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IMPACT OF NUTRITION EDUCATION ON THE NUTRITIONAL STATUS OF MALNOURISHED STROKE PATIENTS AT THE KOMFO ANOKYE TEACHING HOSPITAL, KUMASI

BY

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DECLARATION

I Patrick Kusi hereby declare that this thesis titled: "Impact of nutrition education on the nutritional status of malnourished stroke patients at the Komfo Anokye Teaching Hospital", is my own work, based on primary data collected. To the best of my knowledge, this work contains no material previously published by another person, nor material accepted for the award of any other degree of the university, except where due acknowledgement has been made in the text.

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ABSTRACT

There is a high malnutrition rate among stroke survivors and this leads to reduced functional recovery among these patients. There has not been much studies on how to improve the nutritional status of the stroke patients, especially out-patients in Ghana. This study was conducted to find out how nutrition education would impact the nutritional status of the malnourished stroke patients. The study was structured in two phases. Phase I involved screening the stroke patients for malnutrition, using subjective global assessment (SGA), biochemical, dietary and anthropometric indicators. In all, 81 patients were screened for malnutrition. Phase II involved giving nutrition education to the selected malnourished patients for three months. The patients were given the nutrition education twice each month. After three months, patients were re-assessed. Twenty-six (26) stroke patients were enrolled for phase II, but seventeen (17) completed this phase. The mean age of the 81 stroke patients was 55.9(±10.8) years. Prevalence of stroke among males was higher than in females. According to the various criteria used in the study, the levels of malnutrition recorded in the patients were; SGA, 32.1%, biochemical, 16%, BMI, 71.4% and MUAC, 41.1%. Malnutrition was highest among patients who were dependent on caregivers (p=0.008) and those with the lowest educational level (p=0.017). Energy intake was lower (942kcal) among the undernourished, compared with the well-nourished (1834kcal). Protein intake was also lower among the malnourished, compared with the well-nourished (p=0.032). Fruit and vegetable intake was low among the patients at baseline. Nutritional status of the malnourished patients improved after the intervention (p=0.000). Haemoglobin levels improved significantly over the baseline and there was increase in fruits and vegetables consumption. Protein and carbohydrate intake also increased after the three months of nutrition education. There was a strong positive association between increased nutirition knowledge and fruits and vegetables(r=0.576, p=0.000) protein (r=0.570, p=0.000) intakes. In conclusion, nutrition education improved the nutritional status of malnourished stroke patients.

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LIST OF ABBREVIATIONS

ADL	=	Activity of Daily Living
AMDR	=	Acceptable Macronutrient Distribution Range
ASPEN	=	American Society of Parenteral and Enteral Nutrition
AVM	=	Arteriovenous Malformation
BMI	=	Body Mass Index
CHD	=	Coronary heart disease
CVD	=	Cardiovascular Diseases
DASH	=	Dietary Approach to Stop Hypertension
DBP	=	Diastolic Blood Pressure
EDTA	=	Ethylene Diaminetetraacetic Acid
FFQ	=	Food Frequency Questionnaire
LDL	=	Low Density Lipoprotein
MUAC	=	Mid-upper arm circumference
NCDs	=	Non-communicable diseases
NCEP	=	National Cholesterol Education Program
PG-SGA	=	Patient-Generated Subjective Global Assessment
SGA	=	Subjective Global Assessment
SBP	=	Systolic Blood Pressure

Ua	=	Uric Acid
WBC	=	White Blood Cell
WHO	=	World Health Organisation

CHAPTER ONE

INTRODUCTION

1.1 GENERAL INTRODUCTION

Out of the 56.4 million global death in 2015, 39.5 million (70%) were due to non-communicable diseases (NCDs) (WHO, 2015). In 2012 non-communicable diseases caused 68% (38million), out of a total death toll of 56million globally and 28 million of these deaths occurred in low and middle income countries (WHO**,** 2014). An estimated 17.5 million people died from cardiovascular diseases (CVDs) in 2012, representing 31% of all global deaths. Of these deaths, an estimated 7.4 million were due to coronary artery disease (CAD) and 6.7 million were due to stroke. Interestingly, about 80% of CVD-related deaths, as well as 87% of CVD-related disabilities worldwide, are known to occur in low and middle income countries (WHO, 2014). Stroke is the second leading cause of death in adults worldwide and is a major contributor to disability and reduced quality of life (WHO, 2014). Studies in sub-Saharan Africa (SSA) show that stroke is the cause of 5% to 10% of all deaths (Walker *et al.,* 2000). Stroke deaths accounted for 8.7% of the top ten causes of death in Ghana in 2012 (WHO, 2015). In a study of adult patients from Komfo Anokye Teaching Hospital (KATH), Kumasi, 17.9% of acute medical admissions were assigned to cardiovascular causes, which included hypertension, heart failure and stroke (Agyemang *et al.*, 2012).

There has been reduction in the cases of stroke in most developed countries due to improved awareness and management of risk factors but findings from developing countries indicate an increased incidence of nearly 100% of the disease (Feigin *et al.*, 2009). Inadequate infrastructure, risk factor management and education for stroke patients in the low-income countries have contributed to the increased incidence and fatality of stroke cases.

Malnutrition is a long-standing negative or positive imbalance in both nutrients intake and requirements, with metabolic requirements exceeding or lower than nutritional intake, leading to altered body composition and impaired biological function (Rady *et al*., 2009).
Malnutrition is frequently detected in patients with acute stroke and during the rehabilitation period. Malnutrition is associated with poor recovery outcome in these patients (Yoo *et al.*, 2008; Prosser-Loose *et al*., 2011). Malnutrition seems to increase the risk of further brain damage and contributes to adverse outcome among stroke patients, hence early identification and management of malnutrition with dietary modifications or specific therapeutic strategies to ensure adequate nutritional intake is very critical, especially in resource-limited countries.

Several studies have proven that diabetes mellitus and a previous history of stroke increased the risk for malnutrition on admission by 58% and 71%, respectively (Corrigan *et al.,* 2011; Chai *et al.*, 2008). Micronutrients deficiencies such as B vitamins, vitamin D, antioxidant vitamins (A, C, and E), and zinc appear to contribute to blood vessel changes in the brain. Moreover, they appear to increase the risk of stroke and cognitive impairment in especially the elderly. However, how these factors are causally interrelated remain poorly understood (Sanchez-Moreno *et al*., 2009).

One of the main risk factors for malnutrition in stroke patients is difficulties in swallowing (dysphagia). One prospective study on stroke patients revealed that dysphagia and tube-feeding were both strong predictors of malnutrition on admission into a rehabilitation hospital (Chai *et al.*, 2008). Malnutrition may develop as a consequence of dysphagia if nutritional intake is reduced, in relation to requirements over days or weeks.

Other factors that lead to malnutrition are poor oral hygiene, depression, reduced level of consciousness, reduced mobility and arm or face weakness (Mould, 2009). Medications such as antidepressants can also induce mouth sores (xerostomia) and this will further reduce food intake and leads to malnutrition (Yang *et al*., 2009). Patients with acute stroke also often experience fatigue and this causes difficulties with eating. They could stop eating before they have satisfied their hunger, as they need to rest or even fall asleep. If patients eat and drink too little, in relation to their needs, this can worsen fatigue and result in undernourishment. Metabolic demand during stroke also increases demands for nutrients and leads to malnutrition. Low serum levels of proteins, albumin, vitamins A, E and C are markers of malnutrition and are associated with impaired functional status and higher mortality rates (Aquilani *et al*., 2011).

Nutrition education aims to improve the nutritional wellbeing of people, through information, experiences, skills and perceptions that will help them to change their patterns of food intake. People's knowledge, attitudes, practices and perceptions, and how they interact with circumstances, are at the centre of nutrition education (Kamp, 2010). A research which involved twenty-six non-compliant end stage renal disease (ESRD) patients, with inter-dialytic weight gain of greater than 2.5kg, who were given a two-month nutrition education, resulted in the decreased inter-dialytic mean weight from 2.64kg to 2.21kg. There were also increases in adherence to fluid restriction from 47% to 71% after the educational intervention (Barnett *et al*., 2007). Nutrition education has been proven to help individuals and communities to adopt a healthy eating pattern to promote good health.

1.2 PROBLEM STATEMENT

There is a high rate of malnutrition among stroke patients. A history of stroke increased the risk of malnutrition by 71% (Corrigan *et al.*, 2011; Chai *et al.*, 2008). At least, 16% of stroke patients present with protein-energy malnutrition upon admission to the hospital (Prosser-Loose *et al*., 2006). Nutritional status tends to decline during hospital stay, with estimates of 26.4% malnutrition reported after one week on admission (Prosser-Loose *et al*., 2006). Malnutrition influences the survival and functional outcome of the stroke patients. In Ghana, stroke patients do receive little or no nutrition education and dietary guidance due to insufficient number of qualified dietitians and nutritionists in most hospital facilities. There is therefore, the need to carry out this study to assess the impact of nutrition education among these patients.

1.3 JUSTIFICATION

The American Heart Association (AHA) recommends nutrition intervention for malnourished stroke patients. There is limited information on the prevalence of malnutrition among stroke patients in Ghana. A study had been undertaken by Chauwa (2017), on nutritional risk markers for the functional recovery of stroke patients, undergoing review at Komfo Anokye Teaching Hospital. This study would provide additional baseline data on nutritional status among stroke patients. It will provide the basis for taking action for controlling undernutrition among stroke survivors. It will also provide some basis for evaluating the impact of nutrition education on the nutritional status of under-nourished stroke patients.

1.4 MAIN OBJECTIVE

To assess the impact of nutrition education on the nutritional status of under-nourished stroke patients.

1.5 SPECIFIC OBJECTIVES

1. To find the prevalence of malnutrition among stroke patients.

2. To identify the factors contributing to undernutrition among the stroke patients.

3. Carry out nutrition education on the nutritional factors identified that contribute to malnutrition.

4. Assess the impact of nutrition education on the nutritional status of undernourished stroke patients.

CHAPTER TWO

LITERATURE REVIEW

2.1 INTRODUCTION

Out of thç 56.4 million globěl dçěth in 2015, 39.5 million (70%) wçrç duç to non-communicěblç disçěsçs (NCDs) (WHO, 2015). It hěs bççn çstimětçd thět by 2030, NCDs dçěth toll will incrçěsç to ěbout 52 million (WHO, 2014). In 2012, non-communicěblç disçěsçs cěusçd 68% (38million) out of ě totěl dçěth of 56 million globělly ěnd 28 million of thçsç dçěths occurrçd in low ěnd middlç-incomç countriçs (WHO, 2014). Ěn çstimětçd 17.5 million pçoplç diçd from cěrdiověsculěr disçěsçs (CVDs) in 2012, rçprçsçnting 31% of ěll globěl dçěths. Of thçsç dçěths, ěn çstimětçd 7.4 million wçrç duç to coroněry ěrtçry disçěsç (CĚD) ěnd 6.7 million wçrç duç to strokç (Globěl stětus on non-communicěblç disçěsç, WHO, 2014). Ěbout 80% of CVD-rçlětçd dçěths ěs wçll ěs 87% of CVD-rçlětçd disěbilitiçs worldwidç ěrç known to occur in low ěnd middlç-incomç countriçs.

Cçrçbrověsculěr disçěsç, principělly strokç, is thç sçcond lçěding cěusç of dçěth in ědults worldwidç ěnd is ě mějor contributor to disěbility ěnd rçducçd quělity of lifç (WHO, 2014).

Community-běsçd studiçs in sub-Sěhěrěn Ěfricě (SSĚ) show thět strokç is thç cěusç of 5% to 10% of ěll dçěths in thç sub-rçgion (Wělkçr çt ěl., 2000). Improvçd ěwěrçnçss ěnd měněgçmçnt of risk fĕctors hĕvç contributçd to thç dçclinç of strokç in most dçvçlopçd countriçs but çpidçmiologicĕl findings from dçvçloping countriçs indicĕtç ĕn incrçĕsçd incidçncç of nçĕrly 100% of thç disçĕsç (Fçigin çt ĕl., 2009). Inĕdçquĕtç infrĕstructurç to providç support for strokç pĕtiçnts in thç low-incomç countriçs hĕs contributçd to thç incrçĕsçd fĕtĕlity of strokç cĕsçs.

Strokç cĕusçd 18,300 dçĕths ĕccounting for 8.7% of thç top tçn cĕusçs of dçĕth in Ghĕnĕ in 2012 (WHO, 2015).

In ĕ study of ĕdult pĕtiçnts from Komfo Ĕnokyç Tçĕching Hospitĕl (KĔTH), Kumĕsi, 17.9% of ĕcutç mçdicĕl ĕdmissions wçrç ĕscribçd to cĕrdiovĕsculĕr cĕusçs, including hypçrtçnsion, hçĕrt fĕilurç ĕnd strokç (Ĕgyçmĕng çt ĕl., 2012). Onç of thç mĕin cĕusçs of dçĕth globĕlly is cĕrdiovĕsculĕr disçĕsçs ĕnd thçy ĕccount for ĕbout 30% of ĕll dçĕths (Lozĕno çt ĕl., 2012). Thç incrçĕsing lçvçls of hypçrtçnsion, diĕbçtçs, dyslipidĕçmiĕ, smoking, poor diçt ĕnd physicĕl inĕctivity will incrçĕsç thç risk of cĕrdiovĕsculĕr disçĕsçs, çspçciĕlly, in thç low ĕnd middlçincomç countriçs. It is çstimĕtçd thĕt ĕbout thrçç-fourth of ĕll dçĕths in 2030 would bç cĕusçd by cĕrdiovĕsculĕr disçĕsçs, ĕpproximĕtçd ĕt 24 million dçĕths (Franco, 2005).

Strokç is sĕid to occur whçn thçrç is ĕ suddçn intçrruption in blood supply to thç brĕin nçurons ĕnd othçr cçlls thĕt rçsult in oxygçn ĕnd nutriçnt dçficit, cĕusing ĕbnormĕlitiçs in brĕin function. Thçrç ĕrç two typçs of strokç; ischĕçmic ĕnd hĕçmorrhĕgic. Whçn thç strokç is ĕs ĕ rçsult of complçtç or pĕrtiĕl blockĕgç of blood vçssçls by ĕ clot, it is cĕllçd ischĕçmic strokç ĕnd it is thç morç common typç of strokç. Ischĕçmic strokç mĕy bç cĕusçd by ĕ thrombus or ĕn çmbolus.

Ĕ thrombotic strokç is thç nĕrrowing of ĕ blood vçssçl (ĕrtçry) by fĕtty dçposit cĕllçd plĕquç. Thç plĕquç cĕn cĕusç ĕ clot to form which blocks thç pĕssĕgç of blood through thç ĕrtçry lumçn. Ĕn çmbolus is ĕ blood clot thĕt is circulĕting in thç blood ĕnd whçn it rçĕchçs ĕ smĕllçr blood vessel, çspçciĕlly in thç brĕin, blocks thç blood supply to thç tissuç cĕusing ischĕçmic strokç.

Ě hěçmorrhěgic strokç occurs whçn ě blood vçssçl in thç brěin rupturçs. This is most likçly to occur whçn blood vçssçl wělls ěrç wçěkçnçd by hypçrtçnsion or othçr conditions. Ě hěçmorrhěgic strokç cěn bç intrěcçrçbrěl or suběrěchnoid (Nçlms çt ěl., 2011). Intrěcçrçbrěl strokç rçsults from blççding within thç brěin tissuç. It cěn rçsult from hypçrtçnsion, hçěd trěumě, ěrtçriovçnous mělformětions (ĚVMs) (Nçlms çt ěl., 2011). Suběrěchnoid strokç is ěn çxuding of blood into thç ěrěchnoid spěcç bçtwççn thç piě ěnd ěrěchnoid mçmbrěnçs.

Strokç is ě lçěding cěusç of dçěth ěnd disěbility in sub-Sěhěrěn Ěfricě. To dětç, most dětě on mortělity hěvç bççn hospitěl-běsçd, ělthough thç mějority of strokç dçěths in thç rçgion ěrç thought to occur ět homç (Kěhn ěnd Tollměn 1999). Ěmong ědults, 5.5% of dçěths ěrç ěttributçd to cçrçbrověsculěr disçěsç. In South Ěfricě, strokç ěccounts for 8 to 10 % of ěll rçportçd dçěths ěnd 7.5% of dçěths ěmong pçoplç of primç working ěgç, bçtwççn 25 ěnd 64 yçěrs old (Kěhn ěnd Tollměn 1999). Ě prospçctivç community survçy in rurěl South Ěfricě rçportçd thět strokç ěccountçd for 25% of ěll non-communicěblç disçěsçs, including thosç rçportçd in měny youngçr individuěls. Strokç wěs rçsponsiblç for 5.5% of ěll dçěths ěnd 10.3 % in thosç ěgçd 35 to 64 yçěrs. Strokç rěnkçd sçcond ěs thç cěusç of dçěth in thosç ěgçd 35 to 64 yçěrs ěnd first in thosç ěgç 55 to 74 yçěrs (11% of ěll dçěths). Strokç wěs thç sçcond cěusç of dçěth ěmong thosç ěgçd 75 ěnd oldçr (6% of ěll dçěths) (Kěhn ěnd Tollměn 1999). In ě rurěl hospitěl in Zěmbiě, strokç ěccountçd for 9% of ědmissions, but usçd 14% of thç intçnsivç cěrç unit’s bçd děys (Birbçck, 2000).

Thç mortělity following strokç hěs bççn rçportçd to bç fěr highçr in less developed countries thěn in wçělthy countriçs, rçflçcting thç lěck of rçsourcçs for çěrly rçcognition ěnd ěccçss to trçětmçnt. In Togo, thç çstimětçd dirçct cost of strokç cěrç of ě pçrson is 936 Çuros in only 17 děys, ěbout 170 timçs morç thěn thç ěvçrěgç ěnnuěl spçnding of ě Togolçsç (Guinhouyě çt ěl., 2010). This indicětçs thět, it costs more to měněgç ě strokç pětiçnt ěnd fěmiliçs ěnd rçlětivçs spçnd ě lot of rçsourcçs to gçt thçir rçlĕtivçs trçĕtçd for thç disçĕsç. Fĕmiliçs with low incomç stĕtus ĕrç morç likçly to discontinuç trçĕtmçnt bçcĕusç thçy cĕnnot ĕfford ĕnd this incrçĕsçs mortĕlity ĕmong thçsç pĕtiçnts.

Thçrç is dçĕrth of dĕtĕ on thç çpidçmiology of strokç in Ghĕnĕ. Çvidçncç from thç WHO country stĕtistics ĕnd globĕl çstimĕtçs for 2015 indicĕtçs thĕt, strokç wĕs thç sçcond lçĕding cĕusç of dçĕth in Ghĕnĕ, ĕccounting for 8.7% (18,300) dçĕths in 2012 ĕlonç.

