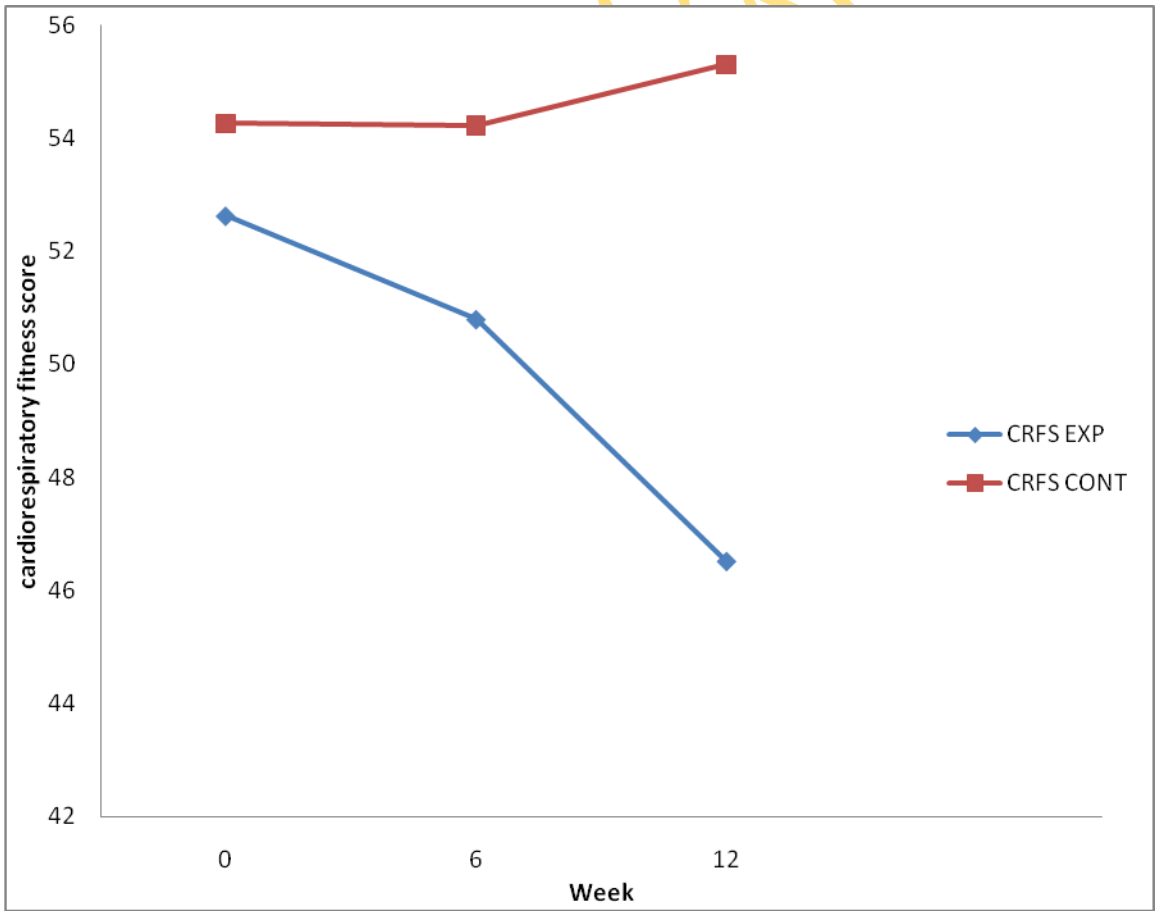

FIGURE 2: changes in the cardiorespiratory fitness scores across the three time frames.

study (Table 3). Although the VC for the two groups were virtually the same at week 0, the VC for the experimental group increased sharply between week 6 and week 12. However, the VC for the control group remained virtually unchanged for the duration of the study (Figure 3).

4.1.4 COMPARISON OF HAEMATOLOGICAL VARIABLES OF PARTICIPANTS IN EXPERIMENTAL AND CONTROL GROUPS AT WEEK 0, WEEK6 AND WEEK 12 OF THE STUDY

4.1.4.1 PACKED CELL VOLUME

The mean packed cell volumes (PCV) of the experimental group at the beginning of week 0, end of the 6th week and end of the 12th week of the study were 24.72±3.16%, 26.06±3.44% and 26.80±3.29% respectively, while the mean PCV of the control group were 24.39±3.42%, 23.84±3.79% and 23.32±3.45% at

the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at $\alpha = 0.05$ showed that the experimental group had significantly greater PCV than the control group at the end of the 6^{th} week ($p= 0.002$) and 12^{th} week ($p= 0.000$) of the study while, the two groups were not statistically different at the beginning of week 0 ($p= 0.614$) (Table 4). The PCV of the experimental group increased for week 0 through week 6 up to week 12 of the study. The PCV of the control group decreased steadily from week 0 through week 6 up to week 12 of the study (Figure 4).

TABLE 4:

COMPARISON OF HAEMATOLOGICAL PARAMETERS OF PARTICIPANTS AT WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Variables	Time Frame	Group: Experimental (n=54) Mean ±	Group: Control (n=50) Mean ±	95% CI	p
PCV%	Week 0	24.72±3.16	24.39±3.42	-0.81 to 1.74	0.614
	Week 6	26.06±3.44	23.84±3.79	1.02 to 3.81	0.002*

	Week 12	26.80±3.29	23.32±3.45	2.48 to 5.04	0.000*
PC×10^9/L,	Week 0	364.61±127.46	343.58±128.81	-28.89 to 70.90	0.405
	Week 6	322.41±100.90	335.20±132.21	-58.34 to 32.75	0.579
	Week 12	269.37±86.29	331.12±122.28	-102.67 to -20.83	0.003*
MCHCg/dl	Week 0	33.67±1.04	33.85±1.06	-0.59 to 0.23	0.384
	Week 6	34.03±1.30	34.27±1.84	-0.86 to 0.37	0.434
	Week 12	34.05±1.01	34.13±1.12	-0.49 to 0.34	0.709

*Significant difference between experimental and control groups at p=0.05
Key: PCV=Packed cell volume PC=Platelet count MCHC=Mean cell haemoglobin concentration

TABLE 5

COMPARISON OF PARTICIPANTS' QUALITY OF LIFE AT WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Time Frame	Group: Experimental (n=54) Mean ± SD	Group: Control (n=50) Mean ± SD	95% CI	p
Week 0	65.06±8.17	65.38±10.13	-3.64 to 3.49	0.860

Week 6	69.22±8.09	65.06±12.49	0.51 to 8.60	0.045*
Week 12	72.90±7.31	64.44±11.15	4.82 to 12.11	0.000*

*Significant difference between experimental and control groups at p=0.05

CI= Confidence Interval

SD= Standard Deviation

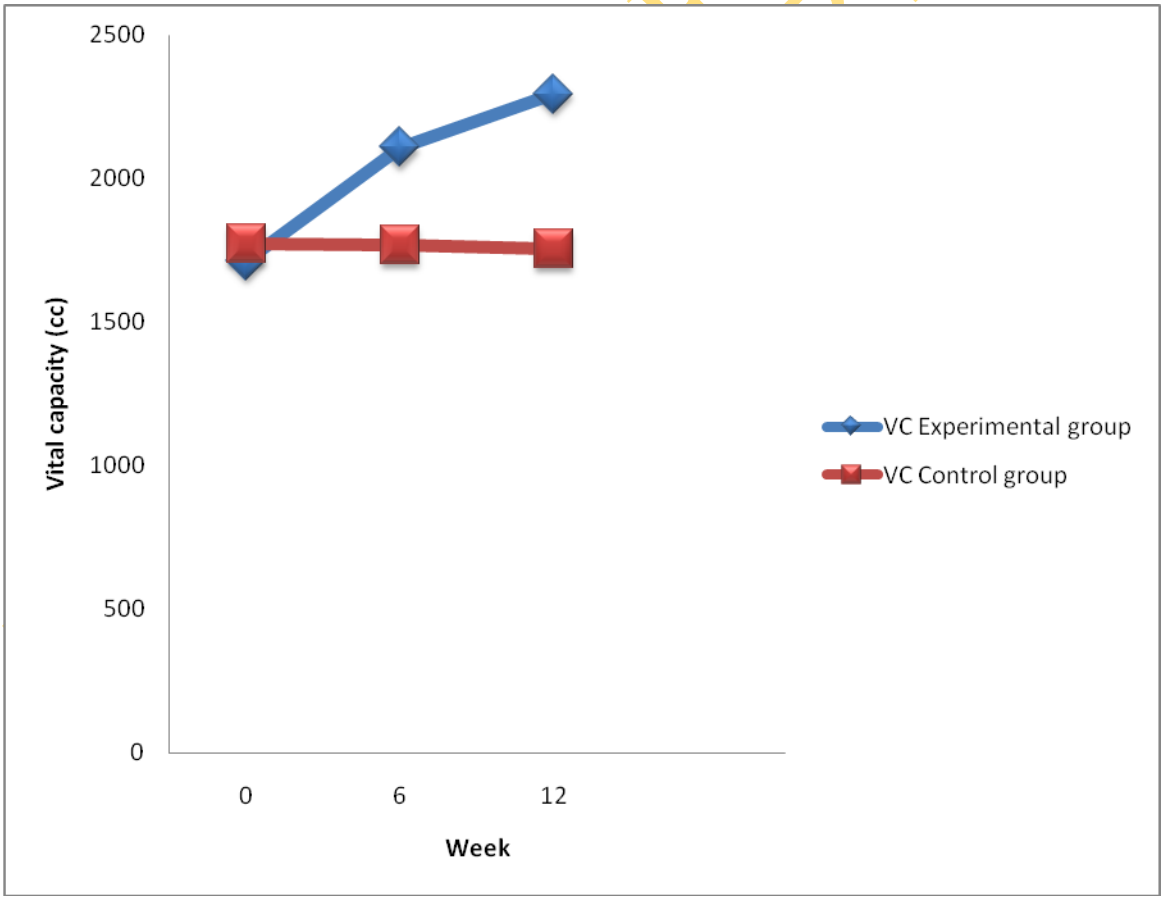

FIGURE 3: changes in vital capacity across the three time frames.

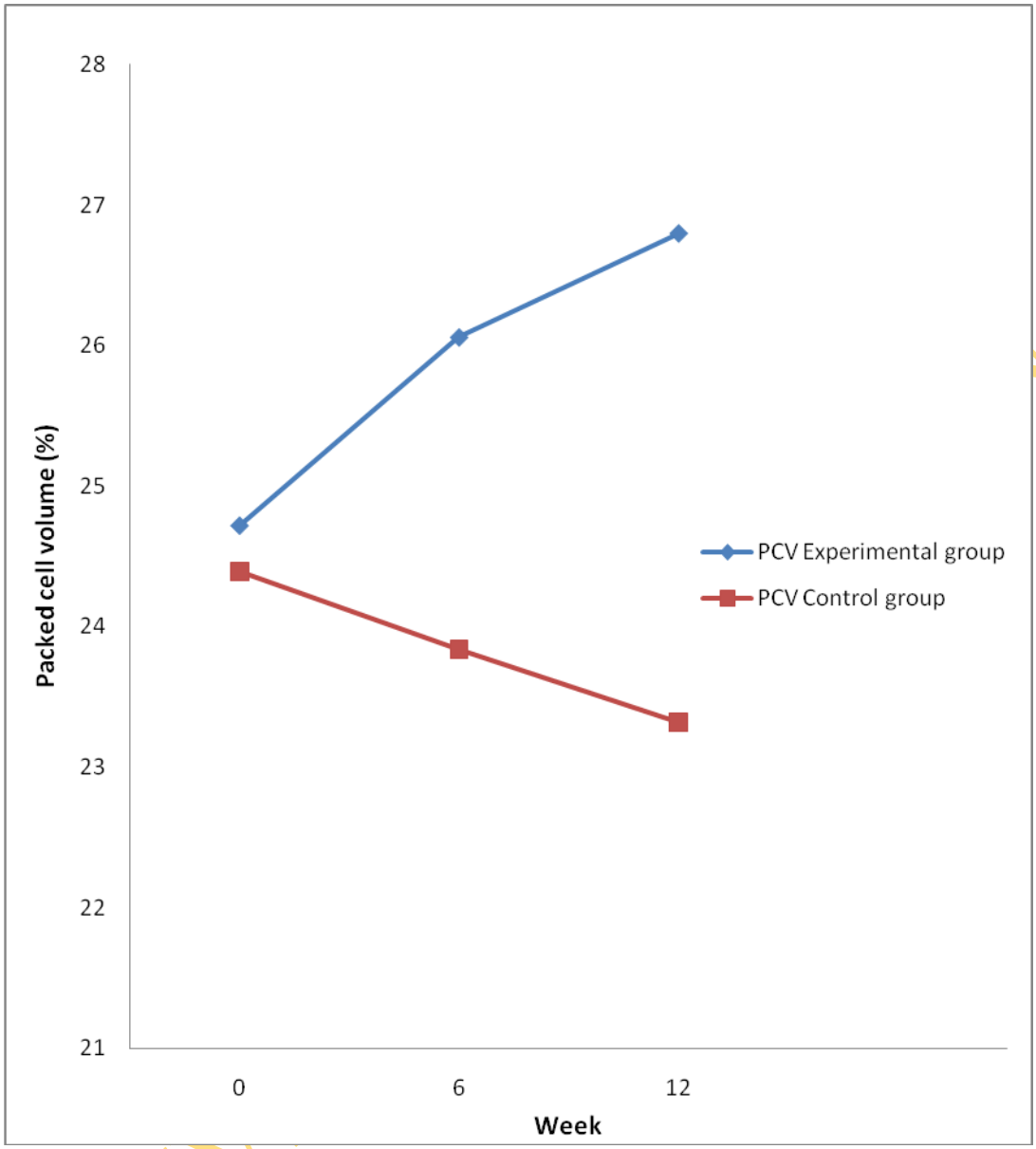

FIGURE 4: changes in packed cell volume across the three time frames

4.1.4.2 PLATELET COUNT

The mean platelet counts (PC) of the experimental group were 364.61±127.46 $\times 10^9$ /L, 322.41±100.90$\times 10^9$ /L and 269.37±86.29$\times 10^9$ /L at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively, while the mean PC for the control group were 343.58±128.81$\times 10^9$ /L, 335.20 ±132.21$\times 10^9$ /L and 331.12±122.28$\times 10^9$ /L at week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at α = 0.05 showed that the experimental group had significantly lesser platelet count than the control groups at the end of the 12th week (p= 0.005) of the study only (Table 4). While the PC for the control group remained virtually unchanged throughout the duration of the study, that of the experimental group reduced steadily during the periods (Figure 5).

