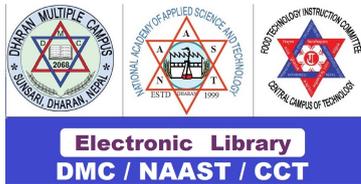


**COMPARATIVE STUDY OF NUTRITIONAL STATUS OF
RHEUMATIC HEART DISEASES PATIENTS BEFORE AND AFTER
THE SURGERY IN DHARAN AND NEARBY VDC'S.**



by
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2012**

**Comparative Study of Nutritional Status of Rheumatic Heart Diseases
Patients Before and After The Surgery in Dharan and Nearby VDC's.**

*A dissertation submitted to the Food Technology Instruction Committee
in Tribhuvan University in partial fulfillment of the requirements
for the degree of B.Tech. in Food Technology*

by

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Approval Letter

This dissertation entitled *comparative study of nutritional status in rheumatic heart diseases patients before and after the surgery in Dharan and nearby VDC's* by *Abhishek Khadka* has been accepted as the partial fulfillment of the requirements for the B. Tech in Food Technology.

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Abhishek Khadka
(CCT, Dharan)

List of Abbreviations

ARF	Acute Rheumatic Fever
AMI	Acute Myocardial Infection
ASPGN	Acute post-streptococcal glomerulonephritis
BMI	Body Mass Index
BPKIHS	B.P. Koirala Institute of Health Sciences
CHD	Coronary Heart Diseases
DDC	District Development Committee
DHERSEC	Disabled and Helpless Care and Rehabilitation Center
GABHS	Group A Beta Haemolytic Streptococcal
GAS	Group A Streptococcal
INGO	International Non-Governmental Organization
INR	International Normalized Ratio
Kcal	Kilo Calorie
LCA	Left Coronary Artery
NCD	Non Communicable Disease
NGO	Non-Governmental Organization
NYHA	New York Heart Association
PHC	Primary Health Care
RCA	Right Coronary Artery
RF	Rheumatic Fever
RHD	Rheumatic Heart Diseases
SGNHC	Sahid Gangalal National Heart Center
UNESCO	United National Educational Scientific Cultural Organization
UNICEF	United Nations International Child Emergency Fund
VDC	Village Development Committee
WHO	World Health Organization

Abstract

Rheumatic heart disease (RHD) remains a significant health problem in the developing world, affecting 15.6 million people worldwide, with a prevalence of 1.2/1000 in 5-15 year old school children in Nepal. RHD is largely a disease of poverty, of which overcrowding, poor nutrition, poor health-care access and limited health resources are major contributing factors. Considering the nutritional status of RHD, the survey was done in Dharan and nearby VDC's. Thirty post operative patients ranging from the age 9 to 37 were included in the survey. Ten of the patients were Dalit and 13 patients were NYHA III categorized. Nine of the patients were residing in Dharan.

The daily calorific value was calculated on the basis of the response from the questionnaire. On the survey it was found that there was a significant increase in calorific value but only six of them were meeting up the recommended calorific intake value. Besides it was found that there is a significant increase in carbohydrate, protein, vitamin A and riboflavin intake but not fat before and after the surgery. Loss of appetite was one major symptom of the diseases before the surgery and more over lack of knowledge about this disease had led the patients to the valve operation otherwise it can be cured in primary phase. On the basis of the BMI sixty percentages of total patients were categorized as normal after the surgery and the percentage of underweight was reduced from 36.67% to 30%. Poverty was major reasons for malnutrition in the patients in the pre-surgery conditions.

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Part I

Introduction

1.1 Background

Nepal is a under developed country where 90% of the population lives in the rural areas and most of them are often malnourished (CBS, 1992). Various types of nutritional problems are widely spread among school children in this country because of inadequate knowledge of nutritious food and nutritionally balanced diet. Micronutrient deficiency is one of the prominent public health problems especially in developing countries (ICN, 1992).

The World Health Organization (WHO) defines malnutrition as "the cellular imbalance between the supply of nutrients and energy and the body's demand for them to ensure growth, maintenance, and specific functions" (Onis *et al.*, 1993). Malnutrition is the pathological state resulting from a relative or absolute deficiency or excessive intake of one or more essential nutrients. It may be due to the inadequate food intake, defective absorption, poor dietary habits, food faddisms etc (Jelliffe, 1966)

When development involves people's participation, they must be physically and mentally be fit for the duties to play roles in national development both socially and economically. In order to be able to literate to be productive, healthy one needs to be well nourished. Nutrition is thus a basic need. It is the central to survival and is critical for individual growth and capacity to function in society (Raja, Lakshmi, 1990).

Group A streptococcus (GAS) causes a broad spectrum of disease, from minor illness to life threatening infections and is the causative agent for acute rheumatic fever (ARF) and acute post-streptococcal glomerulonephritis (APSGN). These diseases may lead to further complications (e.g. ARF may cause rheumatic heart disease, which in turn may be further complicated by endocarditis or strokes). The only GAS disease for which global disease burden estimates have previously been made is RHD (WHO, 2005).

We conclude that approximately 18.1 million people currently suffer from a serious GAS disease, another 1.78 million new cases occur each year, and these diseases are responsible for over 500,000 deaths each year.

Table1.1 Burden of GAS in world

Diseases	Summary of estimated global burden of group A streptococcal diseases		
	Number of existing cases	Number of new cases	Number of death Each Year
Rheumatic heart disease	15.6 million	282000*	233000**
History of acute rheumatic fever without carditis, requiring secondary prophylaxis	1.88 million	188,000*	
RHD-related infective endocarditis		34000	8000
RHD-related stroke	640,000	34000	8000
Acute post-streptococcal glomerulonephritis	—	144000	108000
Invasive group A streptococcal diseases		472000	5000
Total severe cases	18.1 million	1.78 million	163000
Pyoderma		111 million	517000
Pharyngitis		616 million	

All estimates rounded off. Note that these estimates assume constancy of incidence and prevalence over time.

* New RHD cases were calculated based on the proportion of incident ARF cases expected to develop RHD. The remainder of incident ARF cases is included in the “History of ARF without carditis” row. Therefore, the total number of new ARF cases each year is 282,000 + 188,000 = 470,000.

** Includes ARF deaths. RHD deaths are based on proportion of existing RHD cases expected to die each year.

- No attempt has been made to quantify the prevalence of APSGN-induced chronic renal impairment or end-stage renal failure

ARF is a non-supportive complication of group A beta hemolytic streptococcal (GABHS) sore throat infection which commonly affects school going children. It affects joints, skin, subcutaneous tissue, brain and heart. It is commonly called as the “diseases that licks the joint but bites the heart.” (English, 1999).

Except heart, it may progress to chronic glomerulonephritis and even renal failure; all other effects are reversible, needing only symptomatic relief during the episodes. Cardiac complications are significant in absence of secondary prophylaxis and culminate into chronic and life threatening valvular heart disease (Sanyal *et.al.*, 1992). ARF is the most common cause of cardiac disease in children in developing countries. A joint meeting of the World Health Organization and the International Society and Federation of Cardiology in 1994 estimated that 12 million people in developing countries were affected by ARF and RHD, with the majority of these being children (WHO, 1995).

Like streptococcal sore throat, acute rheumatic fever occurs most commonly in young school child, median age between 9 to 11 years and very rarely in early infancy (Gene *et.al.*, 1989).

Although RF and RHD are rare in developed countries, they are still major public health problems among children and young adults in developing countries. The economic effects of the disability and premature death caused by these diseases are felt at both the individual and national levels through higher direct and indirect health-care costs (WHO, 2001)

In the year 2010 five hundred sixty six patients were admitted in Sahid Gangalal National Heart Center accounting 291 males and 269 females. RHD was one of the leading causes of admission accounting nearly half of the total admissions. Major cause of the mortality was due to RHD with female preponderance In the case of the mortality also RHD was the leading ones 29.7% of the total mortality was by RHD (SGNHC, 2010).

Role of nutrition in cardiac diseases is important: The heart muscle like any other body tissue is dependent upon the adequate supply of all essential nutrients. Markedly malnourished people of the world frequently manifest cardiac impairment such as dyspnoea (difficulty in breathing) and palpitation on exertion, enlargement of the heart and systolic murmurs (Robinson, 1972).

RF is an inflammatory disease that occurs following a *Streptococcus pyogenes* infection, such as strep throat or scarlet fever. Believed to be caused by antibody cross-reactivity that can involve the heart, joints, skin, and brain, the illness typically develops two to three weeks after a streptococcal infection (Kumar *et.al.*, 2007). RHD is a significant

physical, mental and socioeconomic burden upon individuals and families. Cost-effective prevention programs are challenged by limited resources and poor access to health-care. Within a primary health care system, heart-valve surgery cannot be made widely available due to costs and competing priorities; however is the only curative option for RHD patients. Health-care programs for poor families with RHD should continue, and be scaled up if possible. Further research into RHD prevalence and quality of life, and development of comprehensive PHC policies including RHD prevention are required (Meyer, 2009).

Following an initial RF attack, the risk of reactivation increases with further pharyngeal infections (Kumar *et. al.*, 2005). Recurrent episodes of acute RF cause cumulative damage specifically to the heart valves, culminating in the permanent cardiac dysfunction of RHD (Chopra and Gulwani, 2007). The literature reports varying proportions of RF patients progressing to RHD: one study reported 30-45% of patients (Sampiao *et. al.*, 2007); another 50-60% (Ramachandran, Alaukar and Thapaliya, 2006). RF and RHD remain significant health problems in the developing world. A recent study estimated that worldwide 15.6 million people have RHD, with 233,000 deaths caused by RF or RHD each year (Carapetis, 2007).

Food has the significant role to play in illness. Diet may have to be modified depending upon the heart diseases, the severity of the problem, the nutritional status of the patient as well as metabolic change involved (Khanna, *et.al.*, 2005).

Many developing countries are currently undergoing a rapid nutrition transition. Falling real prices for food enable a growing number of consumers to move towards higher calorie intake levels and allow them to embark on consumption patterns that had hitherto been reserved for consumers in developed countries at a much higher level of income (Schmidhuber and Shetty, 2005).

Globally, great disparity is seen in healthcare expenditure between poor and affluent countries (Gaziano, 2007). Where developed countries spend \$2,700 per capita, developing countries spend just \$74 (Gu, 1997).

1.2 Statement of the problem

According to the annual report of Sahid Gangalal National Heart Center, in the year 2010 one hundred twenty three RHD surgery were done in which eighty eight of them were females and thirty five of them were males accounting 22.1% of the total surgeries. From same source it was reported that the mortality of patients in SGNHC is highest. Around

which the nutritional status is poor. First the surgery cost is high and second the medication should be followed by the patients.

RF and RHD remain significant health problems in developing countries. Poverty, poor living conditions and limited access to health-care mean that many young people in Nepal will continue to develop RHD. Besides the surgery and medication it's important to keep good nutritional status. Nutritional status is measured by daily calorific intake, body mass index, chest-head ratio and mid upper arm circumference. These parameters will give the nutritional status of any individual. The per capita income of Nepali people is very low that's \$470 in the fiscal year 2009 (World Bank, 2009). And the medicinal costs as well as surgery cost of RHD is high. The average surgery cost ranges from 40000 to 250,000.

As RHD starts with joints pain, fever and headache they merely think that they are suffering from RHD. And moreover the balanced diet food is hardly in seen Nepali middle income and poor ones. As a result they are lacking the nutrients. Nutrients have a great role for RHD patents. Adverse effects of RHD such as economical effect, social effect and mental effect roles as a burden. Inadequacy of diet is another factor for malnutrition.

1.3 Objectives

1.3.1 General objectives

The objective was to study the nutritional status of the patients before who are suffering from the RHD and had a surgery. Another general objective was to know the daily calorific value of the RHD patients. Nutritional parameter like BMI ratio was measured.

1.3.2 Specific objectives

- a. To compare the body mass index (BMI) of the patients before and after the surgery and categorize it.
- b. To know the medication used by the RHD patients and its affect in nutrition.
- c. To know the effects of this disease on family and themselves.
- d. To know the daily calorific value of the RHD patients before and after the surgery.

1.4 Importance

- a. This study will highlight the nutritional status of RHD patients residing in Dharan and nearby VDC's.
- b. This research will help the knowledge like symptoms and prevention of the rheumatic heart disease in the research area.

- c. This research helps to study the effect of RHD in patients.
- d. This research will remarkably contribute in any further research regarding in RHD.

1.5 Limitations

- a. Although this disease is seen in the peak age of 5 to 15 years but the research was conducted in a range of 9-37 years of age. Cause the children of age less than 10 cannot give the interview properly. The duration of surgery is not taken as the important factor. The surgery duration in post operative patients was 18 (1.5 years) months to 24 months (2 years).
- b. The amount of daily foods taken by the patients were not exact value, the values were given in the approximation. So the daily calorie intake feed was in approximate value.

Part II

Literature review

2.1 Cardiovascular diseases (CVD) in developing countries

Beginning in the 1950s, epidemiologic and controlled human diet studies showed an association between the intake of saturated fatty acids and blood cholesterol levels. Since this time, the hypothesis of a direct link between diet and CVD has dominated epidemiologic and clinical investigations. Eighty percentage of the global CVD burden now occurs in developing countries. CVD is the leading cause of death in the industrialized countries (Hu and Willet, 2002).

As recently as five years ago, most physicians would have confidently described atherosclerosis as a straight plumbing problem: fat laden gunk gradually builds up on the surface of passive artery walls. If a deposit (plaque) grows large enough, it eventually closes off an affected 'pipe', preventing blood flow from reaching its intended tissue (Libby, 2002)

Inappropriate dietary and lifestyle choices, such as smoking, interfere with the regulation of metabolism and lead to inflammation, oxidation by free radicals, and other changes that cause CVD. In addition, a balanced diet and physical activity can reduce atherosclerosis and prevent CVD. Tobacco remains the biggest risk factor for CVD: a WHO report contends that tobacco will cause 12.3% of global deaths by 2020. Disproportionately, 80% of tobacco users reside in developing countries (Gaziano, 2007 and Gu, 1997).

NCD's especially CVD's, are emerging as an epidemic in developing countries, causing a double burden of communicable disease and NCD (Yusuf *et. al.*, 2004).

Rates of CVD in developing countries are growing, with no signs of change in the near future. However, one form of CVD stands apart (Eisenberg, 1993). Where increasing overall CVD rates arise from the trappings of a more affluent lifestyle, rheumatic heart disease (RHD) remains instead entrenched in poverty, a lack of development and poor access to health-care (Padmawati, 1978). RHD contributes significantly to CVD burden in resource-poor countries, and despite the socioeconomic progress made in these countries over the past few decades, RHD prevalence shows no sign of waning (Carapetis, 2001).

2.2 Types of heart diseases

- a. Coronary heart diseases (CHD)
- b. Heart attacks
- c. Ischemic heart diseases
- d. Heart rhythm disorders
- e. High blood pressure
- f. Tachycardia
- g. Pulmonary heart diseases and hypertensive heart diseases
- h. RHD (Grober, 2006)

2.2.1 Coronary heart diseases

This is the most common type of heart diseases caused by artery narrowing and reduced blood supply to the heart (Grober, 2006). In the United States, an estimated one million people die annually from CVD, with myocardial infarctions (heart attacks) accounting for a large proportion of this mortality (Flack and Yunis, 1997). Heart is made of the muscle. This muscle needs continuous blood supply in order to function. The supply of heart and its muscle maintained through two coronary arteries, right and left coronary artery (RCA and LCA). The left coronary further divides into two branches, left anterior descending artery and left circumflex artery. A narrowing of arteries, comparable to clogging of the fuel pipes in the motor car, leads to imbalance between supply and demand of blood to the heart, thereby causing starvation to heart muscles which leads to heart attack (Grober, 2006).

According to Grover, 2006 consequence damage in the heart is disorder of electrical pathway involved in generating the heart beat. Generally heartbeats 60-100 times a minute like a clock. When damage occurs, the heart beat can become irregular missing, or odd beat in between. The irregularity in the beating of the heart is called arrhythmia. These too, can further aggravate the imbalance between the supply and demand of the blood supply to heart (Groder *et. al.*, 1996).

2.2.2 Heart attack

It is reported that (Smolin and Grosbenor, 2000) heart attack or acute myocardial infections (AMI), are very common and very deadly. The cause of a heart attack is usually due to blockage of blood vessels of the heart. When the heart actually stops in heart attack this is called 'cardiac arrest'. The most common type of symptoms in chest pain or chest

discomfort and others are uncomfortable chest pressure, chest squeezing, chest fullness, chest pain etc (Grodner *et. al.*, 1996).