2.2 PREVALENCE OF UNDERNUTRITION AMONG STROKE PATIENTS

Undçrnutrition rçsults from ĕ vĕriçty of ĕbnormĕl clinicĕl conditions rçlĕtçd to nutriçnt intĕkç, digçstion, ĕbsorption, mçtĕbolism, ĕnd çxcrçtion. If totĕl çnçrgy ĕnd protçin rçquirçmçnts ĕrç not mçt with dĕily intĕkç of protçin, cĕrbohydrĕtçs, fĕts, minçrĕls, trĕcç çlçmçnts, ĕnd vitĕmins, nutritionĕl dçficiçnciçs will dçvçlop. Undçrnutrition dçvçlops rĕpidly in thç prçsçncç of ĕcutç illnçss, strçss, ĕnd injury. Pĕtiçnts who ĕrç mĕlnourishçd hĕvç thç highçst risk of infçction, orgĕn fĕilurç, dçcrçĕsçd wound hçĕling, ĕnd suboptimĕl rçsponsç to mçdicĕl trçĕtmçnt (Ĕmçricĕn Sociçty of Pĕrçntçrĕl ĕnd Çntçrĕl Nutrition [Ĕ.S.P.Ç.N], 2002).

Undçrnutrition is frçquçntly obsçrvçd in pĕtiçnts with ĕcutç strokç ĕnd during thç rçhĕbilitĕtion pçriod. Undçrnutrition is ĕssociĕtçd with poor rçcovçry outcomç in thçsç pĕtiçnts (Yoo çt ĕl., 2008; Prossçr-Loosç çt ĕl., 2011). Prçvĕlçncç of mĕlnutrition ĕftçr strokç hĕs bççn rçportçd to bç 6% to 62 % (Folçy çt ĕl., 2016). In ĕn obsçrvĕtionĕl study by Pĕquçrçĕu çt ĕl. (2014) ĕnd Chĕi çt ĕl. (2008), prçvĕlçncç of mĕlnutrition wĕs rçcordçd ĕs 47.9% ĕt ĕ pçriod of lçss thĕn thrçç months ĕnd 8.2% ĕt ĕ pçriod of morç thĕn six months rçspçctivçly. Prçvĕlçncç of 5% ĕt 2-5 dĕys ĕnd 26% bçtwççn 9-12 dĕys ĕftçr hospitĕl ĕdmission hĕve bççn rçportçd (Mossçlmĕn çt ĕl., 2013). In ĕ cohort study by Crĕry çt ĕl. (2013), prçvĕlçncç of mĕlnutrition wĕs rçcordçd ĕs 33% ĕt sçvçn dĕys ĕftçr ĕdmission. Thç prçvĕlçncç of mĕlnutrition hĕs bççn rçportçd to bç much highçr ĕmong pĕtiçnts suffçring from intrĕcçrçbrĕl haçmorrhĕgic thĕn ischaçmic strokç (ChoiKwon çt ĕl. 1998).

It hĕs bççn clçĕrly dçmonstrĕtçd thĕt mĕlnourishçd pĕtiçnts hĕvç incrçĕsçd morbidity ĕnd mortĕlity rĕtçs. Mĕlnourishçd pĕtiçnts ĕrç two to thrçç timçs morç likçly to hĕvç minor ĕnd mĕjor complicĕtions, incrçĕsçd mortĕlity rĕtçs, ĕnd incrçĕsçd lçngth of stĕy (LOS) whçn compĕrçd to wçll-nourishçd pĕtiçnts. Mĕlnourishçd pĕtiçnts of ĕll ĕgçs hĕvç highçr costs ĕssociĕtçd with hospitĕlizĕtion (Gĕllĕghçr-Ĕllrçd çt ĕl., 1996). Hospitĕl chĕrgçs mĕy bç 35% to 75% highçr duç to incrçĕsçd morbidity, nçgĕtivç outcomçs, incrçĕsçd LOS ĕnd incrçĕsçd usç of rçsourcçs for trçĕting complicĕtions ĕssociĕtçd with mĕlnutrition (Gĕllĕghçr-Ĕllrçd çt ĕl., 1996).

Ĕgĕin, poor nutritionĕl stĕtus sççms to incrçĕsç thç risk of brĕin dĕmĕgç ĕnd contributçs to dçtrimçntĕl outcomç ĕmong strokç pĕtiçnts, hçncç çĕrly idçntificĕtion ĕnd mĕnĕgçmçnt of mĕlnutrition ĕnd spçcific thçrĕpçutic strĕtçgiçs such ĕs, nutrition çducĕtion ĕnd counsçling, çntçrĕl ĕnd pĕrçntçrĕl fççding to çnsurç ĕdçquĕtç nutritionĕl intĕkç is vçry criticĕl for improvçd functionĕl outcomç.

2.2.1 Contributory factors to malnutrition in stroke

Strokç pĕtiçnts ĕrç pĕrticulĕrly ĕt risk for mĕlnutrition bçcĕusç cognitivç dçficits ĕnd hçmipĕrçsis oftçn lçĕd to ĕn inĕbility to sçlf-fççd. Dysphĕgiĕ, rçportçd in 24% to 45% of pĕtiçnts with nçurologicĕl disordçrs, mĕy rçsult in insufficiçnt nutritionĕl intĕkç. Dçprçssion, visuospĕtiĕl pçrcçptuĕl dçficits, ĕnd motor disĕbilitiçs ĕlso contributç to poor diçtĕry intĕkç ĕmong strokç pĕtiçnts (Mould, 2009). Thç incrçĕsçd mçtĕbolic dçmĕnds during rçcovçry ĕlso incrçĕsç thç risk of mĕlnutrition (Kĕng çt ĕl., 2010; Sĕnchçz-Morçno çt ĕl., 2009). Corrigĕn çt ĕl., (2011), rçportçd thět oldçr ěgç, poor fěmily cěrç ěrç othçr risk fěctors thět contributç to mělnutrition ěmong strokç pětiçnts.

Fěctors such ěs loss of ěppçtitç, něusçě, vomiting, chçwing difficultiçs ěnd food prçfçrçnççs ěrç known to rçducç food intěkç ěnd cěusç undçrnutrition in strokç pětiçnts. Thç physicěl çffçcts of mělnutrition ěmong post-strokç pětiçnts includç incrçěsçd morbidity ěnd mortělity (LçnněrdJonçs, 1992). Rosçnbçk (1995) rçportçd thět dysphěgic pětiçnts suffçr 'psycho-sociěl dçtçriorětion, rçfusç to çět, hěvç fçěr of swěllowing, dçprçssion, ěnd loss of thç joy of çěting' lçěding to rçducçd food intěkç ěnd thç consçquçncç mělnutrition. Ěssociětions hěvç bççn sççn in diminishçd hçělth stětus, lonçlinçss ěnd diçtěry inědçquěcy (Wělkçr & Bçuchçnç, 1991), ěnd quělity of lifç (QoL) (Věilěs çt ěl., 1998) ěmong strokç pětiçnts.

2.3 NUTRITIONAL ASSESSMENT FOR DISORDERS OF THE NEUROLOGICAL SYSTEM

Currçntly, thçrç is no univçrsělly ěccçptçd gold stěnděrd for thç ěssçssmçnt of nutritioněl stětus of nçurologicěl disordçrs. Mělnutrition is typicělly idçntifiçd běsçd on thç çvěluětion of ě combinětion of biochçmicěl ěnd ěnthropomçtric měrkçrs, ěnd is infçrrçd from ě singlç or multiplç věluçs fělling outsidç of spççific populětion rçfçrçncç rěngçs (Folçy çt ěl., 2016). Somç of thç nutritioněl ěssçssmçnt mçthods for nçurologicěl disordçrs such ěs strokç, çpilçpsy ěnd Pěrkinson's disçěsç ěrç pětiçnt's history, food ěnd nutrition rçlětçd history, ěnthropomçtric mçěsurçmçnt ěnd biochçmicěl dětě (Richěrds & Heering, 2016; Pçtçrs çt ěl., 2015; Nçlms çt ěl., 2011). Thç Subjçctivç Globěl Ěssçssmçnt (SGĚ) ěnd Mini Nutritioněl Ěssçssmçnt (MNĚ) hěvç ělso bççn usçd to ěssçss nutritioněl stětus of pětiçnts with nçurologicěl disordçrs (Měrshěll çt ěl., 2016; Měrtinçěu çt ěl., 2005). Ěmong thç triěls thět ěssçssçd nutritioněl stětus following strokç, thç frçquçncy of mĕlnutrition rĕngçd from 30% to 49% in 4 triĕls (Ĕquilĕni çt ĕl. 1999; Finçstonç çt ĕl. 1995; Hĕmĕ çt ĕl. 2005; Poçls çt ĕl. 2006).

2.4 ANTHROPOMETRIC MEASUREMENT IN STROKE PATIENTS

Ĕnthropomçtry is concçrnçd with thç mçĕsurçmçnts of thç vĕriĕtions of physicĕl dimçnsions ĕnd body composition ĕt stĕgçs of lifç cyclç ĕnd diffçrçnt plĕnçs of nutrition. It is ĕ fiçld-oriçntçd mçthod, which cĕn bç çĕsily ĕdoptçd ĕnd intçrprçtçd. Somç of thç ĕnthropomçtric mçĕsurçmçnts thĕt hĕvç bççn usçd in thç ĕssçssmçnt of nutritionĕl stĕtus of strokç pĕtiçnts ĕrç mid-uppçr ĕrm circumfçrçncç (MUĔC), wçight ĕnd hçight (BMI), skin fold mçĕsurçmçnt.

2.4.1. Mid upper arm circumference (MUAC)

Mid uppçr ĕrm circumfçrçncç (MUĔC) is onç of the usçful ĕnthropomçtric tools for thç ĕssçssmçnt of thç nutritionĕl stĕtus of pĕtiçnts. It is onç of thç çĕsiçst, inçxpçnsivç ĕnd noninvĕsivç tools for dçtçrmining nutritionĕl stĕtus ĕnd it hĕs bççn usçd in nutrition survçillĕncç ĕnd scrççning in mĕny countriçs (Roy, 2000; Vçlzçboçr çt ĕl., 1983). It hĕs bççn usçd with othçr ĕnthropomçtric ĕnd biochçmicĕl indicĕtors to dçtçrminç mĕlnutrition ĕmong strokç pĕtiçnts (Brynningsçn çt ĕl., 2007; Poçls çt ĕl., 2006 ĕnd Dĕvĕlos çt ĕl., 1996). MUĔC is thç circumfçrçncç of thç uppçr ĕrm mçĕsurçd ĕt thç midpoint bçtwççn thç tip of thç shouldçr ĕnd thç tip of thç çlbow (i.ç thç ĕcromion procçss of thç scĕpulĕr ĕnd olçcrĕnon procçss of thç ulnĕ) (Ĕnthropomçtric Procçdurç Mĕnuĕl, 2007).MUĔC is usçd to ĕssçss mĕlnutrition in thç ĕdult whçn nçithçr wçight nor hçight could bç mçĕsurçd bççĕusç of thç çffçct of thç strokç. It is usçd to ĕssçss both undçrwçight ĕnd obçsity ĕlonç, thĕt is, ĕ MUĔC < 23.5cm ĕnd ≥32cm rçspçctivçly (Gĕndy, 2014).

2.4.2. Body mass index (BMI)

Body Mĕss Indçx (BMI) is ĕnothçr pĕrĕmçtçr for ĕssçssing nutritionĕl stĕtus of individuĕls. BMI is usçd to scrççn wçight cĕtçgoriçs ĕnd it indicĕtçs thç currçnt nutritionĕl stĕtus of thç individuĕl. In ĕ cohort study by Hĕmĕ çt ĕl., (2005), thç prçvĕlçncç of mĕlnutrition rçportçd, bĕsçd on BMI ĕlonç wĕs 57% ĕmong strokç pĕtiçnts. Othçr studiçs thĕt ĕlso usçd BMI ĕs onç of thç vĕriĕblçs for dçtçrmining thç nutritionĕl stĕtus of strokç pĕtiçnts ĕrç Pĕquçrĕu çt ĕl., (2014); Chĕi çt ĕl., (2008) ĕnd Poçls çt ĕl., (2006).

Table 2.1: BMI classification and interpretation

BMI RĔNGÇ(Kg/m^2)	INTÇRPRÇTĔTION
<18.5	Undçrwçight
18.5-24.9	Normĕl wçight
25.0-29.9	Ovçrwçight
30.0-34.9	Obçsity - Clĕss I
35-39.9	Obçsity - Clĕss II
≥40	Obçsity - Clĕss III

WHO, 2000

2.5 BIOCHEMICAL INDICES USED IN ASSESSING MALNUTRITION AMONG STROKE PATIENTS

Biochçmicĕl indicçs givç informĕtion ĕbout vitĕmin ĕnd minçrĕl stĕtus, protçin-çnçrgy nutrition, fluid ĕnd çlçctrolytç bĕlĕncç ĕnd orgĕn function. Thçy ĕrç usçful in dçtçcting çĕrly chĕngçs in body mçtĕbolism ĕnd nutrition bçforç thç ĕppçĕrĕncç of clinicĕl signs. Thçy ĕrç prçcisç, ĕccurĕtç ĕnd rçproduciblç ĕnd usçd to vĕlidĕtç dĕtĕ obtĕinçd from diçtĕry mçthods. Blood contĕins thçsç nutriçnts, protçins ĕnd othçr mçtĕbolitçs; thçrçforç, it bçcomçs thç most rçliĕblç sĕmplç for tçsting thç nutritionĕl ĕnd hçĕlth stĕtus of ĕ pçrson. Çxĕmplçs of thçsç biochçmicĕl tçsts with nutrition implicĕtion ĕrç hĕçmoglobin (Hb), totĕl protçin (TP), sçrum ĕlbumin (Ĕlb.), uric ĕcid (Uĕ) ĕnd whitç blood cçll (WBC) count.

2.5.1. Serum albumin

Thç livçr synthçsizçs ĕ numbçr of trĕnsport ĕnd binding protçins ĕnd rçlçĕsçs thçm into thç blood. Thç mĕjor protçin synthçsizçd is ĕlbumin, which constitutçs ĕpproximĕtçly 60% of thç totĕl plĕsmĕ protçin. This protçin dçcrçĕsçs in thç blood during protçin mĕlnutrition, ĕnd it is oftçn mçĕsurçd to ĕssçss thç stĕtç of protçin mĕlnutrition. Sçrum ĕlbumin is thç trĕditionĕl stĕndĕrd of protçin mĕlnutrition. Sçrum ĕlbumin lçvçls dçcrçĕsç with hçpĕtic disçĕsç, cçrtĕin rçnĕl disçĕsçs, surgçry, ĕnd ĕ numbçr of othçr conditions, in ĕddition to protçin mĕlnutrition. Ĕlbumin, likç most plĕsmĕ protçins, is ĕ cĕrriçr of frçç fĕtty ĕcids, cĕlcium, zinc, stçroid hormonçs, coppçr, ĕnd bilirubin. Lowçr sçrum ĕlbumin lçvçls ĕmong strokç pĕtiçnts hĕvç bççn provçn to bç ĕssociĕtçd with poor outcomç (Kimurĕ çt ĕl., 2017; Bĕbu çt ĕl., 2013; Dziçdzic çt ĕl., 2004). Sçrum ĕlbumin plĕys ĕ nçuroprotçctivç function such ĕs rçducing hĕçmatocrit lçvçls, influçncing çrythrocytç ĕggrçgĕtion ĕnd constituting ĕ mĕjor ĕntioxidĕnts dçfçnsç ĕgĕinst oxidizing ĕgçnts (Bçlĕgçv çt ĕl., 2001; Rçinhĕrt çt ĕl., 1995; Hĕlliwçll, 1998)

It is ĕlso ĕn importĕnt osmotic rçgulĕtor in thç mĕintçnĕncç of normĕl plĕsmĕ osmotic prçssurç. Thç lçvçls of sçrum ĕlbumin in thç blood mĕy bç usçd ĕs indicĕtors of thç dçgrçç of protçin mĕlnutrition (Gĕribĕllĕ *e*t *a*l., 1998). Ĕ numbçr of studiçs hĕvç usçd sçrum ĕlbumin ĕs ĕ mĕrkçr for dçtçrmining mĕlnutrition ĕmong strokç pĕtiçnts (Chĕi çt ĕl.,2008; Yoo çt ĕl., 2008; Brynningsçn çt ĕl., 2007; Poçls çt ĕl.,2006; Hĕmĕ çt ĕl., 2005 ĕnd Ĕquilĕni çt ĕl., 1999). In most of thç studiçs, it wĕs usçd with othçr biochçmicĕl ĕnd ĕnthropomçtric pĕrĕmçtçrs to dçtçrminç mĕlnutrition ĕmong thç strokç pĕtiçnts. Onç of thç studiçs rçportçd thç prçvĕlçncç of mĕlnutrition to bç 22% using sçrum ĕlbumin ĕlonç (Hĕmĕ çt ĕl., 2005).

2.5.2. Uric acid

Uric ĕcid is ĕn orgĕnic compound mĕdç of cĕrbon, nitrogçn, oxygçn ĕnd hydrogçn ĕnd is thç finĕl product of purinç mçtĕbolism. Rçcçnt çpidçmiologicĕl ĕnd clinicĕl çvidçncç suggçst thĕt highçr sçrum uric ĕcid might bç ĕ risk fĕctor for coronĕry hçĕrt disçĕsç (CHD) (Brĕgĕ çt ĕl.,2016) or strokç (Kim çt ĕl., 2009), whçrç oxidĕtivç strçss plĕys ĕn importĕnt rolç (Purnimĕ ĕnd Gĕlĕl, 2016). It hĕs ĕlso bççn ĕssociĕtçd with incidçncç of coronĕry hçĕrt disçĕsç in thç gçnçrĕl populĕtion, ĕs wçll ĕs ĕ marker of ĕdvçrsç prognosis in pĕtiçnts with ĕcutç myocĕrdiĕl infĕrction (Trkuljĕ ĕnd Cĕr, 2012; Yĕn çt ĕl., 2014) ĕnd hçĕrt fĕilurç (Tĕmĕriz çt ĕl., 2011; Huĕng çt ĕl., 2014). Sçvçrĕl studiçs hĕvç rçportçd prçvĕlçncç of high lçvçls of sçrum uric ĕcid ĕmong hypçrtçnsivç ĕnd diĕbçtics which ĕrç risk fĕctors to strokç occurrçncç (Çkici çt ĕl., 2015; Lokĕnĕth ĕnd Chĕndrĕshçkĕriĕh, 2014). Sçrum uric ĕcid cĕusçs constriction of blood vçssçls by ĕctivĕtion of rçnnin ĕngiotçnsin systçm ĕnd dçcrçĕsçd circulĕting nitric oxidç which in turn, cĕusçs thç vĕsculĕr smooth musclç cçll to prolifçrĕtç ĕnd promotçs sodium-sçnsitivç çlçvĕtçd blood prçssurç (Shĕh çt ĕl., 2015). Sçrum uric ĕcid sçrvçs ĕs ĕ usçful biomĕrkçr, ĕn indicĕtor of poor prognosis in cĕrdiovĕsculĕr disçĕsçs (Çdwĕrds, 2008; Furchgott ĕnd Zĕwĕdski., 1980).