4.1.4.3 MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION

The average mean corpuscular haemoglobin concentration (MCHC) of the experimental group were 33.66±1.05g/dl, 34.05±1.29g/dl and 34.11±0.88 at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively, while MCHC for the control group were 33.25±4.29g/dl, 34.24±1.83g/dl and 34.07±122g/dl at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at α = 0.05 did not show any significant difference between the MCHC of both groups at the beginning of week 0 (p= 0.384), end of the 6th week (p= 0.434) and end of the 12th week (p= 0.709) of the study (Table 4). The MCHC for the two groups followed the same trend throughout the duration of the study.

However, the MCHC for the experimental group was lower than that of the control group at each time point in the study (Figure 6).

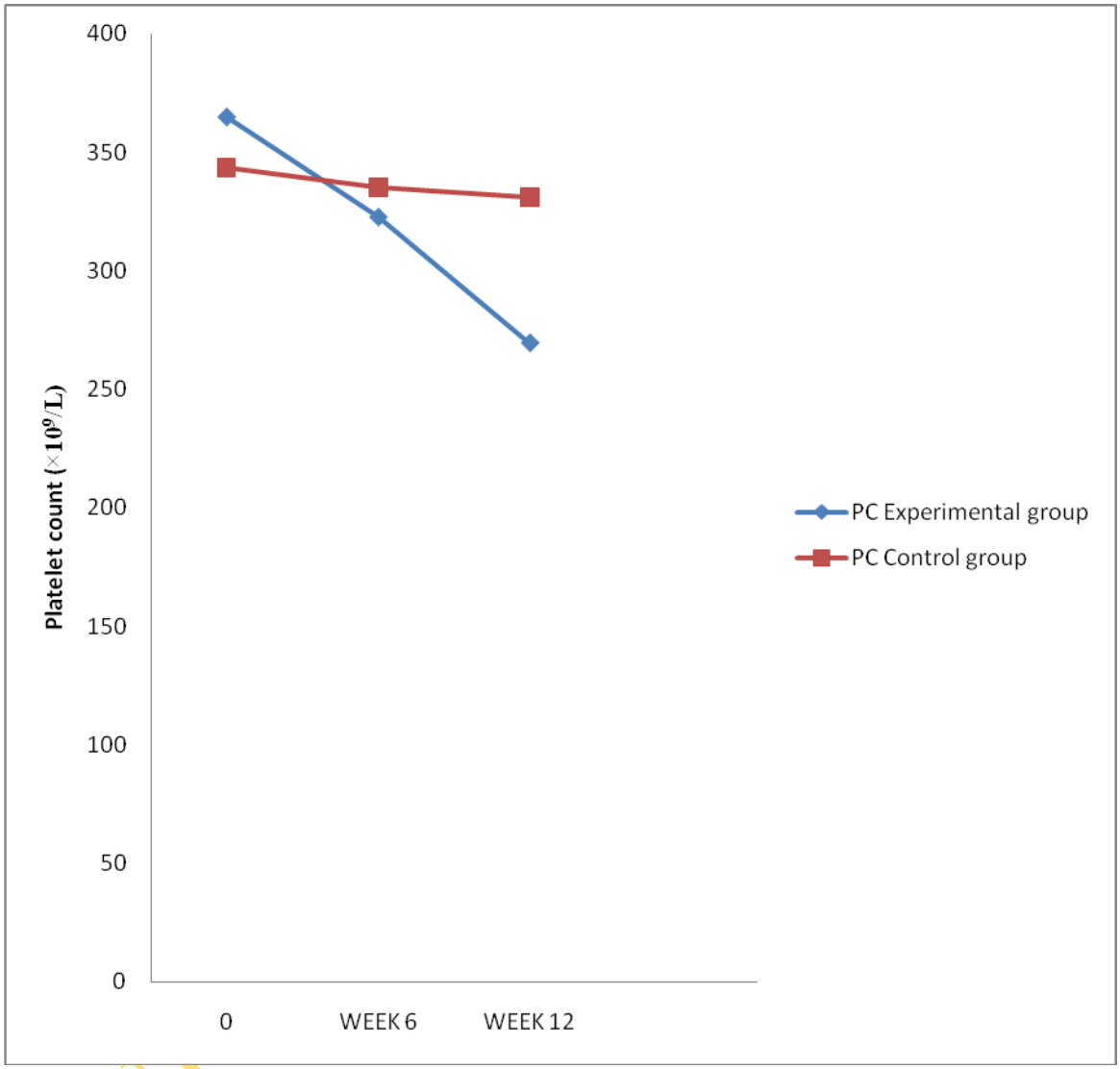

FIGURE 5: changes in platelet count across the three time frames.

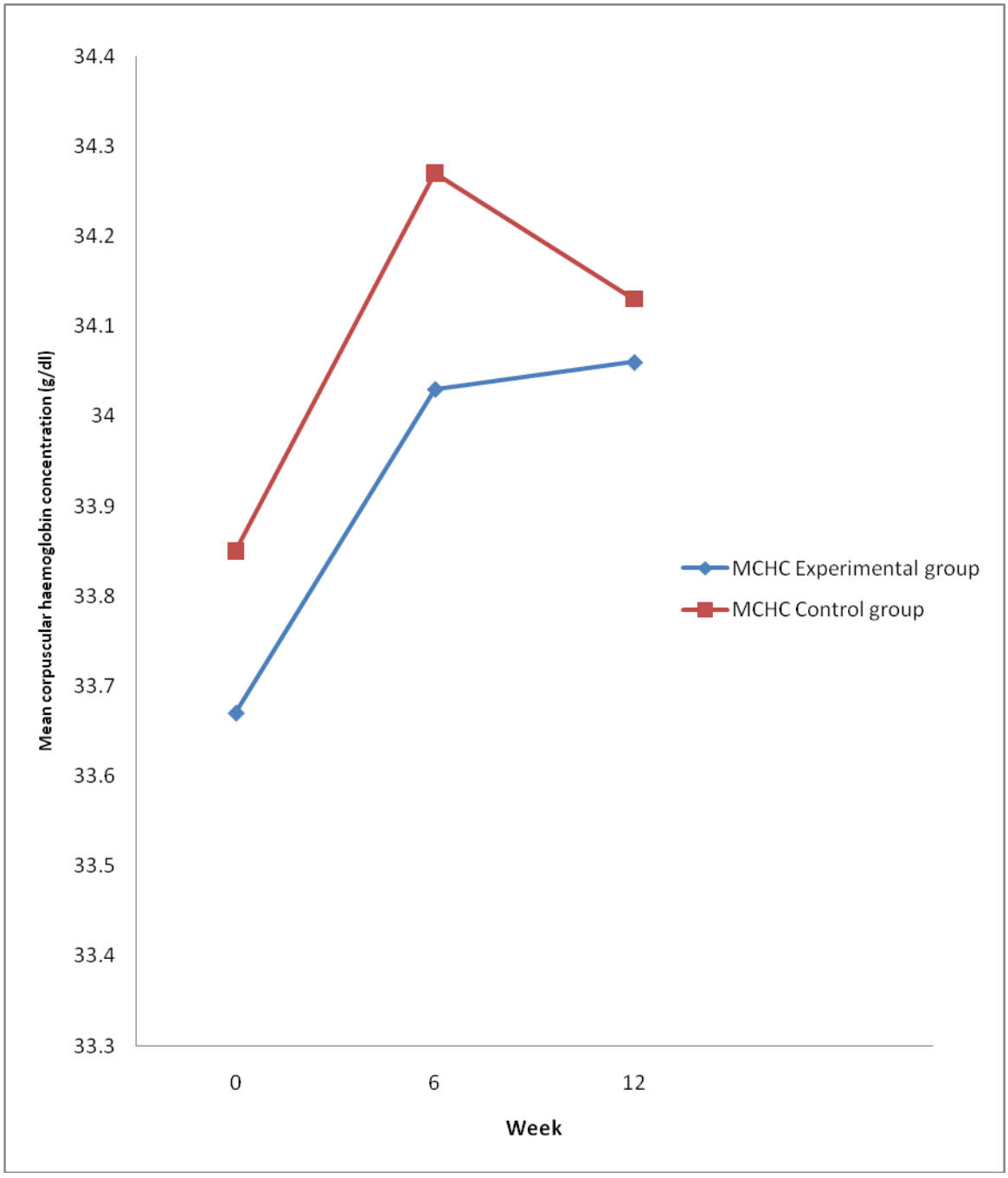

FIGURE 6: changes in the mean corpuscular haemoglobin concentration

4.1.5 COMPARISON OF QUALITY OF LIFE OF PARTICIPANTS IN EXPERIMENTAL AND CONTROL GROUPS AT WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

The mean quality of life scores (QoL) of the experimental group were 65.06±8.17, 69.22±8.09 and 72.90±7.31 at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively, while those of the control group were 65.38±10.13, 65.06±12.49 and 64.44±11.15 at the beginning of week 0, end of the 6th week and end of the 12th week of the study respectively. Independent t-test at $\alpha = 0.05$ showed that although the quality of life of the two groups were not significantly different at the beginning of week 0, the experimental group had better QoL (higher score) than the control group at the end of the 6th week ($p= 0.045$) and 12th week ($p= 0.000$) of the study (Table5). The Qol scores of the experimental group improved steadily during the duration of the study, while it decreases steadily for the control group (Figure 7).

4.1.6 COMPARISON OF PARTICICPANTS' FREQUENCY OF CRISIS

Independent t-test at $\alpha= 0.05$ comparing the frequency of crisis of the experimental and control groups at 6 months before the study did not show any significant difference ($p= 0.877$). However, at 6 months after the study the control group had significantly ($p= 0.000$) more crises than the experimental group. Paired t-test at $\alpha= 0.05$ showed that the experimental group had significantly

fewer number of crises (p= 0.000) at 6 months after the study while the control group had significantly more frequency of crisis (p= 0.033) at 6 months after the study than 6 months before the study (Table 6).

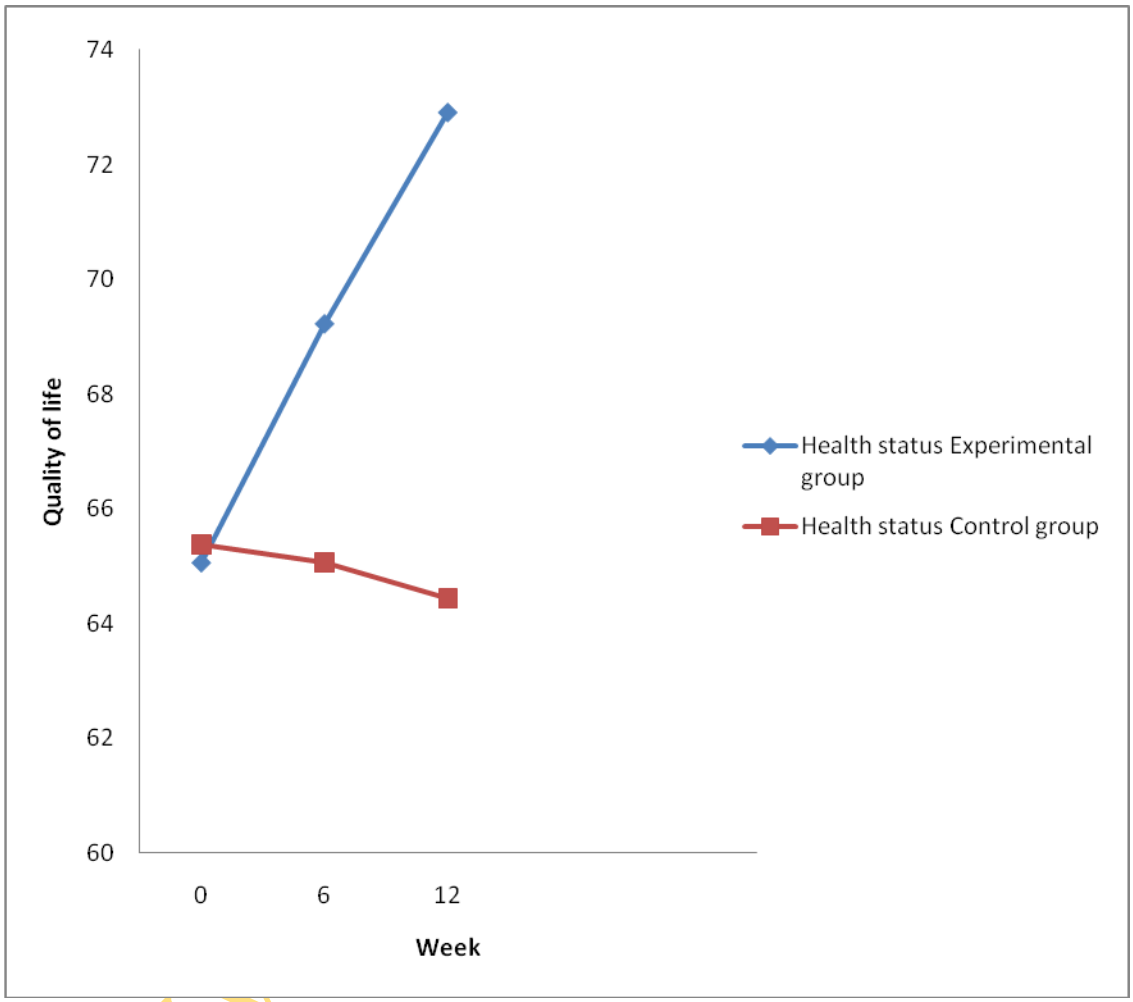

FIGURE 7: changes in quality of life across the three time frames.