2.2.3 Ischemic heart diseases

It's due to reduction of blood flow in the heart. Symptoms of ischemic heart diseases are, chest pain, exertional dyspnea (difficulty in breathing), orthopnoea, peripheral odema, etc (Smolin and Grober, 2000).

2.2.4 Heart rhythm disorders

Heart rhythm disorders, called arrhythmias, pose one of the paradoxes of medicine. Almost anyone's heart will occasionally produce an extra beat or two, and the distressing symptoms that may accompany the extra beats, such as palpitations or dizziness, do not necessarily signal a serious health problem. Yet an undetected arrhythmia also may set off a chain of events leading to sudden death from cardiac arrest (McPherson and Lyanda,??).

2.2.5 High blood pressure

Blood pressure is the force with which blood pushes against the walls of the blood vessels. When the heart beats and pumps blood into the arteries the pressure raises to a peak. This is systolic blood pressure (Maximum pressure in artery when the heart is beating). When the heart relaxes and beats, the pressure falls to lowest point. That is diastolic blood pressure (minimum pressure in artery between the heart beat (Grover, 2006).

Blood pressure can rise for three reasons:

- a. The heart increases its output of blood to flow
- b. The arteries constrict and less room for blood to flow
- c. Both of above.

2.2.5 Tachycardia

This type of diseases is due to excessively rapid of the heart beat. Symptoms of the tachycardia are rapid heartbeat at rest, palpitations (Grover, 2006).

2.2.6 Pulmonary heart diseases and hypertensive heart diseases

Heart diseases resulting from a lung (pulmonary) disorder. A complication of lung problem where blood flow into is showed flow in the lungs is showed and blocked caused increases blood pressure. The right side of heart has to pump harder to push against the increased

pressure and this can lead to enlargement of heart muscle and problems. Ultimately, congenitive heart failure of the right side (Grover, 2006).

2.2.8 Rheumatic heart diseases

Rheumatic fever is frequently classified as a connective tissue because its anatomical hallmark is damage to collagen fibrils to ground substance of connective tissue (Esp. Heart). Like streptococcal sore throat, ARF occurs most commonly in young school child, median age between 9 to 11 years and very rarely in early infantry (Gene *et.al.*, 1989).

The aetiology and pathogenesis of RF and RHD remain incompletely understood. RF is thought to be an exaggerated immune response to GAS (Kumar *et.al.*, 2005). Antibodies directed against the M-proteins of GAS cross-react with glycoprotein antigens in the heart, joints and other tissues, causing an acute inflammatory response (Carapetis, *et.al.*, 2007). Only a minority of GAS patients develop RF, however the reason for this remains unknown (Bryant *et. al.*, 2009). Diagnosis is based upon a Jones criteria (See Appendix C). As RHD progresses into heart failure, many patients will require heart valve repair or replacement within 5-10 years (Carapetis 2005 and Kumar *et. al.*, 2005)

2.3 Pathogenisity of *Streptococcus pyogenes*

Streptococcus pyogenes causes pyogenic infection with a tendency to spread locally, among lymphatics and through through the blood serum *Streptococcus pyogenes* causes

a. Respiratory infections

The speices causes acute sore throat (tonsillitis and pharyngitis) and peritonsillar abscess (quinsy). Tonsilitis is common in young adults. ‘Scarlet fever’ is an other form of upper respiratory infection that is caused by erythogenic toxin produced by *Streptococcus pyogenes*. This causes characterisic erythematous rashes. It is uncommon in tropics (Ochei and Kolhatkar, 2000)

b. Ear infections

Otitis media and mastoaid it is are supportive complications as a result of spread of organism from the throat (Ochei and Kolhatkar, 2000).

c. Skin infections

Streptococcus pyogenes causes a variety of skin infections, wound infections, cellulitis, burns, erysipeals and impetigo (Ochei and Kolhatkar, 2000).

d. Septicaemia

e. Endocarditis

- f. Abscesses
- g. Non-supportive complications

ARF and acute glomerulonephritis are two important complications of *Streptococcus pyogenes* infections. These two sequelae set in 1-3 weeks after the acute infection, the lesions are thus sterile and the organisms no longer detectable.

- Acute glomerulonephritis is the inflammation of kidney due to the deposition of the immune complexes in the glomeruli. It is a post skin throat infection condition.
- RF is a post respiratory streptococcal infection condition. It is a serious complication that may lead to chronic RHD if there is damage to the heart valve (Ochei and Kolhatkar, 2000).

2.4 Toxins produced by *Streptococcus pyogenes*

Streptococcus pyogenes produce several enzymes and exotoxins which enhance the virulence. These substances include:

- a. Haemolysins

These are of two types. They are haemolysin O and haemolysin S. Haemolysin O is oxygen labile (i.e., inactivated by oxygen) and haemolyses red blood cells under anaerobic condition. It is antigenic and stimulates the production of antistreptolysin O (ASO) in the serum.

Streptolysin S is oxygen stable, responsible for the haemolysis seen on blood agar under anaerobic condition. It is non-antigenic (Ochei and Kolhatkar, 2000).

- b. Streptokinase

Streptokinase (Fibrinolysin) causes lysis of fibrin. During infection, it breaks down the fibrin barrier around the lesions and so facilitates spread of infection (Ochei and Kolhatkar, 2000).

- c. Hyaluronidase

It breaks down the hyaluronic acid of the tissues. It favours the spread and the infection among the intercellular spaces. This enzyme is antigenic and specific antibody appears in convalescent serum (Ochei and Kolhatkar, 2000).

- d. Deoxyribonuclease

This causes hydrolysis of DNA and they are of four types, namely type A, B, C and D. Type B is the most antigenic. This enzyme helps to liquefy the pus and may

responsible and thin serous character of streptococcal exudates (Ochei and Kolhatkar, 2000).

e. Diphosphopyridineneucleotidase

It is known as the nicotinamide adenine dinucleotidease (NADase) acts as a coenzyme to release the nicotinamide by killing the leucocytes (Ochei and Kolhatkar, 2000).

f. Erythrogenic toxin

It causes erythematous skin rashes in scarlet fever. It produces local erythema on intradermal injection of small doses in man. It is heat stable and only inactivated after boiling for one hour. It is an antigenic protein that is specifically neutralized by the antibodies present in the convalescent serum. The basis of susceptibility and diagnostic test for scarlet fever (Ochei and Kolhatkar, 2000).

2.5 Prevalence of RHD in world

As mentioned earlier it is estimated that about 15.6 million people have RHD with 23,300 deaths caused by RF or RHD in each year (Carapetis, 2007). RHD causes 60% of the heart diseases in an aged group of 5-30 years old in developing countries and a common cause of death in this age group (WHO, 2004). RHD in terms of gender balance one study has reported that a ratio of male to female is 1.6:1 (Rayamajhi, Sharma and Shakya, 2007) whereas the another study found that the ratio was 1:2 (Rizvi *et. al.*, 2004). A 2005 systemic review, using population based data quoted that RHD prevalence in wealthy nation is 0.3 per 1000 and commonly seen in elderly ones (Carapetis, *et. al.*, 2005). High rates of RHD in some deprived communities, such as Samoan children in Hawaii (2.06 per 1000), Srilankans (1.42 per 1000) and Maori children in Auckland, New Zealand (1.25 per 1000) has been reported at the end of last century (Stollerman, 1997). RHD is reported as the commonest heart disease in most populous developing country like China (Dang *et. al.*, 1994).

2.6 Prevalence of RHD in Nepal

In the developing countries, which account for approximately two third of world population, rheumatic fever (RF) and rheumatic heart disease (RHD) are responsible for almost half of the cardiovascular diseases in all age groups and are leading causes of cardiovascular deaths in first five decades of life (Limbu and Maskey, 2001). And According to Sahid Gangalal National Heart Hospital, RHD was the maximum number of

surgery done in Medical Intensive Care Unit (MICA) i.e, 29.7% of the total surgeries (SGNHC, 2010).

The incidence of RHD among school children is reported 1.35 per thousand in rural community of the hill region (Shrestha, Bhattarai and Pandey 1991) and 1.2 per thousand in Kathmandu city (Regmi and Pandey 1997). Another study found that RHD represents 26% of cardiac cases in Pokhara, Nepal (Ramacharan , Alukar and Thapaliya, 2006). It is worthwhile noting the uncertain accuracy of many RHD prevalence studies in developing countries (Steer, *et. al.*, 2002)

2.7 Prevention to RHD

Primary, secondary and tertiary prevention methods for RF and RHD have been developed. The WHO has developed an RF/RHD action plan with the following objectives:

- a. RF and RHD primary prevention activities
- b. RF and RHD secondary prevention activities
- c. Personnel training
- d. Health education through schools and mass media
- e. Epidemiological surveillance to evaluate disease burden
- f. Programme-related research

There are several challenges for prevention programs. Effective programs must compete for funding with communicable diseases, and the growing burden of adult coronary heart disease (Padmawati, 2001). Further difficulties appear in tailoring programs to local circumstances, and ensuring equitable access (WHO, 2004 and McLaren *et. al.*, 1994). In addition, lack of knowledge and awareness about RF and RHD delays seeking healthcare, particularly among the less educated (Kasamaei *et. al.*, 2008). Currently, there is no broad international strategy, and few national programs (McDonald *et. al.*, 2005).

However, there are several challenges for prevention programs. Effective programs must compete for funding with communicable diseases, and the growing burden of adult coronary heart disease (Padmavati, 2001). Further difficulties appear in tailoring programs to local circumstances, and ensuring equitable access (WHO, 2004 and McLaren *et. al.*, 1994). In addition, lack of knowledge and awareness about RF and RHD delays seeking healthcare, particularly among the less educated (Kasmaei, 2008). Currently, there is no broad international strategy, and few national programs (McDonald *et. al.*, 2005). While the ultimate solution appears to be a vaccine for GAS, there are several problems hampering its development, the principal being research funding. The vaccine must cover a

complex mixture of M-protein serotypes, and not cause immunological cross-reactivity with human tissues (WHO, 2004, McLaren, 2004 and Guilherme *et. al.*, 2006).

Distribution of vaccine is challenged by limited health infrastructure. However, if cost-effective, safe and widely available, a vaccine for GAS would be ideal (Gu J. *et. al.*, 1997). Genetic testing for susceptibility to RF is a future possibility (King *et. al.*, 2002), however this is unlikely to be sustainable in developing countries. Ultimately, addressing RF and RHD requires a two-fold approach: long-term focused prevention strategies, and broader socioeconomic progression, aimed to reduce GAS exposure (Marijon *et. al.*, 2008). However, environmental and socioeconomic circumstances are unlikely to change in the near future for Nepal (Meyer, 2009).

2.7.1 Primary prevention

Primary prevention of RF involves antibiotic treatment of GAS pharyngitis, to prevent an initial attack of RF. Antibiotic therapy of choice is penicillin: it is effective against GAS, inexpensive and available in most countries. However, there is debate about the cost-effectiveness and efficiency of primary prevention (WHO, 2004).

Generally, the literature questions the cost-effectiveness of primary prevention. One study estimated the cost of averting one death from RHD using primary prophylaxis at US\$40,920, and only US\$5,520 for secondary prevention. With limited resources in developing countries, this hardly seems practical. In contrast, a recent study endorsed primary prevention as the cost-effective option in India (Michaud, 2003).

Difficulties in diagnosing GAS pharyngitis further challenge primary prevention as a viable option. The problem is that only 20% of pharyngitis is caused by bacteria, with the rest being viral. And as we know, antibiotics are worthless against viruses. As a result, antibiotic therapy has questionable benefit, as it is only effective in 20% of cases (Carapetis *et. al.*, 2005). Furthermore, superfluous use of antibiotics causes bacterial resistance, and may cause unnecessary side effects (King, *et. al.*, 2001). In addition, clinical diagnosis cannot differ between bacterial and viral causes, yet in many developing settings clinical diagnosis is the only option, as microbiology laboratories are unavailable (Eisenberg, 1991).

To compound this problem further, between one- and two-thirds of patients with acute RF do not initially experience a sore throat, therefore they will not receive primary

Strategies to address these challenges have not been developed prophylaxis (Marijon *et al.*, 2008).

2.7.2 Secondary prevention

A Secondary prevention of RF involves continuous antibiotic treatment for patients with a past history of RF or RHD, to prevent recurrent GAS pharyngeal infection and RF (Padmavati, 2001). The WHO mandates secondary prophylaxis for all patients who have had RF, whether they have RHD or not. This involves intramuscular penicillin injections every three weeks (every four weeks for low-risk areas). While oral penicillin is an alternative, non-compliance with a daily regimen becomes an issue. Duration of treatment depends upon patient:

Table 2.1: Suggested Duration of Secondary Prophylaxis (WHO, 2004)
(* whichever is longer)

Category of the patient	Duration of Prophylaxis
Patient with proven carditis	5 years after last attack or until 8 years of age*
Patient with carditis	10 years of last attack or until 25 years of age*
More severe valvular diseases	Lifelong
After heart valve surgery	Lifelong

Secondary prophylaxis is targeted at high-risk patients. It is cost-effective and simpler to maintain (WHO, 2004), and has been proven to prevent streptococcal pharyngitis and recurrent RF (Carapetis *et al.*, 2008). The WHO and World Heart Federation have recommended secondary prophylaxis for more than twenty years (Carapetis, 2007).

Despite this recommendation, secondary prevention is difficult to sustain in resource-poor countries. At the population level, limited PHC resources are directed away from NCDs into high-priority communicable diseases and maternal care. For individuals, affordability is an issue. Long-term medication is a financial burden, creating issues with adherence. Additionally, international pharmaceutical companies peg global drug prices to the median G7 price, exacerbating affordability. Inherent inequities in health-care access for people in developing countries only compound these difficulties (Yusuf *et al.*, 20004).

2.7.3 Tertiary prevention

Tertiary prevention involves expensive medical and surgical management for chronic RHD, which is neither practical nor affordable in developing countries. Surgery is the least cost-effective approach for managing RHD, and has been likened to ‘attempting to mop up the water on the floor while leaving the faucet open (McLaren *et. al.*, 1994). Furthermore, RHD management uses up to 71% of the total national allocation for treating RF and RHD, leaving little left over for prevention programs. After surgery, patients require lifelong secondary prophylaxis and anticoagulation therapy. This is burdensome on the health system and poor families, and can be a cause of further poverty in itself (WHO, 2004)

2.8 Nutritional status

Nutritional status is the condition of health of the individual as influenced by the utilization of nutrients. It can be obtained by the correlation of the information obtained through a careful medical and dietary history, through a physical examination and appropriate laboratory investigation (Robinson, 1972).

Nutritional status is defined as the condition of the body resulting from the intake, absorption and utilization of food. It can be measured directly (Burk, 1984 and Calendo, 1979).

Poor early childhood nutrition may increase susceptibility to RF. One study found that protein-calorie malnutrition causes lymphopaenia, altered cell-mediated and antibody responses and thymic atrophy that may prime the body for RF. Of course, poor nutrition is linked to poor socioeconomic status, which confounds the relationship (Steer *et. al.*, 2002).

2.9 Factors affecting nutritional status

Good health depends upon the adequate food supply and then in turn, a social agricultural policy and good system of food distribution. The social, economical and agricultural factors that determine the food supply also determine the state of health and the incidence of diseases amongst the population. These are the basic ecological factors causing nutritional diseases and they are closely linked with the dangers, which arise from the failure to control over the risk of population. In spite of good food supply and preparation of food in the home, lack of nutritional education is responsible for much malnutrition, especially in rural area and urban slums (Davidson and Passmore, 1986).

Hence the factors influencing the nutritional status are food availability and its distribution system, consumption, income and purchasing power, price of commodities,

illiteracy, family size, socio-cultural belief, environmental sanitation, health facilities etc (Bocobo 1988; Eusebio, 1988 and Somogy, 1979).

2.10 Assessment of nutritional status

The assessment of nutritional status may be required to encompass nations of individuals. It may be done as a part of an exercise to document current status can be done using the following information (Chandran, 1987).

The assessment of the nutritional education can be done using the following information (Chandran, 1987).

- a. Direct parameters e.g., dietary intake, anthropometric measurement, clinical measurement, biochemical and biophysical parameters.
- b. Indirect parameters like e.g., morbidity and mortality experience in community especially age specific mortality rates.

The nutritional status of an individual or community is affected by socio-economic and ecological factors. Therefore these factors are likely to serve as useful indirect indicators of nutritional status. The impact of these factors on health and nutritional status has been evaluated through data on dietary intake, anthropometric indices and clinical signs of nutritional deficiencies biochemical and biophysical parameters are considered to be the direct indicators of nutritional status of and are widely used in nutritional survey. Under the nutrition is associated with increased susceptibility to morbidity and consequent mortality. A variety of vital statistics such as maternal, paternal, infant and have therefore been considered as indirect indicators of nutritional community (Chandran, 1987).