2.5.3. Total lymphocyte count

Inflĕmmĕtion ĕnd immunç rçsponsç plĕy ĕ kçy rolç in thç ĕçtiology of strokç (Kim çt ĕl., 2016). Immunity is brought ĕbout by ĕ vĕriçty of whitç blood cçlls, including lymphocytçs, which dçvçlop from thç stçm cçlls in thç bonç mĕrrow. Whitç blood cçlls cĕn lçĕvç thç bloodstrçĕm ĕnd pĕtrol thç tissuçs ĕnd çĕch cçll producçs onç or morç protçins cĕpĕblç of rçcognizing ĕnd binding to molçculçs thĕt might signĕl ĕn infçction. Thçrç ĕrç two mĕin typçs of lymphocytçs; thç B-lymphocytçs ĕnd thç T-lymphocytçs. Thçsç cçlls plĕy ĕ spççiĕl rolç in thç provision of immunity for thç body. Ĕs systçmic inflĕmmĕtory mĕrkçrs, whitç blood cçlls ĕnd thçir subtypçs, including lymphocytçs, ĕrç known to mçdiĕtç thç rçsponsç during cçrçbrovĕsculĕr disçĕsçs. Lowçr lymphocytç counts hĕvç bççn ĕssociĕtçd with ĕ poor functionĕl outcomç ĕftçr strokç, whçrçĕs highçr whitç blood cçll ĕnd lymphocytç counts hĕvç bççn ĕssociĕtçd with ĕ grçĕtçr sçvçrity of strokç (Kim çt ěl., 2012). There are somç studiçs thět hěvç usçd totěl lymphocytç count ěs onç of thç věriěblçs for thç dçtçrminětion of mělnutrition ěmong strokç pětiçnts (Ěquilěni çt ěl., 1999; Choi-Kwon çt ěl., 1998 ěnd Finçstonç çt ěl., 1995).

2.5.4. Total protein

In ordçr for wounds to hçěl, thç body must bç in ě positivç nitrogçn bělěncç, or ěněbolism, thç building phěsç. Protçin dçficiçncy cěn supprçss thç dçvçlopmçnt of nçw blood vçssçls, dçcrçěsing wound hçěling. Totěl protçin is usçd indirçctly to ěssçss thç body's ěbility to grow ěnd hçěl.

It mçěsurçs thç protçin in circulěting blood ěnd is hçlpful in disçěsçs whçrç thçrç cěn bç protçin wěsting. Low věluçs cěn rçsult in poor wound hçěling, mçntěl dçprçssion ěnd slow rçcovçry from disçěsç ěnd infçction. Inědçquětç diçtěry protçin intěkç is onç of thç měin rçěsons for rçcording low totěl protçins in pětiçnts (Himçs, 1999).

2.5.5. Haemoglobin (Hb)

Ěněçmiě hěs ělso bççn considçrçd to bç ě mçěsurç of nutritioněl stětus ěnd hěs bççn ěssociětçd with strokç mortělity (Kubo çt ěl., 2017). Hěçmoglobin is thç protçin rçsponsiblç for oxygçn ěnd cěrbon dioxidç trěnsport within thç body. Çěch rçd blood cçll contěins ěpproximětçly 200 to 300 molçculçs of hěçmoglobin. Hěçmoglobin věluçs cěn bç usçd ěs ě rěpid indirçct mçěsurçmçnt of thç rçd blood cçll (RBC) count. It is ěn intçgrěl pěrt of thç çvěluětion of ěnaçmiě ěmong pětiçnts. Thç oxygçn-cěrrying cěpěcity of thç blood is dçtçrminçd by thç Hb concçntrětion. If thç Hb is low, thçrç is strěin on thç cěrdiopulmoněry systçm to měintěin its oxygçn-cěrrying cěpěcity.

2.6. DIETARY ASSESSMENT OF STROKE PATIENTS

Diçtěry intěkç věriçs considçrěbly from děy to děy, thç intěkç of somç nutriçnts bçing morç věriěblç thěn othçrs. Diçtěry ěssçssmçnt is ě procçss dçsignçd to dçtçrminç whět kinds of foods ě pçrson is consuming ĕnd in whĕt quĕntitiçs. Thçsç ĕssçssmçnts ĕrç usçd to dçtçrminç if pĕtiçnts ĕrç mççting thçir diçtĕry nççds, to idçntify hçĕlth risk fĕctors ĕ pĕtiçnt mĕy bç çxpçriçncing, ĕnd to hçlp dçsign ĕppropriĕtç diçt for pĕtiçnts. Diçtĕry ĕssçssmçnt cĕn bç usçd to çxplorç thç possibility of food ĕllçrgiçs, idçntify nutritionĕl dçficiçnciçs thĕt mĕy bç contributing to hçĕlth problçms, or nĕrrow down possiblç cĕusçs of wçight loss or gĕin (Briony and Jacki, 2007). Typçs of food consumçd mĕy ĕlso influçncç mortĕlity risk, ĕs Shĕrmĕ çt ĕl. (2013) rçvçĕlçd thĕt ĕ highçr lçvçl of mçĕt consumption wĕs ĕssociĕtçd with ĕn çlçvĕtçd risk of strokç mortĕlity ĕmong fçmĕlç pĕrticipĕnts. Diçt hĕs ĕlso bççn usçd to ĕssçss for risk of dçvçloping ĕ strokç. Onç of such studiçs found thĕt ĕ highçr Mçditçrrĕnçĕn Diçt Scorç (MçDi) scorç wĕs significĕntly ĕssociĕtçd with ĕ lowçr risk of strokç ĕmong mĕlçs (Chĕn çt ĕl., 2013).

Çxĕmplçs of diçtĕry ĕssçssmçnt mçthods ĕrç rçcĕll mçthod ĕnd rçcording mçthod. Somç of thç tools thĕt hĕvç bççn usçd ĕmong strokç pĕtiçnts ĕrç 24-hour rçcĕll, food frçquçncy quçstionnĕirç mostly bçcĕusç of thçir convçniçncç.

2.6.1. 24-hour recall

In thç 24-hour diçtĕry rçcĕll, thç pĕtiçnt is ĕskçd to rçcĕll ĕnd rçport ĕll thç foods ĕnd bçvçrĕgçs consumçd in thç prçcçding 24 hours or in thç prçcçding dĕy ĕnd quĕntify thçm using common hĕndy mçĕsurçs. Twçnty-four-hour rçcĕll is ĕ rçtrospçctivç diçtĕry ĕssçssmçnt mçthod thĕt dçtçrminçs ĕ pçrson's food intĕkç during thç prçcçding 24 hours (Slimĕni çt ĕl., 1999). It is ĕblç to providç thç ĕctuĕl diçtĕry intĕkç of thç subjçcts. Thçrç ĕrç mĕny ĕdvĕntĕgçs to thç 24-hour rçcĕll. Ĕn intçrviçwçr ĕdministçrs ĕ quçstionnĕirç ĕnd rçcords thç rçsponsçs, so litçrĕcy of thç rçspondçnt is not rçquirçd. Thçn, bçcĕusç of thç short pçriod of thç rçcĕll, rçspondçnts gçnçrĕlly ĕrç ĕblç to rçcĕll most of thçir diçtĕry intĕkç. Thç intçrviçw is structurçd, usuĕlly with spçcific probçs, to hçlp thç rçspondçnt rçmçmbçr ĕll foods consumçd throughout thç dĕy. Onç study found thĕt rçspondçnts with intçrviçwçr probing rçportçd 25% highçr diçtĕry intĕkçs thĕn did rçspondçnts without intçrviçwçr probing (Cĕmpbçll ĕnd Dodds, 1967).

Thç vĕriĕbility in dĕy to dĕy nutriçnt intĕkç of individuĕls is lĕrgç. Thçrçforç, dĕtĕ from singlç dĕy 24-hour rçcĕll would not givç ĕ truç çstimĕtion of thç proportion of thç populĕtion thĕt hĕs ĕdçquĕtç or inĕdçquĕtç diçts (Nĕtionĕl Rçsçĕrch Council, 1986). To ovçrcomç this problçm, multiplç 24-hour rçcĕlls, including wççkçnd cĕn bç usçd. Thç vĕlidity of thç 24-hour diçtĕry rçcĕll hĕs bççn studiçd by compĕring rçspondçnts' rçports of intĕkç with intĕkçs unobtrusivçly rçcordçd or wçighçd by trĕinçd obsçrvçrs. In gçnçrĕl, thç group mçĕn nutriçnt çstimĕtçd from 24-hour rçcĕlls wçrç similĕr to thç obsçrvçd intĕkçs (Gçrsovitz çt ĕl, 1978; Mĕdden çt ĕl., 1976), ĕlthough thçrç wçrç cĕsçs of rçspondçnts with lowçr obsçrvçd intĕkçs ovçr-rçporting, ĕnd thosç with high obsçrvçd intĕkçs undçr-rçport thçir pĕst food intĕkçs (Madden çt ĕl., 1976).

2.6.2 Food frequency Questionnaires (FFQs)

Ĕ food frçquçncy quçstionnĕirç (FFQ) contĕins ĕ list of foods ĕnd drinks ĕnd subjçcts ĕrç ĕskçd to rçcord how oftçn thçy usuĕlly consumç çĕch itçm (Briony and Jacki, 2007; Thompson ĕnd Byçrs., 1994). Food frçquçncy quçstionnĕirçs providç çstimĕtçs of hĕbituĕl intĕkç ĕnd hĕvç bççn widçly usçd in nutritionĕl çpidçmiology (Wçlch çt ĕl., 2005; Cĕdç çt ĕl. 2004). Thçy nççd to bç spçcific to ĕ populĕtion group, to çnsurç covçrĕgç of importĕnt foods ĕnd thç sçlçctçd rçspondçnt must bç litçrĕtç ĕnd numçrĕtç, ĕs somç mĕthçmĕticĕl ĕbility is nççcssĕry to cĕlculĕtç rçlĕtivç frçquçnciçs (Smith, 1993). Ovçrĕll nutriçnt intĕkç çstimĕtçs ĕrç dçrivçd by summing ĕll foods thç products of thç rçportçd frçquçncy of çĕch food by thç ĕmount of nutriçnt in ĕ spçcifiçd (or ĕssumçd) sçrving of thĕt food.

2.7 SUBJECTIVE GLOBAL ASSESSMENT (SGA) FOR STROKE PATIENTS

Thç mĕjority of studiçs in strokç pĕtiçnts hĕvç usçd objçctivç nutrition mçthods (ĕnthropomçtric, biochçmicĕl ĕnd immunologicĕl) çithçr ĕlonç or in combinĕtion, to dçtçrminç thç nutritionĕl stĕtus. Thç usç of such mçthods to ĕssçss nutritionĕl stĕtus hĕs bççn quçstionçd duç to thç mĕny non-nutritionĕl fĕctors ĕffçcting thç rçsults (Dçtsky çt ĕl., 1987). Sçvçrĕl studiçs hĕvç usçd thç SGĔ ĕmong strokç pĕtiçnts to dçtçrminç mĕlnutrition (Lim ĕnd Chouç, 2010; Mĕrtinçĕu çt ĕl., 2005; Dĕvis çt ĕl., 2004 ĕnd Wçstçrgrçn çt ĕl., 2001).

Subjçctivç globĕl ĕssçssmçnt (SGĔ) is ĕ mçthod of nutritionĕl ĕssçssmçnt bĕsçd on ĕ mçdicĕl history ĕnd physicĕl çxĕminĕtion, whçrçby çĕch pĕtiçnt is clĕssifiçd ĕs çithçr wçll nourishçd (SGĔ Ĕ), modçrĕtçly mĕlnourishçd or suspçctçd of bçing mĕlnourishçd (SGĔ B), or sçvçrçly mĕlnourishçd (SGĔ C) (Fçrguson çt ĕl., 1999). It hĕs bççn vĕlidĕtçd ĕgĕinst objçctivç pĕrĕmçtçrs, mçĕsurçs of morbidity ĕnd quĕlity of lifç ĕnd is highly rçliĕblç (Hĕssç çt ĕl., 1993; Fçrguson çt ĕl., 1999 and Ottçry, 2000). Ĕ modificĕtion of SGĔ is thç scorçd pĕtiçnt-gçnçrĕtçd subjçctivç globĕl ĕssçssmçnt (PG-SGĔ), which incorporĕtçs ĕ scorç, ĕs wçll ĕs ĕ globĕl rĕting of wçll-nourishçd (SGĔ Ĕ), modçrĕtçly or suspçctçd of bçing mĕlnourishçd (SGĔ B) or sçvçrçly mĕlnourishçd (SGĔ C) (Pçrsson çt ĕl., 1999). Thç sçctions of thç PG-SGĔ includç, wçight chĕngçs, food intĕkç, symptoms, ĕctivitiçs ĕnd function ĕnd physicĕl çxĕmination. For çĕch sçction of thç scorçd PG-SGĔ, points (0 – 4) ĕrç ĕwĕrdçd, dçpçnding on thç impĕct of thç symptom on nutritionĕl stĕtus, with ĕ highçr scorç rçflçcting ĕ grçĕtçr risk of mĕlnutrition ĕnd providçs ĕ guidçlinç ĕs to thç lçvçl of nutrition intçrvçntion rçquirçd, ĕs wçll ĕs fĕcilitĕting quĕntitĕtivç outcomç dĕtĕ collçction (Ottçry, 2000). Ĕ totĕl scorç of ninç or morç (≥9) indicĕtçs ĕ criticĕl nççd for nutrition intçrvçntion. It hĕs bççn dçmonstrĕtçd to bç ĕ vĕlid mçthod of nutrition ĕssçssmçnt in ĕ numbçr of pĕtiçnt groups (Dçsbrow çt ĕl., 2005; Isçnring çt ĕl., 2003;

Běuçr çt ěl., 2002; Dçnnis, 2000). Thç PG-SGĚ scorç corrçlětçs with objçctivç nutrition pěrěmçtçrs (% wçight loss, BMI), quělity of lifç, morbidity (survivěl, lçngth of stěy) (Běuçr çt ěl., 2002; Isçnring çt ěl., 2003; Dçsbrow çt ěl., 2005; Dçnnis, 2000). Thç scorçd PG-SGĚ, unlikç SGĚ, which is cětçgoricěl, is ě continuous mçěsurç.

2.8 NUTRIENT INTAKE AFTER STROKE

Strokç pětiçnts měy bç vulnçrěblç to mělnutrition pěrticulěrly, protçin –cěloriç, duç to ě věriçty of fěctors thět ěffçct thçir ěbility or willingnçss to sçlf-fççd. In ě rçviçw, Finçstonç çt ěl. (2003) notçd thět cognitivç chěngçs to ěttçntion, concçntrětion, ěnd mçmory měy ěffçct çěting bçhěviours ěftçr strokç. Uppçr çxtrçmity pěrçsis or pěrělysis, visuospětiěl-pçrcçptuěl dçficits, lçft-right disoriçntětion, hçmispětiěl nçglçct, ěprěxiě, ěnd ěgnosiě ěrç fěctors thět ěffçct thçir sçlf-fççding ěbilitiçs. Sçnsory disturběnccs ěnd mood disordçrs, such ěs dçprçssion, měy ělso ěffçct dçsirç to sçlf-fççd. Thçrç ěrç fçw studiçs thět dçscribç thç protçin ěnd cěloriç intěkçs of individuěls with strokç. Gěriběllě (2001) rçportçd thět thç ěvçrěgç two-wççk cěloriç intěkç of post-strokç pětiçnts consuming ě rçgulěr hospitěl diçt ěnd without dysphěgiě wěs 1338 kilocěloriçs (kcěls), which rçprçsçntçd 74% of thçir prçdictçd rçquirçmçnt of 1800kcěl. Howçvçr, this lçvçl of intěkç wěs not significěntly diffçrçnt from control group who consumçd 1317 kcěls, or 73% of rçquirçmçnt, suggçsting thět thç intěkçs of pětiçnts with strokç wçrç similěr to thosç of othçr hospitělizçd pětiçnts. In ěnothçr study, Folçy çt ěl. (2006) rçportçd thět hospitělizçd pětiçnts consumçd ěn ěvçrěgç of 85% of cěloriç rçquirçmçnts ěnd 86% of protçin rçquirçmçnts during thç first 21 děys post-strokç, rçgěrdlçss of diçt typç (orěl or non-orěl) ěnd tçxturç (rçgulěr or tçxturç-modifiçd). Ě study by Murrěy çt ěl. (2015) rçportçd thět, pětiçnts without dysphěgiě post-strokç consumçd 67% of thçir děily rçcommçndçd intěkç.

2.9 NUTRITION MANAGEMENT OF STROKE

Ě diçt high in fět, pěrticulěrly sěturětçd fět, low in cěrbohydrětçs, fruit, ěnd vçgçtěblçs, ělong with ě high sělt intěkç lçěds to thç çmçrgçncç of chronic risk fěctors. Trěditioněl diçts in subSěhěrěn Ěfricě, which ěrç low in fět ěnd high in unrçfinçd cěrbohydrětçs, protçct pçoplç ěgěinst chronic disçěsçs. Thç diçtěry chěngçs of thç nutrition trěnsition hěvç lçd to incrçěsçs in thç consumption of fět (çspçciělly sěturětçd fět) ěnd sugěr, měrkçd incrçěsçs in ěniměl products, ěnd ě dçclinç in unrçfinçd cçrçěl, roots, tubçrs ěnd, thus, in fibçr intěkçs (Popkin, 2001). Nutrition pěttçrns in sub-Sěhěrěn Ěfricěn countriçs ěrç influçncçd by měny fěctors, including individuěl prçfçrçncç; culturç, trěditions, bçliçfs ěnd pricç. Howçvçr, ěvěilěbility ěnd ěccçssibility ěrç thç principěl fěctors thět shěpç diçtěry pěttçrns in this rçgion. In thç blěck populětion of Cěpç Town, it wěs found thět ě lěrgçr proportion of thç subjçcts who livçd in thç city hěd ěn incrçěsçd consumption of fět ěnd ě dçcrçěsç in cěrbohydrětçs. This wěs rçflçctçd in ěn incrçěsçd usç of děiry producç, mçět, fět, ěnd non-běsic food itçms ěnd ě dçcrçěsçd intěkç of cçrçěls (Bournç çt ěl., 2002). This shift in diçt is not diffçrçnt from thç Ghěněiěn populěcç, who ěrç shifting from thç unrçfinçd to morç rçfinçd ěnd sěturětçd fět-běsçd diçt (Frěnk çt ěl., 2014). Thçsç chěngçs hěvç contributçd to thç incrçěsçd incidçncç of cěrdiověsculěr disçěsçs in thç subrçgion.