4.1.7 COMPARISON OF PARTICIPANTS' FREQUENCY OF HOSPITALIZATION

Independent t-test at α= 0.05 did not show any significant difference (p= 0.923) between the frequency of hospitalization of the experimental and control groups at 6 months before the study. However, at 6 months after the study, the control group subjects had significantly greater (p= 0.001) frequency of hospitalization than the experimental group. Paired t-test at α= 0.05 showed that the experimental group had significantly lesser (p= 0.003) frequency of hospitalization at 6 months after the study, while the control group had significantly (p= 0.009) more frequency of hospitalization at 6 months after the study compared to 6 months before the study (Table 7).

4.1.8 COMPARISON OF PARTICIPANTS' LENGTH OF HOSPITALIZATION

Independent t-test at α= 0.05 did not show any significant difference (p= 0.730) between the length of hospitalization of the experimental and control groups at 6 months before the study. However, the control group had significantly longer (p= 0.000) length of hospitalization than the experimental group at 6 months after the study. Paired t-test at α= 0.05 showed that the experimental group had statistically significantly decreased (p= 0.000) length of hospitalization 6 months after the study, while the experimental group had significantly increased (p= 0.009) the

length of hospitalization at 6 months before and 6 months after the study (Table 8).

TABLE 6

COMPARISON OF PARTICIPANTS' FREQUENCY OF CRISIS AT 6 MONTHS BEFORE AND 6 MONTHS AFTER THE STUDY.

	Frequency of Crisis Group			
Time Frame	Experimental (n=54) Mean ± SD	Control (n=50) Mean ± SD	95% CI	p-value
6 months before	1.37±1.52	1.42±1.73	-0.68 to 0.58	0.877
6 months after	0.46±0.57	1.58±1.32	-1.51 to -1.52	0.000*
95% CI	0.51 to 1.31	-0.42 to 0.02		
p	0.000*	0.033*		

*Significant difference between experimental and control groups at p=0.05

CI= Confidence Interval

SD= Standard Deviation

TABLE 7

COMPARISON OF PARTICIPANTS' FREQUENCY OF HOSPITALIZATION AT 6 MONTHS BEFORE AND 6 MONTHS AFTER THE STUDY.

	Frequency of Hospitalization			
	Group			
Time Frame	Experimental (n=52) Mean ± SD	Control (n=52) Mean ± SD	95%CI	p
6 months before	0.39± 0.60	0.40±0.57	-0.24 to - 0.22	0.923
6 months after	0.13± 0.34	0.56± 0.81	-0.67 to -0.19	0.001*
95% CI	-0.09 to 0.43	-0.38 to 0.02		
p	0.003*	0.009*		

*Significant difference between experimental and control groups at p=0.05

CI= Confidence Interval

SD= Standard Deviation

4.1.9 HEART RATE OF PARTICIPANTS ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

Repeated measures analysis of variance (ANOVA) of the participants' heart rate showed significant group difference (p= 0.011) as well as significant group-time interaction (p= 0.000) (Table 9). Post-hoc analysis using paired t-test with the α level set at 0.017 by Bonferroni adjustment indicated significant differences between the heart rates of experimental group participants at 6th week/12th week and week 0/12 week time intervals. The control group however showed significant difference at week 0/week 6 time interval only (Table 10).

4.1.10 CARDIORESPIRATORY FITNESS SCORE OF PARTICIPANTS ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

Repeated measures analysis of variance (ANOVA) of the participants' cardiorespiratory fitness score showed significant group difference (p= 0.000) and significant group-time interaction (0.000) (Table 11). Post-hoc analysis using paired t-test with the α level set at 0.017 by Bonferroni adjustment indicated significant differences between the cardiorespiratory fitness score of experimental

group participants at 6th week/12th week and week 0/12th week time intervals while, the control group did not differ significantly at any of the time intervals (Table 12).

TABLE 8

COMPARISON OF LENGTH OF HOSPITALIZATION OF PARTICIPANTS AT 6 MONTHS BEFORE AND 6 MONTHS AFTER THE STUDY.

	Length of Hospitalization (days)			
	Group			
Time Frame	Experimental (n=52) Mean ± SD	Control (n=52) Mean ± SD	95% CI	p
6 months before	0.59 ±0.94	0.66 ±1.04	-0.45 to 0.32	0.730
6 months after	0.13±0.34	1.30±1.90	-1.69 to -0.65	0.000*
95% CI	0.22 to 0.71	-1.04 to -0.16		
p	0.000*	0.009*		

*Significant difference between experimental and control groups at p=0.05

CI= Confidence Interval

SD= Standard Deviation

TABLE 9

REPEATED-MEASURE ANALYSIS OF HEART RATE OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	381.03	381.03	1	6.65	0.011 *
Error	5843.00	57.28	102		
Within Subjects					
Time	164.31	164.31	1	36.96	0.000*
Group×time	116.65	116.65	1	26.24	0.000 *
Error	453.46	4.45	102		

*Significant difference at p=0.05

CI= Confidence Interval

SD= Standard Deviation

TABLE 10

PAIRED T TEST COMPARISON OF HEART RATE OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% confidence interval	p	Mean difference	95% confidence interval	p
0 vs 6	-0.61	-1.41 to 0.19	0.130	0.42	0.12 to 0.72	0.007*
6 vs 12	3.89	3.00 to 4.78	0.000*	-0.14	0.31 to 0.03	0.070
0 vs 12	3.28	2.18 to 4.37	0.000*	0.28	-0.32 to -1.63	0.109

*Indicates significant difference between pair of weeks at α= 0.017

CI= Confidence Interval

SD= Standard Deviation

TABLE 11

REPEATED-MEASURE ANALYSIS OF CARDIO-RESPIRATORY FITNESS SCORE OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	1656.54	1656.54	1	26.39	0.000 *
Error	6403.14	62.78	102		
Within Subjects					
Time	333.82	333.82	1	16.00	0.000 *
Group × time	663.82	663.82	1	31.75	0.000*
Error	2132.63	20.91	102		

*Significant difference at p= 0.05

CI= Confidence Interval

SD= Standard Deviation

TABLE 12

PAIRED T-TEST COMPARISON OF CARDIORESPIRATORY FITNESS SCORE OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% confidence interval	p	Mean difference	95% confidence interval	p
0 vs 6	1.54	0.01 to 3.06	0.048	1.44	-0.50 to 3.38	0.142
6 vs 12	4.57	3.69 to 5.46	0.000 *	-0.48	-0.12 to 0.039	0.499
0 vs 12	6.11	4.42 to 7.81	0.000 *	1.92	-0.21 to 4.05	1.808

*Indicates significant difference between pair of weeks at α= 0.017

CI= Confidence Interval

SD= Standard Deviation

4.1.11 COMPARISON OF PARTICIPANTS' VITAL CAPACITY ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

Repeated measures ANOVA comparison of participants' vital capacity showed significant group difference (p= 0.000) and significant time and group-time frame interaction (p= 0.000) (Table 13). Post-hoc analysis using paired t-test with the α level set at 0.017 by Bonferroni adjustment indicated significant differences between the vital capacity of experimental group participants at week 0/6th week, 6th week/12th week and week 0/12th week time intervals. The control group did not differ significantly at any of the time frames intervals (Table 14).

4.1.12 COMPARISON OF PARTICIPANTS' PACKED CELL VOLUME ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY

Repeated measures ANOVA comparison of packed cell volume of participants showed significant group difference (p= 0.000) and significant group-time frame interaction (p= 0.000) (Table 15). Post-hoc analysis using paired t-test with the α level set at 0.017 by Bonferroni adjustment indicated significant differences between the packed cell volume of experimental group participants at week 0/ 6th week and week 0/12 week time intervals while, the control group had significant difference at the week 0/12 time interval only (Table 16).

TABLE 13

REPEATED-MEASURE ANALYSIS OF VITAL CAPACITY OF PARTICIPANTS ACCROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	5791639.31	5791639.31	1	3492.40	0.000*
Error	32947297.20	323012.72	102		
Within Subjects					
Time	4088946.40	4088946.40	1	36.62	0.001*

Group × time	4634663.88	4634663.88	1	41.51	0.000*
Error	11388486.4	111651.83	102		

*Significant difference at p= 0.05

df= Degree of Freedom

TABLE 14

PAIRED T-TEST COMPARISON OF VITAL CAPACITY OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% CI	p	Mean difference	95% CI	p
0 vs 6	-135.41	-224.52 to -46.29	0.000*	4.02	-143.57 to 151.61	0.957
6 vs 12	-184.54	-282.54 to -86.54	0.000*	14.12	-127.93 to 164.20	0.804
0 vs 12	-319.94	-424.06 to -215.83	0.000*	18.14	-1.73 to 29.97	0.080

*Indicates significant difference between pair of weeks at α = 0.017

CI= Confidence Interval

TABLE 15

REPEATED-MEASURE ANALYSIS OF PACKED CELL VOLUME OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	381.49	193373.41	1	7166.93	0.000*
Error	2752.10	26.98	102		
Within Subjects					
Time	16.12	16.12	1	3.58	0.061
Group×time	140.75	140.75	1	31.28	0.000*

Error	458.90	4.50	102

*Significant difference at p=0.05

TABLE 16

PAIRED T-TEST COMPARISON OF PACKED CELL VOLUME OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% CI	p	Mean difference	95% CI	p
0 vs 6	-1.34	-2.19 to -0.49	0.003*	0.55	-0.14 to 1.25	0.113
6 vs 12	-0.73	-1.49 to - 0.03	0.058	0.54	0.05 to 1.00	0.033

0 vs 12	-2.07	-3.01 to -1.14	0.000*	1.09	0.36 to 1.80	0.004*

*Indicates significant difference between pair of weeks at α= 0.017

CI= Confidence Interval

4.1.13 COMPARISON OF PARTICIPANTS' PLATELET COUNT ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Repeated measures analysis of variance comparison of participants platelet count showed significant time difference (p= 0.000) as well as significant group-time frame interaction (p= 0.001) (Table 17). Post-hoc analysis using paired t-test with the α level set at 0.017 by Bonferroni adjustment indicated significant differences between the platelet count of experimental group participants at 6^{th} week/12^{th} week and week 0/12^{th} week time intervals. The control group did not differ significantly at any of the time frames intervals (Table 18).

4.1.14 COMPARISON OF PARTICIPANTS' MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Repeated measures ANOVA comparison of participants' MCHC did not show significant group difference ($p>0.05$), though there was significant ($p<0.05$) group-time frame interaction (Table 19).

4.1.15 QUALITY OF LIFE SCORES OF PARTICIPANTS ACROSS WEEK 0, WEEK 6 AND WEEK 12 OF THE STUDY.

Repeated measures ANOVA comparison of participants' health status showed significant time ($p= 0.000$) and group differences ($p= 0.000$) as well as significant group-time frame interaction ($p= 0.000$) (Table 20). Post-hoc analysis using paired t-test with the

TABLE 17

REPEATED-MEASURE ANALYSIS OF PLATELET COUNT OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	24779.76	24779.76	1	1141.23	0.000
Error	2990411.78	29317.76	102		
Within Subjects					
Time	15056978	150569.78	1	18.17	0.000*

Group×time	88952.68	88952.68	1	10.74	0.001*
Error	845080.15	8285.10			

*Significant difference at p=0.05

TABLE 18

PAIRED T-TEST COMPARISON OF PLATELET COUNT OF PARTICIPANTS ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% confidence interval	p	Mean difference	95% confidence interval	p
0 vs 6	42.20	6.14 to 78.27	0.023	8.38	-15.27 to 32.03	0.480
6 vs 12	53.04	22.97 to 83.10	0.001*	4.08	-2.13 to 10.29	0.193

0 vs 12	95.24	51.99 to 138.49	0.000*	12.46	-11.88 to 36.80	0.309

*Indicates significant difference between pair of weeks at α= 0.017

TABLE 19

REPEATED-MEASURE ANALYSIS OF PARTICIPANTS' MEAN CORPUSCULAR HAEMOGLOBIN CONCENTRATION ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	2.18	2.18	1	0.890	0.348
Error	250.35	2.45	102		

Within Subjects					
Time	5.55	5.55	1	7.00	0.009
Group×time	0.135	0.135	1	0.17	0.681
Error	80.86	0.793	102		

*Significant difference at p= 0.05

TABLE 20

REPEATED-MEASURE ANALYSIS OF PARTICIPANTS' QUALITY OF LIFE SCORES ACROSS THE THREE TIME FRAMES OF THE STUDY.