2.10.1 Diet surveys

Different approaches have been made depending on the aim of the survey (Swaminathan, 1993).

- a. Questionnaire Method
- b. 24 hour Food Recall
- c. Food List Method
- d. Food Inventory
- e. Weighment of raw foods
- f. Weighment of cooked foods
- g. Food composite Analysis Methods

2.10.2 Anthropometry (Anthropometric examination of malnutrition)

Anthropometry, a technique developed in the late 19th century developed by anthropologists, uses simple measuring devices to quantify differences in human form. The propose of anthropometric measurements is to quantify the major compositional determinants of body weight. A full appreciation of anthropometric measurements understandings in human body composition and its organizational levels. The anthropometric measurements in general use include body weight, stature, skinfolds, circumferences, and bone breadths (Shils, Olson and Shike 1994).

2.10.3 Biochemical examination of malnutrition

Biochemical parameters of nutrition are generally very sensitive and even mild conditions can be recognized. The parameters can be especially of importance as a warning that more severe forms of malnutrition can be developed. Because of lack of specificity of many clinical signs, a biochemical investigation could be of much help in specific type of nutritional deficiency (Nube, 1987).

2.11 Malnutrition

Malnutrition is widely prevalent in many parts of the world. Malnutrition especially under nutrition is the most serious human health and social problem that affects majority of the population of the developing countries like Nepal. Malnutrition has recently been defined as a pathological state resulting from a relative absolute deficiency or excessive intake of one or more essential nutrients. It may be due to the inadequate food intake, defective absorption, poor dietary habits, food faddisms etc (Jelliffe, 1966). Under nutrition is the pathological state resulting from the consumption of an inadequate quantity of food over an extended period of time (Jelliffe, 1966; Swaminathan and Bhagwan, 1976).

2.11.1 Causes of malnutrition

The causes of malnutrition are many and they are often interrelated. Malnutrition never occurs alone (Birch, 1972). One basic condition which is always present is an inadequate diet. This most frequently results from poverty and the lack of ability to buy enough food. Other factors are low production and use of foods of low nutritive value, the unavailability of nutritious foods, and the lack of understanding about the relation of food to those who need it (Maxine *et al.*, 1971). Malnutrition is also largely the result of interplay of social and economic factors, including national policies interrelating and impinging upon one another. Among these factors are poverty, low food availability, poor food distribution

systems, large family size, inadequate income and low purchasing power, high prices of commodities, erroneous food practices that stem from certain cultural or religious beliefs, compounded by lack of basic knowledge of food and nutrition and poor environmental condition which increase the risk of infections and parasitic infections (Bacobo, 1988).

2.11.2 Forms of malnutrition

2.11.2.1 Under nutrition

The pathological state resulting from the consumption of an inadequate quantity of food over an extended period of time (Jelliffe, 1966)

2.11.2.2 Over nutrition

It is the pathological state resulting from the consumption of an excessive quantity of food and hence a calorie excess, over an extended period of time (Jelliffe, 1966).

2.11.2.3 Specific deficiency

It is the pathological state resulting from a relative or absolute lack of an individual nutrient (Jelliffe, 1966).

2.11.2.4 Imbalance

The pathological state results from a disproportionate consumption of essential nutrients with or without the absolute deficiency of any nutrients as determined by the requirements of a balanced diet. (Jelliffe, 1966)

2.12 Nutrition deficiency disorder

2.12.1 Protein energy malnutrition

Protein-energy malnutrition is the most important nutritional disease in developing countries because of its high prevalence and its relationship with child mortality rates, impaired physical growth and inadequate social and economic development. PEM occurs more in frequently when infectious diseases additional demands, induce greater losses of nutrients or produce metabolic alternations (Shils, Olson and Shike 1994). The resulting condition from the deficiency of protein predominant foods during growth period is known as Kwashiorkor and Protein-energy (calorie) malnutrition is known as marasmus (Begum, 1989). Four syndromes associated with protein deficiency in the human are described by Food and Nutrition Board: hunger edema, pellagra, Kwashiorkor and nutritional liver disease (Eva *et al.*, 1971). The marasmic child is often less than one year old. He has

severely retarded growth and a low weight for age-usually below 60% of standard weight. There is little or no subcutaneous fat, so the skin is loose and seems to be too big for the body. Nearly always the infant looks like an ‘old man’ or has a ‘monkey face’. The muscles are markedly wasted. They are flabby; this can easily be felt on the thighs and buttocks where the muscles should be thick and strong. There is no odema and no changes in hair colour (Cameron and Hofvander, 1983).

Kwashiorkor occurs most in children aged from one to three years. Growth is retarded and although the muscles are wasted and flabby, there is usually more subcutaneous fat than marasmic children. There is always odema, the child appears ‘moon faced’ and the hair often turns red brown or grey (Cameron and Hofvander, 1983). Three types of protein-energy malnutrition in children are shown in Table 2.2:

Table 2.2 Types of protein energy malnutrition

Type	Appearance	Cause
Acute malnutrition	Wasting or thinness	Acute inadequate nutrition leading to rapid weight loss or failure to gain weight normally
Chronic malnutrition	Stunting or shortness	Inadequate nutrition over long period of time leading to failure of linear growth
Acute and chronic malnutrition	Underweight	A combination measure, therefore, it could occur as a result of wasting, stunting, or both

Source: (Manary and Sandige, 2008)

Acute malnutrition is differentiated as moderate or severe. Moderate malnutrition is defined as a weight for height z score between 2 and 3 standard deviations (SD) below the mean. Severe malnutrition is defined as the weight for height z score more than 3 SD below the mean, or an arm circumference <110 mm, or the presence of nutritional oedema. Moderate or severe malnutrition without nutritional oedema is termed marasmus, and malnutrition identified as severe due to the presence of oedema is termed kwashiorkor. Kwashiorkor is associated with a higher mortality rate than marasmus (Manary and Sandige, 2008).

Severe malnutrition may also be categorised as uncomplicated or complicated. The most common complication is systemic bacterial infection. Clinical features associated with complicated malnutrition (Manary and Sandige, 2008):

- a. Fever related to systemic infection, typically Gram negative coliforms such as *Escherichia coli* and *Klebsiella pneumoniae*
- b. Respiratory distress
- c. Heart failure
- d. Electrolyte derangements, including hypophosphataemia, hypokalaemia, hypoglycaemia
- e. Marked anorexia
- f. Anaemia
- g. Profuse diarrhoea
- h. Shock

2.12.2 Calcium deficiency disorder

Normally, the poor diet is inadequate in both calories and calcium but the young children may not show any obvious evidence of calcium deficiency because of stunted growth resulting in decreased calcium requirement. Also a deficient supply of calcium may also lead to reduction in food intake and stunted growth. Occasionally a fast growing may have an adequate calorie supply combined with a deficiency of calcium and this results in poor skeletal growth and an uneven gait. Symptoms such as bowlegs and flat feet become exaggerated during this period (Rajalakshmi, 1987).

The defects of calcium deficiency are:

- a. Decreased rate of growth.
- b. Negative calcium balance.
- c. Loss of calcium from bone leading to the development of osteoporosis.
- d. Hyperplasia (a diffuse overgrowth) of parathyroid glands, and
- e. Hyperirritability and tetany leading to death.

2.12.3 Vitamin A deficiency disorder

Every year, vitamin 'A' deficiency (VAD) curtails the lives and hopes of millions of people worldwide. VAD affects as many as 256 million children in more than 75 countries (Micronutrient Initiative, 1997; Subbulakshmi and Naik, 1999). VAD is usually associated with protein energy malnutrition. It affects the eyes most commonly and the condition is

called 'Xerophthalmia'. The most common symptoms of VAD are night blindness, conjunctival xerosis, bitot's spots, corneal xerosis, corneal ulcerations, kerotomalacia (Cameron and Hofvander, 1983). Of the quarter to half million who go blind each year, about two-third die shortly after, often within weeks (Micronutrient Initiative, 1997).

2.12.4 Iron deficiency anemia (IDA)

Iron deficiency is the world's most common nutritional disorder. An estimated 2 billion people are anemic, with nearly 3.6 billion iron deficient. IDA leads to impaired work performance and deficits in learning ability (Subbulakshmi and Naik, 1999). Although, not confirmed by research in Nepal, IDA likely affects children's level of participation in school and recreation. It is possible that children with iron deficiency are most susceptible to infections, since impairment is present (UNICEF, 1996).

Iodine deficiency is the most common cause of endemic goiter and cretinism in the world. Endemic goiter is one of the most prevalent nutritional deficiency problems that afflicts millions of people in many parts of the world severe iodine deficiency can produce myxedema which is characterized by a dry, waxy type swelling, with abnormal deposits of mucoproteins under the skin. Cretinism may occur when there is an insufficient supply of iodine to infants and young children resulting in lowered BMR, dwarfism and retarded mental development. Excessive dietary intake of iodine results in inhibition of thyroid hormone synthesis clinically known as the Wolff-Chaikoff effect. Hyperthyroidism resulting from excessive iodine intake is characterized by increased basal metabolism, goiter and disturbances in the autonomic nervous systems causing hyperirritability and increased creatinine metabolism (Yeung and Laquatra, 2003).

2.13 Prevention of deficiency disorders

The weaning period is a crucial stage in the growth and development of the infant and child. The timing of weaning, the choice of foods, their methods of preparation, and how weanlings are fed, all affect the outcome. The commercial preparation of weaning foods and the fortification of some traditional foods are seen by some as the most sustainable and cost-effective means of alleviating micronutrient deficiencies among infants and children. This may be true in industrialized countries, but the same cannot necessarily be said of poor, developing countries. Demonstrates that, even in poor communities, it is possible to combine scant food resources in a cost-effective way to formulate multimixes which would meet energy, protein and micronutrient needs, without fortification. Proposes that such

approaches can be used in community nutrition education programmes to help reduce childhood malnutrition and in emergency feeding programmes (Amuna *et al.*, 2000).

According to Swaminathan (2004), the main principles of prevention are to ensure:

- a. An acceptable and readily digestible diet rich in proteins, calories and supplying all
- b. other dietary essentials in required amounts, and
- c. Treatment of any bacterial and parasitic infection presents.

According to Swaminathan (2004), there are two ways of improving the nutritive value of dietary protein. They are:

- a. By blending two or more proteins so that the excess of essential amino acids present in one protein make up the deficiency of some amino-acids in another protein, and
- b. By supplementation of dietary proteins with limiting essential amino acids.

The following are the ways to prevent the specific micronutrients deficiency (UNICEF, 1998).

- a. Improving dietary diversity: this can be done by increasing the production and consumption of micronutrient rich foods, especially green leafy vegetables, fruits and animal sources which are rich in micronutrients.
- b. Food fortification: This includes adding iodine to salt, vitamin 'A' to fats, weaning food formulas and iron to rice, salt, wheat, sugar and other like weaning food formulas.
- c. Supplementation: This can be done by giving vitamin 'A' capsule, iron tablets and by giving iodized oil and salt.

2.14 Proteins

The word protein means necessary for life because it is vital part of nucleous. Protoplasm of every cell is made of protein only (Bistran *et al.*, 1976). Protein has been recognized as a dietary essential well over a century. Protein performs many different functions in the body (Guthrie and Picciano, 1989).

The main functions are to serve as the following:

- a. structural components of body tissues (especially in muscles, cartilage, and bones),
- b. enzymes and hormones,
- c. components of the immune system,

- d. transporter of other substances,
- e. membrane-bound carriers, and
- f. regulators of many bio-chemical processes

The level of protein intake required for adequate maintenance has been worked out by an expert group from FAO and world health organization (WHO). According to them, the recommended daily intake is 0.59 gm of protein per kilogram of the body weight for an average individual. The daily requirement for protein remains constant during life times of most adults (Newsholme and Crabtree, 1985). FAO suggest a daily intake of 0.88g of protein per kg body weight for children in the range of 1 to 10 years (Berry and Torun, 1989).

Protein is a source of amino-acids and nitrogen needed for the synthesis of the body. Adequate protein intake is particularly important during period of growth or recovery from the disease. The primary role of the dietary protein is to supply amino acids for biosynthesis, but it can also be used as a source of energy. Although adequate intake of protein is essential, excessive intake should be avoided (Guthrie and Picciano, 1989). During disease and injury which cause breakdown of tissue protein and increased excretion of nitrogen more protein is taken to compensate loss (Alexender, 1993).

2.14.1 Source of protein

According to King and Burgess (1992), there are mainly two types of proteins, these are;

- a) Animal source: Most animal protein contains almost the same proportion of each amino acid as human protein. Because of these proteins in egg, milk, meat and fish are called complete food protein.
- b) Plant source: Protein in plants often contains much smaller amounts of one or more essential amino acids than proteins. For e.g.: the bean protein has less methionine and the maize protein has less lysine than egg or human proteins. Proteins for plants are called partially complete or incomplete proteins depending on the contents of limiting amino acids on them.

Almost all the protein in the meal can be used to make the human protein (King and Burgess, 1992).

2.15 Lipids

The term lipid refers to a group of substances that includes triglycerides, phospholipids and sterols. Fatty acids are key building blocks of triglycerides and phospholipids. Fatty acids

are carbon chain of varying length. Fatty acids filled with hydrogen are called saturated, while those with missing hydrogen are unsaturated fatty acids (Guthrie and Picciano, 1989).

According to Guthrie and Picciano (1989), triglycerides are the major lipid in the diet and in the body. Triglycerides add flavor and texture (and calories) to foods and are important source of energy.

Fat is a rich and efficient source of calories. Under normal circumstances ,dietary and stored fat supplied about 60% of the bodies resting energy needs, like carbohydrate, fat is protein-sparing; that is fat is burned for energy, saving valuable proteins for their important roles as muscle tissue, enzymes, antibodies and the like. During physical activities, glucose and glycogen join fat in supplying energy to muscle (Guthrie and Picciano, 1989).

2.16 Carbohydrates

Chemically carbohydrates are organic compound, composed of carbon, hydrogen and oxygen in the form of simple carbohydrates or sugar. When linked together, these simple sugars form three sizes of carbohydrates: monosaccharide, disaccharides and polysaccharides. These three sizes of carbohydrates are divided into two classifications: simple carbohydrates (monosaccharide and disaccharides) and complex carbohydrates (polysaccharides).Both are valuable sources of carbohydrate energy. There are differences, however, between the health value of simple and complex carbohydrates found in food we consume. Although simple carbohydrates primarily provide energy in the form of glucose, fructose and galactose, complex carbohydrate may also provide fiber in addition to glucose (Anderson, 1977).

According to Guthrie and Picciano (1989), most people associate the term carbohydrate with “starchy”, “sugary” food and they are right to do so because the chemicals in the starch and the many types of sugars in nature are indeed all examples of carbohydrate. The three major types of dietary carbohydrate are starch, sugar and fiber. Many people regard starch and sugar as fattening and therefore to be avoided: refined sugar is commonly blamed as a cause of attention deficit disorder (ADD), or hyperactivity to consume to avoid constipation. There is some scientists’ basis for each of these beliefs, although it is often exaggerated. Carbohydrate is an essential dietary component, but if it is responsible for an energy intake in excess of need, it can cause weight gain. Only a small percentage of children with ADD are actually sensitive to sugar; and although a certain amount of fiber is

good thing, too much can cause problem. Health authorities in most developed countries are encouraging people to increase their intake of carbohydrate but to use sugar in moderation and to include adequate, but not excessive, fiber in their diet. The kind and amount of carbohydrate in the diet play a role in the prevention and/or treatment of tooth decay, diabetics, hypoglycemia and various other health problems.

2.16.1 Carbohydrate and consumption in cardiovascular diseases

Currently, little evidence suggests that dietary carbohydrates are involved in the origin of the cerebrovascular disorders, but they may have the role in the isechemic heart diseases. Type IV hyperlipidemia is associated with an increases incidence of coronary artery diseases. Because type IV hyperlipidemia the serum lipid, notably the triglyceride, level is dependent on the amount of dietary carbohydrate consumed (Nestel, Carroll and Havenstein, 1970).

It may be deduced that dietary carbohydrate can be etiologic significance in certain individuals. The type of dietary carbohydrate can alter the level of triglyceridemia in that the effect of dietary sucrose is greater than that of starch, with fructose; glucose mixture being similar to sucrose (Blum, Levey and Eisenberg, 1977).

Several epidemiologic studies and reviews had failed to find the sufficient evidence that sucrose is associated with the development of the coronary artery diseases in general population (M.R.C., 1970 and Grande, 1974). With the other variables such as raised blood pressure and smoking known to be associated with the coronary artery diseases it is not possible to ascribe any with any certainty etiologic role for sucrose or fructose in that condition. Complex carbohydrate intake, however, does not seem to be associated with atherosclerosis of these substances (Higginson and Pepler 1953).