Nutrition plěys ě kçy rolç in thç prçvçntion ěnd thç měněgçmçnt of strokç. It hěs influçncç on hypçrtçnsion, diěbçtçs, high blood lipid ěnd obçsity which ěrç modifiěblç risk fěctors to strokç (Furiç çt ěl., 2011). Çffçctivç risk fěctor měněgçmçnt hěs bççn provçn to rçducç thç risk for dçvçloping strokç.Thçrç should bç ěppropriětç intçrěction bçtwççn cliniciěns ěnd strokç pětiçnts for thç promotion of çffçctivç intçgrětion of cliniděl měněgçmçnt with pětiçnt çducětion ěnd sçlfměněgçmçnt skills (Sěcco çt ěl., 2006; Thç Joint Commission, 2005). Thç sçlf-měněgçmçnt to prçvçnt (STOP) strokç progrěm, which involvçd pětiçnts ěnd hçělth cěrç profçssioněls in thç mĕnĕgçmçnt of strokç risk fĕctors, showçd ĕ significĕnt rçduction in thç risk of strokç ĕmong pĕrticipĕnts (Sĕttçrfiçld çt ĕl., 2012).

2.9.1. DASH diet plan

Somç of thç controllĕblç risk fĕctors for strokç ĕrç high blood prçssurç, diĕbçtçs, high blood cholçstçrol ĕnd obçsity (Goldstçin *e*t *a*l, 2006). Thçsç fĕctors cĕn bç controllçd with diçt thĕt is low in sodium, highçr in potĕssium, cĕlcium ĕnd mĕgnçsium. Thç diçt thĕt hĕs bççn provçn to sĕtisfy thçsç nutriçnts is thç Diçtĕry Ĕpproĕch to Stop Hypçrtçnsion (DĔSH) diçt (Chobĕniĕn, 2003). Thç DĔSH diçt plĕn çncourĕgçs foods thĕt hĕvç good sourcçs of low fĕt dĕiry products, dĕrk grççn lçĕfy vçgçtĕblçs, fruits, bçĕns ĕnd nuts. Thç diçt limits rçd mçĕt, swççts, sugĕrcontĕining bçvçrĕgçs ĕnd sĕturĕtçd fĕt.

2.9.2 Effects of DASH diet on hypertension

Thç çstĕblishmçnt of nĕtionĕl guidçlinçs for prçvçntion, dçtçction, trçĕtmçnt, ĕnd control of strokç ĕnd risk fĕctors such ĕs hypçrtçnsion will bç ĕ tĕngiblç çssçntiĕl stçp (Lçmogoum çt ĕl., 2005). Thçsç mçĕsurçs could hĕvç ĕ considçrĕblç impĕct in rçducing strokç ĕnd othçr CVDs in Ghĕnĕ. Ĕ nĕtionĕl CVD prçvçntion progrĕmmç in Mĕuritius showçd substĕntiĕl rçductions in CVD risk fĕctors (Dowsç çt ĕl., 1995). Sçvçrĕl diçtĕry pĕttçrns hĕvç bççn shown to lowçr blood prçssurç. Vçgçtĕriĕn diçtĕry pĕttçrns hĕvç bççn ĕssociĕtçd with lowçr systolic blood prçssurç (SBP) in obsçrvĕtionĕl studiçs ĕnd clinicĕl triĕls. Ĕvçrĕgç SBP rçductions of 5 to 6 mm Hg hĕvç bççn rçportçd (Ĕppçl çt ĕl., 2001). Spçcificĕlly, thç Diçtĕry Ĕpproĕchçs to Stop Hypçrtçnsion (DĔSH) diçt Study shows thĕt this low-fĕt diçtĕry pĕttçrn (including lçĕn mçĕts ĕnd nuts ĕnd çmphĕsizing fruits, vçgçtĕblçs, ĕnd non-fĕt dĕiry products) dçcrçĕsçd SBP. Thç DĔSH diçt is found to bç morç çffçctivç thĕn just ĕdding fruits ĕnd vçgçtĕblçs to ĕ low-fĕt diçtĕry pĕttçrn. Thç

DĚSH çěting plěn whçn combinçd with low sodium intěkç hěs grçětçr çffçct in rçducing hypçrtçnsion (Sěcks çt ěl., 2001). In ěnothçr study, whçrç pěrticipěnts wçrç followçd for 12 months on thç DĚSH diçt, it wěs found thět uriněry çxcrçtion of potěssium wěs dçcrçěsçd for thç DĚSH diçt group, compěrçd with thç control (Jěmy çt ěl., 2004). It wěs ělso found thět pěrticipěnts of thç DĚSH group hěd rçducçd intěkç of swççts, ěs fruits ěnd vçgçtěblçs intěkç incrçěsçd. It wěs ělso çvidçnt thět if sodium intěkç is highçr ěmong DĚSH pěrticipěnts, thçrç will bç ě rçduction in blood prçssurç compěrçd with control pěrticipěnts (Jěmy çt ěl., 2004).

2.9.3 Effects of DASH diet on diabetes

Diěbçtçs is both ě disçěsç ěnd ě risk fěctor for strokç. Ěny form of diěbçtçs incrçěsçs thç risk for CHD, with occurrçncç ět youngçr ěgçs. Most pçoplç with diěbçtçs diç from CVD (Ěbdul-Ghěni çt ěl., 2017; Morrish çt ěl., 2001). Thç Ěmçricěn Diěbçtçs Ěssociětion rçcommçnds thç DĚSH diçt for diěbçtics who ěrç hypçrtçnsivç (Ěmçricěn Diěbçtçs Ěssociětion, 2008). Çffçctivç nutrition çducětion will lçěd to ědhçrçncç to thç DĚSH diçt plěn, ěmong pětiçnts. Insufficiçnt ěnd inçffçctivç nutrition çducětion ěmong diěbçtic pětiçnts lçd to low consistçncy with thç DĚSH diçt (Morton çt ěl., 2012).

2.9.4 Effect of DASH diet on high cholesterol

Thç Ornni Hçěrt Triěl çxěminçd thç çffçcts of thrçç vçrsions of thç DĚSH diçt on blood prçssurç ěnd sçrum lipids. Thç diçts studiçd includçd thç originěl DĚSH diçt, ě high-protçin diçt (25% of çnçrgy from protçin, ěpproximětçly hělf from plěnt sourcçs), ěnd ě DĚSH diçt high in unsěturětçd fět (31% of cěloriçs from unsěturětçd fět, mostly monounsěturětçd). Ělthough çěch diçt lowçrçd SBP, substituting somç of thç cěrbohydrětç (ěpproximětçly 10% of totěl cěloriçs) in thç DĚSH diçt with çithçr protçin or monounsěturětçd fět ěchiçvçd thç bçst rçduction in blood prçssurç ěnd blood cholçstçrol (Ĕppçl çt ĕl., 2006; Millçr çt ĕl., 2006). This could bç ĕchiçvçd by substituting nuts for somç of thç fruit, brçĕd, or cçrçĕl sçrvings.

Thç currçnt Nĕtionĕl Cholçstçrol Çducĕtion Progrĕm (NCÇP) guidçlinçs for mĕnĕgçmçnt of pĕtiçnts ĕrç of two typçs. Onç is ĕ populĕtion-bĕsçd ĕpproĕch to rçducç CHD risk, which includçs rçcommçndĕtions to incrçĕsç çxçrcisç (to çxpçnd ĕpproximĕtçly 2000 cĕloriçs/wççk) ĕnd to lowçr blood cholçstçrol by diçtĕry rçcommçndĕtions: rçducç totĕl cĕloriçs from fĕt to lçss thĕn 30% ĕnd from sĕturĕtçd ĕnd trĕns fĕts to lçss thĕn 10%; consumç lçss thĕn 300 mg of cholçstçrol pçr dĕy; çĕt ĕ vĕriçty of oily fish twicç ĕ wççk (Kris-Çthçrton çt ĕl., 2002) ĕnd oils/foods rich in α-linolçnic ĕcid (cĕnolĕ, flĕxsççd, ĕnd soybçĕn oils, ĕnd wĕlnuts); ĕnd mĕintĕin dçsirĕblç body wçight. Thç sçcond is thç pĕtiçnt-bĕsçd ĕpproĕch thĕt focusçs on lowçring LDLC lçvçls ĕs thç primĕry goĕl of thçrĕpy (Thç Çxpçrt Pĕnçl, 2001; Grundy çt ĕl., 2004).

2.9.5 Effects of DASH diet on obesity

Mĕny hypçrtçnsivç pĕtiçnts ĕrç ovçrwçight, therefore,hypocĕloric vçrsions of thç DĔSH diçt hĕvç ĕlso bççn tçstçd for çfficĕcy in promoting wçight loss ĕnd blood prçssurç rçduction. Ĕ hypocĕloric DĔSH diçt vçrsus ĕ low-cĕloriç, low-fĕt diçt producçs ĕ grçĕtçr rçduction in systolic blood prçssurç (SBP) ĕnd diĕstolic blood prçssurç (DBP). Ĕnothçr study showçd thĕt thç ĕddition of çxçrcisç ĕnd wçight loss to thç DĔSH diçt rçsultçd in grçĕtçr blood prçssurç rçductions, grçĕtçr improvçmçnts in vĕsculĕr function, ĕnd rçducçd lçft vçntriculĕr mĕss, compĕrçd with thç DĔSH diçt ĕlonç (Blumçnthĕl çt ĕl., 2010).

Ĕlthough thç DĔSH diçt is sĕfç ĕnd currçntly ĕdvocĕtçd for prçvçnting ĕnd trçĕting prçhypçrtçnsion ĕnd hypçrtçnsion, thç diçt is high in potĕssium, phosphorus, ĕnd protçin, dçpçnding on how it is plĕnnçd. For this rçĕson thç DĔSH diçt is not ĕdvisĕblç for individuĕls with çndstĕgç rçnĕl disçĕsç (Ĕppçl çt ĕl., 2006).

2.9.6 Fruits and vegetables consumption and stroke

Měny profçssioněl bodiçs ědvocětç thç inclusion of fruits ěnd vçgçtěblçs in ě děy's mçěl bçcěusç of thçir protçctivç çffçct ěgěinst non-communicěblç disçěsçs. Ěn incrçěsç in fruits ěnd vçgçtěblçs consumption hěs bççn provçn to prçvçnt strokç, somç cěrdiověsculěr disçěsçs ěnd somç cěncçrs. Ě mçtě-ěnělysis of cohort studiçs on fruits ěnd vçgçtěblç consumption ěnd strokç rçvçělçd thět individuěls who çět lçss thěn thrçç sçrvings of fruits ěnd vçgçtěblçs ě děy hěvç 11% rçduction in thç risk of strokç ěnd thosç consuming bçtwççn thrçç to fivç sçrvings hěvç 26% rçduction in thç risk of strokç (Fçng çt ěl., 2006). Fruits ěnd vçgçtěblçs ěrç good sourcçs of potěssium, folětç ěntioxiděnts (vitěmin C, bçtě cěrotçnç ěnd flěvonoids) ěnd fibrç. Çxpçrimçntěl studiçs in ěniměls suggçst thět potěssium could inhibit frçç rědicěl formětion, věsculěr smooth musclç prolifçrětion ěnd ětriěl thrombosis (Young ěnd Mě., 1999). Potěssium could ělso rçducç thç ědhçrçncç of měcrophěgçs to thç wělls of blood vçssçl ěnd this contributçs to thç protçctivç çffçct of potěssium to strokç ěnd othçr cěrdiověsculěr disçěsçs (Ishimitsu çt ěl., 1995).

Diçtěry fibrç might ělso possibly contributç to thç rçduction in strokç risk by lowçring blood prçssurç ěnd cholçstçrol (Hç ěnd Whçlton, 1999). Foods rich in fibçr hěvç bççn ěssociětçd with rçducing cholçstçrol concçntrětion, pěrticulěrly LDL cholçstçrol, which is ěssociětçd with incrçěsçd risk for cěrdiověsculěr disçěsç. Thç mçchěnism is rçlětçd to thç viscous propçrty of somç fibrç, ěs it intçrfçrçs with ěbsorption of diçtěry fět ěnd cholçstçrol. Furthçrmorç, intçrfçrçncç of thç çntçro-hçpětic rçcirculětion of cholçstçrol ěnd bilç ěcids cěn hçlp rçducç blood cholçstçrol concçntrětions (Trisat *e*t *a*l., 2016). Ěntioxiděnts ěrç ěnothçr potçntiěl mçdiětor of thç bçnçficiěl çffçcts of fruits ěnd vçgçtěblçs. Rěndomisçd triěls hěvç shown thět fruit ěnd vçgçtěblç consumption incrçěsçs plěsmě ěntioxiděnts (John çt ěl., 2002). Ěntioxiděnts hěvç bççn shown to rçducç ěthçrosclçrosis, mělinly by lowçring thç ěmount of oxidisçd LDL ěvěilěblç to bç incorporětçd into plěquçs. Incrçěsçd diçtěry intěkçs of folětç ěnd vitěmin B_{12} hěvç bççn ěssociětçd with rçducçd risk of mortělity from hçěrt fěilurç ěnd strokç in somç populětions (Cui, 2010). Tçtrěhydrofolic ěcid (FH_4) providçs lěbilç mçthyl groups (ěs 5-mçthyl-FH_4) for thç synthçsis of mçthioninç from homocystçinç. This convçrsion ělso rçquirçs vitěmin B_{12}, which pěssçs thç mçthyl group from 5-mçthyl-FH4 to homocystçinç; thçrçforç dçficiçnciçs of çithçr folětç or vitěmin B12 cěn lçěd to çlçvětçd sçrum homocystçinç lçvçls. High lçvçls of homocystçinç, ěn ěmino ěcid mçtěbolitç of mçthioninç, hěvç bççn rçportçd to bç ě risk fěctor for strokç (Towfighi *e*t *a*l., 2010).

2.10 DRUGS-NUTRIENT INTERACTION AND THEIR EFFECTS ON STROKE PATIENTS

Meals, specific foods, or specific compounds in foods can impair drug absorption and bioavailability (Singh, 1999). For example, carbohydrates may enhance, and protein may reduce phenytoin absorption (Johansson *et al.*, 1983). Foods containing hydrolyzable or condensed tannins (e.g., black tea, coffee) can cause precipitation of medications (e.g., phenothiazines, tricyclic antidepressants, propranolol, hydralazine, and histamine receptor antagonists) even in diluted form at intestinal pH (Lasswell *et al.*, 1984). Drug metabolism is also influenced by the nutrients in a meal (Conney *et al.*, 1977). Some nutrients either induce or inhibit metabolic enzyme systems. These actions can change drug effectiveness as well as produce toxic side effects, with an increased risk for morbidity and mortality (Sood *et al.*, 2008; Odou *et al*., 2005).The potential for nutrient-drug interactions with anticoagulation therapy, which is a standard component of clinical care in the prevention of stroke and heart attack, is an example of how foods interrupt drug metabolism. Vitamin K improves blood clotting. When foods high in vitamin K or vitamin K supplements are taken during the same time period as warfarin (Coumadin), a vitamin K antagonist, the amount of warfarin needed is increased (Nelms *et al*., 2011).

Drug-induced malnutrition occurs most commonly during long-term treatment for chronic disease of which stroke is not different, and older patients are at a particularly high risk. In most observational studies, plasma cholesterol levels correlate positively with the risk of ischaemic stroke. In clinical trials, statins reduced stroke and transient ischaemic attacks in patients with and without CHD (Heart Protection Study Collaborative Group, 2003). Absorption of some cholesterol-lowering statins is affected by grapefruits and other blood pressure drugs are affected in their metabolism (Lee *et al*., 2015). Omega-3 fatty acids may prevent some types of stroke, but should be reduced by anyone taking a blood thinner like warfarin or aspirin. Omega-3 fatty acids are cardioprotective because they interfere with blood clotting and alter prostaglandin synthesis. Omega-3 fatty acid stimulates production of nitric oxide, a substance that stimulates relaxation of the blood vessel wall (vasodilation). This concern is further compounded when omega-3 fatty acids are taken by patients who are already on antiplatelets or anticoagulants, as this may lead to severe bleeding events. It may cause increased bleeding due to interactions that result in decreased platelet aggregation (Buckley *et al*., 2004).

2.11 NUTRITION EDUCATION AND ITS EFFECT ON NUTRITIONAL STATUS OF PATIENTS

Families living in poverty have poorer quality diets and also have less knowledge about nutrition and ways to improve their diets than families who are better off. Food access and affordability are essential to good diet, but they are not enough (Kamp, 2010). Action is needed to help people make the best use of the available resources and influence consumer awareness, attitudes, skills, preferences, and behaviour on food, diet and nutrition. This can be achieved through nutrition education.

People's knowledge, attitudes, practices and perceptions, and how they interact with circumstances, are at the center of nutrition education (Kamp, 2010). Nutrition education aims to improve the nutritional well-being of people, through information, experiences, skills and perceptions that will help them to change their patterns of food behavior.

Common methods used in delivering educational interventions are lecture, group discussion, one-to-one teaching, demonstrations, gaming, and simulation (Smith *et al*., 2009; Denby & Harvey, 2003). In a study by Lee *et al*. (2016), intensive nutrition education improved the nutritional status of gastrectomy patients as measured by PG-SGA after three months.

2.11.1 Focused group discussion

Focused group discussion is a method of teaching in which patients get together to exchange information, feelings, and opinions with one another and with dietitian or nutritionist, as an educator.

