DIET THERAPY AND COUNSELING

M.Sc., FOODS AND NUTRITIONAL SCIENCE,
Second Year, Paper – IV

Specialization-I: Clinical Nutrition and Dietetics

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FOREWORD

Acharya Nagarjuna University, since its establishment in 1976, has been moving ahead in the path of academic excellence, offering a variety of courses and research contributions. The University achieved recognition as one of the eminent universities in the country by gaining A grade from the NAAC 2016. At present Acharya Nagarjuna University is offering educational opportunities at the UG, PG levels to students of 447 affiliated colleges spread over the two districts of Guntur and Prakasam.

The University had started the Centre for Distance Education in 2003-04 with the aim to bring Higher education within the reach of all. The Centre has been extending services to those who cannot join in colleges, cannot afford the exorbitant fees as regular students, and to housewives desirous of pursuing higher studies to study B.A., B.Com, and B.Sc., Courses at the Degree level and M.A., M.Com., M.Sc, M.B.A. and LL.M. courses at the PG level.

For better understanding by students, self-instruction materials have been prepared by eminent and experienced teachers. The lessons have been prepared with care and expertise. However constructive ideas and scholarly suggestions are welcome from students and teachers. Such ideas will be incorporated for the greater efficacy of the distance mode of education. For clarification of doubts and feedback, Weekly classes and contact classes are arranged at UG and PG levels respectively.

I wish the students who pursue higher education through Centre for Distance Education will not only be personally benefited by improving their qualifications but also strive for nation's growth by being a member in Knowledge society I hope that in the years to come, the Centre for Distance Education will grow in strength by introducing new courses, catering to the needs of people. I congratulate all the Directors, Academic coordinators, Editors, Lesson - Writers, and Academic Counsellors and Non-teaching staff of the Centre who have been extending their services in these endeavours.

Prof. Raja Sekhar P.
Vice - Chancellor (FAC)
Acharya Nagarjuna University

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M.Sc (Course Code-139)

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- Nutrition support systems.

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- Febrile Conditions: Types, etiology, metabolic changes, clinical symptoms, diagnostic tests, diet management and counseling.

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- HIV and AIDS, Nutritional cancer, Diet counseling for HIV and AIDS and cancer.

UNIT–I

DIET COUNSELING

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OBJECTIVES

After going through this unit, students will be able to:

- state meaning, process and type of counseling;
- explain the role of counselor and counseling strategies;
- discuss the impact of diet counseling on health;
- describe the role of counseling in hospitals;
- explain the nutrition support systems.

STRUCTURE

1.1 INTRODUCTION

Over the years, the Dietitian's role has definitely evolved. From being a "food police", the role r.ow is to counsel people towards achieving better dietary habits.

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A Dietitian is the qualified person to help people improve their health through nutrition therapy.

The process of nutrition therapy is as follows:

- Assessing nutritional needs based on medical condition, laboratory investigations, dietary and lifestyle patterns.
- Providing personalized information on food preparation, food substitution and portion sizes within the dietary recommendation.
- Following-up on progress and suggest further dietary changes if necessary to achieve outcomes.

Diet counsling is necessary to achieve better nutritional status. The counselor help the people improve their dietary habit through various mechanism. Diet counseling is very much essential for the well being of the people.

1.2 DIET COUNSELING (MEANING AND CHARACTERISTICS)

Diet counseling is an ongoing process in which a health professional, usually a registered dietitian, works with an individual to assess his or her usual dietary intake and identify areas where change is needed. The nutrition counselor provides information, educational materials, support, and follow-up to help the individual make and maintain the needed dietary changes.

Counseling in general is a term describing a process that assists people to learn about themselves, their environment and methods of handling problems. Nutrition counseling is an ongoing process in which a health professional, usually a nutritionist works with an individual to assess his or her usual dietary intake and identify areas where change is needed.

General Characteristics

1. Counseling is a type of assessment made which analyzes various health needs in regard to diet and exercise.
2. A nutritional counselor helps people to set achievable health goals and teaches various ways of maintaining these goals throughout their lifetime.
3. Nutritional counseling is sometimes offered at eating disorder treatment clinics for patients in need of anorexia treatment, bulimia help or binge eating assistance.
4. The nutrition counselor provides information, educational materials, support, and follow-up to help the individual make and maintain the needed dietary changes.
5. Nutritional counseling covers a wide range of assessments and philosophies regarding the quality of nutrition in a diet.

6. Nutritionists offer education in nutritional facts, eating habits, nutrients, allergies, and weight loss.
7. Counselors work with individuals to develop dietary patterns that suits their lifestyle as well as their needs.

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1.3 PURPOSE OF DIET COUNSELING

The goal of nutrition counseling is to help a person make and maintain dietary changes. For a person with a mental disorder, dietary change may be needed to promote healthier eating, to adopt a therapeutic diet, or to avoid nutrient-drug interactions. Nutrition counseling is an integral part of treatment for persons with eating disorders or chemical dependencies. Persons taking certain drugs, such as monoamine oxidase inhibitors, used to treat depression and anxiety disorders, need to follow a tyramine-controlled diet to avoid dietary interference with their medication. Many drugs used to treat mental disorders can cause weight gain or loss, so persons taking these medications may also benefit from nutrition counseling.

The nutrition counselor and individual work together to assess current eating patterns and identify areas where change is needed. Registered dietitians have met certain education and experience standards and are well qualified to provide nutrition counseling, but nurses, physicians, and health educators also provide nutrition counseling.