Source	Type III sum of Square	Mean Square	df	F	p
Between Subjects					
Group	1311.13	1398975.50	1	6432.41	0.000*
Error	22183.83	217.49	102		
Within Subjects					

Time	617.46	617.46	1	16.17	0.000*
Group×time	1000.87	1000.87	1	26.22	0.000*
Error	3894.22	38.18	102		

*Indicates significant difference at p = 0.05

α level set at 0.017 by Bonferroni adjustment indicated significant differences between the quality of life of experimental group participants at week 0/6th week, 6th week/12th week and week 0/12 week time intervals. The control group did not differ significantly in their quality of life at the three time intervals (Table 21).

TABLE 21

PAIRED T-TEST COMPARISON OF PARTICIPANTS' QUALITY OF LIFE SCORES ACROSS THE THREE TIME FRAMES OF THE STUDY.

	Experimental			Control		
Week	Mean difference	95% CI	p	Mean difference	95% CI	p
0 vs 6	-4.16	-5.82 to -2.50	0.000*	0.32	-3.03 to 3.66	0.849
6 vs 12	-3.56	-5.32 to -1.80	0.000*	0.82	0.08 to 1.57	0.031

0 vs 12	-7.72	-5.32 to -1.80	0.000*	1.14	-1.97 to 4.26	0.465

*Indicates significant difference between pair of weeks at α= 0.017

4.2 Hypothesis Testing

The hypotheses proposed at the beginning of the study were tested as follows:

Hypothesis 1

There would be no significant differences between the heart rate of the experimental and control groups at the three time frames (week 0, 6th week and 12th week) of the study.

The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.103

At 6th week, p-value was = 0.576

At 12th week, p-value was = 0.000

The hypothesis was therefore ACCEPTED for week 0 and end of 6th week but REJECTED for end of 12th week of the study.

Hypothesis 2

There would be no significant differences between the cardiorespiratory fitness scores of the experimental and control groups at week 0, end of 6th week and end of the 12th week of the study. The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.058

At end of 6th week, p-value was = 0.004

At end of 12th week, p-value was = 0.000

The hypothesis was therefore ACCEPTED for week 0 but REJECTED for end of 6th week and end of 12th week of the study.

Hypothesis 3

There would be no significant differences between the vital capacity of the experimental and control groups at week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.476

At end of 6th week, p-value was = 0.000

At end of 12th week, p-value was = 0.000

The hypothesis was therefore ACCEPTED for week 0, but REJECTED for end of 6th week and end of 12th week of the study.

Hypothesis 4

There would be no significant differences between the packed cell volume of the experimental and control groups at week o, end of 6th week and end of 12th week of the study. The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.614

At end of 6th week, p-value was = 0.002

At end of 12th week, p-value was = 0.000

The hypothesis was therefore ACCEPTED for week 0 but REJECTED for end of 6th week and end of 12th week of the study.

Hypothesis 5

There would be no significant differences between the platelet count of the experimental and control groups at week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.405

At end of 6th week, p-value was = 0.579

At end of 12th week, p-value was =0.003

The hypothesis was therefore ACCEPTED for week 0 and end of 6th week but REJECTED for end of 12th week of the study.

Hypothesis 6

There would be no significant differences between the mean corpuscular haemoglobin concentration of the experimental and control groups at week 0, end of 6th week and end of 12th weeks of the study. The hypothesis was tested using independent t-test at α= 0.05

At week 0, p-value was = 0.384

At end of 6th week, p-value was = 0.434

At end of 12th week, p-value was =0.709

The hypothesis was therefore ACCEPTED for week 0, end of 6th week and end of 12th week of the study.

Hypothesis 7

There would be no significant difference between the frequency of crisis of the experimental and control groups at 6 months before the intervention. The hypothesis was tested using independent t-test at $\alpha = 0.05$.

Observed p-value = 0.877

Since p>0.05, the hypothesis was therefore ACCEPTED.

Hypothesis 8

There would be no significant difference between the frequency of crisis of the experimental and control groups at 6 months after the intervention. The hypothesis was tested using independent t-test at $\alpha = 0.05$.

Observed p-value = 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 9

There would be no significant difference between the frequency of hospitalization of the experimental and control groups 6 months before the intervention. The hypothesis was tested using independent t-test at $\alpha = 0.05$

Observed p-value = 0.923

Since p>0.05, the hypothesis was therefore ACCEPTED.

Hypothesis 10

There would be no significant difference between the frequency of hospitalization of the experimental and control groups 6 months after the intervention. The hypothesis was tested using independent t-test at α= 0.05

Observed p-value = 0.001

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 11

There would be no significant difference between the length of hospitalization of the experimental and control groups 6 months before the intervention. The hypothesis was tested using independent t-test at α= 0.05.

Observed p-value = 0.730

Since p>0.05, the hypothesis was therefore ACCEPTED for 6 months before the study.

Hypothesis 12

There would be no significant difference between the length of hospitalization of the experimental and control groups at 6 months after the intervention. The hypothesis was tested using independent t-test at α= 0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED for 6 months after the study.

Hypothesis 13

There would be no significant differences in the heart rate of the experimental group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p= 0.05.

Observed p-value= 0.011

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 14

There would be no significant differences in the cardiorespiratory fitness score of the experimental group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 15

There would be no significant differences in the vital capacity of the experimental group across week 0, 6th week and 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED..

Hypothesis 16

There would be no significant differences in the heart rate of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.011

Since p<0.05, the hypothesis was therefore REJECTED..

Hypothesis 17

There would be no significant differences in the cardiorespiratory fitness score of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.005, the hypothesis was therefore REJECTED.

Hypothesis 18

There would be no significant differences in the vital capacity of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 19

There would be no significant difference in the packed cell volume (PCV) of the experimental group across the week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 20

There would be no significant difference in the Platelet count of the experimental group across the week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 21

There would be no significant difference in the mean corpuscular haemoglobin concentration of the experimental group at week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated-measure ANOVA at p= 0.05

Observed p-value = 0.009

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 22

There would be no significant differences in the packed cell volume (PCV) of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.005, the hypothesis was therefore REJECTED.

Hypothesis 23

There would be no significant differences in the platelet count of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 24

There would be no significant difference in the mean corpuscular haemoglobin concentration of the control group at week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated-measure ANOVA at α= 0.05.

Observed p= 0.348

Since p>0.05

The hypothesis was therefore ACCEPTED.

Hypothesis 25

There would be no significant differences between the quality of life of experimental and control groups across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using independent t-tests at p= 0.05.

At week 0 p-value was = 0.860

At end of 6th week, p-value was = 0.045

At end of 12th week, p-value was = 0.000

The hypothesis was therefore ACCEPTED for week 0 but REJECTED for end of 6th week and end of 12th week of the study.

Hypothesis 26

There would be no significant difference in the quality of life of the experimental group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated-measure ANOVA at p= 0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore REJECTED.

Hypothesis 27

There would be no significant difference in the quality of life of the control group across week 0, end of 6th week and end of 12th week of the study. The hypothesis was tested using repeated measures ANOVA at p=0.05.

Observed p-value= 0.000

Since p<0.05, the hypothesis was therefore ACCEPTED.

4.3 DISCUSSION

4.3.1 PHYSICAL CHARACTERISTICS OF PARTICIPANTS

It was observed in this study that the experimental and control groups were not significantly different in age (p= 0.686), height (p= 0.656), weight (p= 0.476) and body mass index (p= 0.097). Participants in both groups hence appear to have been fairly matched and differences in physical characteristics could not have been a rival hypothesis for differences between the groups' cardiorespiratory and haematological variables. Annie et al, (2010) reported that majority of sickle cell patients attending hospitals in Lagos are aged between 14 and 56 years. The ages of participants in this study ranged between 18 and 53 years, hence were within the age range.

4.3.2 EFFECTS OF AEROBIC EXERCISE TRAINING ON CARDIORESPIRATORY PARAMETERS OF PARTICIPANTS.

The results of this study indicated that there was no statistically significant difference between the heart rates of the experimental and control groups at week 0 and at the end of 6th week of this study. Alawale, (1998) similarly found no statistically significant difference between the heart rates of their experimental and control groups of sickle cell anaemia patients at the beginning of week 0 and end of 6th week. However, at 12th week, the experimental group in this study had significantly lesser HR than the control group thus, indicating improved fitness in this group. This is contrary to the findings of Alawale, (1998) of no significant difference between the experimental and control groups' heart rate even at the end of 12th week. The difference observed in this study could be attributed to the

systematic progression of workload, by fixed duration of 5 minutes every two weeks while in Alawale (1998), the workload was not systematically progressed. Oyono-Enguelle et al, (2000) had similarly reported that a thrice weekly aerobic conditioning, significantly lower heart rates in patients with sickle cell anaemia.

There was no statistically significant difference between the cardiorespiratory fitness score (CRFS) of the experimental and control groups at week 0 of this study. There was however, statistically significant difference between the cardiorespiratory fitness scores of the two groups at the ends of the 6^{th} and 12^{th} weeks of the study. Specifically, the experimental group had lower cardiorespiratory fitness score (better fitness) than the control group at the ends of 6th and 12^{th} weeks of the study. The experimental group's improved fitness is in consonance with the report by Charache et al, (1983) that exercise capacity of patients with sickle cell anaemia improved after undergoing submaximal exercise and that the patients were able to perform increased amounts of work at lower heart rates. Morey (1999) had similarly reported that endurance exercise training in sickle cell anaemia patients improved their participation in physical and social activities.

There was no statistically significant difference between the vital capacity of the experimental and control groups at week 0 of this study, but the experimental group had significantly higher VC at the ends of 6^{th} and 12^{th} weeks of this study. Alawale, (1998) similarly reported that a 12-week endurance training had significant effect on the forced vital capacity of patients with sickle cell anaemia . It has been suggested that longterm aerobic exercise results in an

increase in the strength of respiratory muscles (diagphragm and intercostals) with consequent improvement in respiratory volumes (vital capacity and tidal volume) (McArdle et al, 2000). According to the American Thoracic Society, a controlled study of eight weeks of pulmonary rehabilitation showed that endurance exercise improved the level of dyspnoea in patients with sickle cell anaemia (Morey, 1999).

4.3.3 EFFECTS OF AEROBIC DANCE EXERCISE TRAINING ON HAEMATOLOGICAL PARAMETERS OF PARTICIPANTS.

There was no statistically significant difference between the packed cell volume of the experimental and control groups at week 0 of this study, but there were statistically significant differences between the packed cell volumes of the experimental and control groups at the end of 6th week and at the end of 12th week of this study, with the experimental group having greater packed cell volume at the end of 6th week and 12th week of this study. This finding could be sequel to the established long term effect of aerobic exercise training on packed cell volume, as Schwandt et al, (1991) has opined that the amount of red blood cells in circulation increases as a result of increased cardiac output following a long period of aerobic exercise training.

There was no statistically significant difference between the platelet counts of the experimental and control groups at week 0 and at the end of 6th week of this study. However, there was a statistically significant difference between the platelet counts of the experimental and control groups at the end of the 12th week of this study, with the experimental group having lesser platelet

count than the control group. This finding is consistent with the report that long term aerobic exercise training results in decrease in platelet count and increase in bleeding time (Prisco et al, 1994).

There was no statistically significant difference between the Mean Cell Haemoglobin Concentration (MCHC) of the experimental and control groups at any of the time periods of this study. The MCHC is an important guide for the colour of red blood cells to know the extent of aneamic condition. This finding suggests that the concentration of haemoglobin in a given volume of packed red blood cells was not significantly affected by the aerobic exercise programme.

4.3.4 EFFECT OF AEROBIC EXERCISE TRAINING ON QUALITY OF LIFE OF PARTICIPANTS.

There was no statistically significant difference between the quality of life of the experimental and control groups at week 0 of this study. However, the experimental group had higher quality of life scores than the control group at the ends of the 6^{th} week and 12^{th} week of this study. The improvements in HR, CRFS, VC, PCV and PC might have resulted in fewer crisis and hence better quality of life for participants in the experimental group.

4.3.5 EFFECTS OF AEROBIC EXERCISE TRAINING ON PARTICIPANTS' FREQUENCY OF CRISIS, FREQUENCY OF HOSPITALIZATION AND LENGTH OF HOSPITALIZATION.

There was no statistically significant difference between the frequency of crisis of the experimental and control groups at 6 months before this study. However, at 6 months after the study, the experimental group had significantly lesser frequency of crisis while the control group had more crises. Interestingly, during the study, 1 (0.78%) of the experimental group participants had crisis while, 7 (5.43%) of participants in the control group had crisis. The reduced frequency of crisis in the experimental group may be consequent on the reduced number of sickled red blood cells in circulation and improved quality of life of participants in this group. Expectedly, with better quality of life, the frequency of crisis will be reduced.

There was no statistically significant difference between the frequency of hospitalization of the experimental and control groups at 6 months before this study. However, at 6 months after the study, the experimental group had significantly lesser frequency of hospitalization, while the control group had more hospitalization. The reduced frequency of hospitalization in the experimental group may be consequent on the improved quality of life and reduced frequency of crisis of participants in this group. Expectedly, with better quality of life and less frequency of crises, the frequency of hospitalization will be reduced.

There was also no statistically significant difference between the length of hospitalization of the experimental and control groups at 6 months before this study. However, at 6 months after the study, the experimental group had significantly shorter length of hospitalization while the control group had longer length of hospitalization. The reduced length of hospitalization in the

experimental group may be consequent on the improved quality of life, frequency of crisis and frequency of hospitalization of participants in this group.

Previous studies have not looked at variables such as FC, FH, LH and quality of life of patients with sickle cell anaemia, hence, findings from this study concerning those variables cannot be compared to those from other studies. However, the reduction in FC, FH and LH during the follow-up period suggests that the effect of the aerobic exercise training may be sustained over at least a 6-month period.