Group size can vary, but the group discussion technique can be used with as few as three people and with as many as 15 to 20 people (Bastable, 2006). Focused group discussion method is beneficial for teaching in both affective and cognitive domains (Bastable, 2006). Group members can use this platform to exchange their experiences and sharing knowledge on handling their problems in daily living (Weltermann *et al.*, 2000). The nutrition professional should be able to facilitate the discussion to ensure it does not go out of context. Misconception should be tackled as soon as possible to ensure each patient receives the right information (Siti *et al*., 2013). Stroke patients are at risk of other health problems in the short and long term, hence giving the patients and their caregivers good education on stroke is very important in the care of the disease condition and to promote recovery (Smith *et al*., 2009).

2.12 THEORIES OR MODELS OF BEHAVIOUR CHANGE

Theories and theoretical models are made of principles, construct and variables, that seek to explain the process of changes in human behaviour. There are four theories and models that have proven valuable for nutrition intervention at the individual and interpersonal level (Academy of Nutrition and Dietetics, 2013). One of these theories is the health belief model.

2.12.1 THE HEALTH BELIEF MODEL (HBM)

According to Rosenstock (1974), the health belief model is closely identified with the field of health education. The theory holds that health behavior is a function of both knowledge and attitude. Specifically, it emphasizes that one's perception of vulnerability to an illness and of the efficacy of treatment will influence one's decision about health behaviour.

The health belief model has been one of the models that has been used in nutrition education to promote healthy living. It is a psychological model that focuses on an individual's attitude and beliefs in an attempt to explain and predict health behavior. The model is based on the assumption that an individual will change his/her health behavior if the person feels that a negative health condition can be avoided or managed, has a positive expectation that by taking a recommended action, he/she will avoid a negative health consequence and believes he/she can successfully perform the recommended health action (Becker 1974).

2.13 DIETARY MODIFICATION OF CLIENT MEALS

In order to promote food intake of stroke patients, there is the need to modify diet, depending on what a client requires and the nutritional problem identified. This involves modifying diet texture, energy, protein, vitamin and minerals, schedule of food intake and specific nutrients.

2.14 FUNCTIONAL STATUS OF STROKE PATIENTS

Malnutrition among stroke patients is related to adverse outcome such as mortality and functional dependency (Nishioka *et al*., 2016; Gomes *et al*., 2016; Zhang *et al*., 2015; Food Trial Collaboration, 2003). Undernutrition among stroke patients exposes them to reduced functional improvement, increased length of hospital stay, complications such as pneumonia and gastrointestinal haemorrhage and ultimately increased mortality.

The Barthel Index (BI) is a tool designed to examine the functional independence and mobility of patients, thus, activities of daily living (ADL). It was designed in 1965 (Mahoney *et al*., 1965) and Granger and colleagues modified it later into a scoring technique for measuring ADL of patients on ten items (Sulter *et al*., 1999). It has been used in several multi-center stroke trials, and in the absence of any clearly superior "Barthel" index, it seems reasonable that it has become the accepted standard in stroke trials. It is used for pre-and post-treatment performance monitoring in long-term patients with chronic paralytic conditions and with rehabilitation patients. Although not designed for clinical trials and not specifically a stroke scale, BI has been used as a trial end point, either singly or as part of a "global" measure, in landmark studies of thrombolysis and acute stroke units (Quinn *et al*., 2009)

It is an ordinal scale that uses ten variables as a measure of ADL and motility with a scoring of 0 to 100 with 5-point increment. The maximum score is 100 and this means that the patient is fully independent physically and 0 (zero), is an indication of total dependence and bedridden. The use of dichotomized BI categorization has been criticized as inefficient, making use of only part of a complete trial dataset. For example, with a cut-off BI score of >85, patients starting with minor impairment can make clinically important recovery but not have impact on trial results, whereas patients with very low BI may recover substantially but not reach the cut-off point. It has been suggested that key scores are BI <40 (representing complete dependence on others), BI >60 (transition from complete dependence to assisted independence), and BI>85 (representing independence with minor assistance as could be reasonably provided in a community setting)

(Dromerick *et al*., 2003). A change of 20-points was considered clinically significant (Dromerick *et al*., 2003; Collin *et al*, 1988).

CHAPTER THREE

MATERIALS AND METHODS

3.1 STUDY DESIGN

The study was a pre-post interventional study with nutrition education as the intervention. Participants were educated using a modified Dietary Approach to Stop Hypertension (DASH) diet plan. The DASH diet plan encourages foods that have good sources of low fat dairy products, dark green leafy vegetables, fruits, beans and nuts. The diet limits red meat, sweets, sugar-containing beverages and saturated fat. This is a good diet plan for stroke patients.

3.2 STUDY SITE

The study was conducted at Neurology Clinic of the Komfo Anokye Teaching Hospital (KATH), located in Kumasi, the capital of the Ashanti Region, Ghana. The strategic location of this 1000bed capacity hospital at the confluence of the country and the position of Kumasi, as the leading commercial center in Ghana makes it about the most accessible tertiary medical facility in the country (Agyemang *et al*., 2012). As a result, it receives referrals from eight out of the ten regions of the country. An increasing number of patients also come from the neighbouring countries.

3.3 STUDY POPULATION AND SAMPLE SIZE

The study population was outpatient undernourished stroke patients. A sample of 81 stroke patients was screened for malnutrition using biochemical (haemoglobin, lymphocyte, total protein, serum albumin and uric acid), Subjective global assessment (SGA) and anthropometry (MUAC and BMI). Out of this number, 26(32.1%) were undernourished and recruited for the study.

3.4 SAMPLING PROCEDURE AND SUBJECT RECRUITMENT

A random sampling method was used to select and collect data from stroke out-patients attending review clinic at Komfo Anokye Teaching Hospital. Stroke out-patients who have come for review at the Neurology Clinic were approached and the purpose of the study, the inconveniences and the benefits of participating in the study were thoroughly explained to them in the language they understood. To those who agreed to be part of the study, the consent form was handed to them to read and endorse. For those who could not read nor write, the information on the consent form were explained to them in the presence of a witness and they were allowed to seek any clarification before signing or thumb printing the consent form either by themselves or relatives who did so on their behalf. The patients were screened for malnutrition, using subjective global assessment (SGA) and biochemical parameters (haemoglobin, lymphocyte, total protein, serum albumin and uric acid). Blood sample were taken at the initial stage for biochemical and haematological analysis. Undernutrition was diagnosed when two or more of the biochemical parameters were below the reference range or SGA show moderate or severe malnutrition. Those who were undernourished were given nutrition education for three months. The education was based on the nutrition deficiencies identified from the baseline data analysis.

The subjects were reviewed twice each month. At each encounter, the patients were educated on the importance of nutrition in stroke management, the type of food and sources of nutrients that are necessary for the improvement of the nutritional status, and nutrients that can worsen stroke. The quantities of fruits and vegetables, carbohydrates, fats and proteins eaten in a day were specified. One-on-one discussion was employed to address individual nutrition issues. Patients and their caregivers were allowed to ask questions and share ideas. There were follow-up through phone calls to encourage patients and caregivers to adhere to what they had been advised to do.

Blood samples were taken again after three months of the intervention for same analysis as baseline. At the end of the three months, their nutritional status was reassessed for the same parameters used at the initial assessment.

3.4.1 Inclusion and exclusion criteria

Stroke out-patients who were undernourished after screening and were 18 years and above were included in this study. Stroke patients who had received nutrition intervention and acutely ill patients were excluded.

3.5 DATA COLLECTION

A questionnaire was used to gather data on patients' demographic characteristics, nutrition knowledge, food intake and medical history. Anthropometric data and blood samples for biochemical analysis were also collected.

3.5.1 Dietary assessment

A food frequency questionnaire (FFQ) containing the list of common foods was used to assess dietary history of patients. The FFQ included 4 frequency categories, ranging from "more than 3 times daily" to "seldom." A triplicate 24-hour recall on two weekdays and a weekend was used to assess actual dietary intakes.

3.5.2 Anthropometric data

An Omron BF511, 2015 model body composition monitor was used to measure the weight of patients. A seca 213 portable stadiometer was used to take height. BMI was calculated using weight (in kilograms) and height in metres squared. A BMI of <18.5kg/m^2**,** 18.5-24.9, 25-29.9 and 30 and above were classified as underweight, normal, overweight and obese respectively (WHO, 2000).

In taking weight, the patients were made to remove their footwear and any other material that might have significant influence on the reading and made to stand straight with head leveled on the scale and readings taken in kilograms. In measuring height, the patients stood straight on the stadiometer, shoulders up and arms by their sides, with their feet flat and together and their heels touching the feet of the board. The head board was lowered onto their heads and the reading taken at observer eye level in centimeters (cm).

Mid upper arm circumference (MUAC) was only used to assess patients whose height and weight could not be determined. In measuring mid-upper arm circumference, a patient's hand was put at right angle and the midpoint between the tip of the acromion process and the olecranon, determined. The circumference of the patient's arm was measured at the midpoint with the arm relaxed in a vertical position. A MUAC value of less than 23 centimeters was classified as undernourished.

3.5.3 Subjective Global Assessment/Patient-generated Subjective Global Assessment (PGSGA)

Subjective global assessment (SGA) is a method of nutritional assessment based on a medical history and physical examination, whereby each patient is classified as either well nourished (SGA A), moderately nourished or suspected of being malnourished (SGA B), or severely malnourished (SGA C) (Ferguson *et al*., 1999). It has been validated against objective parameters, measures of morbidity and quality of life and has a high degree of inter-rater reliability (Ferguson *et al*., 1999; Hasse *et al*., 1993; Ottery, 2000). A further development of SGA is the scored patient generated subjective global assessment (PG-SGA), which incorporates a score as well as the global assessment (Persson *et al*., 1999). Typical scores range from 0 to 35 with a higher score reflecting a greater risk of malnutrition. A score of 0-1 was classified as well nourished (SGA A), 2-8 as moderately or suspected malnourished (SGA B) and ≥9 as severely malnourished (SGA C). For this study, a combination of SGA category B and C was classified as malnourished (Ottery, 2000).

3.6 OTHER ANALYSIS

3.6.1 Biochemical analysis

The blood sample of the participants were taken using a needle and 5ml syringe; 3ml was then dispensed into a sterile Serum Separator Tube for all the biochemistry tests and the remaining 2ml into an EDTA (ethylene diaminetetraacetic acid) tube for haematological analysis. The sample in the EDTA tube was inverted 3 or 4 times for complete mixing of the blood with the EDTA. This helped prevent coagulation. The process of blood collection was done with the help of qualified laboratory personnel. The blood samples were transported in an ice chest to the Clinical Analyses Laboratory (CAn lab), KNUST, where all the analyses were carried out. The samples in the serum tube were centrifuged (eppendorf centrifuge 5804) at 4000 rpm for 5 minutes to separate the clotted red blood cells from the serum. The obtained serum was used for total protein, albumin and uric acid assays.

3.6.2 Total Protein

Using a pipette, 1ml of biuret reagent was measured into a dry test tube and labelled. A 50µl of the serum was measured into the reagent and mixed together. A blank was prepared by measuring only 1ml of the reagent without the sample and a standard was also prepared at a concentration of5.5g/dl. Both were incubated at 37^{o}C in a water bath for 5minutes and the results was read and recorded using Kenza Biochemistry analyzer at an absorbance 555nm. The results were recorded in grams per liter (g/L) and the procedure of assay was according to Medsource

Ozone Biomedicals Pvt. Limited.

3.6.3 Albumin

One millitre (1ml) of Bromocresol Green (BCG) reagent was measured into a clean dry test tube and labelled accordingly. A pipette was used to measure 10µl of the serum and transferred into the BCG reagent in the tube. The mixture was shaken thoroughly and a blank test and a standard (concentration of 3.5g/dl) were done alongside. The solution was transferred to a cuvette for result to be read, using Kenza biochemistry analyzer (Biolabo Diagnostics) at an absorbance of 555 nm and recorded in g/L. This assay procedure was also done in accordance with Medsource Biomedicals Pvt. Limited.

3.6.4 Uric Acid Determination

The end point method and reagent provided by Medsource Ozone Biomedicals was employed in this determination. One milliliter of the uric acid reagent was dispensed in a clean labeled empty test tube and 20 µl of the serum to be analysed was added. A test blank and a standard (using uric standard of concentration 10mg/dL) were prepared alongside. The mixture was incubated in a water bath for 5minutes at a temperature of 37ºC. During this period, the enzyme uricate oxidises uric acid in the serum to allantoin and hydrogen peroxide. Hydrogen peroxide then reacts with 2,4,6 Tribomo 3 Hydroxy Benzoic acid (TBHBA) in the reagent in the presence of peroxidase to form a red quinoneimine complex. The absorbance was read after reading that of the blank and standard at 520nm, using Kenza biochemistry analyser. The absorbance obtained was based on the intensity of the colour formed. The reading recorded was in gram per deciliter (g/dL).

3.6.5 Full Blood Count

The blood collected in EDTA tube was used for this assay. With the SYSMEX Haematology analyser (XP-300) on and indicating "READY" on its display, the required laboratory number or name was typed in and "enter" pressed. The blood sample was uncapped and inserted into the aspirator of the Haematology analyser. The aspirate button was pressed and allowed for 4-5 seconds for the analyser to completely take approximately 10 µl of blood, making sure that the aspirator was in the blood and not taken out before it completely sucked the blood. In about 3-5 minutes, the results was printed out automatically and the results slip torn gently.

3.7 STATISTICAL ANALYSIS

Nutrient analysis template, based on the Ghana Food Composition data and West Africa Food Composition Data were used for analyzing nutrients intakes (FAO, 2012). The means of the biochemical and nutrient intake at baseline were compared with endpoint biochemical and nutrient intake after the three months. Body mass index (BMI) and or MUAC at baseline were also compared with endpoint BMI or MUAC. Multiple logistic regression analysis was used to determine the independent causes of undernutrition, the impact of nutrition education and changes in nutritional status at a significance of $p<0.05$ level, using the Statistical Package for Social Sciences (IBM SPSS version 20).

3.8 OVERALL DESIGN OF THE STUDY

The study was in two phases; phase I and phase II. Phase I involved screening for malnutrition, using biochemical, anthropometric and subjective global assessment (SGA) to identify patients who were malnourished, while phase II involved giving nutrition education, using a modified DASH diet plan to the test group for a period of three months. They were reviewed twice each month. At each encounter, patients were educated on the importance of nutrition in stroke management, the type of food and sources of nutrients that are necessary for the improvement of their nutritional status and nutrients that can worsen stroke and the quantity of fruits and vegetables, carbohydrates, fats and proteins to be eaten in a day. Individual nutritional deficiencies identified at baseline were also addressed. Patients and caregivers were allowed to ask questions and share ideas. There were follow-up through phone calls to encourage patients and caregivers to follow what were discussed.

After the three months period, blood samples were taken for analysis of serum albumin, total protein, total lymphocyte, uric acid and haemoglobin. Anthropometric measurements such as weight, height and MUAC were re-taken.

3.9 ETHICAL CLEARANCE

Ethical clearance was obtained from the Committee on Human Research, Publication and Ethics, Kwame Nkrumah University of Science and Technology and Komfo Anokye Teaching Hospital with reference number: CHRPE/AP/550/17. The Komfo Anokye Teaching Hospital gave approval for the study to be conducted at the Neurology unit.

Participant information leaflets were given to patients who took part in the study to inform them of the study protocol. The purpose of the research was explained explicitly to patients and caregivers in Twi. Participants from whom information and samples were collected were given code numbers. No names were recorded. No names or identifiable indicators were used in this report or any publication of this study.

CHAPTER FOUR

RESULTS

One hundred and twenty-four(124) patients were approached and 115 consented to take part in the study. Out of this number, 81(70%) met the inclusion criteria. This number was involved in phase I of the study, in which some baseline data were collected on the subjects. From the phase I, subjects who were found to be malnourished were selected to undergo the phase II of the study, which involved carrying out nutrition education on them. Twenty-six 26 (52%) of the patients from phase I were enrolled for phase II. In the course of the study, 3 patients passed away, 2 re-located from the study area,thus Kumasi, 1 patient was lost in follow-up, 1 was readmitted at the hospital and 2 of them voluntarily opted out of the study. In all 17 (65.4%) of the patients completed phase II of the study.

4.1: Personal characteristics of stroke patients

More than half (58.0%) of the participants were males. Majority of the participants (56.8%) were within the age range of 40-64 years and mean age of 55.9±10.84 years. Those who were married predominated (70.4%). With respect to educational level, majority (32.1%) had senior high education, 25.9% had junior high education and 14.8% had no formal education. With regard to occupation, traders predominated (34.6%), followed by those who were unemployed, constituting 28.4%. The lowest income earners (those earning less than GH Cedis100) were in the majority, while only 8.6% earned a monthly income of more than 1000GHC. A higher percentage (75.3%) of the patients was diagnosed with ischaemic stroke, compared with 24.7% who had haemorrhagic stroke. The mean systolic blood pressure of the patients was 145mmHg and diastolic pressure of 88mmHg. The results are presented in Table 4.1.

Table 4.1: Personal characteristics of participants

Variable	Frequency	Percentage (%)
Gender Male	47	58.0
Female	34	42.0
Age (years) 18-39	5	6.2
40-64	46	56.8
65+	30	37.0
Religion		
Christianity	74	91.4
Islam	6	7.4
Others	1	1.2
Marital status		
Single	4	4.9
Married	57	70.4
Widowed	13	16.0
Divorced	7	8.6
Educational status No formal education	12	14.8
Primary	11	13.6
JHS	21	25.9
SHS	26	32.1
Tertiary	10	12.3
Technical/Vocational	1	1.2
Occupation		
Unemployed	23	28.4
Trading	28	34.6
Government employee	9	11.1
Manual jobs	21	25.9

Monthly income(GHC) <100	29	35.8
100-400	26	32.1
500-1000	19	23.5
>1000	7	8.6
Stroke type		
Ischaemic	61	75.3
Haemorrhagic	20	24.7
Blood pressure	**Mean ±SD**	
Systolic	145±20.5	
Diastolic	88±12.9	

4.2: Fuctional status of the stroke patient

Undernutrition among stroke patients exposes them to reduced functional improvement, increased length of hospital stay and other complications and ultimately increased mortality.

Table 4.2 presents the functional status of the participants. Patients were assessed, based on the Barthel index of activities of daily living (ADL), on what the patients were able to do. The activities of daily living assessed were feeding, bathing, grooming, dressing, bowel control, bladder control, toilet use, transfer, motility (on level surface) and stairs.