Dietary change may be needed to promote healthier eating, to adopt a therapeutic diet, or to avoid nutrient-drug interactions. Nutrition counseling is an integral part of treatment for persons with eating disorders or chemical dependencies. Many drugs used to treat mental disorders can cause weight gain or loss, so persons taking these medications may also benefit from nutrition counseling.

Assessing Dietary Habits

Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake.

The 24-hour recall method is a listing of all the foods and beverages a person consumed within the previous 24-hour period. The nutrition counselor may ask a person to recall the first thing he or she ate or drank the previous morning. The counselor then records the estimated amounts of all the foods and beverages the person consumed the rest of the day. The 24-hour food recall can be used to provide an estimate of energy and nutrient intake. However, people tend to over- or underestimate intake of certain foods, and food intake on one day may not accurately represent typical food intake.

A food frequency questionnaire can sometimes provide a more accurate picture of a person's typical eating patterns. The nutrition counselor may ask the client how often he or she consumes certain food groups. For example, the counselor may ask a person how many servings of dairy products, fruits, vegetables, grains and cereals, meats, or fats he or she consumes in a typical day, week, or month.

Daily food records are also useful in assessing food intake. An individual keeps a written record of the amounts of all foods and beverages consumed over a given period of time. The nutrition counselor can then use the food records to analyze actual energy and nutrient intake. Three-day food records kept over two weekdays and one weekend day are often used.

Assessing Body Weight

Nutrition counselors may assess an individual's body weight by comparing his or her weight to various weight-for-height tables. A rough rule of thumb for determining a woman's ideal body weight is to allow 100 lb (45 kg) for the first 5 ft (1.5 m) of height plus 5 lb (2.3 kg) for every additional inch. A man is allowed 106 lb (48 kg) for the first 5 ft (1.5 m) of height plus 6 lb (2.7 kg) for every additional inch. However, this guide does not take into account a person's frame size.

Body mass index, or BMI, is another indicator used to assess body weight. BMI is calculated as weight in kilograms divided by height in meters squared. A BMI of 20 to 25 is considered normal weight, a BMI of less than 20 is considered underweight, and a BMI of greater than 25 is considered overweight.

Identifying Changes Needed

The initial dietary assessment and interview provide the basis for identifying behaviours that need to be changed. Sometimes a person already has a good idea of what dietary changes are needed, but may require help making the changes. Other times the nutrition counselor can help educate a person on the health effects of different dietary choices. The nutrition counselor and client work together to identify areas where change is needed, prioritize changes, and problem-solve as to how to make the changes.

Making dietary change is a gradual process. An individual may start with one or two easier dietary changes the first few weeks and gradually make additional or more difficult changes over several weeks or months. For example, an easy change for a person might be switching from 2% to skim milk, or taking time for a quick yogurt or granola bar in the morning instead of skipping breakfast. More difficult changes might be learning to replace high-fat meat choices with leaner ones, or including more servings of vegetables daily.

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In making dietary changes, each individual's situation and background must be carefully considered. Factors that affect food decisions include an individual's ethnic background, religion, group affiliation, socioeconomic status, and world view.

Identifying Barriers to Change

Once the needed changes have been identified, the client and nutrition counselor think through potential problems that may arise. For example, changing eating behaviours may mean involving others, purchasing different foods, planning ahead for social events, or bringing special foods to work. Some common barriers to changing eating habits include:

- inconvenience
- social gatherings
- food preferences
- lack of knowledge or time
- cost

Setting Goals

The nutrition counselor and client set behaviour-oriented goals together. Goals should focus on the behaviours needed to achieve the desired dietary change, not on an absolute value, such as achieving a certain body weight. For a person working to prevent weight gain associated with certain medications, for example, his or her goals might be to increase the amount of fruits, vegetables, and whole grains consumed each day. Such changes would help prevent weight gain while placing the emphasis on needed behaviours rather than on actual weight.

Finding Support

Family members are encouraged to attend nutrition counseling sessions with the client, especially if they share responsibility for food selection and preparation. Although the individual must make food choices and take responsibility for dietary changes, having the support and understanding of family and friends makes success more likely.

Maintaining Changes

The challenge for the nutrition client lies not in making the initial dietary changes, but in maintaining them over the long term. Self-monitoring, realistic expectations, and continued follow-up can help a person maintain dietary changes.

Self-monitoring involves regularly checking eating habits against desired goals and keeping track of eating behaviours. Keeping a food diary on a daily or periodic basis helps the individual be more aware of his or her eating behaviours

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and provides a ready tool to analyze eating habits. Sometimes a simplified checklist to assure adequate intake of different food groups may be used.

Individuals and nutrition counselors should not expect perfect dietary compliance—slips inevitably occur. The goal is to keep small slips, such as eating a few extra cookies, from becoming big slips, like total abandonment of dietary change. The counselor can help the client identify situations that may lead to relapse and plan ways to handle the situations ahead of time.

Nutrition counseling is an ongoing process that can take months or years. In follow-up nutrition counseling sessions, the individual and counselor analyze food records together and problem-solve behaviours that are especially difficult to change. Follow-up counseling also allows the opportunity to reevaluate goals and strategies for achieving those goals.