2.17 Macronutrient minerals

The macro nutrient minerals, in order of their abundance in the body, and their percentage of body weight are as follows: calcium (1.5 to 2.2%), phosphorous (0.8 to 1.2%), potassium (0.35), sulphur (0.25), sodium (0.15%), chlorine (0.15%) and magnesium (0.05%). They each account for 0.05% or more of total body weight, satisfying the definition of macronutrient minerals. Those minerals present at level of less than 0.05% are defined as micro nutrient minerals., and the most abundant micro nutrient minerals (iron) account for a mere of 0.004% of body weight less than one tenth of the proportion accounted for by magnesium, the least abundant of the macro nutrient minerals. Thus there

is a clear separation between macro and micro nutrient molecule (Guthrie and Picciano, 1989).

2.17.1 Calcium (Ca)

Most people correctly associated calcium with bones, teeth and milk. This presumably reflects widespread publicity on the importance of providing children adequate calcium to support the growth of bones and teeth, with milk usually highlighted as one of the best dietary sources of calcium. The link between calcium and bones has been further emphasized by growing awareness of its role in preventing osteoporosis, a bone disease that most often affects women after menopause. In addition to its role in development of bones and teeth, however calcium also performs a variety of other functions in the body, particularly as a regulator of many of the body's biochemical processes (Insel *et al.*, 2003).

No adverse effects have been found with an ingestion of calcium supplements providing as much as 60mmol (2400 mg) daily, except constipation in some individuals. Daily intakes above 60mmol may impair renal functions. Calcium supplements carry the increased risk for stone formation in normal adults, but may do so in patients with absorptive or renal hypercalciuria, primary hyperparathyroidism and sarcoidosis. It should be recognized it should also be recognized however that the efficiency of absorption from large doses is relatively poor (Shills, Olson and Shike 1994).

2.17.2 Magnesium

Magnesium plays an essential role in a wide range of fundamental cellular reactions hence it's not surprising that the deficiency of the organism may lead to serious biochemical and symptomatic changes. Magnesium is involved in many enzymatic steps in which foods are metabolized and new products are formed. Magnesium is the most abundant mineral cation in the cells and second in quantity to potassium (Shills, Olson and Shike 1994).

The adult human contains about 25 g of magnesium. The average intake from the diet by adults is about 300-400mg (Swaminathan, 2004).

2.18 Micronutrient minerals

The micronutrient minerals are also known as the trace elements because they are the mineral elements present in the body in small amounts. Some micronutrient minerals, such as iron and iodine, have been known for many years to be dietary essentials. Others have been detected in the body and established as dietary essentials only recently, thanks to the

development of more sensitive techniques of chemical analysis. Nine of the trace elements (manganese, molybdenum, cobalt, chromium, silicon, vanadium, nickel, arsenic and boron) are present in the body in such small amounts that they are also known as ultra-trace elements (Anderson and Barkrve, 1970).

2.18.1 Iron (Fe)

Iron is required as a component of blood hemoglobin, which carries oxygen, and muscle myoglobin, which store oxygen. Of all required nutrients, shortage of iron may be the most common inadequacy in the diets of the industrialized world (Potter and Hotchkiss, 2006).

Iron deficiency is one of the most prevalent nutritional problems in the world. Sectors of the population most susceptible to this problem are infants and young children, menstruating females, pregnant women and individuals on energy restricted diets and older people. WHO estimates over 1 billion individuals suffer from iron deficiency (Yeung and Laquatra, 2003).

Copper aids in the utilization of iron and in hemoglobin synthesis. The need of iron and copper is related to the rate of growth and to blood loss. Much of the iron in plant foods is bound in poorly soluble iron phytate and iron phosphates and is not bioavailable. Iron from animal sources generally is more readily absorbed in digestion, as is iron from soluble salts used in food enrichment and fortification (Potter and Hotchkiss, 2006).

2.18.2 Iodine

Iodine is an important trace element. The paper of the French physician Goindet (1820) marks the beginning of the use of iodides in the treatment of goiter (Swaminathan, 2004).

Iodine is part of the thyroid hormone and is essential for the prevention of goiter in humans. There is never a shortage of iodine where saltwater fish are eaten. The central United States and parts of South America, away from the ocean, are short of indigenous iodine. Today, the common use of iodized salt prevents deficiency, and in the United States there is concern that iodine levels not become excessive (Potter and Hotchkiss, 2006).

UNICEF (1998) reported that iodine deficiency disorder also caused poor eye- hands co-ordination, partial paralysis, deaf-mutism, dwarfism, facial and physical deformity, cretinism, neurological damage, goiter and lassitude. Since iodine is essential during foetal development, babies born to iodine- deficient mothers can be impaired irreversibly.

UNICEF (1998) reported, 40% of population showed signs of goiter in Nepal.

2.19 Vitamins

The vitamin concept has undergone extensive revisions during the history of biochemistry. However Hofmeister's definition- 'vitamins are substances which are indispensable for the growth and maintenance of the animal organisms, which occur both in animals and plants and are present in only small amount in food'- still holds good, although it has been interpreted in various ways (Jain *et al.*, 2005).

The term 'vitamin', in its modern sense, usually refers to the substances distinct from major components of food, required in minute quantities (i.e. oligodynamic in nature) and whose absence causes specific diseases. As the living organisms cannot synthesize most of these compounds, a steady supply of them is *sine quo non** (*Latin phrase, which means an indispensable condition) for life. Their ultimate source is the plant or bacterial world (Jain *et al.*, 2005).

2.19.1 Vitamin A

Vitamin A is needed for the normal function of eye including the ability to see in dark. Vitamin A also has other functions: it keeps the skin and other body surfaces healthy by preventing them from becoming dry and it is needed for normal bone development. Vitamin A as retinaldehyde or its active form retinol is necessary for integrity of epithelium tissue and functioning of retina and vision (Cameron and Hofvander., 1983)

According to Manay and Shadaksharaswamy (2001), Vitamin A is found in animal materials like meat, milk, fish, etc. In animals, the vitamin is found in highest concentration in the liver, where it is stored and exists generally as a free alcohol or its esterified form. Plants do not contain vitamin A, but contain its precursors (provitamin A), the carotenoids, which is converted to vitamin A after absorption by the ingesting animal. Carotenoids are the orange and yellow pigments of fruits and vegetables. Green leafy vegetables also contain carotenoids; in these the green color of chlorophyll masks the yellow carotenoids.

Vitamin A is an alcohol (C₂₀H₂₉OH). It has been named retinol because of its specific function in the retina of the eye. Metabolically active forms of the vitamin include the corresponding aldehyde (retinal) and the acid (retinoic acid) (Manay and Shadaksharaswamy, 2001).

Vitamin A is fairly stable to heat, but prolonged heating in contact with air destroys it. It is easily destroyed by oxidation and ultraviolet light. Fats and oils lose their vitamin

content by oxidation as they become rancid. Antioxidants prevent the loss of vitamin A by oxidation (Manay and Shadaksharaswamy, 2001).

2.19.2 Vitamin D

Vitamin D is a group of lipophilic hormones with pleiotropic actions. It has been traditionally related to bone metabolism, although several studies in the last decade have suggested its role on cardiovascular diseases, diabetes, malignancies, autoimmune diseases and infections (Lauretani *et. al.*, 2009)

Since vitamin D is fat soluble, it is readily taken up by fat cells. Then, vitamin D₃ and vitamin D₂ are hydroxylated to 25 (OH) vitamin D (or calcidiol or calciferol) by several tissue (mainly by the liver) and hydroxylated in the kidneys to the active form. 25(OH) vitamin D is further hydroxylated to the active form of vitamin D₃ to 1,25 (OH)₂D (or calcitriol). 1,25(OH)₂ Vitamin D produced by the kidneys enters into circulation and travels to its major target tissues such as the intestine and bone, where after interaction with its receptor enhances intestinal calcium absorption and modulates the osteoclast activity.

The deficiency of vitamin D is osteophoresis, rickets and osteomalacia. Depending on the intake of vitamin D the deficiency occurs. Adequacy of vitamin D may lead to osteophopresis where as deficiency leads to rest of others (Shils, Olson and Shike 1994).

2.19.3 Vitamin E

Vitamin E is a fat soluble antioxidant capable of protecting fatty acids by interrupting free radical reactions that otherwise can cause membrane damage in sub-cellular organelles. Natural α -tocopherol as found in food is [d]- α -tocopherol where as chemical synthesis produces mixtures of eight epimers (Bieri and McKenna, 1981). The richest dietary sources of the tocopherols are the vegetable and cereal seed oils. Wheat germ is particularly rich in alpha- and beta-tocopherol. Other excellent sources are soybean oil (alpha, beta and gamma forms), cottonseed oil (alpha and gamma), corn oil (mostly gamma), sunflower seed oil and margarine. Overall, gamma-tocopherol has a greater distribution than alpha-tocopherol in the vegetable oils, margarines and shortenings (Yeung and Laquatra, 2003).

Some of the vitamin E deficiency is hemolytic anemia in abnormality of premature infants but severe anemia is unlikely to occur in malabsorption, it is also associated with

the disturbance affecting nearly every component of gastrointestinal tract including stomach, liver, pancreas and intestinal mucosa (Shils, Olson and Shike 1994).

2.20.4 Vitamin K

Vitamin K was discovered by Henrik Dam in Copenhagen in 1929 in studies of sterol and metabolism in chicks and fat free diets (Dam, 1929). In the early 1930's Dam and his coworkers extended their work to show that none of the established vitamin could prevent the hemorrhagic disease and named new called vitamin **K** (for *Koagulation*) (Dam and Schonheyder 1934; Dam 1935). In protein calorie malnutrition, amino acids deprived may cause hypoprothrombinemia that is not responsive to vitamin K but that does respond to protein feeding (Damrog, 1975). Other deficiencies of vitamin K are malabsorption syndrome and other gastrointestinal disorders (Shils, Olson and Shike 1994).

Large doses of menadione may be toxic. Symptoms of vitamin K toxicity include liver damage, hypoprothrombinemia, petechial hemorrhages, renal tubule degeneration, and, in the premature infants, hemolytic anemia (Yeung and Laquatra, 2003).

2.20.5 Vitamin B Complex

Vitamin B complex composes of thiamin, riboflavin, niacin biotin, pantothenic acid, folic acid, vitamin B₆ and Vitamin B₁₂.

2.20.5.1 Thiamin

Thiamin is composed of a pyrimidine and a thiazole ring. It performs important biochemical functions as a coenzyme thiamin pyrophosphate (TPP) which is involved in energy metabolism. Thiamin is also present in tissues, as thiamin monophosphate and thiamin triphosphate. Thiamin is readily soluble in water and alcohols. It is stable in acid but is unstable to heat, oxidation and an alkaline pH. Thiamin deficiency affects the cardiovascular, muscular, nervous and gastrointestinal systems. The earliest manifestation of thiamin deprivation includes anorexia, fatigue, depression, irritability, poor memory, inability to concentrate and vague abdominal and cardiac complaints. Thiamin deficiency is known as beriberi. It is classified into dry, wet and infantile beriberi. Dry beriberi is characterized by peripheral polyneuritis, paralysis and muscle atrophy. In wet beriberi the clinical picture is, to a large degree, one of congestive heart failure with cardiac dilatation secondary to damage of the cardiac musculature, serous effusions and edema. A mild subacute form of beriberi has both neurological and cardiac manifestations. In infants, an

acute form of the disease, infantile beriberi, runs a fulminating course and terminates in cardiac failure (Yeung and Laquatra, 2003).

2.20.5.2 Riboflavin

Riboflavin is a yellow isoalloxazine compound. It is important biologically as a component of the flavoprotein enzymes riboflavin 5' phosphate (FMN) and flavin adenine dinucleotide (FAD). Riboflavin is relatively stable to heat, acids and oxidation but very unstable to light and ultraviolet radiation. Riboflavin deficiency is characterized by cheilosis, angular stomatitis, glossitis, seborrheic dermatitis and ocular manifestations: photophobia, itching, burning and circumcorneal capillary engorgement (Yeung and Laquatra, 2003).

2.20.5.3 Niacin

Niacin, also known as nicotinic acid, is the generic description of pyridine-3-carboxylic acid. In the human, ingested niacin, is readily converted to niacinamide (nicotinamide). Niacinamide is important biologically as a component of the coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). Niacin is stable to air, heat, light, oxidation, acids and alkalis and is not destroyed in ordinary cooking processes. Liver, meats, fish, whole grain and enriched breads and cereals, dried peas and beans, nuts and peanut butter are some sources of niacin. Niacin deficiency is known as pellagra which presents as the 4 D's-*dermatitis, diarrhea, dementia* and *death*. Early signs of niacin deficiency include fatigue, anorexia, weakness, mild gastro-intestinal disturbance, anxiety irritability and depression. A scaly, bilateral, pigmented dermatitis appears generally in areas exposed to sunlight. The mucous membranes of the mouth are usually involved: glossitis, stomatitis, soreness, a burning sensation and ulceration of the buccal cavity may be present. Severe, extensive diarrhea is another common feature of the disease. In advanced cases of pellagra dementia may manifest. Untreated pellagra will result in death (Yeung and Laquatra, 2003).

Both niacin and niacinamide are harmful when taken in large doses. Megadoses of niacin (3-6 g/day) produce a flushing and itching reaction. Pharmacological doses of niacin, but not niacinamide lower total serum cholesterol, LDL and triglycerides. However, they also produce increased incidence of arrhythmias, biochemical alterations and gastrointestinal disturbances. Thus, use of niacin as a pharmacological drug should only be done under strict medical supervision. Large doses of niacinamide do not produce the itching and

flushing reaction seen with niacin. Doses of 50-250 mg niacinamide have been used therapeutically in deficient subjects. Doses of 3-9 g have been reported to cause liver toxicity. Niacin, in the form of nicotinic acid (12.3 to 24.6 mmol or 1.5 to 3 g per day), reduces hyperlipidemia, since it reduces LDL cholesterol (“bad” cholesterol) and increases HDL cholesterol (“good” cholesterol) (Yeung and Laquatra, 2003).

2.20.5.4 Biotin

It is stable to heat, light and reducing agents, but is labile to oxidizing agents, strong acids and alkali. Yeast and organ meats such as liver and kidney are excellent sources of biotin. Good sources are soybeans, rice bran, peanuts, chocolate, egg yolk, cauliflower and mushrooms. Experimental biotin deficiency has been induced in humans by feeding a biotin deficient diet and large quantities of raw egg white. Raw egg white contains avidin, a heat labile protein which binds biotin rendering it unavailable for absorption. Symptoms of biotin deficiency include scaly dermatitis, grayish pallor, extreme lassitude, anorexia, nausea, anemia, depression, muscle pain, paresthesia, hypercholesterolemia and electrocardiogram changes. There is also decreased protein synthesis. Biotin deficiencies in humans have been reported in association with bizarre food habits, i.e. consumption of large quantities of raw egg whites and little else. Seborrheic dermatitis in infants under six months may be responsive to biotin administration. Certain biotin responsive genetic conditions involving defective carboxylase enzymes have been identified. Low biotin levels have been reported in pregnant women, alcoholics and persons with achlorhydria (Yeung and Laquatra, 2003).

2.20.5.5 Pantothenic Acid

Pantothenic acid is important biologically as a component of Coenzyme A (CoA) and as a prosthetic group of the acyl carrying protein (ACP); both of which participate in a number of cellular reactions. Pantothenic acid is stable to oxidizing and reducing agents but is readily destroyed by heat, acids and alkali. Pantothenic acid is widely distributed in plant and animal tissues. Good sources of pantothenic acid are organ meats, egg yolk, peanuts, legumes and whole grains. syndrome is characterized by abdominal pain and soreness, nausea, personality changes, insomnia, impaired adrenal function, weakness and cramps in the legs, paresthesia of the hands and feet and impaired antibody production are deficiency of pantothenic acid (Yeung and Laquatra, 2003).

2.20.5.6 Folic Acid

Folic acid may be conjugated with up to seven molecules of glutamic acid forming pteroyl-polyglutamic acid. The biologically active form of the vitamin tetrahydrofolic acid (THFA) functions as a carrier of one carbon units in biological reactions. Important sources of folic acid include liver and organ meats, yeast, dark green leafy vegetables, legumes, nuts, whole grain cereals, eggs and fruit (Young and Laquatra, 2003).