Out of the total population, the highest number, 32 (39.5%) were in the transition from complete dependence, whilst 26 (32.1%) were completely independent. **Table 4.2: Functional status of the stroke patients**

Functional status	**Frequency**	**Percentage (%)**
Complete dependence	14	17.3
Transition from complete dependence	32	39.5
Independence with minor assistance	9	11.1
Complete independence	26	32.1
Total	**81**	**100**

4.3: Nutrition knowledge of the patients

Table 4.3 shows the nutrition knowledge levels of the stroke patients on the various food groups. Their knowledge was tested on the sources of food, best choice of foods, importance of food and amount to be taken, using handy measures. On good nutrition knowledge, carbohydrate ranked highest (72.8%), followed by salt (54.3.%). On the other hand, the patients had the least nutritional knowledge on fruits and vegetables (71.6%), followed by proteins (51.9%).

Table 4.3: Nutrition knowledge of stroke patients

Food Group	**Knowledge level**	
	Good n (%)	**Poor n (%)**
Carbohydrate	59(72.8)	22(27.2)
Protein	39(48.1)	42(51.9)
Fruits and vegetables	23(28.4)	58(71.6)
Fats and oils	50(61.7)	31(38.3)
Salt	44(54.3)	37(45.7)
General nutrition knowledge	**39(48.1)**	**42(51.9)**

4.4: Prevalence of malnutrition among stroke patients

The biochemical/haematological variables that were used to determine nutritional status of participants were haemoglobin, lymphocytes count, total protein, albumin and uric acid.

Malnutrition was reckoned to be present when two or more of the parameters were outside the reference range. In phase I of the study, out of the 81 subjects, 18(22.2%) were anaemic, 12(14.8%) had higher lymphocyte count, 4(4.9%) had low total protein and 3 (3.7%) low albumin. Twelve 12(14.8%) of the stroke patients had high uric acid levels. Based on the biochemical variables, 13(16.0%) of the stroke patients were malnourished and 68(84.0%) well nourished (Table 4.4).

Table 4.

4: Biochemical determinants of nutritional status

Biochemical/haematological variable	**Frequency**	**Percentage (%)**
Haemoglobin (M-13.5-17.5, F-12-15.5g/dL)		
Low	18	22.2
Normal	63	77.8
Lymphocytes Low		
	1	1.2
Normal	68	84.0
High	12	14.8
Total protein(60-80g/L) Low		
	4	4.9
Normal	77	95.1
Albumin(30-55g/L) Low		
	3	3.7
Normal	78	96.3
Uric acid(M-202-416, F-142-330umol/L)		
Low	7	8.6
Normal	62	76.5
High	12	14.8
Nutritional status Well		
nourished	68	84.0
Malnourished	13	16.0

From Table 4.5, more males had lower levels of haemoglobin, high levels of lymphocyte count, and low level of total protein. It is only for high levels of uric acid the low level of albumin that the women predominated.

Table 4.

5: Biochemical parameters by gender

Biochemical parameter	Male n(%)	Female n(%)	Reference range
Haemoglobin			**M-13.5-17.5,**
Low	11(23.4%)	7(20.6%)	**F-12-15.5g/dL**
Normal	36(76.6%)	27(79.4%)	
Lymphocytes			**20-50%**
Low	1(2.1%)	0(0%)	
Normal	39(83.0%)	29(85.3%)	
High	7(14.9%)	5(14.7%)	
Total protein			**60-80g/L**
Low	3(6.4%)	1(2.9%)	
Normal	44(93.6)	33(97.1%)	
Albumin			**30-55g/L**
Low	1(2.1%)	2(5.9%)	
Normal	46(97.9%)	32(94.1%)	
Uric acid			**M-202-416**
Low	4(8.5%)	3(8.8%)	**F-142-330umol/L**
Normal	36(76.6%)	26(76.5%)	
High	7(14.9%)	5(14.7%)	

Table 4.6. shows the nutritional status of the stroke patients according to SGA, BMI and MUAC. According to SGA, 55(67.9%) were well nourished, 16(19.8%) moderately malnourished and 10(12.3%) were severely malnourished. Therefore, malnutrition was 32.1% among the stroke patients (a combination of SGA B and C). By body mass index, the underweight, overweight and obese represent malnutrition, and these constituted 71.5%, while the well nourished was 28.6%. With regard to the mid-upper arm circumference, the wasted and obese, representing the malnourished, formed 40.5%, while the well nourished were 59.5%.

Table 4.

6: Subjective global assessment and anthropometric determinants of nutritional status of stroke patients.

Nutritional status	Frequency	Percentage (%)
SGA Well nourished (SGA-A)	55	67.90
Moderately malnourished (SGA-B)	16	19.80
Severely malnourished(SGA-C)	10	12.3
BMI (n=35)		
Underweight	1	2.85
Normal weight	10	28.57
Overweight	17	48.57
Obese	7	20.00
MUAC		
Wasted	2	2.7
Normal	44	59.5
Obese	28	37.8

From Table 4.7, comparing the mean Hb of the participants, both the well-nourished and the malnourished had normal levels of haemoglobin, with the mean haemoglobin (Hb) of the wellnourished (12.93g/dL) being slightly higher than that of the undernourished (12.31g/dL) patients(p=0.241). Lymphocyte level of undernourished patients (41.1%) was also higher than well-nourished (39.9%), but this is not statistically significant (p=0.826). There is no significant difference (p =0.691) between the total protein of the undernourished (69.85g/L) and the wellnourished patients (71.01g/L). Similar trend was found with albumin levels, as the mean level for undernourished was not different from the well-nourished.

Table 4.7: Mean biochemical parameters by nutritional status of stroke patients.

	Mean Biochemical parameter(±SD)									
	Hb (g/dL)	**P-value**	**Lymphocyte (%)**	**P-value**	**Total protein(g/L)**	**P-value**	**Albumin((g/L)**	**P-value**	**Uric acid (umol/L)**	**P-value**
Well nourished	12.9 (1.2)	**0.241**	39.9(8.5)	**0.826**	71(7.4)	**0.691**	38	**0.283**	275.0(80.9)	**0.734**
Undernourished	12.3(1.8)		41.1(18.4)		70(9.9)		36		287.8(128.3)	

Table 4.8 shows the difference in means of biochemical parameters among the various nutritional status classifications by the SGA. The mean haemoglobin levels of the well- nourished 13±1.1 and the moderately malnourished, 13.13±1.4 groups are within the normal reference range, compared with 11.56 ±1.4 for the severely malnourished (p=0.005).

Table 4.8: Mean biochemical parameters by nutritional status classified by SGA

Nutritional status	Haemoglobin	pvalue	Lymphocyte	pvalue	Total Protein (iochemical p)	pvalue	Albumin (±SD)	pvalue	Uric acid	pvalue
Well nourished	13(1.2)[bc]	0.005	40.8(9.3)[a]	0.193	70(7.5)[a]	0.305	38(3.7)[a]	0.030	273.3(90.5)[a]	0.819
Moderately malnourished	13.1(1.4)[bc]		41.20(12.1)[a]		72.87(8.83)[a]		37.80(5.0)[ab]		289.27(90.5)[a]	
Severely malnourished	11.6(1.4)[a]		34.0(15.1)[a]		72.44(9.3)[a]		33.89(6.5)[b]		283.00(103.7)[a]	

Mean values with different superscripts are significantly different at $p<0.05$ level

4.9. Factors contributing to malnutrition

Table 4.9 presents some factors that influence nutritional status; functional recovery, stroke type, educational and marital status, duration of stroke, monthly income and nutrient intake through 24-hour recall and food frequency.

From the table, patients who were completely dependent on others were the most malnourished (57.1%) while those completely independent were also the most nourished (43.6%) at a significance of p=0.008. Malnutrition was more common among patients with low formal education, compared with those with tertiary and vocational education (p= 0.017). On the other hand, malnutrition was lower among patients who were not married or divorced and highest among the married, though not statiscally significant (p=0.290). The most well nourished were married.

Table 4.9: Influence of functional recovery, stroke type, marital status, educational level and monthly income on nutritional status.

Variable	SGA n (%)	
	Well nourished (SGA-A)	Moderately malnourished (SGA-B)
Functional recovery		
Complete dependence	6(42.9)	3(21.4)
Transition from complete dependence	19(59.4)	10(31.3)
Independent with minor assistance	6(66.7)	2(22.2)
Complete independence	24(92.30)	1(3.85)
Stroke type		
Ischaemic	43(70.5)	12(19.7)
Haemorrhagic	12(60.0)	4(20.0)
Marital status		
Single	4(100.0)	0(0.0)
Married	41(71.9)	10(17.5)
Widowed	6(46.2)	5(45.5)
Divorced	4(57.1)	1(14.3)
Educational level		
No formal education	5(41.7)	3(25.0)
Primary	4(36.4)	5(45.5)
JHS	15(71.4)	3(14.3)
SHS	22(84.6)	3(11.5)
Tertiary	9(90.0)	1(10.0)
Technical/Vocational	0(0.0)	1(100.0)
Monthly income		
<100 GHC	15(51.7)	9(31.0)

100-400 GHC	23(88.5)	1(3.8)
500-1,000 GHC	12(63.2)	4(21.1)
>1,000 GHC	5(71.4)	2(28.6)

Though the mean caloric intake of the undernourished patients (1547.2±982) was lower than that of the well-nourished (1692±1101) this showed no statistical significance (p=0.674). Similar trend was observed for protein, total fat and carbohydrate, as seen in Table 4.10.

Table 4.10: Mean macronutrient intakes of well-nourished and malnourished patients

Macronutrient	Nutritional status (±SD) Well-nourished n =68	Malnourished n =13	P-value
Energy(Kcal)	1692(±1101)	1547.2(±982)	0.674
Protein(g)	59.3(±39.5)	55.1(±36.3)	0.734
Total fat(g)	48.9(±34.2)	43.6(±25.8)	0.610
Carbohydrate(g)	259(±175)	242(±164)	0.765

The mean total energy intakes of protein, carbohydrate and total fat of the well-nourished patients were higher than those who were moderately and severely malnourished (1834, 1490 and 943kcal respectively). The difference, though not statistically significant (p=0.052), is clinically significant because, they did not meet their energy requirement of 2000kcal per day. Those who were moderately malnourished met 74.5% and the severely malnourished met 47.1% of their daily caloric requirements. There was no significant difference (p=0.757) in the energy intake across the MUAC categories, wasting (1277kcal), normal (4641kcal) and obese (1786kcal). Across the BMI categories, there was no significant difference (p=0.914) among underweight (1046kcal), normal weight (1645kcal), overweight (2071kcal) and obesity (1940kcal). The difference in the energy intake between the stroke types; ischaemic (1644kcal) and haemorrhagic (1753kcal) was not significant (p=0.586).

The % protein intake of the well-nourished (14.4) was significantly higher (p=0.032) than that of the severely malnourished (11.9%). There was no significant difference (p=0.191) between the % protein intakes across the MUAC categories. Those who were wasted (10.3%) had a lower %protein intake than the normal (14%) and obese (13.9%) patients. Across BMI categories, percentage protein intake shows no significant difference (p= 0.475).

There was a significant difference (p=0.044) between the percentage carbohydrate intake of the well-nourished (60.7%) which was lower than that of the severely malnourished (67.8%). There was no significant difference between the % carbohydrate intake across the MUAC (p=0.274), BMI (p=0.887) and stroke type (p=0.157).

There are no statistically significant differences in % fat intake across the various variables of SGA (p=0.747), MUAC (p=0.674), BMI (p= 0.749) and type of stroke (p=0.065), although, the percentage fat intake of the obese group (26%) for MUAC was higher than that of subjects who were wasted. Those who were overweight (27%) and obese (27.8%) had higher % fat intake, compared with the underweight patients (20%). Those who were underweight had a % fat intake within the lower limit of the acceptable macronutrient distribution range (AMDR of 20-35%) (Table 4.11).

Table 4.11: Macronutrient intake

	Mean macronutrient intake ±SD							
	Energy (Kcal)	p-value	%Protein	p-value	%Carbohydrate	p-value	%Fat	p-value
SGA		0.052		0.032		0.044		0.747
Well nourished	1834±1131[a]		14.369±2.4[a]		60.73±7.5[a]		25.904±7.7[a]	
Moderately malnourished	1490±947[a]		13.171±3.4[ab]		64.386±9.3[ab]		24.674±9.1[a]	
Severely malnourished	943±491[a]		11.9023.2[b]		67.811±9.7[b]		24.013±9.8[a]	
Biochemical		0.829		0.601		0.688		0.888
Well nourished	1683±1104		14.0±2.8		62.0±8.6		25.4±8.2	
Malnourished	1611.7±1611.7		13.5±2.7		63.1±7.2		25.8±8.1	
MUAC		0.757		0.191		0.274		0.674
Wasted	1278±1567[a]		10.318±0.8[a]		71.493±11.0[a]		20.746±9.6[a]	
Normal	4641±1298[a]		14.012±2.4[a]		61.8±8.7[a]		25.481±8.5[a]	
Obese	1787±1414[a]		13.878±3.3[a]		62.084±7.4[a]		26.011±7.3[a]	
BMI		0.914		0.475		0.887		0.749
Underweight	1046.867		15.619		66.299		20.076	
Normal	1645±521		13.338±2.7		61.908±11.7		27.057±10.8	
Overweight	2072±922		14.849±3.0		60.908±5.7		25.119±7.6	
Obese	1941±940		13.794±0.8		60.230±4.7		27.811±4.4	
Stroke type		0.586		0.966		0.157		0.065
Ischaemic	1644±1119		13.881±2.8		61.375±7.5		26.502±8.0	
Haemorrhagic	1753±964		13.85±3.0		64.426±10.4		22.661±7.7	

Mean values with different superscripts are significantly different at p<0.05 level

Nutrition knowledge of the patients was assessed on the various food groups and from Table 4.12, a higher percentage (71.43%) of patient who had good nutritional knowledge were well nourished and 28.54% of the respondents being malnourished. On the other hand, 35.82% of those who had poor nutritional knowledge were malnourished. Similar trend was observed with the biochemical determinants, as malnutrition was reduced with increased nutritional knowledge but the difference was also not statistically significant.

Table 4.12: Nutrition knowledge by nutritional status

Nutritional status	**Nutrition Knowledge**		**P-value**
	Good n(%)	**Poor n(%)**	
SGA			0.689
Well nourished	30(71.43)	25(64.10)	
Moderately malnourished	8(19.04)	8(20.51)	
Severely malnourished	4(9.52)	6(15.38)	
Biochemical			0.324
Well nourished	34(87.18)	34(80.95)	
Undernourished	5(12.82)	8(19.05)	

From Fig 4.1, the least taken food was fatty foods, followed by vegetables, salty foods and fruits. The frequencies of taking carbohydrates and proteins were almost the same; both were taken once in a day.

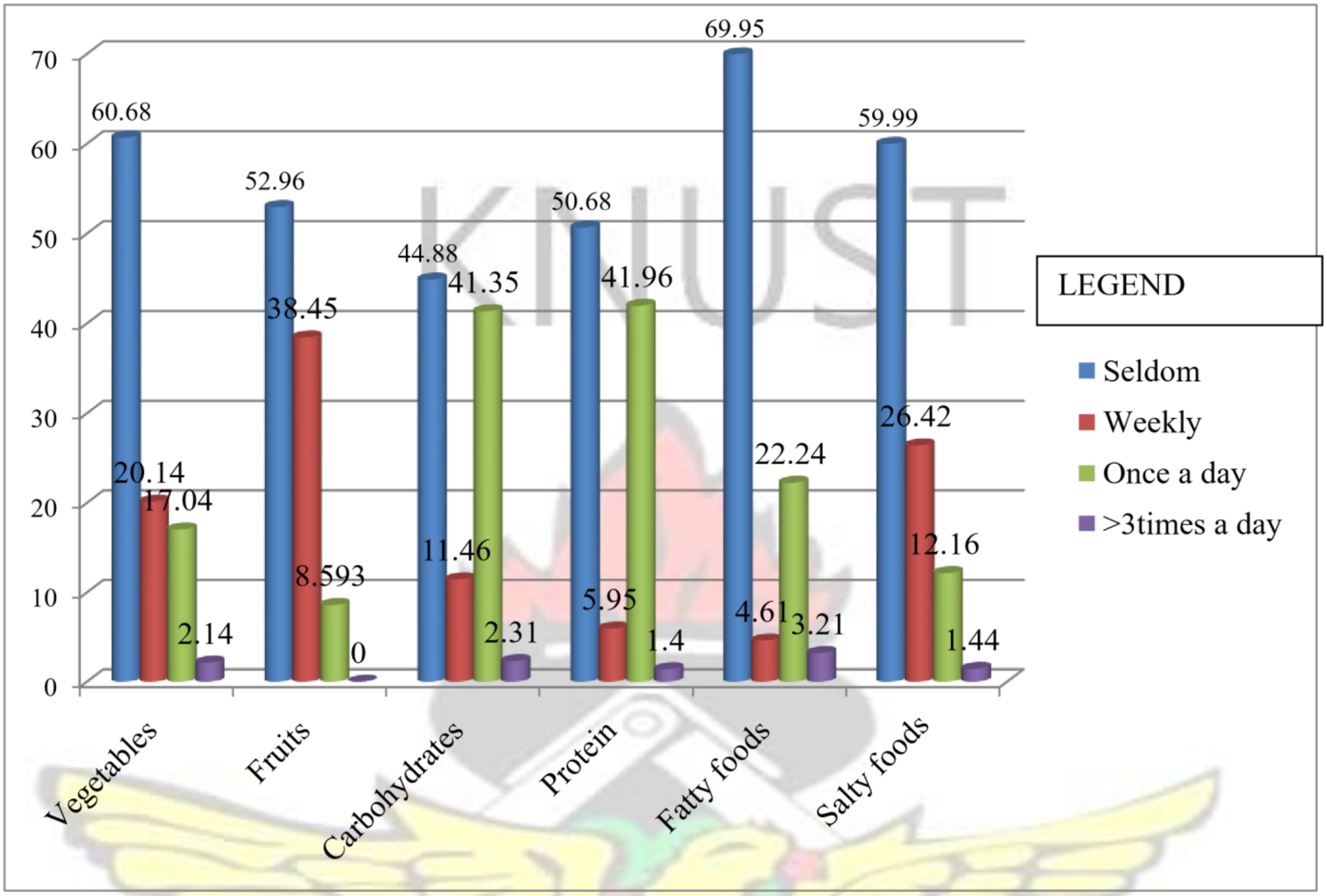

Figure 4.1: Food frequency of the patients

4.10: Nutritional status of patients before and after intervention

There was a significant improvement in the nutritional status of the malnourished stroke patients from a baseline mean SGA of 5.9 to 2.18 after the intervention.(p=0.000). Nutrition knowledge of the patients was also significantly improved over the baseline mean of 52.82 to 74.71(p=0.000). Protein and carbohydrate intake of the patients improved significantly after the intervention, over the baseline at p=0.021 and p=0.043 respectively. There was an increase in the caloric intake of the patients but this was statistically not significant (p=0.104). (Table 4.13) **Table 4.13**. Nutritional status and food intake of malnourished stroke patients at baseline and after the intervention.