1.4 FIELDS OF COUNSELING, ITS PRINCIPLES, BENEFITS AND BARRIERS

The need for nutrition counseling has grown with the increase in chronic conditions such as Cancer, Diabetes, Eating Disorders, Excess Weight Gain, Food Allergies, Gastrointestinal Disorders etc. Vegetarian nutrition, sports nutrition, and childhood nutrition. Healthy lifestyles in the prevention of cardiovascular diseases are of utmost importance for people with non insulin-dependent diabetes mellitus, hypertension, and/or dyslipidemia.

Frame of Work

(A) ***On the spot information and advice.*** The provider may give advice about a particular question or problem. The purpose of the advice is to find a useful or helpful course of action that the recipient can pursue independently. The advice is specific to the situation, usually requiring 10 to 20 minutes (or less). Many practitioners and patients expect that matters concerning lifestyle risks, such as physical inactivity, can be dealt with in such a manner. Sometimes this assumption is correct; most often it is not.

(B) ***A few repeated contacts on the same topic.*** A contact with a provider may lead to further visits for the same reason. If the timing of the new visits is known beforehand, it will also be possible to make some advanced plans. The provider can monitor what aspects of the advice the recipient has put into use, what their experience has been, how their needs have changed. This kind of advice and guidance may be given by a range of health professionals and can be linked to monitoring of symptoms.

(C) ***Planned period of structured counseling sessions.*** The contacts between the provider and the recipient may have their beginning in certain problems and needs related to habits and quality of life. The sessions are planned ahead, they have a fixed duration and several sessions are

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implemented at fairly short intervals (*e.g.* weekly). This kind of counseling gives the opportunity to use the full range of methods, outlined in the models section. This form of counseling, also, assumes that the provider has special skills and training, and that each session requires a longer time frame than situations where only advice is given.

Key Principles for Counseling

(i) Counseling is a cooperative mode of work demanding active participation from both the provider and the recipient.

(ii) Counseling is goal-oriented as the aim is for the recipient to find an answer to an identified question or a solution to his/her specific problem and through counseling, the recipient will learn to apply knowledge in new ways, may acquire new skills, or change some of his/her beliefs or behaviours.

(iii) Counseling is best characterized by client-centeredness. The needs and views of the recipient have to be respected. The provider must always be sensitive to introducing only culturally appropriate interventions and avoid ordering, patronizing, or acting on the client.

Who Provide It

Because of their continuous contact with almost all segments of the population, nutritionist can play an important role in initiating and encouraging dietary change. Nutrition counseling is to be provided by specialized nutritionist. Qualified and trained physicians, general practitioners can provide NC. Specified role of NC may also be provided through guided nurses and health educators. The nutrition counselor and individual work together to assess current eating patterns and identify areas where change is needed.

Targets for Nutritional Counseling

A variety of people benefit from nutritional counseling. This type of counseling assists all ages, ranging from pregnant women, to vegetarians, to the elderly and people with different medical complications. Those who may benefit include those with all types of health problems and diseases. For many people with eating disorders, this type of advice is very beneficial.

Benefits of Nutritional Counseling

Various benefits of nutritional counseling include:

Increase in energy, Healthier lifestyle and food choices , Better quality sleep, Reduced symptoms of disease, Less drastic range, of emotions, Happiness, Possible increase in longevity, Strengthened immune system, Lesser chance of disorders

and disease appearing, Better ability to concentrate. Such an assessment is beneficial to a wide variety of people and can help those with numerous disorders. During this assessment, the counselor provides information based on a person's current status, helping to improve overall health. Many nutritional counselors recommend fitness programs and nutritional supplements. By seeing a nutritional counselor instead of looking and testing out various products, time is saved. Through nutritional counseling, basic health needs are met and specialized information is given to fit each person's everyday lifestyle.

Tasks of the Counselor

Proceeding by steps: Five markers: "Assess, Advise, Agree, Assist, Arrange" ("5 A's" step- model) are helpful in managing the progression through the successive sessions. These steps organize the tasks for the provider. They remind him/her of where the emphasis is meant to be during any given session. To move on to the next step, it is necessary to have completed the specific tasks for the current step, and thus, to have brought about the necessary conditions for moving forward.

Step l: Assess - The provider first collects, selects and analyses information to make decisions.

Counseling calls for information that the recipient alone can provide. The provider always begins at the recipient's level of understanding. In the case of life-style problems, it is important to assess the chronicity of the recipient's relevant behaviours. The provider works to involve the recipient in the process of defining targets and determining goals and objectives.

Step 2: Advice - It is not practical to take on too many targets at the same time. Generally, counseling is more effective when the recipient selects the target behaviour on which to focus. The first targets should be highly concrete and the recipient's own actions should lead to fairly quick and clearly visible changes in them. When needed, the provider should help the decision making process with his/her knowledge of the condition/situation.

Step 3: Agree - The provider has responsibility for the correct assessment of problems, for the formulation of the targets, and for the suitability of the suggested management. A plan or an agreement should state, in the least, the short-term objectives, and trace a route most likely to lead away from the current situation. Often a written agreement or contract clarifies the respective responsibilities.

Step 4: Assist - Courses of action should be pondered together with the recipient, always concentrating on the route from the current situation to the nearest immediate objective. At this stage, the responsibility for actions rests with the recipient; s/he should try out the suggestions agreed upon, according to the plan,

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and bring back experiences for discussion and evaluation together. Common elements in many behaviour change counseling situations include moral support, skill training, environmental change, relapse prevention, and maintenance techniques. The provider's task lies in identifying the recipient's particular needs for instruction, and practical training. This step of implementation may take several sessions.