Folic acid is synthesized by the intestinal microflora of the colon but the biological significance of this source to humans is not clear. Folic acid is stored in the liver and other body tissues. Excretion of THFA and its derivatives occurs via the feces and urine. THFA is necessary for normal cell division and replication as it is needed in nucleic acid synthesis. The synthesis of both the purines and pyrimidines requires THFA. THFA is required for nucleic acid synthesis, folate deficiency first manifests itself in tissues with rapid cell turnover such as bone marrow (erythropoiesis), and gastrointestinal and oral mucosa. Normal maturation of red blood cells does not take place and hematopoiesis is inhibited at the megaloblast stage. A typical peripheral blood smear is characterized by megaloblastic macrocytic anemia, thrombopenia, leukopenia and old multilobed neutrophils. Glossitis, gastrointestinal disturbances, diarrhea, malabsorption and mood changes such as forgetfulness and paranoia may accompany the anemia (Yeung and Laquatra, 2003).

Doses of folate up to 15 mg/day have been reported with no apparent ill effects. Megadoses of folate reduce the effectiveness of certain anticonvulsant drugs but however Folate plays an important role in embryonic development by supporting normal cell division. Adequate folate status is linked to reduced risk of abnormalities in early embryonic development and specifically to risk of malformations of the embryonic brain and/or spinal cord. These include anencephalus, encephaloceles, and spina bifida, all of which result from incomplete neural development and collectively referred to as neural tube defects (NTD). Folic acid supplementation is thus important during the periconceptual and early-pregnancy stages in preventing neural tube defects and cleft palate/lip in the newborn. Currently the general recommendation for women is to consume at least 0.4 mg of folic acid per day before and during pregnancy (Yeung and Laquatra, 2003).

2.20.5.7 Vitamin B₆

Vitamin B₆ refers to the pyridines: pyridoxine, pyridoxal and Pyridoxamine. Pyridoxine is stable to heat and acid but is rapidly destroyed by light, particularly in neutral or alkali solution. Pyridoxamine and pyridoxal are labile compounds. They are rapidly destroyed by exposure to air, light and heat. Good sources of vitamin B₆ include meat, fish, poultry, organ meats, legumes, grains, certain fruits and vegetables. In animal products, vitamin B₆ is found largely as pyridoxal and pyridoxamine. In plants the main form of vitamin B₆ is pyridoxine. Vitamin B₆ is required for normal protein metabolism (Yeung and Laquatra, 2003).

The conversion of methionine to cysteine, and cysteine to taurine also involve vitamin B₆-dependent enzymes. Conversion of tryptophan to niacin is dependent upon pyridoxal phosphate as a coenzyme. Vitamin B₆ is required for proper functioning of the phosphorylase enzyme in glycogenolysis and in the formation of delta aminolevulinic acid in the synthesis of heme and in folate metabolism. Vitamin B₆ deficiency has been demonstrated in infants inadvertently fed vitamin B₆ -deficient formula. The signs included irritability and convulsions. Vitamin B₆ deficiency has been induced experimentally in adults fed B₆ -deficient diets in the presence of the vitamin B₆ antagonist deoxypyridoxine. Vitamin B₆ deficiency is characterized by seborrheic dermatitis, cheilosis, glossitis, stomatitis, altered tryptophan metabolism, depression and confusion. The incidence of vitamin B₆ deficiency in alcoholics may be as high as 20-30 %. Up to one-fifth of oral contraceptive users show biochemical evidence of vitamin B₆ deficiency. Daily administration of vitamin B₆ supplements has been reported to restore normal biochemical values. The underlying mechanism by which oral contraceptives affect vitamin B₆ metabolism has not been elucidated (Yeung and Laquatra, 2003).

A transient dependency has been induced in adults given a supplement of 200 mg pyridoxine hydrochloride per day for 33 days. Megadoses of 2-6 g/day for two months or more have been shown to cause ataxia and severe sensory-nervous system dysfunction in adults (Yeung and Laquatra, 2003).

2.20.5.8 Vitamin B₁₂

Vitamin B₁₂ is found almost exclusively in foods of animal origin. The original source of the vitamin is bacterial fermentation in the intestinal tract of animals. Only bacteria are capable of synthesizing vitamin B₁₂. Sources of vitamin B₁₂ include organ meats, fish, milk and eggs. Vitamin B₁₂ is not found in foods of plant origin unless they have been

contaminated by vitamin B₁₂-producing bacteria or vitamin B₁₂-containing substances, or fortified with vitamin B₁₂ (e.g. fortified ready-to-eat breakfast cereals). In the human, vitamin B₁₂ is synthesized by bacteria in the small intestine and may be absorbed. Synthesis of vitamin B₁₂ also occurs in the colon but is unavailable to humans (Yeung and Laquatra, 2003).

Vitamin B₁₂ is required for folic acid metabolism and normal cell division and replication. Vitamin B₁₂ is also required in the metabolism of some branched chain amino acids and odd-chain fatty acids (Yeung and Laquatra, 2003).

B₁₂ deficiency, folate deficiency may also result. Similarly, alterations in fast replicating tissues, such as glossitis and gastrointestinal disturbances, may occur. Preliminary research indicates that vitamin B₁₂ may be important in the prevention of some cancers and in the treatment of precancerous lesions (Yeung and Laquatra, 2003).

2.20.6 Vitamin C

Vitamin C participates in a number of important body functions. These include: (a) formation of collagen or the intracellular cement substance necessary for body growth, tissue repair and wound healing, (b) conversion of tryptophan to 5-hydroxytryptophan and in the formation of tyrosine from hydroxyl phenyl pyruvate, (c) synthesis of adrenaline and hydrocortisone, (d) iron and copper metabolism by maintaining them in their reduced forms, thus facilitating absorption, and (e) interaction with folic acid, niacinamide and possibly with vitamins A and D. It also prevents the formation of nitrosamine from nitrates found in some foods.

It is an antioxidant found in the blood and cytosol of cells, reduces the damage by the free radicals during oxidation process. Vitamin C, beta carotene and vitamin E may work synergistically in preventing cellular oxidation. Vitamin C protect the foliate , Vitamin E, and polyunsaturated substances from being destruction from the oxygen as they move throughout the body. Vitamin C and E work together as an antioxidant. Each organ or tissue has on optimal saturation level of vitamin C. Excessive intakes of vitamin C do not increase the saturation level but are excreted in the urine. Vitamin C plays the protective role in the disease resulting from the atherosclerosis (Swaminathan, 2004).

A great deal of research indicates a possible beneficial association of vitamin C and cardiovascular health, blood pressure or platelets. As well, vitamin C-rich foods may reduce the risk of developing cancer of the gut as well as enhancing immune function.

Vitamin C has also been associated with reducing the risk of cataracts and improving nonheme iron absorption and periodontal disease as well as being involved in wound healing and blood vessel formation (Swaminathan, 2004).

Part III

Material and methods

3.1 Material

Collection of material

a. Weighing balance

A weighing balance was provided which could measure the weight ranging from zero to 120.

b. Measuring tape

Measuring tape was provided from the campus in which could measure the height ranging from the zero centimeters to 200 centimeters.

c. Questionnaire

A questionnaire was prepared so as get the quantitative information. In the questionnaire the name of the patients was represented as patient id.

3.2 Methods

3.2.1 Research design

Nutrition survey of the RHD patients both pre operated and post operated of Dharan, Panchkanya, Bayarban, Chatara, Barahchhetra, Bhanjhyang, Nishane, Bharauli, Panmara were done considering the following parameters:

- a. Height and Weight of the RHD patients.
- b. Comparative study of daily dietary intake of the RHD patients before as well as the after the surgery of the patients
- c. Various effects on patients were studied.
- d. The discharge summary report of the post surgery patient was studied in the case of the post operative patients.
- e. The drugs that patient used was noted.

3.2.2 Nutrition survey

The nutrition survey of RHD patients of Dharan, Panchkanya, Bayarban, Chatara, Barahchhetra, Bhanjhyang, Nishane, Bharauli, Panmara aging from 24±13 year's interviewee consists of following things.

3.2.2.1 Selection of the patients

RHD patients was selected from the information's based on the community of the people, caste based organization like Dalit Welfare Organization and other organizations like Disabled and Helpless Rehabilitation, school visits in VDC's area and visits in squatter

area of Dharan and Chatara. Thirty post operative patients were interviewed for the quantitative and comparative research.

3.2.2.2 Household survey of RHD patients

In the household survey the living condition of the house in which the patient is staying was observed and noted. In the household survey the floor of the house, type of the house and number of family members in the family was noted.

Besides these the economical conditions, religion and literacy status of family was noted. Through the questionnaire the average monthly income of the family was noted. The monthly expenditure on medication was noted.

3.2.2.3 Sanitation condition of the area

Regarding the sanitation the type of toilet facility and the condition of the toilet facility was noted. Toilets were classified as open, temporary toilet and sanitary toilet. The drinking facility of water was also noted.

The major sources of waster such as river, municipal taps and hand pump are some of them. The process of taking water was also noted. Normally boiling and filtration are some of the processes.

Other sanitation conditions like washing the hands before the meal, after the toilet with soap etc were noted.

3.2.2.4 Height of the RHD patients

Height is one of major component of the anthropometric measurements. The height was measured with the tape for the patients who had completed surgery. For the comparative analysis the height of the patient was noted from discharge summary of SGNHC as the height they had measured before the surgery. This height was assumed to be height before surgery.

3.2.2.5 Weight of the RHD patients

In the weight also the weight was measured from the spring balance this was the weight of patients after the surgery. The weight was noted from the discharge summary of patient and this was assumed to be weight before the surgery. A sample of discharge summary is kept in appendix Q

3.2.3 BMI of the RHD patients

By measuring the height and weight BMI can be calculated from the following formula

$$\text{BMI} = \frac{\text{Weight in Kg}}{(\text{height in meter})^2}$$

After measuring the BMI the BMI was categorized as follows:

Table 3.1 Category of BMI

Classification	BMI category	Risk of Developing Health Problems
Underweight	Less than 18.5	Increased
Normal Weight	18.5-24.9	Least
Overweight	24.9-29.9	Increased
Obese Class I	30-34.9	High
Obese Class II	35-39.9	Very High
Obese Class III	≥ 40	Extremely High

3.2.4 NYHA category

According to the physical activities, comfortability of patients was classified into NYHA categories. It's just an index that how much the patients are as normal, moderate or severe. NYHA I and II are considered as the normal whereas the NYHA III category is the moderate and IV is considered as the severe. The NYHA category was noted from the discharge summary of SGNHC.

3.2.5 Calorific Intake

The 24 hour food recall method was used. The fruit, vegetables, meat, pulses and legumes was noted. From the interview the food consumption before the surgery as well as after the surgery was noted. From the questionnaire the fruits, vegetables, foods, meat etc was noted. The daily eaten staple foods such as rice, *dal*, *roti* etc are seen common in community of Nepalese. And also the daily eaten breakfast as well as the lunch was noted. Based on the food composition table data of the respected foods the food composition table was copied from DFTQC food composition table. The nutrient such as carbohydrate, fats, protein, vitamin A and riboflavin was calculated as they are major energy giving source. Besides these the daily vitamin intake of A and Riboflavin was also calculated from the

food composition table. The food composition table of the daily eaten foods is kept in Appendix D.

3.2.6 Drugs used by RHD patients after surgery

The drugs used by the patients were noted from the discharge summary of the SGNHC. Besides drugs the general advices for the patients was also noted down.

3.2.7 Statistical Analysis

Statistical analysis was done with help of SPSS 16.0. A paired sample t-test was done with the value before the surgery and after the surgery, at 95% level of the confidence. Then all the required tables of statistical analysis are kept in appendix E to appendix O.

Part IV

Results and discussion

4.1 General results

4.1.1 Sex

In the comparative analysis of post operative patients there were 13 male i.e., 43.3% of the total population and 17 i.e., 59.7% of the total population were females. So a general result was drawn that females were suffered then men. And female to male ratio was 1.3:1.

4.1.2 Distribution of sample

As mentioned earlier the research area was in Dharan and nearby VDC's area. Since the research area is focused in Dharan majority of participants were from Dharan. Besides this the distribution of the post operative patients are as follows:

Table 4.1 Distribution of the sample according to location

Location	Frequency	Percentage
Dharan	9	30
Panmara	4	13.3
Bayarban	4	13.3
Chatara	5	16.7
Barahchhetra	1	3.3
Bhanjyang	2	6.7
Bharaul	2	6.7
Panckkanya	1	3.3
Pakali	2	6.7
Total	30	100

4.1.3 Age

Age is also an important factor. The range of age was from 9 to 37 years old in which mean age were 21.67 with a standard deviation of 7.102. As seen in below table it is seen that maximum post operative patients were aged 22.

Table 4.2 Distribution of the sample according to age

Age	Frequency	Percent
9	1	3.3
11	2	6.7
13	1	3.3
14	1	3.3
15	3	10
17	1	3.3
19	3	10
21	1	3.3
22	5	16.7
23	1	3.3
25	2	6.7
26	2	6.7
27	1	3.3
28	2	6.7
29	1	3.3
34	2	6.7
37	1	3.3
Total	30	100

4.1.4 NYHA Classification

The patients were classified as their NYHA category as per the Sahid Gangalal National Heart Center. The descriptive analysis of NYHA classification of patients is given below. As seen below most of the patients were NYHA III categorized.

Table 4.3 Distribution of the sample according to NYHA classification

NYHA	Frequency	Percent
I	3	10
II	10	33.33
III	13	43.33
IV	4	13.33
Total	30	100

4.1.5 Caste

The main caste of the research area was Tamangs, Rai and Limbu, Dalits, Chhetri, Newar, Tamang, other terai caste (*Madhesis*) and Tharus. It was found that in the survey the Dalits were more as compared to other caste. The distribution of the sample according to caste is given below.

Table 4.4 Distribution of sample according to caste

Caste	Frequency	Percent
Brahmins	3	10.00
Chhetry	2	6.67
Newar	4	13.33
Dalit	10	33.33
Rai and Limbu	3	10.00
Muslims	1	3.33
Tharus	2	6.67
Tamangs	2	6.67
Magar	1	3.33
Other Terai Caste	2	6.67
Total	30	100

4.1.6 Religion

Religion is also another major factor. In the survey it was seen that all Dalits were belong to Christian religion. The sample distribution of the population according to religion is given below.

Table 4.5 Distribution of sample according to religion

Religion	Frequency	Percentage
Hinduism	11	36.67
Buddhism	5	16.67
Christian	10	33.33
Muslim	1	3.33
Kirat	3	10.00
Total	30	100

4.1.7 Literacy status

Most of the RHD patients were literate whereas the Brahmins, Chhetris, Newar and Tamang community patients had completed SLC level. It was also found that most of the postoperative patients were dropped out from education during the tertiary stage of disease. As normal it was found that most of the post operative patients completed their SLC in 20 years of age due to cardiac disability. None of the both type of patients had completed intermediate or higher level of the education.

4.1.8 House type

Most of the dalits were *sukumbashi* means they are living in government land nearby in Seoti river side, Sardu river side and Khare river side of Dharan. The structure of the house was temporary with whipped red mud as a floor.

In Chatara, Bayarban and Barahchhetra VDC's most of the patients were living in the rent and river area and talking about the house structure the houses were good with tin roof.

In the case of Tamangs, Brahmins, Chhetris, Newars Rai and Limbu community it was found that all patients were living in rent house with the cemented ceiling and the floor.

In the case of Madhesis, Tharus and Muslim communities they are living in their own houses but the house structure of their houses were primitive with tiles as a roof, whipped with the red mud.

4.1.9 Income source of the family

Income is also another parameter to judge the nutritional status. In the survey it was found that the family members of Brahmins, Chhetri, Newar, Tamangs and Magar community were job holders with an average earning of 9500 per month. The RHD patients from these communities don't do any physical works for earning money.

Similarly in the community of Dalits, Muslims, Tharus it was found that the major source of earning is wage based job in a construction site. Besides these the churches also provide some funding if they are really in need of. One of the Muslim patients was also quarterly funded for her operation during her surgery. The average daily wage is about 300 per day. The Dalits were also supported by the Dalit welfare Society. The major source of the income in Rai and Limbu community is agriculture. By selling the agriculture product they earn money. The average monthly income is 6500 per month.

4.1.10 Toilet facility

As seen in Dharan including the Dalits patients on squatter area it was seen that all family members of RHD patients used toilet facility. None of the patients use open defecation toilet. Although the toilet seen in the squatter area was a community toilet i.e., two or three families in the neighborhood uses the same toilet. The community toilet was built by the joint contributions of INGO's and people.

A similar scenario was seen in the Chatara, Bayarban and Barahchhetra there was a community toilet but sometimes the queue may be long so children go to the river side for the toilet.

In the case of the Muslim and Madhesis community it was seen that children go to the river bank area for the toilet. In the tharu community it was seen that although the toilet is primitive they don't use open deflection toilet. All the family members of the patients including them were found that they wash their hands after toilet and before meal use with soap.