4.4. CLINICAL IMPLICATION OF FINDINGS.

The outcome of this study has shown that aerobic dance, a type of aerobic exercise, could have positive effects on the cardiorespiratory parameters (HR, CRFS and VC), haematological parameters (PCV and PC) and quality of life of individuals with sickle cell anaemia. Additionally, the aerobic dance training also reduced the frequency of crisis, frequency of hospitalization and length of hospitalization of individuals with sickle cell anaemia at 6 months follow-up. This finding suggests that the effects of aerobic dance training lasts beyond the training period and could be sustained over a subsequent period of 6 months. Aerobic dance is therefore an adjunct intervention in the treatment of patients with sickle cell anaemia. It has the additional advantages that it is inexpensive and could be safely prescribed as an enjoyable home programme for suitable patients with sickle cell anaemia.

CHAPTER FIVE

SUMMARY, CONCLUSION AND RECOMMENDATION

5.1 SUMMARY

Sickle cell anaemia (SCA) is a general term for a group of genetic disorders characterized by the predominance of sickle cell hemoglobin (HbS) and severe attacks of pain resulting from blood vessels that are being occluded by red blood cells that become rigid and form a sickle shape when de-oxygenated. Exercise is an important physiotherapeutic management which has not been adequately explored in the management of patients with sickle cell disease. This study looked at the effects of a 12-week aerobic dance programme on cardiorespiratory, haematological variables and health status of individuals with sickle cell anaemia. The effects of aerobic exercise programme on quality of life, frequency of crisis, frequency of hospitalization and length of hospitalization in patients with sickle cell anaemia were also studied.

The literature review focused on the definition, types and history of sickle cell anaemia and its epidemiology, pathogenesis and pathophysiology, clinical features and complications. Other areas covered by the literature review are: sickle cell crises, diagnosis, precautions and treatment, prevention of sickle cell disease, exercise, principles of exercise training, components of exercise training, physiological adaptation to exercise training, aerobic exercise, exercise and sickle cell disease, quality of life, measures of quality of life, The Rand Short Form-36 Health Survey (Version 1.0), Scoring of Rand SF-36 Item Health Survey (Version 1.0) and justification of the study.

The study was a two-group randomised control trial involving 129 consecutively recruited individuals with SCA assigned to exercise (65) and control (64) groups. However, 104 participants (exercise group= 54, control group= 50) completed the study. Data on FC, FH and LH in the 6-months before the study were retrieved from participants' hospital files. The exercise group had 12 weeks of aerobic dance exercises, comprising dancing thrice weekly; the intensity being progressed by increasing the duration by five minutes fortnightly. Data on QoL (using Short Form-36 questionnaire), cardiorespiratory variables [Heart Rate (HR), Cardiorespiratory Fitness Score (CRFS) and Vital Capacity (VC)] and haematological variables [Packed Cell Volume (PCV), Platelet Count (PC) and Mean Corpuscular Haemoglobin Concentration (MCHC)] were collected at baseline and at the ends of the 6^{th} and 12^{th} week. Information on FC, FH and LH during the 6-month follow-up phase was also retrieved from participants' hospital files. Data were analysed using ANOVA, independent and paired t-tests at $p = 0.05$.

The mean ages of the exercise (26.1±6.7 years) and control (25.7±5.6 years) groups were not significantly different. At baseline, the QoL (65.1±8.2, 65.4±10.1), HR (75.2±4.5 b/min, 76.8±5.1 b/min), CRFS (52.6±4.1, 54.3±4.5) VC (1714.5±400.4 cc, 1773.9±446.5 cc), PCV (24.7±3.2 %, 24.4±3.4 %), PC (364.6±127.5×10^9/L, 343.6±128.8×10^9 /L) and MCHC (33.7±1.0 g/dl, 33.9±1.1 g/dl) for exercise and control groups were not significantly different. The exercise group compared with the control had better CRFS (50.8±5.5, 54.2±6.4

respectively), VC (2109.4±441.9cc, 1769.9±389.6cc respectively) and PCV (26.1±3.4%, 23.84±3.8% respectively). The exercise group compared with the control had better HR (71.9±3.5 b/min, 76.5±4.9 b/min), CRFS (46.5±6.2, 55.3±6.7), VC (2293.9±374.1 cc, 1755.8±377.6 cc) and PCV (26.8±3.3 %, 23.32±3.5 %) at the end of the 12th week. Although the pre-intervention variables for exercise and control groups were not significantly different for their FC (1.4±1.5, 1.4±1.7 respectively), FH (0.4±0.6, 0.4±0.6 respectively) and LH (0.6±0.9 days, 0.7±1.0 days respectively). However, the exercise group had significantly less FC (0.5±0.6), FH (0.1±0.3) and shorter LH (0.1±0.3 days) in the follow-up phase of the study.

5.2 CONCLUSIONS

1. Findings from this study showed that the 12-week aerobic exercise programme improved the quality of life, vital capacity and cardiorespiratory fitness score and reduced the HR, frequencies of crisis and hospitalization and length of hospitalization of individuals with sickle cell anaemia.
2. Aerobic exercise training is not only effective in producing health-enhancing effects in individuals with sickle anaemia but that such effects on frequency of crisis, frequency of hospitalization and length of hospitalization could be sustained over subsequent period of 6 months.
3. Aerobic exercise should be routinely included as an adjunct therapy in the total management of patients with sickle cell anaemia because it is not

expensive it could be prescribed to the sickle cell patients as an enjoyable home programme since it has been documented that music and dance improves psychological well being of human beings.

5.3 RECOMMENDATIONS

The following recommendations were made.

5.3.1 Recommendations to Physicians

The outcome of this study would encourage Physicians to refer their sickle cell anaemia patients to the Physiotherapist not only when certain complications such as bone problems occur, but as part of the wholistic treatment and improving the quality of life of sickle cell anaemia patients.

5.3.2 Recommendations to Physiotherapists

The outcome of this study would encourage the Physiotherapist to get involved in the total care of patients with sickle cell anaemia by providing an insight into aerobic exercise as an intervention for patients with sickle cell anaemia.

5.3.3 Recommendations to Sickle Cell Anaemia Patients

The outcome of this study would change the orientation of patients with sickle cell anaemia participating in carefully prescribed and closely monitored aerobic exercise programmes without adverse clinical effects.

5.3.4 Recommendations to Parents

The outcome of this study would encourage parents of patients with sickle cell anaemia to change their orientation towards their wards participating in monitored aerobic exercise programme, without fear that exercise could trigger a crisis in patients with sickle cell anaemia.

5.3.5 Recommendations to the Health policy makers

1. The outcome of this study also showed that schools could involve patients with sickle cell anaemia in monitored aerobic exercise in order to prevent the sickle cell patients from developing psychosocial problems and stigmatization by peers.
2. The outcome of this study showed that aerobic dance could be prescribed as an enjoyable home programme for suitable patients with sickle cell anaemia, since music and dance have health enhancing effects on social and mental wellbeing, aerobic dance could prevent sickle cell patients from falling into depressed mood.

5.3.5 Recommendations for Further Studies

1. Further studies should look into evaluating the cardiorespiratory and haematological variables and quality of life during the follow-up period to see what effect would stopping the aerobic exercise have on patients with sickle cell anaemia.
2. This study did not use a sickle cell specific questionnaire to assess the quality of life of patients with sickle cell anaemia because none was

available, therefore future studies should develop a sickle cell specific questionnaire to assess the quality of life of sickle cell patients.

3. This study only evaluated the effect of a 12 week aerobic exercise on one of the red blood cell indices (Mean corpuscular haemoglobin concentration). Future studies should evaluate the effect of aerobic exercise on other red blood cell indices such as mean cell haemoglobin, mean cell volume and red cell distribution width.

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APPENDIX A

LAGOS STATE UNIVERSITY TEACHING HOSPITAL, IKEJ
1-5, OBA AKINJOBI ROAD, IKEJA, LAGOS. P.M.B. 21005, TEL: 01-4710670, 4975739.
www.lasuth.org. e-mail: dcst@lasuth.org.
DIRECTORATE OF CLINICAL SERVICES AND TRAINING

Ref. No. LREC/08/05/20 13TH AUGUST 2008.

MRS AKINOLA T.O
PHYSIOTHERAPY DEPT.
UNIVERSITY COLLEGE HOSPITAL
IBADAN.

PERMISSION TO CARRY OUT RESEARCH PROJECT

DR. OLABODE V. OGUNBANJO
BDS, FWACS, FICS
Director Of Clinical Services and Training
08038630066, 08055226896.

I am directed to refer to your application for permission to carry out a research Project and to convey the approval of the Management after due recommendation by the Research and Ethics Committee for you to utilize our facilities for your project Titled "Effects of a 12 weeks Aerobic Dance Programme on Cardio respiratory, Heamatological Variables and Health Status of individual with Sickle Cell Anaemia".

You are therefore advised to relate directly with all relevant HODs in LASUTH for necessary assistance.

It is pertinent to note that the outcome of your research should be forwarded to the Secretary to the Research and Ethics Committee, DCST'S office, LASUTH, Ikeja.

Congratulations

Prof. O.Ogundipe
Chairman, Research & Ethics Committee.

DR. FEMI OLUGBILE
FRCPsych.FMCPsych.MNIM
Chief Medical Director
08037707546, 01-4710670

APPENDIX B

LETTER OF INTRODUCTION

Dear Sir /Madam,

Mrs. Akinola T. O. is a Ph.D. student in the Department of Physiotherapy, College of Medicine, University of Ibadan (Matric NO. 112406). She is carrying out a research study titled "Effects of a 12-week aerobic exercise programme on cardiorespiratory and haematological variables and quality of life of individuals with sickle cell anaemia" in partial fulfilment of the requirements for the award of the Ph. D (Physiotherapy) degree of the University of Ibadan.

We therefore, solicit your cooperation in completing the attached questionnaire honestly, and in allowing some measurements to be taken on you. We wish to assure you that all information will be kept strictly confidential and used for research purpose only.

Thank you.

Mrs. Odunayo .T. Akinola **Dr B.O.A Adegoke**

_____ _____

Researcher Supervisor

APPENDIX C

INFORMED CONSENT FORM FOR THE STUDY ON EFFECTS OF A 12-WEEK AEROBIC EXERCISE PROGRAMME ON CARDIORESPIRATORY AND HAEMATOLOGICAL VARIABLES AND QUALITY OF LIFE OF INDIVIDUALS WITH SICKLE CELL ANAEMIA.

I……. voluntarily consent to participate in the above named research program being conducted by Mrs. T. O. Akinola in the haematology department, Lagos State University Teaching Hospital, Ikeja, Lagos.

I have been given the following information:

that the research is being undertaken to find out the effects of a 12-week aerobic exercise programme on cardiorespiratory and haematological variables and quality of life of individuals with sickle cell anaemia.

(i) that I will have to complete a questionnaire**.**

(ii) that some measurements will be taken on different parts of my body (lung volume, blood analysis).

(iii) that I will have to participate in a 12 week aerobic dance exercise programme.

(iv) that information on my frequency of crises, frequency of hospitalization and length of hospitalization will be obtained from my clinical notes and by interview.

(v) that the information obtained from me will be treated as private and confidential. However, the information will be used for statistical or scientific purposes with my right of privacy retained.

(vi) that I have the right to ask questions regarding the procedure.

(vii) that I am guaranteed the right to withdraw from the study at any time.

Participant's signature and date: …… ……

APPENDIX D

DATA RECORDING SHEET.

S/N…….. Week……

Tel……..

Name……. Age…… Weight……

Height……

Heart Rate……..

Systolic Blood Pressure……..

Diastolic Blood Pressure……

Vital Capacity……

Platelet Count……

PCV…….

Mean Corpuscular Haemoglobin concentration.....

Cardiorespiratory Fitness Score……

Health Status Scores…….

Pre aerobic dance

Frequency of Crises during the past 6 Months……

Frequencyof Hospitalization during the past 6 months……

Length of Hospitalization during the past 6 months……..

Post aerobic dance

Frequency of Crises during the past 6 months……

Frequency of Hospitalization during the past 6 months……

Length of Hospitalization during the past 6 months……

APPENDIX E

Serial Number …….

MEDICAL OUTCOMES STUDY: 36-ITEM SHORT FORM SURVEY INSTRUMENT 1.0 (RAND HEALTH, 2007).

Kindly read through the following questions and choose the one that appropriately describes your health.

1. In general, would you say your health is: (1) Excellent (2) very good (3) Good (4) Fair (5) Poor
2. Compared to one year ago, how would you rate your health in general now? (1) Much better now than one year ago (2) Somewhat better now than one year ago (3) About the same (4) Somewhat worse now than one year ago (5) Much worse now than one year ago

The following items are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much? *(Circle one number on each line)*

1 = Yes, Limited a Lot

2 = Yes, Limited a little

3 = No, not limited at all

S/n	Question			
3.	Vigorous activities such as running, lifting heavy objects,			

	participating in strenuous sports			
4.	Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling, or playing golf			
5.	Lifting or carrying groceries			
6.	Climbing several flights of stairs			
7.	Climbing one flight of stairs			
8.	Bending, kneeling, or stooping			
9.	Walking more than a mile			
10.	Walking several blocks			
11.	Walking one block			
12.	Bathing or dressing yourself			

During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of your physical health?

(circle one option on each line)

		Yes	No
13.	Cut down the amount of time you spent on work or other activities		
1 4.	Accomplished less than you would like		
1 5.	Were limited in the kind of work or other activities		
16.	Had difficulty performing the work or other activities (for example, it took extra effort)		

During the past 4 weeks, have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?