Step 5: Arrange - Counseling requires that the actions taken undergo continuous monitoring for evidence of change. It is often necessary to arrange for additional learning or skill development through community services or programs. It is essential to find out throughout the duration of the counseling, to what extent the recipient has followed the course of action agreed upon in the previous sessions. By the final session, the recipient needs to have a clear picture of the kind of journey she/he has made and of what his/her own efforts have achieved.

BARRIERS

(1) *Top-down approach:* The traditional approach in nutrition education is a top-down approach; the counselor assumes the role of expert and dictates to the client.

(2) *Resistance:* Tension between educator and client or two people with two different points of view and wills and an absence of collaborative direction towards goal.

Resistance occurs when the counselor is moving beyond the client's stage of change. Resistance is occurring when any of the signs listed below appear during the session:

- Challenges or discounts counselor's advice.
- Interrupts or cuts off counselor's advice.
- Minimizes need to change, making excuses, blaming others, or pessimism (yes, but).
- Inattentiveness or non responsiveness.

(3) *Noncompliance:* The person is expected to make permanent changes to remain in optimum health; diet may be only one of several changes the individual is expected to effect. the individual must adopt dietary changes and sustain them over a period of time, often a lifetime if the condition is a chronic one such as diabetes mellitus, cardiovascular, or renal disease. Compliance may be defined as the extent to which the individual's food and dietary behaviors coincide with the dietary recommendations and prescriptions. It tells the extent to which individuals have been successful in integrating dietary changes and selfcare behaviours into their day-to-day activities.

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Factors Affecting Compliance

(1) Client age, sex, socioeconomic status, marital status, and the like, have not found that these variables predict compliance, although better comprehension of the regimen tends to be associated with higher educational levels. When areas in need of improvement are identified by self-monitoring, they can be translated into short-term, attainable goals for change. the most effective element of intervention in producing dietary adherence to goals was the process of "self monitoring" of daily intake. It enhanced patient self-management by allowing individuals to identify targeted nutrient sources in their diets and then to develop strategies for modifying their eating behaviours to achieve desired goals. The person's beliefs about health are determinants of his or her readiness to take action. Psychosocial factors related to adherence included knowledge, attitudes, support, satisfaction, and self-perceptions of success.

(2) *The relationship between the health care provider and the patient.* What strategies can a counselor use to enhance client adherence along the path to long-term change and a healthy outcome? For long-term dietary change, Using the stages of change model discussed earlier, as well as relapse prevention strategies and self-efficacy. were also recommended. Trust, respect, sharing responsibilities and the acknowledgment of expertise and experience are fundamentals. Social support by family members included in counseling sessions was also suggested.

(3) *The clinical setting.* The characteristics of the clinic are also important. People kept waiting for long periods of time often fail to return for future appointments. A warm and caring environment, created by not only the counselor but also the entire office staff, puts clients into a frame of mind that enables them to benefit from their counseling.

(4) *The treatment regimen characteristics.* The characteristics of the regimen are the most important factors in adherence. Of these

(i) Complexity, the more communicable something is (*i.e.*, the easier to describe), the more clearly it is understood. Complexity of a regimen has been negatively associated with adherence, perhaps related to the difficulty of fitting the regimen into one's daily routine

(ii) Simplicity is an advantage, as increasing complexity of a regimen is associated with less readily adopted changes.

(iii) compatibility, the change must be compatible with the person's existing values and beliefs. If divisible into parts, a change may be tried out on a small scale so that any barriers can be worked out.

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(5) changes in lifestyle required, which tend to be restrictive, last a long duration or for a lifetime, and interfere with family habits.

(6) high cost of the diet, lack of access to the proper foods, or extra effort, time, and skill required in preparing the diet, the likelihood of no adherence increases.

(7) Advantage, the change should have a relative advantage, or be perceived as preferable in efficiency, health, pleasure, economics, prestige, and the like.

(8) Pleasure, people want food that tastes good. Pleasure or the absence of it may change the rate of acceptance of new practices. One problem is that many of the less nutritious foods, such as rich desserts, snack foods, and beverages, have the highest prestige value. Pleasure may be a major factor in some cases while cost or ease of food preparation is an important factor in another.

(9) *The features of the disease.* The nature of the illness is another variable. A serious, life-threatening problem, such as a heart attack, may convince an individual to make dietary and exercise changes, at least in the near term. A person with few overt symptoms, as in hypertension, may not see the need for adherence to a dietary regimen.

(10) *The professional.* Although noncompliance may be viewed as failure on the patient's part to cooperate with recommendations, counselors are not excused from responsibility for other variables that are under their control, such as the quality of the client-counselor relationship and the use of appropriate influence strategies. Satisfaction with the level of care and with the attitude of the counselor has been reported to influence adherence. Adherence may be more satisfactory if the patient sees the same counselor at each visit, and if clear-cut communication occurs so that the individual fully understands what is best and what is expected.

(11) *Practice Implications.* Dietary change programs that may be successful on the short-term, often do not lead to long-term success. Some situations respond to brief on-the-spot advice, others require a few repeated counseling sessions utilizing concepts from behavioural theory, and certain ones need referral to a structured counseling program that employs a longer time-frame and allows for the opportunity to use a range of methods.

1.5 THE NUTRITION PROFESSION

In the past, nutrition has been taught and applied as a secondary health helping discipline that offer basic knowledge and general guidance regarding food and nutrients and their possible effect on the health of individuals, groups and societies. Furthermore, due to the well established traditional medical

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profession and its advanced therapeutic oriented system, the mainly known nutrition specialty was clinic/dietetic field which overcome other nutrition specialties that been collectively designated the name of general nutrition.