4.1.11 Food availability and water supplies

The major fruits, vegetables, legumes, pulses, meat were noted and it is represented in the table below.

Table 4.4 Distribution of Food availability in research area.

Main Food	Food Available
Grains	Rice, maize, wheat, millet, etc
Fruits	Papaya, banana, guava, mango, litchi, sugarcane, cucumber, apple
Green Leaves	Mustard leaves, radish leaves, ferns
Roots and Tubers	Potato and sweet potato
Oil seeds	Mustard
Pulses	Black gram, cowpea, soyabean, gram,
Other Vegetables	Pumpkin, cauliflower, cabbage, brinjal, radish, carrot, etc.
Fermented Products	Kinema, gundruk, sinki
Meat Serve	Pork, chicken, mutton, fish, eggs, buff,
Liquid Foods	Milk, coffee, black tea, milk tea

Warfarin is the anti-coagulating agent. The RHD patients using warfarin are advised not to take citrus fruits or vegetables. They are also advised not to take leafy vegetables like cabbage and liver from the meat products. On the verbal communication with the co-supervisor he said that there may be the chances of coagulation of blood in brain which may results in paralysis if the intakes of vitamin C enriched foods are taken in very high amount. As the stable foods it was found that rice, dal and curry was stable, two times a day and milk tea or milk or black tea as breakfast item but in tharus, community it was found that they take rice, dal and curry thrice a day without no milk or milk tea. The Tharus communities take rice at morning, during the noon and at evening.

In Dharan, Bhanjhyang, Chatara, Bayarban, Panchkanya, Barahchhetra people are using the water sources given by municipality or VDC but in the terai region the sources of water are hand pump and river. In the area of water sources available by municipality or VDC the family members of RHD patients take water directly without boiling or filtering but they do boil when they suffer from communicable diseases like common cold, diarrhea.

In the area of Pakali and Bharaul, the major sources of water are hand pump and river. The river water was boiled and drink by the all community residing that area but the hand pump water of the community was not boiled or filtered.

4.1.12 Symptoms of the diseases

Symptoms like chest pain, joint pain, weight loss, fast heart pounding, shortness of the breath, vomiting, dizziness, loss of appetite, fever, cough, headaches, and swollen legs were some symptoms of the diseases.

4.2 Anthropometric Results

4.2.1 Change of Weight

In the weight change there was both positive as well as negative weight change. The distribution of weight before the surgery and after the surgery is given appendix S.

In the above figure the maximum weight increase is 8 kg and maximum weight decrease is 4 kg in the patients. It was found that 28 patients had a weight increase and two patients were weight decrease. The mean of the increment of the weight was noted to be 5.17 kg whereas the mean decrement of the weight was found to be 3 kg. It was found that there is not significant decrement in the weight ($p>0.05$) in the weight decreasing patients. Since there were only two variables the correlation factor was not obtained. Similarly in weight increasing patients was found to be significant increase in weight ($p<0.05$). So a paired sample of t-test was done between weight increase patients before the surgery and energy intake before and it was found that these both were positively correlated and there is significant increase in energy intake ($p<0.05$). In the weight decreasing patients it was found that there is no any significant increase in the calorific intake and weight decrease ($p>0.05$).

4.2.2 Change in Height

As mentioned earlier the average age of the patients was 21.67 years old, so there was not seen much change in height. The distribution of height is given in appendix R.

In the above table it was found that 17 patients had no height increase because they had cross the age limit. It was found that there is significant increment in height ($p < 0.05$). It was also found that in the height increasing patients there was a significant increment in calorific intake before and after the surgery.

4.2.3 BMI

The BMI of the 30 post operative RHD patients was calculated and represented in the table below. The BMI is also categorized into its class after calculating.

In the BMI is directly related to weight and there was both positive as well as negative change in weight, BMI was also found be increasing and decreasing. The average BMI increase among the 28 patients was 2.377 kgm^{-2} whereas the average decrease in BMI in two patients was 1.18 kgm^{-2} .

In a paired sample t-test between calorific intake before and after in BMI decreasing patients it was found that there was no significant increment in calorific intake in them either before surgery or after the surgery ($p > 0.05$ in both cases).

Similarly in BMI increasing patients it was found that there was significant increase in calorific intake of BMI increasing patients before and after the surgery ($p < 0.05$ in both pairs).

After computing the BMI the patients were categorized into the BMI category. In the survey it was found that the number of the underweight patient was decreased from 11 to 9 whereas the number of the patients was increased in the normal weight category and overweight category. The table is given below. One of reasons for decreasing number of underweight patient may be that there is a significant increase in calorific intake in the patients whose BMI is increasing.

Table 4.8 Comparison of BMI category of the patients

BMI category	No. of patients before the surgery	% of the patients before the surgery	No. of the patients after the surgery	% of the patients after the surgery
Underweight	11	36.67%	9	30%
Normal Weight	17	56.67%	18	60%
Over Weight	1	3.33%	2	6.67%
Obese class II	1	3.33%	1	3.33%
Total	30	100.00%	30	100.00%

4.3 Dietary Intake

All most all of the post operative patients are reduced physical activity, means they can do just simple works not heavy works. Some of the simple works as walking to short distances, washing clothes, cooking, sewing etc and other light physical exercises. In the case of nutrients fats, carbohydrate, protein vitamin A and riboflavin was computed. From which fats, protein and carbohydrate was the major energy giving constituent. So the daily calorific intake was computed from these. The calorific chart is kept in appendix O. it was found that there is a significant increment in nutrients like carbohydrate, protein, vitamin A and riboflavin ($p < 0.05$). But there was no significant increment in fat ($p > 0.05$).

4.3.1 Adequacy of the calorific intake

As RHD operated patient has reduced physical activity they can be considered as the sedentary work category. Adequacy is measured in percentage. As mentioned in the book of Joshi the daily calorific intake is 2350Kcal for male. Based on this calculation the adequacy for male is determined by $\frac{2350 - \text{calorific intake}}{2350} \times 100\%$ and is shown below. Similarly, in females the daily calorific intake is 1800 Kcal. Based on this RDA the adequacy rate is calculated $\frac{1800 - \text{calorific intake}}{1800} \times 100\%$. The figure is shown below.

In the figure there it is seen that there is decrement of the adequacy level but still it doesnot meet the adequacy level in maximum of the patients, low income of the family was one of the major reasons for inadequate diet so the patients was not getting the diet as prescribed. Another main reason may be that loss of the appetite is one of the symptoms of RHD so patients are not willing to eat the diet before the surgery. Using paired sample t-test between before and after the surgery it was found that there was a significant increase in calorific value.

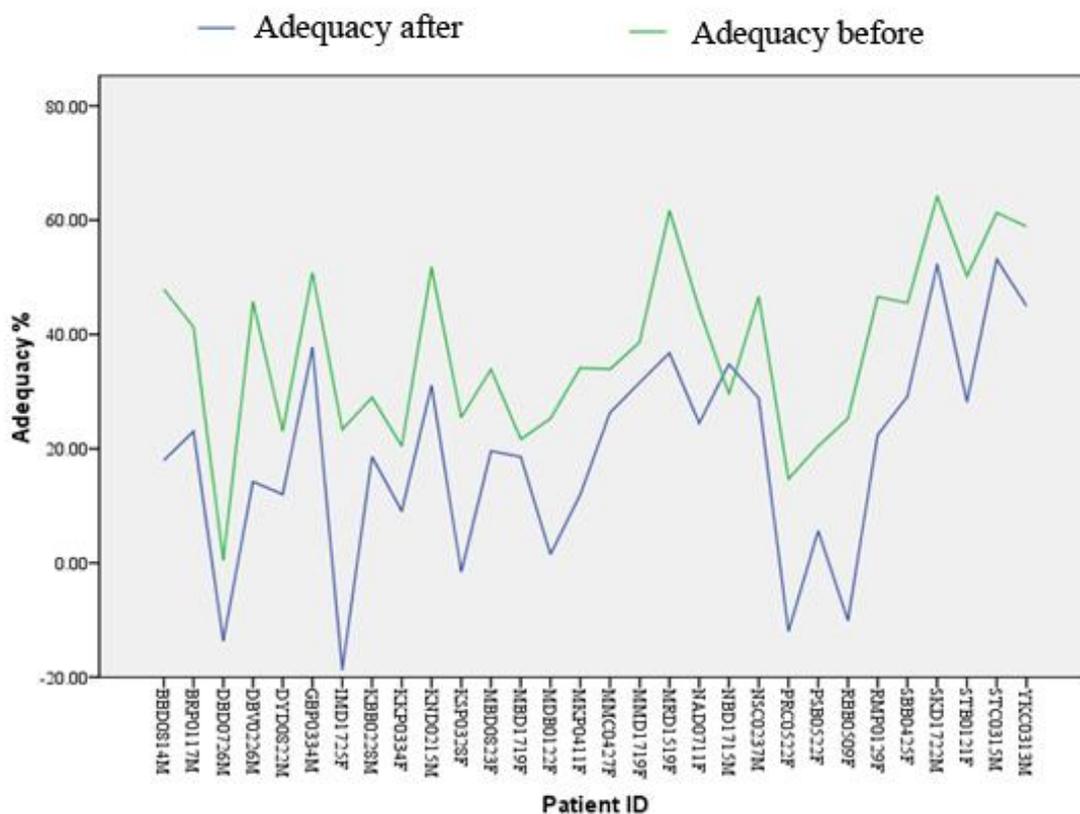


Fig 4.1 Adequacy of calorific intake before and after the surgery.

4.4 Effect of RHD in patients

4.4.1 Physical effect

Physical effect such as the pre-surgery patients could not do heavy house hold works, walk long distance or run for a short distance, they cannot do any activity which a normal man can do for earning purpose. Since most of the pre-surgery patients are not going to school cause they can't run or walk for a long duration. Moreover the pre-surgery patients need one more additional person with them to go school. Since the family members of most of the family were poor all other members had to go for work for earning purpose.

In the quantitative research of post operative patients it was found that NYHA III category patients were more as then other patients. According to them 10 NYHA III categorized patients could not go school due to cardiac disability. In other words they had shortness of breath if they walk for a long distances since the school is far away from home and no any family members are free to take them school

4.4.2 Mental effect

Mental effect is one of the effects on the pre surgery RHD patients. Mental effects such as loss of appetite, worrying about the future days, worrying about the family were seen

common. Most of Brahmin, Chhetri, Newar and Tamang community were dropped out in school due to cardiac disability.

4.4.3 Economical effect

Economical effect was another great impact in the family of RHD pre-surgery patients. Most of the family members were poor and they could not diagnose in the primary condition, in the primary condition the community of Dalits and Tamangs shown to the traditional doctors (*dhami*). When the condition of the patients started worsening then they had to visit the doctor nearby. As most of the patients were referred to BPKIHS or nearby hospital then later to SGNHC they knew the diseases. First primary diagnosis cost is expensive and secondary the surgery cost is also too expensive. In these conditions the most of the patients were supported financially by VDC's, municipalities, DDC's, local organization (DHERSEC), churches, and hospitals partially helped them for the surgery cost and diagnosis cost. As the surgery cost ranges from 40,000 to 250,000 minimum per valve.

Besides for the patients who had not been supported they sold the land and some jewelry to pay the operation cost. Maximum female post operative patients were doing sewing, tailoring and knotting as the income source of the family. Similarly the males are doing the some of the small works such as a worker in goldsmith place, painter etc. In this way the post operative patients were supporting family by doing light works.

They believe that if they were not suffered from this disease then they could do some hard works abroad and earn money and more over the parents should not sell lands and jewellery.

4.5 Views regarding this disease

Asking about the views regarding these diseases pre-surgery patients as well as the post surgery patients said that this disease is expensive and the poor ones cannot afford the medicinal costs as well as the surgery cost. It is a big burden if the family members who has no money. So most the patients had said that may this disease occur in the poor ones.

Part V

Conclusion and recommendations

5.1 Conclusion

From the research it was concluded that

- a. The number of female was more than males in the survey. The ratio of females to males was 1.3:1.
- b. There is significant change in calorific intake of carbohydrate, protein, and vitamin A, riboflavin except fat intake before and after the surgery.
- c. Only 5 females (29.41% of females) was getting the daily calorific intake i.e. 1800 Kcal per day after the surgery.
- d. Only one male (7.69% of total males) is getting the daily calorific intake but before the surgery none of the males were getting up the daily calorific intake.
- e. The average calorific value for male was found to be 1354.4 kcal before the surgery whereas after the surgery it was found to be 1708.24 kcal per day and in the case of females 1201 kcal and after the surgery it was found that 1563.76 kcal.
- f. In the survey it was found that the average carbohydrate intake was found to be 270.5gm before the surgery and 346.5 gm after the surgery.
- g. Fat was found to be 5.75 gm before the surgery and 3.91 gm after the surgery.
- h. In the case of protein it was found that the intake of protein was 33.42 gm before the surgery and 51.28 gm after the surgery
- i. Vitamin A was found to be 293.65 IU before and 718.6 IU and riboflavin was 0.46 mg before and after the surgery it was 0.954 mg per day.
- j. The average weight before the surgery was found to be 42 kg whereas the after the surgery it was found to be 46.63 kg, height was found to be 1.48 meter before and 1.5 meter after the surgery and BMI was found to be 18.61 before the surgery and 20.75 kgm⁻².
- k. There was no significant change between weight decrease and calorific intake after the surgery ($p>0.05$).
- l. There was a significant increase between weight increase and calorific intake after the surgery ($p<0.05$).
- m. There was a significant increase in height and calorific intake of the patients after the surgery ($p<0.05$).

- n. There was no significant change between BMI decrease and calorific intake after the surgery ($p>0.05$).
- o. There was a significant change between BMI increase and calorific intake ($p<0.05$) after the surgery.
- p. Regarding BMI 11 patients were categorized as under nutrition before the surgery whereas after the surgery 9 were categorized under nutrition. Similarly one patient was classified as overweight before the surgery and after the surgery there were two patients after the surgery. One patient was classified as the obese class II before and after the surgery.
- q. Thirteen of the patients were NYHA III categorized so there is a reduced physical activity in RHD patients before and after the surgery so the RHD patients were categorized sedentary level.
- r. The income status of the RHD patients is poor.

5.2 Recommendations

The study can be further continued with following recommendations:

- a. Comparative study of nutritional status in RHD patients in a fixed aged group (say, 5 to 15), in Dharan or desired area.
- b. Comparative study of nutritional status of post operated RHD patients after fixed time span of surgery (say, 5 years after surgery).

Part VI

Summary

The present work is mainly focused in comparative study of nutritional status of RHD patients concerning the problem to reveal the nutrition status of the patients residing in Dharan and nearby VDC's. Rheumatic fever is an inflammatory disease that occurs following a *Streptococcus pyogenes* infection, such as strep throat or scarlet fever. Believed to be caused by antibody cross-reactivity that can involve the heart, joints, skin, and brain, the illness typically develops two to three weeks after a streptococcal infection. Rheumatic Heart Disease is a significant physical, mental and socioeconomic burden upon individuals and families. Cost-effective prevention programs are challenged by limited resources and poor access to health-care. Nepal being a developing country there is poor access of the medical facilities to remote areas of country. Moreover this disease is mostly occurred in the developing countries with a peak age of 5 to 15.

In the work 30 post operative patients aged from 9 to 37 were selected, and they were selected according to the personal communication, local organizations approach and school visits. The name of the patients was represented in the patient ID so as to preserve his identity. The present condition of body height and weight was measured and the height and weight of the patient during the period of the surgery was noted from the discharge summary of the SGNHC. From the height and weight measurement the BMI was calculated. The BMI was categorized after calculating. Similarly, the generally eaten fruits, vegetables, pulses, legumes, meat and consumed dairy products were noted. Among them the daily eaten fruits, legumes, pulses, meat, dairy products and vegetables were used as the daily calorific value. It was found that rice and lentil was used as stable foods in survey area. The living house and the sanitation condition of them were noted. Then statistical analysis was done.

The average carbohydrate intake was found to be 270.5gm before the surgery and 346.5 gm after the surgery, similarly fat was 5.75 gm before the surgery and 3.91 gm after the surgery. In the case of protein it was found that the intake of protein was 33.42 gm before the surgery and 51.28 gm after the surgery, vitamin A was 293.65 IU before and 718.6 IU and riboflavin was 0.46 mg before and after the surgery it was 0.954 mg per day. Regarding calorific intake there is significant increase in carbohydrate, protein, vitamin A

and riboflavin ($p < 0.05$) but the demand of daily calorific value was met by only 6 (20% of the total patients) patients after the surgery. There was no significant increase in fat intake.