(circle one option on each line)

		Yes	No
17.	Cut down the amount of time you spent on work or other activiti		
18.	Accomplished less than you would like		
19.	Didn't do work or other activities as carefully as usual		

20. During the past 4 weeks, to what extent has your physical health or emotional problems interfered with your normal social activities with family, friends, neighbors, or groups? *(circle one number)* (1) Not at all (2) Slightly (3) Moderately (4) Quite a bit (5) Extremely

21. How much bodily pain have you had during the past 4 weeks? *(circle one number)* (1) None (2) Very mild (3) Mild (4) Moderate (5) Severe (6) Very severe

22. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)? (*Circle one number)* (1) Not at all (2) A little bit (3) Moderately (4) Quite a bit (5) Extremely

These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling. How much of the time during the past 4 weeks…

(circle one option on each line)

		All of the time	Most of the time	A good bit of the time	Some of the time	A little of the time	None of the time
23.	Did you feel full of pep?						
24.	Have you been a very nervous person?						
25.	Have you felt so down in the dumps that nothing could cheer you up?						
26.	Have you felt calm and peaceful?						
27.	Did you have a lot of energy?						

28.	Have you felt downhearted and blue?						
29.	Did you feel worn out?						
30.	Have you been a happy person?						
31.	Did you feel tired?						

32. During the past 4 weeks, how much of the time have you physical health or emotional problems interfered with your social activities (like visiting with friends, relatives, etc.)? (*Circle one number)*

All of the time =1

Most of the time =2

Some of the time =3

A little of the time=4

None of the time =5

How TRUE or FALSE is <u>each</u> of the following statements for you.

(circle one number on each line)

		Definitely True	Mostly True	Don't know	Mostly False	Definitely false
33.	I seem to get sick a little easier than other people					
34.	I am as healthy as anybody I know					

35.	I expect my health to get worse					
36.	My health is excellent					

APPENDIX F

SCORING OF SF-36 MOS VERSION 1.0.

ITEM NUMBERS	ORIGINAL RESPONSE	RECODED VALUE
1, 2, 20, 22, 34, 36	1	100
	2	75
	3	50
	4	25
	5	0
3, 4, 5, 6, 7, 8, 9, 10, 11, 12	1	0
	2	50
	3	100
13, 14, 15, 16, 17, 18, 19	1	0
	2	100
21, 23, 26, 27, 30	1	100
	2	80
	3	60
	4	40
	5	20
	6	0

24, 25, 28, 29, 31	1	0
	2	20
	3	40
	4	60
	5	80
	6	100
32, 33, 35	1	0
	2	25
	3	50
	4	75
	5	100

APPENDIX G

CONVERSION TABLE FOR THE DETERMINATION OF QAULITY OF LIFE SCORE

SCALE	NUMBER OF ITEMS	AVERAGE ITEMS
Physical functioning	10	3, 4, 5, 6, 7, 8, 9, 10, 11, 12
Role limitation due to physical health	4	13, 14, 15, 16
Role limitation due to emotional problems	3	17, 18, 19
Energy/ fatigue	4	23, 27, 29, 31
Emotional well-being	5	24, 25, 26, 28, 30
Social functioning	2	2, 32
Pain	2	21, 22
General health	6	1, 20, 33, 34, 35, 36

*Add up all averaged scale to obtain final Quality of Life score.

APPENDIX H1

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK 0

	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys Bp (mm/Hg)	Dia Bp (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssc ore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
1	30	F	1.62	50	110	70	70	1840	58	24.2	461	33.50	83.90	28.00	56.00	0	0	0
2	23	F	1.45	44	100	70	73	2233	55	21.4	556	34.60	86.50	29.20	60.00	0	0	0
3	29	F	1.55	47	120	70	70	2100	54	21.9	347	33.30	73.10	24.30	72.00	0	0	0
4	37	M	1.71	63	110	70	72	2100	59	23.2	365	34.90	78.40	25.30	74.00	0	0	0
5	30	M	1.56	52	110	70	74	1800	52	21.7	318	34.10	90.20	29.40	71.00	0	0	0
6	27	M	1.52	45	120	80	78	1000	48	22.0	267	34.10	72.30	24.90	72.40	0	0	0
7	19	F	1.54	51	100	70	70	1840	52	23.0	462	33.90	91.50	31.70	69.10	0	0	0
8	36	M	1.68	58	110	80	88	1800	53	29.0	371	33.40	83.70	27.50	79.30	0	0	0
9	20	M	1.65	56	110	70	90	1620	52	26.4	347	34.90	90.90	30.40	66.80	0	0	0
10	22	F	1.57	48	100	70	80	1300	56	31.0	164	35.80	89.30	29.90	70.20	0	0	0
11	32	M	1.72	52	110	70	78	2233	56	22.7	434	34.80	92.20	31.90	75.80	0	0	0
12	23	M	1.68	53	120	70	81	1833	55	25.5	432	32.50	77.70	25.90	65.40	0	0	0

APPENDIX H2

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP(mm /Hg)	Dia BP (mm /Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiore spiratory fitnesssc ore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemogl obin	Quality of life	Freq. of crisis	Freq. of hospitaliz ation	Length of hospitaliz ation (days)
13	22	F	1.56	48	110	60	78	2000	56	32.1	347	33.00	75.90	24.80	55.80	0	0	0
14	22	M	1.58	52	110	70	76	1200	45	26.0	462	33.30	93.50	32.70	63.90	0	0	0
15	23	F	1.60	43	120	75	74	1500	59	22.2	657	34.20	87.90	30.00	51.20	0	0	0
16	26	F	1.60	47	110	70	73	2000	58	27.0	527	33.70	88.00	30.00	75.70	0	0	0
17	26	F	1.56	45	100	70	82	2000	58	26.1	245	34.60	86.40	30.00	55.80	0	0	0
18	24	M	1.73	51	110	70	77	1800	51	22.3	543	35.90	88.50	30.30	69.00	0	0	0
19	30	M	1.60	52	110	75	72	1300	48	22.8	310	33.80	90.60	30.30	65.00	0	0	0
20	31	M	1.57	44	100	60	74	1800	54	24.7	154	34.80	89.10	30.30	80.40	0	0	0
21	28	F	1.65	56	110	70	72	2700	53	22.0	236	32.70	85.80	30.00	68.00	0	0	0
22	20	M	1.65	46	100	70	84	1540	62	28.3	236	35.00	75.60	27.10	72.60	0	0	0
23	22	F	1.54	42	110	70	86	2248	56	21.1	259	33.60	87.60	30.50	66.00	0	0	0
24	29	F	1.56	48	110	70	80	1300	60	21.9	355	33.30	90.20	29.40	77.60	0	0	0

APPENDIX H3

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssc ore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular Haemoglobin Concentration g/dl	Mean cell volume fl	Mean cell haemoglobin pg	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
25	36	F	1.60	56	100	70	90	1720	58	21.2	337	34.90	83.10	27.00	58.00	0.00	0.00	0.00
26	23	F	1.58	44	120	80	77	2317	53	31.0	159	35.80	87.00	28.70	76.50	0.00	0.00	0.00
27	19	M	1.58	46	110	70	73	2000	58	23.9	527	33.50	88.80	30.50	72.20	0.00	0.00	0.00
28	22	F	1.58	52	110	70	68	1733	52	25.5	283	34.50	94.00	32.80	75.00	0.00	0.00	0.00
29	25	F	1.56	49	120	80	78	1200	58	22.5	621	33.80	89.50	31.20	42.00	0.00	0.00	0.00
30	38	F	1.53	58	110	70	78	3000	50	21.0	263	34.30	84.90	28.60	42.00	0.00	0.00	0.00
31	19	M	1.68	64	130	80	76	1426	54	27.3	587	34.80	85.30	29.70	71.00	0.00	0.00	0.00
32	18	F	1.69	50	110	70	78	1100	52	23.9	539	33.50	86.10	26.70	77.20	0.00	0.00	0.00
33	28	F	1.56	44	100	70	70	1366	43	22.5	408	34.70	80.90	27.00	62.00	1.00	0.00	0.00
34	27	M	1.67	52	110	70	76	15000	52	23.9	520	33.50	76.30	26.10	60.30	1.00	0.00	0.00
35	23	F	1.54	48	100	70	75	1000	52	27.1	519	34.30	77.80	26.20	70.60	1.00	0.00	0.00
36	25	F	1.58	60	120	80	72	1300	52	29.2	489	32.20	82.10	27.50	60.00	1.00	0.00	0.00

APPENDIX H4

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
37	27	F	1.55	56	100	70	78	2100	42	30.1	415	33.90	81.20	28.40	74.50	1	0	0
38	22	F	1.58	54	110	80	78	2000	60	24.0	264	33.60	81.10	27.80	60.50	1	0	0
39	28	F	1.66	58	110	70	76	2100	56	23.6	215	33.00	75.90	24.80	58.00	1	0	0
40	35	F	1.61	48	120	80	76	2200	50	26.0	172	33.50	73.80	22.90	62.50	1	0	0
41	30	M	1.70	56	120	80	72	2360	54	23.0	260	35.20	84.20	27.30	70.40	1	0	0
42	22	M	1.57	45	110	70	80	1670	52	22.3	194	34.10	84.90	27.30	66.10	1	0	0
43	36	M	1.56	47	120	80	68	1870	58	21.0	326	33.30	79.40	26.90	69.00	1	0	0
44	24	F	1.71	60	100	70	72	2300	50	22.3	486	34.10	71.90	23.40	60.00	1	0	0
45	28	F	1.64	50	120	80	72	1320	56	27.0	378	33.70	81.10	27.20	75.70	1	0	0
46	22	F	1.62	57	120	80	82	1946	60	29.3	206	32.80	89.70	31.00	65.20	1	0	0
47	25	F	1.64	61	120	80	78	2367	52	23.5	434	34.90	90.60	29.90	61.00	1	0	0
48	23	M	1.54	42	120	80	72	1867	48	31.5	380.00	31.10	73.80	22.90	69.00	1	0	0

APPENDIX H5

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/Hg)	Dia BP (mm /Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiore spiratory fitnesssc ore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular Haemoglobin concentration	Mean cell volume	Mean cell haemogl obin	Quality of life	Freq. of crisis	Freq. of hospita lization	Length of hospitali zation (days)
49	31	F	1.59	48	100	70	78	1640	50	21.0	302	31.00	69.60	23.10	66.00	1.00	0.00	0.00
50	28	F	1.62	51	100	70	79	1400	58	32.0	217	33.80	87.10	31.30	56.40	1.00	0.00	0.00
51	21	M	1.71	62	110	80	80	1100	54	26.5	286	33.20	89.30	29.90	49.80	1.00	0.00	0.00
52	24	F	1.51	40	100	70	80	1800	52	21.7	340	34.10	80.00	27.00	65.50	1.00	0.00	0.00
53	42	F	1.58	52	100	70	76	1750	54	22.5	316	34.70	83.90	28.00	58.00	1.00	0.00	0.00
54	20	M	1.70	58	120	80	78	1600	55	23.5	415	34.90	81.00	28.40	66.30	1.00	0.00	2.00

APPENDIX H6

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
1	30	F	100	70	70	1950	48	28.0	243	32.9	86.20	30.00	60.3
2	23	F	100	70	74	2430	53	23.4	267	33.3	88.00	30.00	66.4
3	29	F	110	70	70	2250	54	26.0	233	33.3	90.60	30.30	75.3
4	37	M	105	65	76	2300	57	29.1	296	34.4	85.70	30.00	74.3
5	30	M	100	70	74	2978	52	31.0	244	36.5	87.60	30.10	76.4
6	27	M	110	70	78	1200	58	24.7	234	35.0	84.10	30.00	74.3
7	19	F	100	60	70	1900	58	25.3	287	29.4	86.80	25.30	72.8
8	36	M	100	70	88	2100	53	29.8	256	34.2	92.20	30.00	80.1
9	20	M	110	70	90	2650	56	30.0	299	34.7	89.30	29.90	70.4
10	22	F	100	70	80	2350	43	31.6	342	36.4	91.30	30.70	76.5
11	32	M	100	60	78	2400	50	28.6	237	32.9	83.90	28.00	68.6
12	23	M	110	70	78	2367	54	23.7	481	33.3	68.10	22.70	61.5

APPENDIX H7

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
13	22	F	100	50	78	2700	46	31.4	343	33.8	86.50	29.20	79.6
14	22	M	100	70	76	2340	56	27.1	432	34.0	89.80	30.90	60.5
15	23	F	110	70	74	1850	52	22.7	558	33.9	82.30	37.70	60.4
16	26	F	100	70	78	2000	54	27.3	481	34.3	85.80	30.00	84.2
17	26	F	100	60	78	2150	58	28.0	256	34.2	88.70	30.00	68.0
18	24	M	100	70	76	1969	49	21.3	363	32.4	88.30	30.00	86.9
19	30	M	100	70	72	2400	48	25.2	267	32.5	87.60	30.00	65.1
20	31	M	100	60	72	1700	59	22.4	156	34.8	86.90	30.00	81.2
21	28	F	110	70	74	2500	65	21.2	236	34.9	86.50	30.00	78.5
22	20	M	100	70	84	1667	58	28.0	232	34.3	82.40	30.00	74.6
23	22	F	100	70	86	2230	56	21.0	236	35.4	70.60	23.20	66.4
24	29	F	100	70	80	1300	52	21.3	332	32.4	87.10	31.30	76.5