Even though clinical/dietetics were relatively advanced compared to general nutrition specially in the developed countries, both require specific qualifications and feasibly designed training program in specified food or health institutions. The nutrition profession outcome on health has been evaluated to be unsatisfactory as people continue to raise fundamental issues and questions while the specialties thrive with their limited knowledge and skillfulness, to provide practical solution and reliable answers. In recent years, however, tremendous shift has taken place that introduced the nutrition in a more specified discipline and in newer prospective, position, involvement, adequacy and effectiveness.

New Era of Profession

Today, nutrition considered as a health oriented profession and one of the vast developing and advancing discipline. It becomes under focus as a fundamental alternative approach in preventing as well as curing human illnesses and diseases and in resolving today health-nutrition problems. Resolving such concerns and questions are dealt with, not any more, simple, shallow, general and unreliable approaches. It requires far more than providing general information on nutrition as such information can be obtained from the many books, magazines, newspapers, television, computer software, the web, family, and friends and one does not need a professional to provide it. The resolution of problems, however relies on facing the profession challenge through adequacy of qualification, self advancing in today issues, skillfulness and effective job filling posed by the nutrition professional.

The recent specialties within nutrition profession include the followings:

Consultant nutritionists: Work within or under contract with health care facilities or in their own private practice. They perform nutrition screenings for their clients and offer preventative advices and treatment programs on diet-related diseases and concerns.

Therapy nutritionist: The therapeutic nutrition is a new specialty that introduced nutrition as a fundamental alternative-integrative approach. Professional through designated clinics provide direct curative services to clients with all sort of health complains and ill health.

Community nutritionists: Counsel Individuals and groups on nutritional practices designed to prevent disease and promote health. Work in public health clinics, home health agencies, and health maintenance organizations and other group facilities.

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Management nutritionists: manage and operate food-service systems, oversee large-scale meal planning and preparation in hospitals, health care facilities, company cafeterias, schools and prisons. They hire, train, and direct other specialists and food service workers; budget for and purchase food, equipment, and supplies; enforce sanitary and safety regulations; and prepare records and reports.

Clinical nutritionist/dietitians: These tow branches of nutrition discipline form the helping hand to the traditional medical system. Specialists confer with doctors and other health care professionals to coordinate medical and nutritional needs their job includes providing nutritional services to hospital outpatients, inpatients, nursing care facilities, and other medical institutions. They assess patients' nutritional needs, develop and implement nutrition programs, and evaluate and report the results.

Profession Task

Health oriented fields involve tasks that relate to the profession, professionals and the targeted care receivers. Nutrition concerned primarily with providing services beneficial to individuals and society, dedicating to improving the health and nutritional status of people and applying nutritional science to their language and lifestyles directed to the benefit of health. At present, nutritionists need to be professionally active and up to date in their knowledge, skills and recent field advancements. Working and communication skills are an important key to leadership and managerial effectiveness as well to handling and resolving nutrition health related problems. In addition to having the requisite scientific and technical skills, nutrition professionals must be able to relate effectively to others. Their jobs require effective interaction with clients, patients, employees, colleagues, other health professionals and the public. They are expected to apprehend from traditional unfruitful approaches to deliver health services with most professionalism, reliability and hard based scientific evidence.

The nutrition professional may be viewed as a "change agent" who needs the ability to intervene to promote change in behaviour. Knowledge of the social, cultural, psychological, and other forces affecting motivation for change in individuals or in groups, either positively or negatively, is necessary. As a health profession, numbers of skills are needed by nutrition specialists. They include techniques of planning, managing, interviewing and counseling ability to relate to groups, individuals, and communities; effectiveness in bringing about change; capacity in establishment of professional, interdisciplinary relationships and knowledge in personality, group, and societal dynamics.

Finally, it is the task of nutritionist to deal effectively with past general concepts such as people habits and individual awareness. Relying on such terms

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take the individual nowhere and keep the process in a circle of excuses. It is their challenging responsibility to fuse the sold dietary habits to correctable behaviours and the undefined awareness into measurable goals.

1.6 NUTRITION BEHAVIOUR (BEVAVIOURAL COUNSELING)

Behaviours related to nutrition are the most challenging issue facing the nutrition profession. People, even though, knowledgeable, aware and hoping to change are faced with life nutritional habits and with many factors in forcing them and barriers in the direction against change. The professional, especially in counseling, is expected to use a variety of intervention strategies to promote desired change. To be an effective agent of behavioral change, four categories of necessary skills were identified: *(a)* relationship-building skills, *(b)* interviewing and assessment skills, *(c)* problem-diagnosis skills, and *(d)* behavioral intervention skills. Furthermore, before applying change strategies, the professional should establish that the individual is an informed and at the determining stage for change. For effective interventions and counseling results, reviewing and comprehending factors and barriers related to change is needed.