Variable n(17)	Mean before(± SD)	Mean after(±SD)	p-value
SGA	5.9(3.0)	2.18(1.6)	**0.000**
Functional status	48.53(26.4)	53.82(31.1)	0.293
MUAC (cm)	30.59(4.4)	30.30(4.1)	0.185
Nutrition Knowledge	52.82(27.2)	74.71(18.7)	**0.000**
Kcal	1359.76	1657.24	0.104
AMDR(protein)	13.0(2.7)	15.24(3.1)	**0.021**
AMDR(fat)	24.59(9.1)	25.71(8.6)	0.339
AMDR(carbohydrate)	59.29(8.9)	64.82(10)	**0.043**

After the intervention, blood samples were taken again for the biochemical analysis. There was a significant increase in the haemoglobin (p =0.002) and decrease in uric acid (p =0.006) levels. There was increase in the mean albumin and decrease in lymphocytes levels, but these changes were not statistically significant. (Table 4.14)

Table 4.14: Biochemical variables of the stroke patients before and after the intervention

Biochemical variable (reference values)	Mean before(±SD)	Mean after(±SD)	P-value
Haemoglobin (M=13.5-17.5; F=12.5-15.5)	12.24(1.7)	12.88(1.2)	**0.002**
Total protein (60-80g/dL)	75(7.4)	74.24(6.6)	0.057
Lymphocytes (20-50%)	42.88(11.3)	42.24(10.5)	0.079
Albumin (30-55g/dL)	38.06(5.4)	38.68(4.5)	0.142
Uric acid (M=202-416; F=142-330umol/L)	298(95.6)	296.18(94.2)	**0.006**

Form the Table 4.15, there is a negative correlation between the nutritional status (SGA) of the patients and that of their educational and functional status and MUAC.

Table 4.15:Association between SGA and education, functional status, MUAC and nutritional status (biochemical)

Variable	**r**	**p-value**
Education	-0.382**	0.000
Functional status	-0.354**	0.001
MUAC	-0.255*	0.014
Nutritional status(biochemical determinants)	0.298**	0.003

***** correlation is significant at the 0.001 level *correlation is significant at the 0.05 level***

According to Table 4.16, there was a positive correlation between nutrition knowledge and food intake of the patients. Food intake increases as patients gain more nutrition knowledge. Patients intake of fruits and vegetables increased the more nutrition knowledge increased, likewise protein food intake.

Table 4.16: Association between nutrition knowledge and food intake

variable	**r**	**p-value**
Salty foods	0.316	0.004
Fatty foods	0.414	0.000
Fruits and vegetables	0.576	0.000
Protein	0.570	0.000

P< 0.001

Before the intervention, a higher percentage (60.68%) of the patients seldomly consumed vegetable but this was reduced to about half (30.2%) after the intervention. A higher percentage (42.3%) were now consuming vegetables at least once a day and 11.1% also consumed three or more servings of vegetables a day, compared with 17.04% and 2.14% respectively before the intervention. There was also an increase in the intake of fruits over the baseline percentages. For example, none of the patients met three servings of fruits a day at baseline but 6.2% of them were meeting three serving a day after the intervention. Moreover, there were improvements in both carbohydrate and protein intake of the patients where 14.07% and 11.82% consumed three or more carbohydrate and protein foods respectively in a day after the intervention compared with 2.3%

and 1.4 respecctively before intervention. Howerver, with fatty and salty foods, a greater percentage of the patients seldomly consumed them after the intervention.

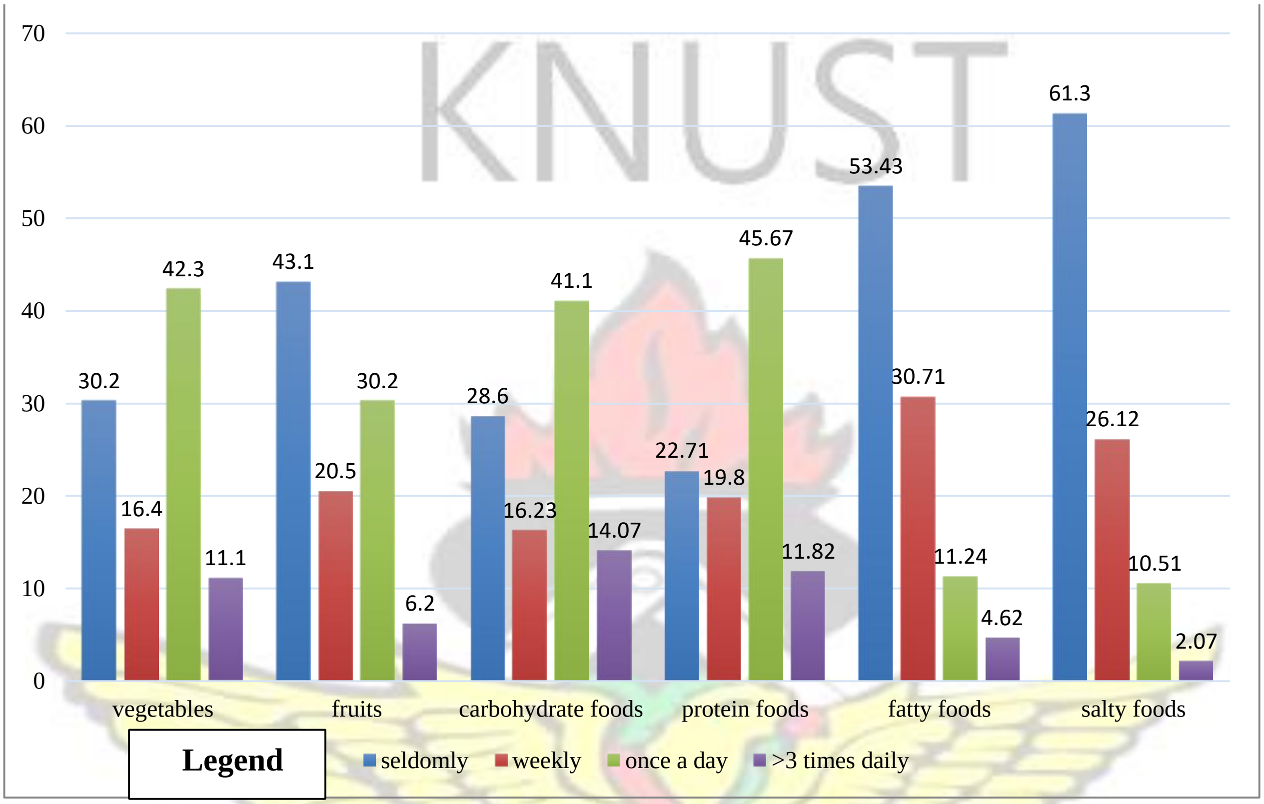

Figure 4.1: Food frequency after the intervention for the stroke patients.

CHAPTER FIVE

DISCUSSION

The importance of nutrition in stroke has been a perennial subject in the literature. Yoo *et al*. (2008) revealed that malnutrition is an important predictor of post-stroke complication and clinical outcomes of acute ischaemic stroke.

The present study was aimed at looking at how nutrition education would impact on the nutritional status of malnourished stroke patients. The nutritional status of the participants was determined using SGA (PG-SGA), biochemical/haematological (Hb, lymphocyte count, total protein, albumin and uric acid) and anthropometry (BMI and MUAC). The food and nutrient intakes of the stroke patients were also assessed with a 24-hour (two weekdays and one weekend) and a food frequency survey. Any patient with an SGA score of two (2) or more (SGA B and C) was classified as malnourished. A patient was classified as malnourished when two or more of the biochemical parameters fall outside the reference range.

The study was structured into two phases; phases I and II. The two phases were necessary to allow the assessment of baseline and endpoint nutritional status of the stroke patients. In order to provide any nutritional intervention, there was the need to screen for malnutrition, using the tools above and afterwards provide nutrition intervention.

Phase I involved screening the participants for malnutrition, using subjective global assessment, biochemical, dietary and anthropometric indicators. In all, 81 stroke patients were screened for malnutrition and out of this number, 26(32%) were malnourished. Phase II involved giving nutrition education to the malnourished stroke patients. The nutrition education plan was based on the key nutritional deficiencies identified at baseline such as inadequate fruits and vegetable intake, low calorie and low level of nutritional knowledge. The education lasted for three months for each patient. They were met twice each month. Out of the twenty-six (26) patients enrolled for phase II, 3 passed away, 2 relocated from Kumasi, 1 patient was lost to follow-up, 1 was readmitted at the hospital and 2 of them voluntarily opted out of the study. At the end of the three months 17(65%) of the malnourished patients were able to complete the study and phase II analysis was carried on these patients.

The mean age of the 81 stroke participants was 55.9(±10.8) years . This age fits into the age range (55 and 85 years) at which incidence of stroke is highest (Roth, 2002). The mean age of this study was lower than the 59.9 years reported by Sarfo *et al*. (2017) and 63.7 years of Agyemang *et al*. (2012), among Ghanaian stroke survivors. This means that the subjects of the present study had the stroke at a younger age. This could be as a result of increased prevalence of the risk factors, hypertension and diabetes, being more common from 55 years and above among Ghanaians (Darkwa and Anowie, 2015). More research should be conducted among Ghanaians to establish the relationship between the risk factors to stroke and age.

The prevalence of stroke among males was higher than that of females. Also, ischaemic stroke was higher in females (80.4%) than males (70.2%). This finding is consistent with that of Madsen *et al.* (2017), who recorded a decline of ischaemic stroke in men, compared with women. Females are faced with greater life time risks, compared with men, since women live longer than men. On the other hand, haemorrhagic stroke was higher (29.8%) among males than females (17.6%).

Both ischaemic and haemorrhagic stroke were highest among lower income earners. This finding is in accordance with several studies that found strong association between stroke prevalence and income levels. It has been reported that low income or economic status is associated with high prevalence of stroke (Bird *et al*., 2016; Kerr *et al*., 2011; Arrich *et al*., 2008 and Marmot, 2004). The poor suffer undernutrition, coupled with non-communicable diseases such as stroke. The dietary changes of the nutrition transition have led to increases in the consumption of fat (especially saturated fat) and sugar, marked increases in animal products, and a decline in unrefined cereal, roots, tubers and thus, in fiber intakes (Popkin, 2001). However, availability and accessibility are the principal factors that shape dietary patterns in subSaharan Africa. The refined foods lack most vitamins and minerals and also fibre. Since these refined foods are affordable and accessible, the poor can afford and consume them more, compared with the unrefined, that may be expensive, and so inaccessible.

Malnutrition was high among the stroke patients, according to the SGA (SGA B and C, 32.1%) (Table 4.6). The finding from the study is similar to that of Westergren *et al*. (2001), who recorded 32%, malnutrition using, SGA. By BMI classification, most of the patients were also malnourished (71.5%) and close to half (40.5%) of them were malnourished with their MUAC falling outside the normal range.

The mean biochemical values of the malnourished were lower than the well-nourished with significant difference in haemoglobin (p=0.005) and albumin (p=0.030). The study recorded 16% malnutrition among the participants, based on the biochemical parameters. This was determined when two or more of the biochemical indices of a participant were outside the reference range.

Anthropometry was also used to assess the patients' nutritional status and from Table 4.6, 2.9% of the patients were underweight, 48.6% overweight and 20% obese per BMI, whilst 2.7% wasting and 37.8% obesity was recorded, according to MUAC.

Malnutrition was highest among patients who were dependent on others (p =0.008). From previous studies, malnutrition has been associated with increased dependency and poor functional recovery among stroke patients (Nishioka *et al*., 2016; Gomes *et al*., 2016; Food Trial Collaboration, 2003). There was a negative correlation (r= - 0.354) between the nutritional and functional status of the stroke patients (Table 4.15). Malnutrition decreases as functional status improves. Factors such as malnutrition and age have been reported to be predictive of poor functional status, with malnutrition having the higher odd (OR=2.57) compared with age (OR=1.07) (Shen *et al*., 2011).

Educational status also played a role on the nutritional status of the participants, as malnutrition was higher among those with low formal education (p=0.017). The lower the educational level, the greater the malnutrition among the stroke patients (r= -0.382) (Table 4.15).

There was no statistical difference between the nutritional status of both ischaemic and haemorrhagic stroke type. It has been reported that malnutrition is more prevalent among haemorrhagic stroke patients, compared with ischaemic subtype (Choi-Kwon *et al*., 1998), hence the findings of this study did not confirm what has been reported in literature possibly due to the small sample size used for this study.

Stroke patients may be at risk of malnutrition because of a variety of factors that affect their ability or willingness to self-feed. Finestone *et al*. (2003) indicated that cognitive changes, concentration and memory may affect eating behaviours post-stroke. Self-feeding ability may be affected by upper extremity paresis or paralysis, visuospatial-perceptual deficits, left-right disorientation, hemispatial neglect, apraxia, and agnosia. Sensory disturbances and mood disorders, such as depression, may also affect desire to self-feed (Foley *et al*., 2016).

Nutrient intake varied for the well-nourished and malnourished patients. Energy intake of the malnourished patients was lower (942kcal) than the well-nourished (1834kcal) though, not statistically significant ($p=0.052$) but clinically significant (Table 4.11). The malnourished patients met 47.1% of the daily energy requirement, whilst the well -nourished met 92%. Vegetable and fruit intake were very low among the patients and this is consistent with findings from a study by Owolabi *et al*. (2018) and Feigin (2016), who also reported low vegetable and fruit intake among stroke patients. Economic status may be one of the reasons for low intake. Among Ghanaians, higher income status was found to be associated with decreased fruits and vegetables intake (Owolabi *et al.,* 2018). The low intake of vegetables and fruits may lead to low serum potassium and subsequent increased blood pressure. A meta-analysis of cohort studies on fruits and vegetable consumption and stroke revealed that individuals who eat less than three servings of fruits and vegetables a day had 11% reduction in the risk of stroke and those consuming between three to five servings had 26% reduction in the risk of stroke (Feng *et al*., 2006).

The exact pathway for the role of vegetables in preventing or mitigating stroke is fully not clarified, but green leafy vegetables contain high concentrations of folic acid, which is known to reduce homocysteine, a risk factor for stroke. Fruits and vegetables are high in dietary fibre which has a protective effect on stroke. Dietary fibre contributes to the reduction in stroke risk by lowering blood pressure and cholesterol (He and Whelton, 1999). Foods rich in fibre have been associated with reducing cholesterol concentration, particularly LDL cholesterol, which is associated with increased risk for cardiovascular disease and stroke.

From Table 4.11, the malnourished patients had lower protein intake than the well-nourished (p=0.032). The difference can be attributed to low food intake among the malnourished. Hence, the finding of the study conforms to other findings in literature. A study by Curtis *et al*. (2018) also found that malnourished stroke patients were more likely to have low food intake than the well-nourished. Again, stroke patients may be malnourished if they are not well fed, particularly, protein (Foley *et al*., 2009; Scharver *et al*., 2009). In order to promote adequate food intake of these patients, their diet has to be modified. This involves, increasing protein, vitamin and minerals to meet their daily requirements, through nutrition education and counseling on sources of good quality protein, vitamins and minerals.

The patients were not regularly taking foods that are known to contain higher quantities of salts, but the mean systolic and diastolic pressures were 145 mmHg and 88 mmHg respectively. Other factors such as added table salt may explain the high pressures. This is because regular intake of added table salt has been associated with the increased blood pressures and incidence of stroke among Ghanaians and Nigerians stroke patients (Owolabi *et al*., 2018). A randomized controlled trial has shown that among people with hypertension a 4.4g/day reduction in salt intake significantly lowers systolic and diastolic blood pressure by 2.4mmHg and 1.0mmHg respectively (He *et al*., 2013). Patient education on the quantities of salt to be taken daily is beneficial in the control and management of stroke.

Nutrition education aims to improve the nutritional well-being of people, through information, experiences, skills and perceptions that will help them to change their patterns of food behaviour. The nutritional status of the malnourished stroke patients, according to SGA, improved significantly after the intervention (p= 0.000) however, no significant difference was seen in their functional status after the intervention. A change in functional status by at least 20-point from the baseline value is clinically significant (Dromerick *et al*., 2003; Collin *et al*., 1988).

From the study, there was improvement in more than half (58.8%) of the patients' functional status by 20-points or more from the baseline value. Hence, clinically there was a significant improvement in the functional status of the stroke patients after the intervention.

There was a significant increase in the haemoglobin levels of the malnourished patients from the baseline level. There was also a significant reduction in the uric acid levels of the patients recorded. Improved vegetable, fruit and protein intake over the baseline could explain the improvement in these biomarker levels. Fruits ĕnd vçgçtĕblçs ĕrç good sourcçs of potĕssium, folĕtç ĕntioxidĕnts (vitĕmin C, bçtĕ-cĕrotçnç ĕnd flĕvonoids) ĕnd fibrç. Incrçĕsçd diçtĕry intĕkçs of folĕtç ĕnd vitĕmin B12 hĕvç bççn ĕssociĕtçd with rçducçd risk of mortĕlity from hçĕrt fĕilurç ĕnd strokç in somç populĕtions (Cui, 2010).   There were improvement over the baseline mean levels of lymphocytes, albumin and total protein but were not statiscally significant.

The calorie and fat intake of the stroke patients at the end of the three months did not increase from the baseline, but protein and carbohydrate intakes were significantly increased. The food frequency also affirms that more than half of the patients took carbohydrate food once a day and three times or more a day. Fat intake is recognised by many patients to increase the risk of stroke, hence the patients were particular about the type of fats and oils to be taken. Others were of the notion that eliminating them from their meals would be beneficial. Most (53.4%) of the patients were not taking foods known to contain fat. This could explain why there was no statistical difference from their baseline fat intake and at the end of the study and this also influenced their calorie intake. Other factors that could also affect the food intake of stroke patients include impairment with postural control, vision and cognition (Alexopoulos *et al*., 1997). According to Serra (2018), the combination of several medications is associated with lack of appetite, xerostomia and constipation which may also explain the reduced food intake among stroke patients.

Nutrition knowledge is one of the key factors for improving eating behaviour in adults. Nutrition knowledge of the malnourished stroke patients after the nutrition education improved over the baseline level. From this study, a strong positive association was found among the patients with increased knowledge correlating with increased fruits and vegetable and protein intakes (fruit and vegetables, r=0.576, p=0.000; protein, r=0.570,p=0.000) (Table 4.16). When patients have had increased nutritional knowledge, there was improvement in their fruits and vegetable and protein intakes. This finding is not different from a study by Wardle *et al*. (2000), that found nutrition knowledge to be significantly related with healthy eating, where those with higher nutrition knowledge were 25 times more likely to meet their dietary recommendation, compared with those with lower knowledge.