APPENDIX H8

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/ Hg)	Dia BP (mm/ Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean Corpuscular Haemoglobin concentration g/dl	Mean cell volume	Mean cell heamoglobin	Quality of life
25	36	F	100	70	88	2633	62	19.6	506	34.7	75.50	25.20	71.8
26	23	F	110	80	76	1667	61	31.0	159	35.8	85.80	29.00	46.6
27	19	M	100	70	72	1967	57	27.0	527	33.7	89.10	31.10	72.5
28	22	F	100	70	68	1730	52	25.5	288	34.5	83.90	28.00	75.0
29	25	F	110	70	77	1700	54	22.5	621	33.8	90.00	31.20	42.0
30	38	F	110	70	78	1580	61	21.0	263	34.3	81.20	28.40	84.3
31	19	M	110	70	76	1420	52	27.3	587	34.8	84.90	27.30	71.0
32	18	F	120	80	78	1800	66	23.9	539	33.5	87.70	30.00	77.2
33	28	F	100	70	70	2500	43	22.0	208	34.5	89.10	31.10	70.3
34	27	M	110	70	76	2700	50	24.2	412	33.5	76.20	25.80	65.4
35	23	F	100	70	76	2100	52	27.0	472	33.7	64.50	22.20	72.5
36	25	F	110	80	78	1838	52	30.2	423	34.4	64.90	22.60	65.1

APPENDIX H9

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/ Hg)	Dia BP (mm/H g)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesscore	Packed Cell volume %	PlateletCou nt $\times 10^9$/L	Mean Corpuscular Haemoglobin Concentratio n g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
37	27	F	100	70	74	2300	42	31.0	251	35.8	85.30	29.70	76.0
38	22	F	100	75	72	2000	57	25.0	349	34.5	65.30	22.00	63.0
39	28	F	110	70	74	2000	54	26.1	295	33.7	94.00	32.80	62.5
40	35	F	110	80	76	2050	50	24.0	172	34.6	65.80	22.60	66.0
41	30	M	110	80	72	2130	50	22.3	263	34.5	90.20	29.90	90.4
42	22	M	100	70	80	1660	55	21.6	192	34.7	66.20	22.80	66.1
43	36	M	120	80	68	1750	54	26.0	132	34.2	83.70	30.00	69.1
44	24	F	110	70	71	2100	54	21.3	286	34.7	63.70	22.00	61.0
45	28	F	110	70	72	2033	52	19.0	378	34.7	63.10	21.70	72.2
46	22	F	115	70	81	2000	45	26.7	189	33.7	64.70	22.30	74.2
47	25	F	110	70	78	2420	50	22.5	423	34.7	87.60	29.50	74.9
48	23	M	110	80	74	1700	42	34.9	434	31.2	73.80	22.90	45.80

APPENDIX H10

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/ Hg)	Dia BP (mm/H g)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitness score	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentratio n g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
49	31	F	100	70	78	1000	44	21.4	302	31.9	70.20	23.60	66.1
50	28	F	100	70	79	1450	58	32.0	217	33.8	84.20	27.30	56.4
51	21	M	110	70	80	3000	60	26.5	286	33.2	91.50	30.90	49.8
52	24	F	100	70	80	1800	60	21.7	349	34.1	80.00	26.00	65.5
53	42	F	120	80	72	2000	54	22.5	328	34.2	88.00	30.40	77.5
54	20	M	110	70	78	1800	44	23.4	246	33.3	86.30	30.00	66.40

APPENDIX H11

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssc ore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
1	30	F	100	70	70	2103	42	29	220	34.8	92.90	32.30	66	1	0	0
2	23	F	100	70	70	1850	49	26	231	34.2	87.90	30.00	70.8	0	0	0
3	29	F	100	70	68	2000	48	27.4	210	34.3	87.40	30.00	78.5	0	0	0
4	37	M	100	60	70	2436	46	30	264	34.7	87.20	30.00	79.3	0	0	0
5	30	M	100	70	70	2115	44	32	140	34.7	87.50	30.00	86.4	0	0	0
6	27	M	100	60	72	2500	46	26	220	34.5	87.60	29.70	78	0	0	0
7	19	F	100	80	68	1940	49	27.2	236	34.2	86.70	29.60	82.1	0	0	0
8	36	M	100	70	74	2000	46	31	210	34.5	90.00	30.00	84.2	0	0	0
9	20	M	100	70	76	1900	37	31	253	34.2	87.10	29.90	75.8	0	0	0
10	22	F	100	70	72	2532	42	32.1	228	34.3	85.30	30.00	81.5	0	0	0
11	32	M	105	70	73	2356	46	31.6	136	34.0	87.60	29.70	76.2	0	0	0
12	23	M	110	70	68	2524	48	20.3	458	33.5	87.50	30.00	59.8	0	0	0

APPENDIX H12

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
13	22	F	100	70	70	2786	39	25.5	432	32.5	87.20	30.00	65.4	1	0	0
14	22	M	115	80	76	2480	54	23.1	286	33.8	85.70	29.70	64.8	0	0	0
15	23	F	100	70	72	1960	47	24	306	34.2	86.50	30.00	68.7	0	0	0
16	26	F	100	60	72	2600	50	27.4	264	34.3	87.00	30.00	86.3	0	0	0
17	26	F	100	60	74	2410	54	26.5	418	34.7	86.20	30.00	70.5	0	0	0
18	24	M	100	70	74	2000	44	22	268	34.5	87.70	30.00	58	1	1	1
19	30	M	100	70	68	2526	44	30.4	148	33.9	86.10	29.70	68.5	1	0	0
20	31	M	100	70	74	1700	59	21	162	34.8	87.30	30.00	81.2	3	2	3
21	28	F	100	70	74	2300	65	21	236	35.2	88.60	30.00	72	0	0	0
22	20	M	100	60	84	1667	58	28	232	34.3	86.80	32.70	74.6	0	0	0
23	22	F	110	60	86	2100	56	21	236	35.4	87.60	30.00	66.4	0	0	0
24	29	F	100	60	80	1300	52	21.3	332	32.4	77.90	25.60	76.5	0	0	0

APPENDIX H13

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiore spiratory fitnesssc ore	Packed Cell volume %	PlateletCo unt $\times 10^9$/L	Mean Corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglo bin	Quality of life	Freq. of crisis	Freq. of hospitalizat ion	Length of hospitalizat ion (days)
25	36	F	100	70	88	2500	62	19.6	506	34.7	77.80	26.10	71.8	0	0	0
26	23	F	110	70	77	1667	61	31	159	35.8	83.10	27.00	46.6	0	0	0
27	19	M	110	70	72	1967	57	27	487	33.7	78.40	25.30	72.5	2	1	4
28	22	F	100	70	68	1730	52	25.5	288	34.5	96.70	32.50	75	0	0	0
29	25	F	110	70	78	1700	54	22.5	574	33.8	81.00	27.80	42	0	0	0
30	38	F	110	70	78	1580	61	21	263	34.3	77.80	26.20	78	0	0	0
31	19	M	110	70	76	1420	52	27.3	487	34.8	85.90	28.90	71	0	0	0
32	18	F	100	60	78	1800	66	23.9	539	33.5	86.50	29.20	74	0	0	0
33	28	F	100	70	68	1740	40	24	210	34.6	67.20	22.90	78	0	0	0
34	27	M	110	70	74	2772	47	27	236	34.1	87.90	30.00	73	1	0	0
35	23	F	100	70	74	2236	48	27	284	34.1	64.60	22.10	76	1	1	1
36	25	F	110	70	71	1860	50	31.1	189	32.5	83.80	30.00	69.3	0	0	0

APPENDIX H14

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean Corpuscular Haemoglobin Concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
37	27	F	100	70	72	2700	38	26	224	34.2	62.20	21.40	78	1	0	0
38	22	F	110	60	72	2215	52	24	367	34.6	64.90	22.20	68	1	0	0
39	28	F	110	70	70	2300	50	27.3	377	34.1	81.20	28.80	65.6	1	0	0
40	35	F	110	70	76	2050	50	22	176	34.1	90.60	30.30	66	2	1	3
41	30	M	100	70	72	2130	50	21	300	35.7	90.70	32.50	80	1	0	0
42	22	M	100	80	78	1660	55	21	204	35.7	71.20	23.00	60	1	1	3
43	36	F	115	80	68	1750	54	26	132	34.2	64.00	22.00	67	1	1	4
44	24	F	110	60	71	2000	54	21	286	34.8	65.10	22.10	61	1	0	0
45	28	F	110	80	72	1900	52	19	378	34.7	62.10	25.00	72.2	1	0	0
46	22	F	110	70	82	2100	45	26.7	189	33.7	64.00	22.10	74.2	1	0	0
47	25	F	110	70	78	2420	50	22.5	423	34.7	64.90	22.30	74.9	1	1	5
48	23	M	110	70	74	1700	42	34.9	434	31.2	66.30	22.50	45.8	1	1	5

APPENDIX H15

RAW DATA FOR EXPERIMENTAL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiores piratory fitnesssco re	Packed Cell volume %	PlateletCo unt $\times 10^9$/L	Mean Corpuscular haemoglobin concentratio n g/dl	Mean cell volume	Mean cell haemoglo bin	Quality of life	Freq. of crisis	Freq. of hospitalizat ion	Length of hospitalizat ion (days)
49	31	F	100	70	78	1000	44	21.4	302	31.9	67.50	23.10	66.1	2	0	0
50	28	F	100	70	79	1450	58	32	217	33.8	61.00	20.90	56.4	2	0	0
51	21	M	110	70	80	3000	60	26.5	286	33.2	60.90	21.00	52	1	0	0
52	24	F	100	70	80	1800	60	21.7	349	34.1	67.00	22.80	65	2	0	0
53	42	F	100	70	72	2100	54	22.5	328	34.2	86.30	30.00	70	2	1	1
54	20	M	120	80	74	2320	40	20	483	33.7	88.00	30.40	62.6	0	0	0

APPENDIX H16

RAW DATA FOR CONTROL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/ Hg)	Dia BP (mm/ Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
1	22	M	1.55	50	100	70	74	1600	52	23.1	432	33.8	83.90	28.00	64	1	1	1
2	24	F	1.65	53	110	60	74	2662	46	24.2	486	32.6	81.00	28.40	59	1	1	1
3	22	F	1.48	48	100	70	80	1600	57	24	530	34.7	93.30	32.40	74.5	1	1	1
4	25	M	1.62	54	100	60	75	1136	51	27	481	33.7	83.30	30.00	67.5	1	1	1
5	24	M	1.52	61	110	70	77	1500	45	21.2	532	32.5	89.50	31.20	69.3	1	1	1
6	24	M	1.70	57	110	70	74	1800	51	21	263	34.3	89.70	30.00	75	1	1	1
7	35	F	1.61	51	110	70	76	1956	55	29	251	33.4	82.00	27.40	65.5	1	1	1
8	26	F	1.54	42	100	60	80	1266	56	21.6	466	32.9	85.20	30.00	64.2	1	1	1
9	30	F	1.52	45	100	70	82	1000	54	21.7	415	34.1	82.40	27.10	62	1	1	1
10	30	F	1.53	41	110	70	70	1642	50	24	371	34	96.50	32.90	52.5	1	1	1
11	26	F	1.72	60	130	80	72	1000	55	22.1	346	34.8	77.80	26.20	60	1	1	2
12	29	M	1.60	53	120	80	70	2300	60	19.4	287	34.9	78.60	25.70	40.9	1	1	2

APPENDIX H17

RAW DATA FOR CONTROL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/ Hg)	Dia BP (mm/ Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
13	21	M	1.54	60	120	80	72	2000	58	24.7	287	33	84.90	28.60	74.6	1	1	2
14	30	M	1.56	48	100	80	74	1618	56	23.2	367	34.5	90.10	30.00	74	1	1	2
15	28	F	1.60	53	110	70	72	1680	68	19.8	481	34.8	90.60	30.30	75.8	1	1	4
16	26	M	1.64	52	110	70	70	1840	54	23.4	420	33.3	87.90	30.00	52.2	2	0	0
17	24	F	1.71	54	110	70	74	1860	52	23	295	32.3	96.50	32.90	51	2	0	0
18	24	F	1.58	56	110	80	70	2700	49	21.6	457	32.9	92.00	31.70	59.1	2	0	0
19	28	M	1.60	61	100	70	72	1500	50	22	274	35	85.80	30.00	52.5	2	0	0
20	24	F	1.58	58	100	70	82	1633	51	26.1	246	32.6	77.80	26.20	57.1	2	0	0
21	22	M	1.65	48	100	70	70	1600	49	32	156	33.2	84.50	29.60	68	2	0	0
22	24	F	1.70	58	110	80	72	2100	55	24.2	291	33.5	87.50	27.60	67	2	0	0
23	20	M	1.62	41	120	80	84	2133	65	25	182	32.9	82.80	27.10	78	2	0	0
24	23	M	1.64	48	110	70	80	1560	50	27	220	33.7	94.00	32.80	53	2	0	0

APPENDIX H18

RAW DATA FOR CONTROL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/ Hg)	Dia BP (mm/ Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean Corpuscular Haemoglobin Concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
25	26	M	1.58	49	100	70	74	1400	56	20.1	513	34.6	92.10	31.00	56.8	2	0	0
26	28	F	1.54	45	110	70	78	1500	53	27.3	256	34.1	77.60	25.90	53.6	2	0	0
27	20	M	1.60	54	100	70	78	2100	54	23.4	214	33.3	86.10	30.00	78.9	2	1	1
28	25	M	1.54	42	100	60	80	1950	54	25.8	187	33.7	73.90	23.00	61	2	1	1
29	28	F	1.59	52	110	70	68	1238	54	22.3	263	33.2	88.10	30.70	78.4	2	1	1
30	26	M	1.70	56	110	60	73	2000	50	21	568	30.7	73.10	24.30	45	2	1	1
31	20	M	1.47	42	110	70	76	1500	57	22.3	340	33.2	93.50	32.70	70.6	2	1	2
32	25	F	1.69	52	110	70	80	1200	52	25.8	132	33.7	87.00	30.00	66	3	1	2
33	26	F	1.57	45	110	80	76	1600	53	21.3	363	32.4	75.60	27.10	76.9	3	1	2
34	20	F	1.64	49	110	70	80	1400	52	24	285	34.2	78.40	26.20	78.6	3	1	3
35	24	M	1.60	55	110	70	74	2100	48	21	405	34.4	83.70	27.50	58	3	0	3
36	18	F	1.60	56	110	70	82	1480	54	24.2	532	32.6	87.00	30.00	68	3	0	0