Factors Affecting BC

- Factors that affect primarily individuals include their personal attitudes toward health activity and healthy eating; knowledge of nutrition; personal belief systems that impact eating and health activity; reasons for desiring behaviour change; elfperceived ability to make and sustain behaviour changes; support and financial resources if needed.
- Interpersonal Factors (Parental and family influence) including family eating and activity attitudes and behaviors, family views/knowledge of nutrition health association and family role modeling and family structure.
- Factors that primarily affect groups, such as families or peers, include cultural beliefs that impact eating and health activities; availability of adequate time within daily routines or school schedules to make and sustain behaviour changes; financial resources necessary to provide healthier food and activity choices; peer attitudes towards health, nutrition, body shape/ size and exercise; presence of supportive individuals; peer and parental modeling of healthy or unhealthy behaviours; and attempts to sabotage healthy food choices by family members or peers.
- Factors affecting community include the environments that can either promote or hinder behaviour changes made by families or individuals. The presence or lack of safe places and food venues that offer healthy food choices in reasonable portion sizes can either support or deter an

individual's attempts to improve their health. Policies related to food served and food availability and advertising, social acceptability and cultural norms.

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Working Toward Behavioural Change

How does behaviour change occur? This question probably has as many answers as there are diverse populations and cultures. The common approach of professionals who dispense more and more information to get people to change to more healthful eating behaviours is useless and needs modification. Giving out information does not always result in gains in knowledge, and nutrition knowledge does not necessarily lead to healthy food choices. The extent to which patients change their dietary behaviours after a nutrition counseling program, however, did not depend on how much their levels of awareness and motivation had changed. The counseling models has been shown to increase program effectiveness as nutrition counseling activities tailored to patients' different levels of awareness and motivation.

Experience has shown that change is a process and that people go through stages in their intentional changes. Furthermore, Readiness to change is the cornerstone stage for effective treatment approaches. Successful interventions, including education and counseling, is to plan them around the stage the individual is in currently to enhance motivation and desired outcomes. Thus it is important to assess and identify the client's readiness to change and match the treatment intervention to it and to implement defined working strategies in order to achieve desired outcomes and success.

There is a need to focus on the process of delivering nutrition services in treating illness, injury, and chronic health conditions through nutrition therapy. The two phases of nutrition therapy are *(a)* "assessment of the nutritional status of the patient or client" and *(b)* "treatment, which includes diet therapy, counseling, or use of specialized nutrition supplements". Two basic questions to consider include *(a)* what sort of change do we wish to bring about, and *(b)* how can this be accomplished?

1.7 THEORIES AND CONCEPTUAL MODELS

(1) Counseling Theories (Behavioural Change)

Four of the most commonly cited theories have been outlined: The AIDS Risk Reduction Model, The Theory of Reasoned Action, The Health Belief Model and The Stages of Change Theory. The first two theories are more to the psychology field and have yet to be extensively applied in research and they may not capture the elements necessary for behaviour change in every culture or population. The Stages of Change Theory and The Health Belief Model have been

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applied in nutrition education and counseling with defined implementation strategies.

1. AIDS Risk Reduction Model (ARRM)

The AIDS Risk Reduction Model (ARRM) is a three-stage model, introduced in 1990, provides a framework for explaining and predicting the behaviour change efforts of individuals specifically in relationship to the sexual transmission of HIV/AIDS.

2. Theory of Reasoned Action (TRA)

The TRA theory has explained and predicted a variety of human behaviours based on the premise that humans are rational and that the behaviours being explored are under volitional control. The theory provides a construct that links individual beliefs, attitudes, intentions, and behaviour variables together. Again, TRA demonstrated the inability, due to its individualistic approach, to consider the role of environmental and structural issues.

3. Health Belief Model (HBM)

The Health Belief Model (HBM) is a psychological model that attempts to explain and predict health behaviors by focusing on the attitudes and beliefs of individuals. People hold various beliefs about their health that may influence adherence to a dietary regimen more than their state of knowledge. The "Health Belief Model," which attempts to explain preventive health behaviour was developed originally to interpret the decisions of people not currently suffering disease, but wishing to prevent health problems. It has been extended to more general adherence as one of several theories attempting to explain dietary behaviour change. The model postulates that the person's beliefs about health are determinants of his or her readiness to take action. The three key beliefs are: *(a)* The extent to which the person believes that he is "susceptible" to contracting a specific disease, "has the disease" now, or is "susceptible" in the case of an illness from which he has recovered. *(b)* How serious he thinks the disease is or its consequences are in having a negative effect on his life? *(c)* what he perceives are the benefits of changing health behaviors in terms of reducing susceptibility to or severity of the disease, as compared to the psychological costs and barriers to taking action.

4. Stages of Change Theory

The Stages of Change Theory (Trans-theoretical Model) is a psychological one which includes five components of the Stages in a cyclical process that varies for each individual. The rationale behind "staging" people was to tailor therapy to a person's needs at his/her particular point in the change process. The Stages of Change Theory is a method for characterizing individuals along a change continuum with the intent of enhancing the effectiveness of interventions. In

addition, the theory offers a method for evaluating programs by measuring individual change.The stages of change is as follows:

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1. *Precomtemplation:* Individual has the problem (whether he/she recognizes it or not) and has no intention for change.
2. *Contemplation:* Individual recognizes the problem and is seriously thinking about changing.
3. *Preparation for Action:* Individual recognizes the problem and intends to change the behavior within the next month.
4. *Action:* Individual has enacted consistent behaviour change (*i.e.*, consistent condom usage) for less than six months.
5. *Maintenance:* Individual maintains new behaviour for six months or more.
6. *Termination:* This is the ultimate goal stage that dependent on the type of problem and the time length require for the maintenance stage.