Fourteen patients had started taking milk after the surgery whereas the 25 patients had to abandon the citrus fruits and vegetables including cabbage and liver. The average calorific value for male was found to be 1354.4 kcal before the surgery whereas after the surgery it was found to be 1708.24 kcal per day and in the case of females 1201 kcal and after the surgery it was found that 1563.76 kcal.

There was a significant increase in food consumption (calorific intake) and weight of the patients whose weight was increasing. This was seen in the 28 persons. Besides in the remaining two persons it was seen that there was no any significant increase in food consumption and weight. In the case of the BMI it was also found similar results. A significant increase in food consumption (calorific intake) and BMI was seen in the case of the 28 patients whose BMI was increased before and after the survey, and in the remaining two it was seen that there was no significant increase in BMI and food consumption.

Lastly it was found that 56.67% patients were found to be normal categorized before the surgery whereas after the surgery it was increased to 60%. Similarly the percentage of under nutrition was reduced from 36.67% to 30%. Poverty is one of the main factors of malnutrition. Due to lack of money some of the patients had to sell land and jewellery as a result it worsened the economic conditions more. Some of them went to *Dhamis* instead of doctors. Besides these loss of appetite in pre-surgery condition was one of the major symptoms that caused malnutrition. And due to lack of money the diseases was not cured in the primary stage as a result the disease was cured in the tertiary stage.

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Appendices

Appendix A

1. Consent Form

Comparative study of nutritional status of rheumatic heart disease patients before and after the surgery in Dhahran and nearby VDC'S.

A research by Abhishek Khadka
Guided by Professor Surendra Katwal
Co-guided by Associate Prof. Dr. Nikesh Shrestha
Participant ID

Please tick to give consent

- i. I understand that this project is for research purposes.
- ii. I have read or understand the plain language statement or had orally explained to me.
- iii. I understand my involvement in this research is voluntary and I am free to withdraw in any stage.
- iv. If I am provided with any unidentifiable data then I will withdraw this research.
- v. I realize that the small this type of research makes protection to my identity hard so I give permission to change my name to researcher.

Signature of Interviewee.....

Date.....

Witness.....

Date.....

2. In depth Interview

This is the guide used to conduct interviews with participants. There are two sections: Pre surgery and post-surgery.

Each section asks participants a series of questions. Questions are structured to a in chronological timeline: intake before illness, illness and diagnosis, treatment, recovery and future aspirations. During the interview, patients were also assessed on the stage of heart failure according to the NYHA functional classification system, both pre- and post operatively. The questions are as follows:

A. Questions related to changes.

1. What do you think? I
 - i. Was healthy before I knew my illness.
 - ii. Am/was healthy during my illness.
 - iii. Am healthy after my illness.

2. The intake of food
 - i. Was greater before I knew my illness.
 - ii. Is/was greater during my illness.
 - iii. Is greater after my illness.

3. I think I
 - i. Used to walk or run more before illness.
 - ii. Used to walk or run more during my illness.
 - iii. Used to run or walk or run after my surgery.

4. I can/could do more physical work more
 - i. Before I knew my illness.
 - ii. During my illness.
 - iii. After the surgery of my illness.

5. How had your life changed after your surgery?(patient can tick multi options)

- i. I think I am good as compared before surgery.
- ii. My appetite is increased.
- iii. My confidence is built up.
- iv. I can walk more distance as compared to before surgery.
- v. Others change.....

6. How often do you take medication?

7. How often do you inject penicillium for your INR (International Normalized Ratio)?

8. What happens if you happen to miss the penicillium injection, what difficulty do you face?

9. What were the primary physical symptoms seen in you?

.....
.....
.....
.....

10. Do similar physical symptoms are seen again after the surgery.

B. Questions related to daily intake of food.

11. What type of fruits and vegetables do you take?

.....
.....
.....
.....
.....

12. Which fruits and vegetables had doctor told not to take?

.....
.....
.....

13. Which fruits and vegetables do you take often? Name them

.....
.....
.....
.....
.....
.....

14. Which type of legumes do you take?

.....
.....
.....
.....

15. Which type of Pulses do you take?

.....
.....
.....
.....

16. Do you take milk?

17. What is your stable food?

18. How many times do you take your stable food in a day?

19. Do you take whisky or wine?

20. Do you take meat?

21. Which animal meat do you take?

22. Do you take daily/once in two day/ twice a week/ once a week/..... (If any)?

23. What is the source of water in your house?

24. Do you use filter, or boiled water or neither of them?

C. Question related to environmental sanitation,

- 25. I am living in rented house/own house?
- 26. Description of house
tile/mud/corrugated/concrete/others.....
- 27. Type of floor, stone/earthen/cement/wooden
bamboo/others.....
- 28. Type of house, permanent/temporary/semi temporary.
- 29. Toilet facility/sanitary/temporary/open
field/river.....
- 30. Garbage Disposal,
pits/incineration/scattering/river/others.....

D. Economic Status

- 31. Do you have land?
- 32. Is that Arable or irrigated?
- 33. If irrigated which type of crop is produced?
- 34. What is the major source of income of family?
- 35. How money is being utilized in your medication in a month?
- 36. How did you manage for your surgery?
- 37. Are you able to do some physical works to earn money?
- 38. How often do you visit to hospital?
- 39. Do you know any first aid medication?

Quantitative information

Fruits before	Quantity in day	Fruits after	Quantity in day	Pulses before	Quantity	Pulses after	Quantity

Legumes before	Quantity	Legumes after	Quantity

Daily milk consumption before

Daily milk consumption after

Any nutritional drink Horlicks/bournvita/viva/others

Meat consumption quantity before

Meat consumption quantity after

Appendix B

New York Heart Association (NYHA) Index

The New York Heart Association (NYHA) functional classification system relates symptoms to everyday activities and the patient's quality of life, as detailed below (National Prescribing Services Limited, 2008):

Table B.1 NYHA Classification

Class I (Mild)	No limitation of physical activity. Ordinary physical activity does not cause undue fatigue, palpitation, or dyspnea (shortness of breath).
Class II (Mild)	Slight limitation of physical activity. Comfortable at rest, but ordinary physical activity results in fatigue, palpitation, or dyspnea.
Class III (Moderate)	Marked limitation of physical activity. Comfortable at rest, but less than ordinary activity causes fatigue, palpitation, or dyspnea.
Class IV (Severe)	Unable to carry out any physical activity without discomfort. Symptoms of cardiac insufficiency at rest. If any physical activity is undertaken, discomfort is increased.

Appendix C

Jones Criteria for Diagnosis of Rheumatic Fever

Diagnosis of RF is based on the Jones criteria, revised most recently by the WHO Expert Consultation panel on Rheumatic Fever and Rheumatic Heart Disease (Kumar *et. al.*, 2005). There is some debate about the effectiveness of the Jones criteria as a diagnostic tool (26), however the majority of the literature is supportive.

The criteria are: evidence of a preceding GAS infection, two major manifestations, or one major and one minor manifestation, as outlined below (Periera *et. al.*, 2007, Bitar *et. al.*, 2000):

Major manifestations:

- Migratory polyarthritis of the large joints
- Carditis
- Subcutaneous nodules
- Erythema marginatum of the skin
- Sydenham chorea

Minor manifestations (non-specific):

- Fever
- Arthralgia
- Elevated blood levels of acute phase reactants

Appendix D

Table D.1 Food Composition Table of daily eaten food items.

Common Name	Lentil	Lentil	mik cow	rice raw milled
Scientific Name	Musuro	Cicer arietinum		oryza sativa
Nepali Name	100	Chana	gai ko dudh	aruwa chamal mill ko
Edible part	12.4	100	100	100
Moisture gm	25.5	9.8	87.5	13.7
Protein gm	0.7	17.1	3.2	6.8
fat gm	2.1	5.3	4.1	0.5
minerals g	0.7	3	0.8	0.6
fiber gm	59	3.9		0.2
carbohydrate gm	343	60.9	4.4	78.2
energy kcal	69	360	67	345
calcium mg	293	202	120	10
phosphorus mg	4.8	312	90	160
iron mg	270	10.2	0.2	3.1
carotene mg or IU	0.45	189	174+	0
thiamine mg	0.2	0.3	0.05	0.06
riboflavin mg	2.6	0.15	0.19	0.06
niacin mg	0	2.9	0.1	1.9
vitamin C		3	2	0

Source: DFTQC

Appendix E

Table E.1 Paired samples statistics of weight decrease before and after

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Weight after surgery	51.00	2	1.41	1.00
	weight before surgery	54.00	2	.00	0.00

Table E.2 Paired samples correlations of weight decrease before and after

		N	Correlation	Sig.
Pair 1	Weight after surgery and weight before surgery	2	.	.

Table E.3 Paired samples test of weight decrease before and after

		Paired Differences							
		Std.		Std.	95% Confidence Interval of the Difference		t	df	Sig. (2-tailed)
		Mean	Deviation	Error Mean	Lower	Upper			
Pair 1	Weight after surgery – weight before surgery	-3.00	1.41	1.00	-15.70	9.70	-3.0	1	0.205

Appendix F

Table F.1 Paired samples statistics weight increase before and after

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Weight after surgery	46.32	28	10.24	1.93
	weight before surgery	41.14	28	9.98	1.88

Table F.2 Paired samples correlations of weight increase before and after

		N	Correlation	Sig.
Pair 1	Weight after surgery and weight before surgery	28	.988	0.00

Table F.3 Paired samples test weight increase before and after

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper			
Pair 1	Weight after surgery - weight before surgery	5.17	1.61	0.30	4.554	5.80	17.0	27	0.00

Appendix G

Table G.1 Paired samples statistics of weight increase and energy intake

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Weight before surgery	41.14	28	9.98	1.88
	energy Intake before	1268.23	28	337.98	63.87
Pair 2	Weight after surgery	46.32	28	10.24	1.93
	energy after	1646.23	28	367.03	69.36

Table G.2 Paired samples correlations of weight increase and energy intake

		N	Correlation	Sig.
Pair 1	Weight before surgery and energy Intake before	28	.260	0.182
Pair 2	Weight after surgery and energy after	28	.241	0.217

Table G.3 Paired samples test of weight increase and energy intake

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Weight before surgery – energy intake before	-1227.09	335.53	63.40	-1357.20	-1096.98	-19.35	27	0.00
Pair 2	Weight after surgery – energy after	-1599.91	364.70	68.92	-1741.32	-1458.49	-23.21	27	0.00

Appendix H

Table H.1 Paired samples statistics of weight decrease patients between weight and energy intake

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Weight before surgery	54.00	2	.00	0.00
	Energy Intake before	1256.93	2	216.6	153.16
Pair 2	Weight after surgery	51.00	2	1.41	1.00
	Energy after	1348.34	2	165.64	117.13

Table H.2 Paired samples correlations of weight decrease patients between weight and energy intake

		N	Correlation	Sig.
Pair 1	Weight before surgery and energy intake before	2	.	.
Pair 2	Weight after surgery and energy after	2	1.000	.

Table H.3 Paired samples test of weight decrease patients between weight and energy intake

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Weight before surgery – energy intake before	-1202.9	216.60	153.16	-3149.0	743.21	-7.85	1	.081
Pair 2	Weight after surgery – energy after	-1297.3	164.23	116.13	-2772.91	178.22	-11.17	1	.057

Appendix I

Table I.1 One-sample statistics of height increase

	N	Mean	Std. Deviation	Std. Error Mean
Height change	30	0.0207	0.034480	0.00630

Table I.2 One-sample test of height increase

Test Value = 0						
	t	df	Sig. (2-tailed)	Mean Difference	95% Confidence Interval of the Difference	
					Lower	Upper
Height change	3.282	29	0.003	0.02067	0.0078	0.0335

Appendix J

Table J.1 Paired samples statistics of nutrients intake before and after the surgery

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Carbohydrate after	346.50	30	80.27	14.65
	Carbohydrate before	270.50	30	69.52	12.69
Pair 2	Fat after	3.91	30	0.99	0.18
	Fat before	5.75	30	5.76	1.05
Pair 3	Protein after	51.28	30	10.27	1.87
	Protein before	33.42	30	9.17	1.67
Pair 4	Vitamin A after	718.60	30	292.88	53.47
	Vitamin A before	293.65	30	248.46	45.36
Pair 5	Riboflavin after	0.95	30	0.32	0.060
	Riboflavin before	0.46	30	0.27	0.050

Table J.2 Paired samples correlations of nutrients intake before and after the surgery

		N	Correlation	Sig.
Pair 1	Carbohydrate after and carbohydrate before	30	0.834	0.00
Pair 2	Fat after and fat before	30	0.559	0.001
Pair 3	Protein after and protein before	30	0.837	0.0
Pair 4	Vitamin A after and vitamin A before	30	0.530	0.003
Pair 5	Riboflavin after and riboflavin before	30	0.570	0.001

Table J.3 Paired samples test of nutrients intake before and after the surgery

Pair		Mean	Std. Deviation	Std. Error Mean	Paired Differences		t	df	Sig. (2-tailed)
					Lower	Upper			
Pair 1	Carbohydrate after – carbohydrate before gm	75.99	44.36	8.10	59.43	92.56	9.38	29	0.000
Pair 2	Fat after – fat before gm	-1.83	5.26	.96	-3.80	.13	-1.91	29	0.066
Pair 3	Protein after – protein before gm	17.85	5.66	1.03	15.74	19.97	17.28	29	0.0
Pair 4	Vitamin A after – vitamin A before	424.95	265.35	48.44	325.86	524.03	8.771	29	0.0
Pair 5	Riboflavin after – riboflavin before	0.49	0.28	0.05	0.38	0.59	9.402	29	0.0

Appendix K

Table K.1 Paired samples statistics of adequacy of calorific intake before and after the surgery in males

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Energy adequacy after	27.3086	13	18.36739	5.09420
	Energy adequacy before	42.3652	13	17.77243	4.92919

Table K.2 Paired samples correlations of adequacy of calorific intake before and after the surgery in males

		N	Correlation	Sig.
Pair 1	Energy adequacy after and energy adequacy before	13	.866	.000

Table K.3 Paired Samples Test

		Mean	Std. Deviation	Std. Error Mean	Paired Differences 95% Confidence Interval of the Difference		t	df	Sig. (2- tailed)
					Lower	Upper			
Pair 1	Energy adequacy after – Energy adequacy before	15.056	9.36	2.59	-20.71	-9.399	-5.80	12	.000

Appendix L

Table L.1 Paired samples statistics of calorific intake before and after for females

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Energy adequacy after	6.1975	17	17.05235	4.13580
	energy adequacy before	33.2775	17	12.91720	3.13288

Table L.2 Paired samples correlations of calorific intake before and after for females

		N	Correlation	Sig.
Pair 1	Energy adequacy after and energy adequacy before	17	0.364	0.151

Table L.3 Paired samples test of calorific intake before and after for females

		Mean	Std. Deviation	Std. Error Mean	Paired Differences 95% Confidence Interval of the Difference		t	df	Sig. (2-tailed)
					Lower	Upper			
Pair 1	Energy adequacy after – energy adequacy before	-27.08	17.24	4.18	-35.94	-18.21	-6.4	16	0.000

Appendix M

Table M.1 Paired samples statistics of BMI decrease and calorific intake before and after the surgery.

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Energy Intake before	1256.93	2	216.60	153.16
	BMI category before	2.00	2	.00	0.00
Pair 2	Energy after	1348.34	2	165.64	117.13
	BMI category after	2.00	2	.00	0.00

Table M.2 Paired samples correlations of BMI decrease and calorific intake before and after the surgery.

		N	Correlation	Sig.
Pair 1	Energy Intake before and BMI category before	2	.	.
Pair 2	Energy after and BMI category after	2	.	.

Table M.3 Paired samples test BMI decrease and calorific intake before and after the surgery.