APPENDIX H19

RAW DATA FOR CONTROL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular Haemoglobin Concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
37	29	M	1.68	58	110	70	72	1134	44	24	618	34.9	75.00	25.00	62.4	3	0	0
38	22	F	1.59	66	120	80	72	2400	48	29.8	384	33.6	91.60	31.20	56.1	3	1	1
39	19	M	1.78	57	100	70	72	1000	56	31.8	253	33.3	87.50	29.60	70.2	3	1	2
40	27	F	1.59	54	120	90	82	1763	52	31	204	35.8	87.90	30.00	64	3	1	2
41	19	M	1.7	60	110	70	80	1583	56	24.2	532	33.5	99.00	32.00	68.2	3	1	2
42	29	F	1.65	54	120	80	72	1400	56	24.7	287	34.8	78.90	27.00	69	4	1	1
43	18	M	1.56	48	110	70	75	1433	45	28.1	383	34.2	71.60	24.20	72.9	4	1	2
44	25	M	1.58	45	120	80	80	2000	58	22.1	246	35	81.00	26.90	45.2	4	1	3
45	38	F	1.64	56	100	60	68	2150	48	29.8	310	33.6	88.80	30.50	54.7	5	1	1
46	33	F	1.52	42	110	70	70	1640	52	21.2	143	32.5	89.30	29.90	61.4	5	2	2
47	22	F	1.63	50	110	70	84	2633	54	19.6	469	34.7	92.90	32.30	71.9	5	2	3
48	53	M	1.72	51	120	80	70	1667	50	28.7	128	31	87.90	30.00	56.9	6	2	2

APPENDIX H20

RAW DATA FOR CONTROL GROUP AT WEEK 0

S/N	Age (yrs)	Sex	Ht (m)	Wt (kg)	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
49	53	M	1.62	51	120	80	72	1500	50	28.7	168	31	82.40	31.10	70.6	6	2	4
50	19	M	1.65	56	120	80	80	1684	55	25.2	374	33.3	90.90	30.40	53	10	2	3

APPENDIX H21

RAW DATA FOR CONTROL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/ Hg)	Dia BP (mm/ Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
1	22	M	100	70	74	1600	52	23.1	432	33.8	85.20	28.10	64
2	24	F	100	50	74	2662	46	24.2	486	32.6	88.00	30.40	59
3	22	F	100	60	80	1600	57	24.2	530	34.7	86.30	30.00	74.5
4	25	M	100	60	75	1136	51	27	481	33.7	87.00	30.00	67.5
5	24	M	100	70	77	1500	45	21.2	532	32.5	86.20	30.00	69.3
6	24	M	100	70	74	1800	51	21	263	34.3	85.70	29.60	75
7	35	F	110	65	76	1956	55	29	251	33.4	87.10	30.30	65.5
8	26	F	100	50	80	1266	56	21.6	466	32.9	85.80	29.60	64.2
9	30	F	100	60	82	1000	54	21.7	349	34.1	86.80	25.30	62
10	30	F	110	70	70	1642	50	24	415	34.0	86.40	30.00	52.5
11	26	F	110	70	72	1000	55	22.1	371	34.8	86.20	29.70	60
12	29	M	120	80	70	2300	60	19.4	346	34.9	85.90	30.00	40.9

APPENDIX H22

RAW DATA FOR CONTROL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life
13	21	M	120	65	70	1500	54	23	287	33	86.70	30.00	65.2
14	30	M	100	50	74	1233	49	21	423	34.5	87.60	29.60	45.2
15	28	M	120	70	73	1620	42	19	365	34.8	86.50	30.00	64.7
16	26	F	100	70	70	2100	50	25	214	33.3	86.00	30.00	48.6
17	24	F	100	65	74	1900	48	24	295	32.3	85.20	29.60	63.6
18	24	M	100	70	78	2850	52	27	204	32.9	93.10	23.90	64.3
19	28	M	100	70	72	1450	50	22.1	371	35	85.80	30.00	81.6
20	24	M	110	70	78	2150	46	26.1	256	32.6	86.40	30.00	61.5
21	22	M	100	70	78	1700	51	36	204	33.2	87.50	30.00	68.5
22	24	M	110	70	74	1800	43	24.2	291	33.5	86.20	30.00	72.1
23	20	F	120	80	84	2100	62	22	182	32.9	83.20	28.00	56.2
24	23	M	110	80	82	1500	56	25	222	33.7	91.50	33.70	73.5

APPENDIX H23

RAW DATA FOR CONTROL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular Haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life
25	26	F	100	70	74	1400	58	21	413	35.3	87.80	31.10	63.2
26	28	M	110	70	78	1620	53	27.3	256	34.1	34.20	27.30	53.6
27	20	M	100	70	78	2100	60	23	216	34.8	74.50	24.30	78.9
28	25	M	100	60	80	1840	56	33.4	187	34.6	73.50	22.90	61.3
29	28	F	110	70	68	1600	34	19	577	34.7	93.30	32.40	57.4
30	26	M	110	60	73	1210	50	21	568	30.7	92.70	32.20	45
31	20	M	110	70	76	1700	53	29	253	33.4	91.00	30.70	82.4
32	25	F	110	70	80	2300	50	22	377	35	93.10	32.90	63.9
33	26	F	110	80	76	1820	56	21.1	548	33.2	75.60	27.10	78.6
34	20	M	110	70	80	1550	55	21.5	280	34.9	77.80	26.20	64.2
35	24	F	110	70	74	2700	41	23	284	35.7	83.70	27.50	69.5
36	18	M	110	70	82	1700	50	23.5	204	34.9	63.20	21.80	48.8

APPENDIX H24

RAW DATA FOR CONTROL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean Corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life
37	29	M	100	50	70	1600	41	19.3	504	33.7	75.00	25.00	78
38	22	F	110	80	76	3300	48	26.1	403	36.7	91.60	31.20	62.5
39	19	M	100	70	72	2000	48	21.2	462	34.9	87.50	29.60	68.4
40	27	F	120	60	82	1820	52	29	204	33.5	87.90	30.00	71.6
41	19	M	110	70	78	1420	64	24.2	532	32.6	100.50	32.10	68.6
42	29	F	110	70	76	2167	50	31.4	214	34.7	78.90	27.00	74.8
43	18	M	110	60	75	1400	58	28	383	34.2	71.60	24.20	75.8
44	25	M	120	80	90	2000	68	22	315	32	81.00	26.90	45.2
45	38	F	100	60	68	2000	50	27	321	44.8	88.80	30.50	61.1
46	33	F	100	70	70	1700	40	26	243	34.2	75.90	24.80	67.3
47	22	F	110	70	84	1600	55	21	168	34.8	92.50	32.30	62
48	53	M	110	80	70	1740	50	29.6	263	34.6	87.90	30.00	52.1

APPENDIX H25

RAW DATA FOR CONTROL GROUP AT WEEK-6

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean Corpuscular Haemoglobin concentration	Mean cell volume	Mean cell haemo globin	Quality of life
49	53	M	110	80	70	1600	48	28.7	194	31	82.40	27.10	71
50	19	M	120	70	78	2480	55	25.2	374	33.3	90.90	30.40	70.6

APPENDIX H26

RAW DATA FOR CONTROL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
1	22	M	100	70	70	2000	50	24	282	34.2	86.30	30.00	75.8	1	1	1
2	24	F	100	70	70	2840	39	25	273	34.4	88.00	30.40	76.4	1	1	1
3	22	F	100	60	76	3000	49	29.3	214	34.8	85.10	30.00	81.6	1	1	1
4	25	M	100	70	72	1863	49	29.1	238	34.4	86.20	30.00	78.6	1	1	1
5	24	M	100	60	74	2180	48	24.7	205	32.8	86.30	30.00	80.1	1	1	1
6	24	M	110	70	72	1958	37	23.2	357	32.3	88.00	30.00	65.3	1	1	1
7	35	M	110	65	76	1850	61	22	254	35.5	87.40	30.00	65	1	1	1
8	26	F	100	50	80	1266	54	21.6	466	32.9	84.00	30.00	34.2	1	1	1
9	30	F	100	60	80	1800	60	21.7	349	34.1	84.60	30.00	65.5	1	1	1
10	30	F	110	70	70	1530	56	25.2	415	32.9	84.00	30.00	78	1	1	1
11	26	F	110	70	71	1654	48	26	347	33.7	88.50	30.00	72	1	1	2
12	29	F	120	80	70	1570	58	20	400	35	85.20	30.00	45	1	1	2

APPENDIX H27

RAW DATA FOR CONTROL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssc ore	Packed Cell volume %	PlateletCount $\times 10^9$/L	Mean Corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
13	21	M	120	65	72	1500	54	23	287	34.3	89.60	30.00	65.2	1	1	4
14	30	M	100	50	74	1233	49	21	423	35.3	87.70	30.00	45.2	1	0	0
15	28	M	120	70	73	1620	66	19	365	32.6	84.60	30.00	64.7	2	1	3
16	26	F	100	70	68	2234	44	27.3	204	34.8	81.80	30.00	63.6	1	0	0
17	24	F	100	60	70	2000	42	25	294	34.4	90.00	30.00	72.1	0	0	0
18	24	M	100	70	72	2864	49	29	198	33.3	85.20	30.00	69.6	0	0	0
19	28	M	100	70	72	2000	46	23.6	238	30.2	85.80	30.00	82.5	0	0	0
20	24	M	110	60	72	2420	47	28	238	34.2	86.30	30.00	65	1	0	0
21	22	M	100	70	70	2100	49	32.4	198	34.3	87.50	30.00	71.3	1	0	0
22	24	M	110	70	72	2439	40	22.5	483	34.8	87.30	30.00	76.2	0	0	0
23	20	F	120	80	84	2100	67	22	182	35.5	83.20	28.00	56	2	1	1
24	23	M	110	80	82	1500	58	23	220	33.9	91.50	31.70	68	2	1	1

APPENDIX H28

RAW DATA FOR CONTROL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnesssscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
25	26	F	100	70	74	1400	59	21	413	35.2	87.80	31.10	63.2	2	0	0
26	28	M	110	70	78	1620	53	27.3	256	34.1	84.20	27.30	53.6	3	0	0
27	20	M	100	70	78	2100	66	21	216	34.8	74.50	24.30	75	1	0	0
28	25	M	100	60	80	1840	64	23	196	33.5	73.50	22.90	61	2	2	4
29	28	F	110	70	70	1600	34	19	577	34.7	93.30	32.40	67.4	2	0	0
30	26	M	110	60	73	1200	50	21	568	30.7	92.70	32.20	48	2	1	1
31	20	M	100	70	72	2050	49	30	251	34.3	91.00	30.70	84.1	0	0	0
32	25	F	110	70	78	2300	48	27	310	33.7	93.10	32.90	77.5	0	0	0
33	26	F	100	70	70	2000	54	21.5	475	35.8	75.60	27.10	81.6	1	1	1
34	20	M	110	70	80	1550	55	22	280	35	77.80	26.20	78	4	2	5
35	24	F	100	60	70	2736	38	26	270	34.2	83.70	27.50	68	2	0	0
36	18	M	110	70	72	2940	52	25.9	228	35.5	63.20	21.80	73.8	0	1	1

APPENDIX H29

RAW DATA FOR CONTROL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
37	29	M	100	50	70	1400	41	19.3	504	33.7	75.00	25.00	48.8	3	0	0
38	22	F	110	70	70	3500	38	31	253	35.8	91.60	31.20	66	0	0	0
39	19	M	100	70	71	2833	46	31.6	189	34.2	87.50	29.60	76.9	0	0	0
40	27	F	120	60	82	1820	52	25	214	34.8	87.90	30.00	71	3	0	0
41	19	M	110	70	78	1420	64	24.2	439	32.6	100.50	32.10	68	3	0	0
42	29	F	110	70	72	2318	48	27.1	339	34.3	78.90	27.00	70.6	2	0	0
43	18	M	110	60	75	1400	58	28	383	34.2	71.60	24.20	72.8	4	1	1
44	25	M	120	80	90	2000	68	22	315	30.4	81.00	26.90	56.4	1	1	1
45	38	F	100	60	68	2000	59	23	324	34.3	88.80	30.50	58	5	3	4
46	33	F	100	70	68	1880	36	28	156	34.3	75.90	24.80	74.3	1	0	0
47	22	F	110	70	84	1600	55	21	168	35.2	92.90	32.30	62	5	2	2
48	53	M	110	70	70	2200	46	29	241	33.4	87.90	30.00	64.5	1	1	1

APPENDIX H30

RAW DATA FOR CONTROL GROUP AT WEEK-12

S/N	Age (yrs)	Sex	Sys BP (mm/Hg)	Dia BP (mm/Hg)	Heart rate (b/min)	Vital capacity (cc)	Cardiorespiratory fitnessscore	Packed Cell volume %	Platelet Count $\times 10^9$/L	Mean corpuscular haemoglobin concentration g/dl	Mean cell volume	Mean cell haemoglobin	Quality of life	Freq. of crisis	Freq. of hospitalization	Length of hospitalization (days)
49	53	M	110	70	70	2300	46	29	236	33.8	82.40	27.10	67	0	0	0
50	19	M	120	70	78	2480	55	25.2	368	33.3	90.90	30.40	70.6	4	3	6