Characteristics of Stage Change

1. People proceed through the stages in a spiral, rather than linear, fashion. Because lapses and relapse are common problems, regression to an earlier stage may be expected several times as people struggle to modify or cease behaviors and negative emotional reactions (guilt, shame, failure) may rise.
2. The processes of change involve shifts in behaviours, attitudes, and intentions.
3. In the early stages, focusing on the benefits of making a change and how that change can improve the individual's life is suggested.
4. In precontemplation, consciousness raising techniques, such as about the individual's risk for chronic disease based on dietary habits, and revaluation of values, problems, self, and environment are appropriate.
5. Moving individual from precontemplation to contemplation can be enhanced through communication methods such as posters, flyers, paycheck inserts, and electronic mail.
6. In the contemplation stage cognitive and affective self-reevaluation, in addition to consciousness rising, is suggested and.
7. In the preparation stage self-liberation (a belief that one can change) and behavioral goals should be initiated.
8. In action and maintenance stages, behavioral techniques of stimulus control, reinforcement management, recipe modification, coping responses during conditions when relapse is likely, and support from helping relationships are useful.

(2) CONCEPTUAL MODELS

Four broad categories of behavioural models have contributed to the understanding of lifestyle change and compliance with recommendations during counseling practice, these include:

1. communication.
2. rational belief.
3. self-regulative system.
4. social learning models.

Each of these models views the problem of behaviour associated with increased health risks from a different perspective. A counseling intervention may have one or more of these models as its theoretical framework.

Communications Models: These models highlight the importance of the generation of the message, the reception of the message, message comprehension, and belief in the substance of the message. The approach can avoid inappropriate techniques, for example trying to persuade an already-motivated patient that change is necessary. As is evident, communications models have been used for a wide range of counseling interventions.

Rational belief models: According to these theories, objective, logical thought processes determine behaviour; providing one has appropriate information on both the health risks and benefits, and the consequences of various behaviours.

Self-regulative systems models: The self-regulation process consists of three components: self-monitoring, self-evaluation, and self-reinforcement. A basic assumption is that people are rational and will act in accordance with their interest, once it is known to them. This model highlights the impact of social and cultural values and norms of the surrounding environment.

Operant and social learning models: These models focus on the stimuli that elicit or reinforce a specific behaviour. New ways of behaving occur through imitation and modeling, and by observing the behaviour of others.

1.8 NUTRITION COUNSELING STRATEGIES

MOTIVATIONAL NEGOTIATION

The top-down approach only results in knowledge change, but hardly ever does behaviour change. Nutrition educators have been exploring new strategies based on the need for the counselor to change from the traditional medical approach to a partnership where the client plays an active role. The new approach introduces Motivational negotiation, a term used for a client-centered approach used in an effort to promote positive changes in behaviour based on the person's

own motivation to change. Differences exist in the roles of the counselor and the client as the client and the counselor are seen as experts in their own areas.

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Strategies for Motivational Negotiation

Motivational negotiation uses a variety of strategies that stimulate the client to think about change which in turn enhances self motivation to change behaviors.

- Draws on values and goals of participant's helps to link current, past and future behaviour to concerns most relevant to the individual.
- Discrepancy between behaviour and values is acknowledged as "normal".
- Counselor and client are a team: Client is expert of own life; counselor is expert of facts and experiences of others.
- Client assumes active role, drives the situation, selects what information is appropriate and needed at that time and initiate Ideas for change when ready. The counselor will know when this happens by one or more of the following events:
  - —Client asks questions about change methods.
  - —Client expresses optimism about change.
  - —Client asks what the next step should be.
  - —Client experiments with change methods.

Essential Principles

There are five essential principles involved in MN:

1. *Express empathy:* Empathy is not sympathy. Counselors should reflect a level an understanding regarding the challenges faced by the client. They should not feel sorry for the client nor his or her situation.
2. *Avoid argumentation:* Arguing is destructive to the counseling environment and will force the client to shut down.
3. *Roll with resistance:* Resistance is a natural consequence of a turn in the conversation caused by something the counselor said. The counselor must stop immediately and redirect responses.
4. *Support self-efficacy:* Helping the client to believe in him/her and abilities to cope with new situation is a significant role of the counselor. It is more important than giving information about diet and exercise etc.
5. *Develop discrepancy:* Clients know that behaviors and beliefs are often at odds. They question this in adults; guiding young clients to recognize they are responsible for the same patterns will facilitate change.

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Techniques in MN

(1) Reflective Listening

The counselor only reflects back what is heard in order to elicit more input from the client. Counselor makes no assumptions about what the client means, encourages the client to make a personal exploration and conveys empathy. As the counselor becomes more comfortable with the technique, the reflective listening responses can incorporate deeper reflections. This is done by trying to draw out emotions or direct the client toward positive change statements.

(2) Session initiation

In order to initiate the counseling session, questions or as statements can be phrased, but none of these can be answered with a "yes" or "no" response. Once the conversation is started, the counselor may ask direct questions and allow adequate silence for response from the client.

(a) Phrases for Open-Ended Questions:

- Tell me why.
- What other things.
- How often do you.
- Why do you think?
- Let me see if I understand correctly.
- Give me some examples of.
- Tell me about.
- What is a typical day like for you?

(b) Phrases " a statement, not a question " :

- It sounds like you...
- It's difficult/easy for you to...
- You realize that...
- You're having trouble/success with...
- You understand that...
- You feel that...
- You do/don't see the need to ...

Once the client responds to the open-ended question, the counselor reflects back what was said. These phrases can be used to begin the reflection.

(3) *Dealing with Resistance*

- Acknowledge it exists.
- Reflect discrepancy.
- Change the focus or the subject.
- Change meaning.
- Emphasize personal control.
- Allow client to leave with pride.