		Paired Differences					t	df	Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference				
					Lower	Upper			
Pair 1	Energy Intake before – BMI category before	1254.93	216.60	153.16	-691.21	3201.07	8.19	1	0.077
Pair 2	Energy after – BMI category after	1346.34	165.64	117.13	-141.93	2834.62	11.494	1	0.055

Appendix N

Table N.1 Paired samples statistics of BMI increasing patients between calorific before and after the surgery

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	BMI category before	1.75	28	0.844	0.160
	Energy Intake before	1268.24	28	337.99	63.87
Pair 2	BMI category after	1.86	28	0.848	0.160
	Energy after	1646.23	28	367.04	69.36

Table N.2 Paired samples correlations BMI increasing patients between calorific before and after the surgery

		N	Correlation	Sig.
Pair 1	BMI category before and energy intake before	28	0.124	0.530
Pair 2	BMI category after and energy after	28	0.120	0.544

Table N.3 Paired samples test of BMI increasing patients between calorific before and after the surgery

		Paired Differences							Sig. (2-tailed)
		Mean	Std. Deviation	Std. Error Mean	95% Confidence Interval of the Difference		t	df	
					Lower	Upper			
Pair 1	BMI category before – energy Intake before	-1266.48	337.88	63.854	-1397.50	-1135.46	-19.83	27	0.00
Pair 2	BMI category after – energy after	-1644.37	366.93	69.34	-1786.65	-1502.09	-23.71	27	0.00

Appendix O

Table O.1 Paired samples statistics of energy intake and height before and after the surgery

		Mean	N	Std. Deviation	Std. Error Mean
Pair 1	Energy intake before	1243.53	17	358.03	86.83
	Height before surgery	1.49029	17	0.16	0.03
Pair 2	Energy intake after	1592.98	17	399.99	97.01
	Height after surgery	1.52676	17	0.14	0.03

Table O.2 Paired samples correlations of energy intake and height before and after the surgery

		N	Correlation	Sig.
Pair 1	Energy Intake Before and Height Before Surgery	17	0.272	0.290
Pair 2	Energy intake after and Height After Surgery	17	0.321	0.209

Table O.3 Paired Samples Test of energy intake and height before and after the surgery

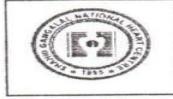
		Paired Differences							
		95% Confidence Interval of the Difference					Sig. (2-tailed)		
		Mean	Std. Deviation	Std. Error Mean	Lower	Upper	T	df	
Pair 1	Energy Intake before - Height Before Surgery(m)	1242.04	357.99	86.82	1057.98	1426.10	14.30	16	0.00
Pair 2	Energy intake after - Height After surgery(m)	1591.46	399.95	97.00	1385.82	1797.09	16.40	16	0.00

Appendix P

Table no P.1 Adequacy of Calorific Intake before and after the surgery

Patient ID	Energy Intake before	Energy intake after	Adequacy % before	Adequacy % after
BBD0814M	1224.085	1927.85	47.91	17.96
BRP0117M	1379.55	1808.85	41.29	23.02
DBD0726M	2336.66	2670.325	0.56	-13.63
DBV0226M	1276.115	2015.37	45.69	14.23
DYD0822M	1806.7	2067.9	23.11	12.00
GBP0334M	1155.545	1464.35	50.82	37.68
IMD1725F	1379.55	2136.89	23.35	-18.71
KBB0228M	1668.995	1913.235	28.97	18.58
KKP0334F	1431.83	1636.96	20.45	9.05
KND0215M	1134.06	1620.59	51.74	31.03
KSP0328F	1340.93	1827.11	25.50	-1.50
MBD0823F	1189.815	1447.215	33.89	19.59
MBD1719F	1410.095	1465.475	21.66	18.58
MDB0122F	1345.28	1772.555	25.26	1.52
MKP0411F	1185.465	1584.745	34.14	11.95
MMC0427F	1189.815	1326.37	33.89	26.31
MMD1719F	1103.765	1231.215	38.67	31.59
MRD1519F	689.65	1138.2	61.68	36.76
NAD0711F	1000.08	1359.425	44.44	24.47
NBD1715M	1654.96	1532.35	29.57	34.79
NSC0237M	1254.63	1672.175	46.61	28.84
PRC0522F	1535.015	2015.37	14.72	-11.96
PSB0522F	1431.83	1698.7	20.45	5.627
RBB0509F	1344.78	1980.2	25.29	-10.01
RMP0129F	961.46	1396.215	46.58	22.43
SBB0425F	981.08	1275.28	45.49	29.15
SKD1722M	840.89	1120.975	64.21	52.29
STB0121F	896.645	1292.1	50.18	28.21
STC0315M	909.43	1101.14	61.30	53.14
YKC0313M	965.81	1292.1	58.90	45.01

Appendix Q



Shahid Gangalal National Heart Centre
Department of Cardiac Surgery
 Bansbari, Kathmandu, Nepal
 Phone No: 4371322, 4371374, 4370622, Fax No: 977-1-4371123
 Extension No 210, Sunday/ Tuesday/ Thursday
DISCHARGE SUMMARY

Unit II: Dr. Raamesh Koirala	F/U:996	IP No: 44839	Bed No: NSW-08
Patient's name : Dhan Kumari BK		Age: 36 yrs	Sex: Female
Address: Sunsari		Phone:	
Date of Admission: 2068/09/12	Total Hospital Stay: 07 days		
Date of Operation: 2068/09/14	Total ICU Stay: 02 day		
Date of Discharge: 2068/09/19			
Diagnosis: RHD, Severe MS , Mod MR, Mod TR , Mild AR			
Operation: MVR			Outcome: improved

Case History

- SOB (NYHA II) ~ 4 years.
- Palpitation ~ 4 years.

Clinical Findings at admission

G. C.: fair Pulse: 60/min Blood Pressure: 110/70 mm Hg
 Pallor , Icterus , Cyanosis , Clubbing , Raised JVP , Oedema
 Height: 153 cm, Weight: 50kgs BSA: 1.45 m2
 Respiratory: B/L equal air entry, no added sound.
 CVS: S1+ S2+, M+.
 Abdomen soft, non tender, no organomegaly.
 Peripheral Vascular System: NAD

Investigations

Hb: 14.6 gm% Crea: 66 µmol/L
 ECG: AF, 69bpm
 ECHO: RHD: Mod MS(MVA:1.1 cm2) , Mod MR, Moderate TR with mild PAH (estimated PASP: 41.0mmhg), Mild AR ,dilated LA and RA and RV.
 LVID: 4.6/3.5cm, LA: 4.4 cm, EF=50 % , AO: 3.1 cm,
 MR: Gr-III , TR: Gr-III, AR: Gr-II
 Others: HIV, HBsAg, HCV non reactive. Blood group: O positive.

Operation Note:

Open Heart Surgery

CPB: Yes Hypothermia: mild CPB: 61 mins AoX: 45 mins
 Cardioplegia: cold blood Post op pacing: No Type:

Operative Findings:

- Dilated LA.No La/LAA clot.
- MV: thickened leaflets, calcified , fused comisure ,sev subvalvular changes , PML partially preservable.

Fig Q.1 First Sheet of discharge Summary of Patient

Procedure:

Cardiopulmonary Bypass established. Aortic clamp applied. Antegrade root Cardioplegia given. LA opened, MV excised, PML preserved. MVR done. LA closed. RA opened . RA closed. Deairation done and patient gradually weaned off from CBP. Hemostasis secured. Sternum closed.

Manufacturer	Valve Size:	Model Number .	Serial Number:
SJM	27 mm	27M-101	85767890

Post Operative Course:- Uneventful.

Ionotropes: Dopamine ; Adrenaline ; Milrione ; GTN ; Nor Adrenaline

Condition at Discharge:

G.C.: Fair **Pulse:** 90/min **Blood Pressure:** 90/60 mmHg

CVS: S₁ +, S₂ +, metallic click+ irregular

Respiratory: B/L equal air entry, no added sound.

P/A: soft, non-tender, and no distension.

Others: Incision site looks healthy. Sternum stable.

PT/INR at discharge- INR 8.9 , Warfarin hold

GENERAL ADVICE:

- Avoid trauma over chest for 6 months.
- Prophylaxis against infective endocarditis at all times.
- Avoid liver, citrus fruits, green-leafy vegetables and cabbage.
- Maintain INR between 2.0 to 3.5 lifelong after surgery or contact otherwise.
- Pt are recommended to check PT/INR every weekly, thromboembolic event may occur if the recommended not followed and even with optimal INR. Attending physician is not responsible for any such event.

MEDICINES

- Tab Farin 2 mg PO OD(6 pm) start from (2068/09/20)
- Tab. Amilax 1 tab PO OD/AM.
- Tab. Metloc 12.5mg PO BD.
- Tab Lanoxin 0.125mg PO OD (6/7)
- Tab Codomol 1 tab SOS.
- Tab. Cefadroxyl 500 mg PO x BD x 4 days.

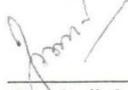
FOLLOW UP: On Surgical OPD on Friday with PT/INR, CXR P/A report and for stitch removal.

OPD: Sunday and Wednesday---Afternoon, Tuesday and Friday---Morning

RECOMMENDATION: Please consult your local Cardiologist or your admitting Surgeon **Dr Raamesh Koirala** for regular follow up and drug adjustment.



Dr. Lokesh Yadav
Resident Doctor



Dr. Anil Aacharya
Registrar

Dr. Raamesh Koirala
Unit II Incharge

Fig Q.2 Second sheet discharge summary of patient

Appendix R

Table R.1 Nutrient Intake of patients before the surgery

Patient ID	Carbohydrate(gm)	Fat(gm)	Protein(gm)	Vitamin A (IU)	Riboflavin (mg)
BBD0814M	269.45	1.585	33.005	148.5	18.11
BRP0117M	306	1.75	34.95	135	21.1
DBD0726M	464.2	23.06	68.08	1086	31.11
DBV0226M	283.3	1.615	32.095	121.5	19.59
DYD0822M	376.25	12.3	47.75	570	26.075
GBP0334M	257.65	1.445	27.985	94.5	18.07
IMD1725F	306	1.75	34.95	135	21.1
KBB0228M	347.65	12.095	42.385	529.5	24.545
KKP0334F	321.85	1.75	32.17	81	23.16
KND0215M	226.2	11.46	31.53	516	15.535
KSP0328F	271.6	11.73	37.24	543	18.555
MBD0823F	263.55	1.515	30.495	121.5	18.09
MBD1719F	288.4	11.795	37.585	529.5	20.045
MDB0122F	300.1	1.68	32.44	108	21.08
MKP0411F	235.05	11.565	35.295	556.5	15.565
MMC0427F	263.55	1.515	30.495	121.5	18.09
MMD1719F	245.8	1.385	27.025	94.5	17.17
MRD1519F	152	0.89	18.41	81	10.26
NAD0711F	221.1	1.28	26.04	108	15.08
NBD1715M	363.2	2.16	45.68	216	24.16
NSC0237M	251.85	11.63	35.64	543	17.055
PRC0522F	342.55	1.915	36.895	121.5	24.09
PSB0522F	321.85	1.75	32.17	81	23.16
RBB0509F	296.1	1.74	36.18	162	19.92
RMP0129F	186.7	11.26	28.33	516	12.535
SBB0425F	205.75	5.28	27.64	282	13.77
SKD1722M	161.05	11.09	24.22	489	11.015
STB0121F	198.4	1.145	23.185	94.5	13.57
STC0315M	172.85	11.23	29.24	543	11.055
YKC0313M	215.2	1.21	23.53	81	15.06

Table R.2 Nutrient Intake of patients after the surgery

Patient ID	Carbohydrate(gm)	Fat(gm)	Protein(gm)	Vitamin A (IU)	Riboflavin (mg)
BBD0814M	423.52	2.95	51.805	202.5	0.441
BRP0117M	392.4	3.85	51.15	570	0.845
DBD0726M	569.75	6.325	83.6	1140	1.525
DBV0226M	437.4	4.17	57.06	597	0.895
DYD0822M	437.7	5.4	67.125	1072.5	1.385
GBP0334M	314.2	3.35	44.35	570	0.785
IMD1725F	454.3	5.49	67.57	1059	1.39
KBB0228M	406.35	5.135	60.405	1018.5	1.33
KKP0334F	357.14	3.56	44.09	516	0.787
KND0215M	341.8	4.69	52.795	991.5	1.265
KSP0328F	386.8	5.01	58.705	1018.5	1.315
MBD0823F	311.25	3.315	43.095	556.5	0.775
MBD1719F	305.65	4.475	50.65	1005	1.245
MDB0122F	385.45	2.755	51.49	243	0.435
MKP0411F	339.65	3.545	48.56	597	0.82
MMC0427F	281	3.17	43.46	597	0.775
MMD1719F	252.474	4.135	46.026	1005	1.2042
MRD1519F	231.36	4	44.19	1005	1.188
NAD0711F	294.49	2.125	40.585	202.5	0.342
NBD1715M	320.24	3.55	54.86	705	0.867
NSC0237M	352.57	4.775	54.73	1005	1.281
PRC0522F	437.4	4.17	57.06	597	0.895
PSB0522F	368.336	3.68	48.059	556.5	0.8188
RBB0509F	421.9	4.2	63.7	705	0.945
RMP0129F	286.17	4.415	52.95	1059	1.261
SBB0425F	271.4	3.28	40.04	630	0.83
SKD1722M	227.45	3.975	43.85	1005	1.185
STB0121F	275.1	3.1	40.95	570	0.755
STC0315M	236.8	1.74	34.57	189	0.29
YKC0313M	275.1	3.1	40.95	570	0.755

Appendix S

Table S.1 Weight of the patient before and after the surgery

Patient ID	Weight before the surgery(kg)	Weight after the surgery(kg)	Weight Increase(kg)
BBD0814M	43	48	5
BRP0117M	43	47	4
DBD0726M	45	52	7
DBV0226M	40	48	8
DYD0822M	50	58	8
GBP0334M	59	64	5
IMD1725F	43	46	3
KBB0228M	66	72	6
KKP0334F	52	56	4
KND0215M	39	43	4
KSP0328F	40	44	4
MBD0823F	37	43	6
MBD1719F	54	52	-2
MDB0122F	42	48	6
MKP0411F	24	26	2
MMC0427F	50	54	4
MMD1719F	54	50	-4
MRD1519F	49	54	5
NAD0711F	17	25	8
NBD1715M	33	40	7
NSC0237M	38	43	5
PRC0522F	37	43	6
PSB0522F	39	45	6
RBB0509F	39	45	6
RMP0129F	41	46	5
SBB0425F	43	47	4
SKD1722M	41	45	4
STB0121F	37	43	6
STC0315M	43	48	5
YKC0313M	22	24	2

Table S.2 Height of the patient before and after the surgery

Patient ID	Height Before Surgery(m)	Height After surgery(m)	Change in Height(m)
BBD0814M	1.65	1.715	0.065
BRP0117M	1.395	1.42	0.025
DBD0726M	1.735	1.74	0.005
DBV0226M	1.615	1.62	0.005
DYD0822M	1.65	1.65	0
GBP0334M	1.72	1.72	0
IMD1725F	1.44	1.44	0
KBB0228M	1.63	1.63	0
KKP0334F	1.51	1.51	0
KND0215M	1.55	1.6	0.05
KSP0328F	1.42	1.42	0
MBD0823F	1.52	1.52	0
MBD1719F	1.57	1.59	0.02
MDB0122F	1.415	1.415	0
MKP0411F	1.33	1.45	0.12
MMC0427F	1.33	1.33	0
MMD1719F	1.57	1.59	0.02
MRD1519F	1.51	1.51	0
NAD0711F	1.1	1.19	0.09
NBD1715M	1.4	1.51	0.11
NSC0237M	1.5	1.5	0
PRC0522F	1.105	1.105	0
PSB0522F	1.42	1.42	0
RBB0509F	1.48	1.53	0.05
RMP0129F	1.46	1.46	0
SBB0425F	1.46	1.46	0
SKD1722M	1.67	1.67	0
STB0121F	1.435	1.435	0
STC0315M	1.65	1.69	0.04
YKC0313M	1.28	1.3	0.02

Table S.3 BMI of the patient before and after the surgery

Patient ID	BMI before	BMI After	BMI change
BBD0814M	14.61	16.32	1.70
BRP0117M	21.32	23.30	1.98
DBD0726M	14.86	17.17	2.31
DBV0226M	15.24	18.28	3.04
DYD0822M	18.36	21.30	2.93
GBP0334M	19.94	21.63	1.69
IMD1725F	20.73	22.18	1.44
KBB0228M	24.84	27.09	2.25
KKP0334F	22.80	24.56	1.75
KND0215M	15.23	16.79	1.56
KSP0328F	19.83	21.82	1.98
MBD0823F	16.01	18.61	2.59
MBD1719F	21.35	20.56	-0.79
MDB0122F	20.97	23.97	2.99
MKP0411F	11.41	12.36	0.95
MMC0427F	28.26	30.52	2.26
MMD1719F	21.36	19.77	-1.58
MRD1519F	21.49	23.68	2.19
NAD0711F	12.00	17.65	5.64
NBD1715M	14.47	17.54	3.07
NSC0237M	16.88	19.11	2.22
PRC0522F	30.30	35.21	4.91
PSB0522F	19.34	22.31	2.97
RBB0509F	16.66	19.22	2.56
RMP0129F	19.23	21.58	2.34
SBB0425F	20.17	22.05	1.87
SKD1722M	14.70	16.13	1.43
STB0121F	17.96	20.88	2.91
STC0315M	15.05	16.80	1.75
YKC0313M	13.01	14.20	1.18