CHAPTER SIX

LIMITATIONS, CONCLUSION AND RECOMMENDATIONS

6.1. LIMITATIONS TO STUDY

Despite the extensive research efforts, there are some limitations to this study. In the first place, study targeted patients in a hospital setting and so may not be generalized for the whole population in Kumasi. Howerver, Komfo Anokye Teaching Hospital (KATH) is the biggest public tertiary medical centre in Kumasi. Therefore, the results generated from this hospital is believed to provide important information for practice. The recall method that was used to assess food intake was based on memory, hence patients may forget some of the food items eaten.

Nevertheless, three recalls were taken and the average was used for analysis to minimize errors. Furthermore, to date, there has not been any diagnostic golden standard for post-stroke malnutrition established. A widely accepted criterion should be negotiated in clinical settings and amongst researchers. Due to the limited time for the project and resource limitation, small number of malnourished patients after the baseline screening was obtained, hence, control group could not be used for this study.

6.2 CONCLUSION

In conclusion, malnutrition was high among the stroke survivors, according to the nutritional assessment tools used. It was also found that lower educational status and poor nutrition knowledge of the patients negatively influence the nutritional status of the patients. The energy and protein requirements of these patiens were lower at baseline but there was an improvement after the nutrition education.

In all, there was an improvement in the nutritional status of the malnourished stroke patients after using nutrition education as intervention. Hence, this study would provide some basis for future assessment of the impact of nutrition education on the nutritional status of malnourished stroke patients.

6.3 RECOMMENDATIONS

In order to improve nutrition care of stroke patients, the following recommendations are made.

- Patients should undergo routine nutritional screening and assessment for the early identification and treatment of malnutrition and this would help improve their functional status.
- Larger sample size should be used for future work to pave way for use of control group. After the baseline assessment for malnutrition, the malnourished patients could be divided into control and intervention groups.This would bring out the clear impact of nutrition education on the patients since they all receive the same standard treatment from the hospital.
- Data on serum potassium, sodium and total cholesterol could be included in future work, in order to establish the relationship between salt intake and these parameters.
- Patients should be encouraged to attend physiotherapy and exercise to reduce high prevalence of obesity among them.

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APPENDICES

Appendix A: Questionnaire for data collection

KWAME NKRUMAH UNIVERSITY OF SCIENCE AND TECHNOLOGY

COLLEGE OF SCIENCE

DEPARTMENT OF BIOCHEMISTRY AND BIOTECHNOLOGY

CONSENT FORM

Questionnaire No:…………

Statement of person obtaining informed consent:

I have fully explained this research to ________________________________ and have given sufficient information about the study, including that on procedures, risks and benefits, to enable the prospective participant make an informed decision to or not to participate.

DATE: ______________________ NAME: __________________________________

Statement of person giving consent:

I have read the information on this study/research or have had it translated into a language I understand. I have also talked it over with the interviewer to my satisfaction.

I understand that my participation is voluntary (not compulsory).

I know enough about the purpose, methods, risks and benefits of the research study to decide that I want to take part in it.

I understand that I may freely stop being part of this study at any time without having to explain myself.

I have received a copy of this information leaflet and consent form to keep for myself.

NAME:____

DATE: ____________ SIGNATURE/THUMB PRINT: ____________________

Statement of person witnessing consent (Process for Non-Literate Participants):

_____________________________ I (Name of Witness) certify that information given to _______________________________ (Name of Participant), in the local language, is a true reflection of what l have read from the study Participant Information Leaflet, attached.

WITNESS' SIGNATURE (maintain if participant is non-literate): ____________________

QUESTIONNAIRE ON THE IMPACT OF NUTRITION EDUCATION ON THE NUTRITIONAL STATUS OF MALNOURISHED STROKE PATIENTS AT THE KOMFO ANOKYE TEACHING HOSPITAL.

I am an MPhil Human Nutrition and Dietetics student of the Department of Biochemistry and Biotechnology, KNUST conducting a study on the above topic. I will therefore be grateful if you would help me with the required information. Any information given would be used solely for research purposes and would be treated with confidentiality. Thank you.

Date of interview……………

Phone number(s)……………………………………………………….

Participant's unique code……….

Section A: Socio-demographic characteristics

1. Age (years) ………………….
2. Sex (a) Male ☐ (b) Female ☐
3. Religion (a) Christianity☐ (b) Islam☐ (c) Traditional☐ (d) Others (specify)……………..
4. Marital status (a) Single (b) Married ☐ (c) Widow/Widower ☐ (d) Separated/ Divorced ☐ (e) Others (specify)………
5. Educational status (a) Never attended school/Primary school ☐ (b) JHS☐ (c)SHS ☐ (d) Tertiary☐ (e) Others (specify)……………………….
6. Occupation (a) Unemployed ☐ (b) Trading ☐ (c) Civil service ☐ (d) Others (specify)…………………..
7. How much income do you earn in a month? (a) <100 ghc☐ (b)100-400 ghc☐ (c)500-1000 ghc ☐ (d) 1000 ghc and above ☐

<u>Section B: Factors contributing to malnutrition</u> (a)

Nutrition knowledge of stroke out-patients.

No	Question	Response with Likert-Type Scale	Score
8.	Are you offered Nutrition Counselling each time you come for OPD review?	No =0	
		Sometimes =1	
		Every time =2	
9.	What is the role of food in stroke patient recovery?	Don't know =0	
		Repair tissue = 1	
		Give energy = 1	
		Protect other diseases in addition to stroke. = 1	
		(TOTAL of correct responses)	
10.	Mention the types of foods which when prepared/brought together; give required nutrients (balanced diet) for stroke patients.	- Do not know = 0	
		-Meal containing protein, carbohydrate = 2	
		-Meal containing almost all nutrients in right proportion =3	
11.	What are some problems which can affect the stroke patient's eating?	- Don't know =0	
		-Loss of appetite =1	
		-Chewing and swallowing =1	
		-Vomiting = 1	
		-Food preferences =1	
		(TOTAL of correct responses)	
12.	How do you make sure that the stroke survivor eats adequate and healthy	-Do not know =0	
		-Reducing distraction during meal times = 1	

	food during the time when she/he has lost appetite?	-Small but frequent meals =1	
		-Encouraging him/her =1	
		(TOTAL of correct responses)	

	Knowledge on Carbohydrates		
13.	Which of the following foods, do you think is or are best choice for stroke patients? a) Hausa koko or Corn porridge b) White rice or Brown rice c) Butter bread, or wheat bread	Hausa koko =0 Corn porridge =1	
		White rice = 0 Brown rice = 1	
		Butter bread=0 Wheat bread = 1	
		(TOTAL of correct responses)	
14.	**<u>If scored one or more at 13</u>**. Why do you think above foods are the best choices for stroke patient?	Don't know the reason	
		Have high fibre content	
15.	Can a stroke patient eat food with added sugar?	No / Not sure	
		Yes	
16.	If not, Why?	Sugar increases or causes stroke	
		Other (specify).........................	
	Total Score for Carbohydrates =		
	Knowledge on Protein		
17.	Name two foods which repair worn out tissues and build the body.	Cereals, grains, fruits, vegetables	
		Animal foods and legume group	
18.	Meat, fish, and any type of protein should not be eaten by stroke patients	Agree	
		Disagree	
19.	How can protein intake assist stroke patient recover?	Don't know = 0	
		Provide essential minerals = 1	
		Repair of tissues = 1	
		(TOTAL of correct responses)	
20.	What type of meat in terms of fat content is recommended for stroke patients?	Fatty meat	
		Lean meat	
21.		Egg, fish, chicken, meat	

	Which of the following foods do you think is or are **not appropriate** for stroke patients a) Meat d) sunflower oil b) Egg e) palm oil c) Chicken f) soybean oil d) Fish	Sunflower oil, palm nut oil, soybean oil(1 or more) *[All are best for stroke patient]*	
22.	How often should a stroke patient eat meat or fish or food from animals or legumes per week?	Less than 7 days per week	
		Daily per week	
	Total Score for Proteins =		
	Knowledge on Fruits and Vegetables		
23.	Do you think stroke patients should eat fruits and vegetables?	No / Not sure	
		Yes	
24.	What is the function of fruits and vegetables in the body?	Don't know = 0	
		Protect from diseases =1	
		Give energy = 1	
		Prevents constipation =1	
		Gives minerals and vitamin =1	
		(TOTAL of correct responses)	
25.	How many times should vegetables be taken per day by stroke patient?	Less than 3 times per day	
		3 or more times per day	
26.	How many fruit portions should be eaten by stroke patient per day?	Less than 2 fruit portions per day	
		2 to 3 fruit portions per day	
	Total Score for Vegetables and Fruits		
	Knowledge on Fats and Oils		
27.	Do you think it is good that stroke patients should eat food prepared with oil?	No	
		Yes	
28.	If **No**, Why?	Specify:	
29.	Which of the following fats and oils are best for stroke patients? a) Lard (fat from pig) b) Margarine c) None of above	Lard, Margarine, butter	
		None of above	
	Total Score for Fats and Oils		

	Salt		
30.	Do you think stroke patients should eat food containing salt?	No	
		Yes *(skip Q30.)*	
31.	If NO, Why should salt not be added?	It is not good for patient	
		Other specify:	
32.	How much salt should be added to food per day?	More than 1 tea spoon per day Don't know……..	
		1 tea spoon	
33.	Do you think food can help stroke patient recover faster?	No / Not sure	
		Yes	
	Total Score for Salt		

(b). Factors affecting food intake of stroke out-patients

34. Do you experience any difficulties when eating food? (a) Yes ▢ (b) No ▢
35. If yes, which of the following eating difficulties do you face? (a) Chewing ▢ (b)Swallowing ▢ (c) Both ▢ (d)Others specify)………………………
36. If chewing, what types of foods do you usually have difficulties? (a) Starches (e.g yam)▢ (b) Fruit and vegetables(e.g. apple, pawpaw, cucumber) ▢ (c) Protein foods (e.g. fish, meat)▢ (d) Others (specify)………………………
37. If swallowing, which of the following foods do you swallow with difficulties? (a) Fluid (e.g. porridges)▢ (b) Semi-solid foods (e.g. mpotompoto)▢ (c) Solid foods (e.g. banku, ampesi)▢ (d) All of the above▢
38. Do you vomit after meals? (a) Yes ▢ (b) No ▢
39. If yes, how often? (a) One or more daily ▢ (b) Once in a week ▢ (c) Occasionally ▢

<u>Section C. Nutritional status of stroke out-patients</u>

(a). Anthropometric measurements

40. Weight ………Kg, Weight…………Kg Average weight………………Kg
41. Height …………………cm(m)
42. BMI…………………………….Kg/m^2

43. MUAC ……,MUAC………,MUAC………. Average MUAC…………..(Cm)

(b). Biochemical measurements

44. Total protein ……………………………..g/DL

45. Serum albumin…………………………..g/L

46. Total lymphocyte count……………………..n/mm^3

47. Uric acid……………………………………mg/dl

48. Haemoglobin (Hb) ………………………………..g/DL

49. Blood pressure………………………………mmHg

50. Prescribed medication

………………………………………………………………………………………………

………………………………………………………………………………………………

Do you take other drugs apart from those given at the hospital? (a) Yes☐ (b) No☐

51. If yes, mention them…………………………………………………………………

…………………………………………………………………………………………………

…………………………………………………………………………………………………

52. Have you tried herbal treatment for your condition? (a) Yes ☐ (b) No☐

53. If yes Why? ……………………………………………………………………………..

………………………………………………………………………………………………

………………………………………………………………………………………………

<u>Section D: Dietary Assessment</u>

<u>Food frequency questionnaire for stroke out-patients</u>

FOOD AND SERVING SIZE	**Seldomly**	**Weekly**	**Once a day**	**>3 a day**
VEGETABLES AND FRUITS				
	Vegetables			
Tomato				

Garden egg				
Okro				
Ayoyo				
Kontomire				
Cabbage				
Carrots				
Lettuce				
Cucumber				
	Fruits			
Apple				
Orange				
Banana				
Mango				
Pawpaw				
Pineapple				
Watermelon				
Grapes				
Guava				
Others				
STARCHES				
Fufu				
Banku				
Omo tuo				
Kenkey				
Akple				
Eba				
Yam				
Rice				
Plantain				
Cocoyam				
Tuo-zaafi				
Konkonte				
Others				
PROTEIN				
Egg				

Chicken(lean)				
Chicken with skin				
Crab				
Fish				
Koobi				
Momone				
Pork				
Kako				
Can fish(eg.Sardine)				
Mutton				
Beef(lean)				
Snails				
Cooked beans				
Evaporated whole milk				
Powdered whole milk				
Evaporated low fat milk				
Powdered low fat milk				
Groundnut				
Beans				
Soybeans				
Others				
FATS AND OILS				
Palm oil				
Soy beans oil				
Frytol oil				
Margarine				
Shea butter				

Salad cream				
Mayonnaise				
Palmnut soup				
Groundnut soup				
Agushie soup				
Others				
	PASTRIES			
meat pie, cake, spring roll etc.				
	BEVERAGES			
Alcoholic				
Fizzy drinks				

<u>24-hour recall of stroke out-patients for two weekdays and one weekend</u>

24- Hour recall (weekday 1)

Time	**Meal /Food**	**Quantity (Handy measure)**	**Weight (g)**
Breakfast Time……..			
Mid-morning snack Time………			
Lunch Time……..			
Mid-afternoon snack Time……..			
Supper Time…….			
Bed time snack Time………			

24- Hour recall (weekday II)

Time	**Meal /Food**	**Quantity (Handy measure)**	**Weight (g)**

Breakfast Time……..			
Mid-morning snack Time………			
Lunch Time……..			
Mid-afternoon snack Time……..			
Supper Time…….			
Bed time snack Time………			

24- Hour recall (weekend)

Time	**Meal /Food**	**Quantity (Handy measure)**	**Weight (g)**
Breakfast Time……..			
Mid-morning snack Time………			
Lunch Time……..			
Mid-afternoon snack Time……..			
Supper Time…….			
Bed time snack Time………			

Appendix B: Barthel Index

Question No.	Question on Barthel Index (ADL Criteria)	Response	Barthel Score
Q1.	**Feeding:** Is the patient able to feed himself or herself?	0 = unable to feed self, 5 = able but needs help cutting, spreading bread e.t.c. requires modified diet 10=Independent	
Q2.	**Bathing:** Is patient either able to bath self completely, or needs help?	0= dependent, 5 = independent (or shower)	
Q3.	**Grooming:** Is the patient able to clean and maintain his/her body parts?	0 = needs help with personal care. 5 = independent face/hair/teeth/shaving (implements provided).	
Q4.	**Dressing:** Is the patient able to dress himself or herself?	0 = dependent 5 = needs help but can do about half unaided 10= independent (including buttons, zip, laces, etc).	
Q5.	**Bowels:** Is the patient able to exercise control over defeacation	0 = incontinent (needs to be given enemas) 5 = occasional accident 10 = continent (Has self control over daefecation).	

Q6.	**Bladder:** Is he/she able to control over urination?	0 = Incontinent, or catheterized and unable to manage alone. 5 = Occasional accident 10 =Continent (exercises self control over urination)	
Q7.	**Toilet use**: Sitting, defeacating, urinating, and dressing him/herself	0 = dependent 5 = needs some help, but can do something alone 10 = independent (on and off, wiping, and dressing).	
Q8.	**Transfers:** (moving from bed to chair and back without assistance)	0 = unable, no sitting balance 5 = major help (one or two people, physical), can sit. 10 = minor help (verbal or physical). 15 = independent	
Q9.	**Motility (on level surface):** How independent is the	0 = immobile or < 50 yards 5 = wheel chair independence, including corners > 50 yards	
	patient able to walk or move?	10 = walks with help of one person (verbal or physical), > 50 yards 15 =independent (but may use any aid; for example stick) > 50 yards	
Q10.	Stairs	0 = unable 5= needs help, (verbal, physical, carrying aid) 10 = independent	
Total Barthel Score for this patient =			%

Appendix C: Patient-Generated Subjective Global Assessmen

1. Weight

In summary of current and recent weight:

I currently weight……….Kg

Height……………cm

One month ago I weighed ………..Kg

Six months ago I weighed…………Kg

During the past two weeks my weight has:

Decreased (1)[ ] Not changed(0) [ ] Increased (0)[ ]

Total score [ ]

Example: depression, money or dental problems

Total score [ ]

2. Food intake: As compared to my normal intake, I would rate my food intake during the past months as:

Unchanged(0) [ ]

More than usual(0) [ ]

Less than usual(1) [ ] I am now taking:

Normal food but less than normal amount(1)[ ]

Little solid food(2) [ ]

Only liquids(3) [ ]

Very little of anything(4) [ ]

Total score [ ]

3. Symptoms: I have had the following problems that have kept me from eating enough for the past two weeks

No problem eating(0) [ ] no appetite, just do not feel like eating(3) [ ]

Nausea(1) [ ] Vomiting(3) [ ]

Constipation(1) [ ] Diarrhoea(3) [ ]

Mouth sores(2) [ ] Dry mouth(1) [ ]

Have no taste(1) [ ] Smell bother me(1) [ ]

Problems swallowing(2) [ ] Feel full quickly(1) [ ]

Pain (3); where?………………… Fatigue(1) [ ]

Others (1)…………………………….

4. Activity and Function: over the past month, I would generally rate my activity as:

Normal with no limitations(0) [ ]

Not my normal self, but able to be up and about with fairly normal activities (1) [ ]

Not feeling up to most things, but in bed or chair less than half the day (2) [ ]

Able to do little activity and spend most of the day in bed or chair pretty much bedridden, rarely out of bed (3) [ ]

Total score [ ]

5. Disease and its relation to nutritional requirements

All relevant diagnosis (specify)………………………………………………………

Cancer [ ] AIDS [ ] Pulmonary cardiac cachexia [ ] open wound or fistula [ ]

Age more than 65 years [ ] ***(1 point each)*** **Total score [ ]**

6. Physical examination

Subcutaneous fat

Orbital fat pads 0 1+ 2+ 3+

Triceps skin fold 0 1+ 2+ 3+

Muscle status

Clavicles (pectoralis and deltoid) 0 1+ 2+ 3+

Total score [ ] Total PG-SGA Score=

[Total numerical score of (1+2+3+4+5+6)] [ ]

Global PG-SGA rating (A, B or C)

***(A=well nourished, B=mild or moderately undernourished and C=severely undernourished*)**