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STUDENT ACTIVITY

1. Point out the important characteristics of diet counseling.

2. Discuss the tasks of the counselor.

3. Discuss the features of the conceptual model of counseling.

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1.9 THE PROCESS OF COUNSELING

(1) Steps of Counseling

(i) Assessing dietary habits

Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake and records the estimated amounts of all the foods and beverages the person consumed. The 24-hour food recall can be used to provide an estimate of energy and nutrient intake. As people tend to over or underestimate intake of certain foods, and food intake on one day may not accurately represent typical food intake. The food frequency questionnaire can sometimes provide a more accurate picture of a person's typical eating patterns.

The nutrition counselor may ask the client how often he or she consumes certain food groups or the consumed servings of dairy products, fruits, vegetables, grains and cereals, meats, or fats he or she consumes in a typical day, week, or month. Daily food records can be useful in assessing food intake as individual keeps a written record of the amounts of all foods and beverages consumed over a given period of time. Also the three-day food records kept over two weekdays and one weekend day are often used. The nutrition counselor can then use the food records to analyze actual energy and nutrient intake.

(ii) Identifying changes needed

The initial dietary assessment and interview provide the basis for identifying behaviours that need to be changed. Sometimes a person already has a good idea of what dietary changes are needed, but may require help in making the changes. Other times the nutrition counselor can help educate a person on the health effects of different dietary choices. The nutrition counselor and client work together to identify areas where change is needed prioritize changes and problem-solve as to how to make the changes.

(iii) Setting goals

The nutrition counselor and client set behaviour-oriented goals together. Goals should focus on the behaviours needed to achieve the desired dietary change, not on an absolute value, such as achieving a certain body weight. For a person working to prevent weight gain associated with certain medications, for example, his or her goals might be to increase the amount of fruits, vegetables, and whole grains consumed each day. Such changes would help prevent weight gain while placing the emphasis on needed behaviours rather than on actual weight.

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(iv) Making dietary change

In making dietary changes, each individual's situation, background and the factors that affect food decisions must be carefully considered. An individual, in gradual process, may start with one or two easier dietary changes the first few weeks and gradually make additional or more difficult changes over several weeks or months.

(v) Identifying barriers to change

Once the needed changes have been identified, the client and nutrition counselor think through potential problems that may arise. For example, changing eating behaviours may mean involving others, purchasing different foods, planning ahead for social events, or bringing special foods to work. Some common barriers to changing eating habits include: inconvenience, social gatherings, food preferences, lack of knowledge or time, cost etc.

(vi) Finding support

Family members are encouraged to attend nutrition counseling sessions with the client, especially if they share responsibility for food selection and preparation. Although the individual must make food choices and take responsibility for dietary changes, having the support and understanding of family and friends makes success more likely.

(vii) Maintaining changes

The challenge for the nutrition client lies not in making the initial dietary changes, but in maintaining them over the long-term. Self-monitoring, realistic expectations, and continued follow-up can help a person maintain dietary changes. Self-monitoring involves regularly checking eating habits against desired goals and keeping track of eating behaviours. Keeping a food diary on a daily or periodic basis helps the individual be more aware of his or her eating behaviours and provides a ready tool to analyze eating habits. Sometimes a simplified checklist to assure adequate intake of different food groups may be used. Individuals and nutrition counselors should not expect perfect dietary compliance—slips inevitably occur. The goal is to keep small slips, such as eating a few extra cookies, from becoming big slips, like total abandonment of dietary change. The counselor can help the client identify situations that may lead to relapse and plan ways to handle the situations ahead of time.

(2) Responsibility Sharing

(i) The relationship is a key to the effectiveness of problem solving.

(ii) The interaction between the helper and the individual is a goal-oriented process through which change occurs in the form of learning new information,

knowledge, or skills; gaining new insights and perspectives; modifying feelings; changing behaviours; and developing new resources as decisions are made and problems resolved. This can be imitated through an environment of respect, trust, concern, understanding, careful listening and practicing nonjudgmental responses.

(iii) Clients and patients should not be perceived as passive recipients of services. They are active participants in their treatment, working with the professional to restore or optimize their health. Ultimately, clients or patients are responsible for managing their own nutrition and health. The acceptance of help is voluntary, and the aim of the professional is to make people self-sufficient so that eventually they can manage on their own, solving future problems alone.

(iv) Those being counseled should be given an opportunity to discuss changes and to ask questions, because the more they internalize the new ideas and solutions, the greater is the likelihood of their being committed to them.

(v) Some people are more resistant than others to making changes in lifestyles, and such resistance to modifying their old patterns of behaviour is normal.

(vi) Change upsets the established ways of doing things, creates uncertainty and anxiety, and forces the need for adjustments. Because attitudes are thought to be the pre-disposing agents of practice, they should be explored as well.

(vii) Every dietary problem has two aspects—what the client thinks about it and how he or she feels about it. A client may think, for example, that a dietary regimen would be beneficial, but feel that it would be too difficult to follow. Both thoughts and feelings must be considered and dealt with for problem solving and change to occur.

(viii) The client's priorities take precedence over those of the professional. Although helpers may see exactly what needs changing, they should bear in mind that the client is the one who decides which changes to make and who ensures that they continue.

(ix) When an individual deals with the necessity of change in food patterns and behaviours, knowledge and education are not in themselves sufficient to motivate change. Many people already know what they should eat! But they do not always act on their knowledge.

(x) The health professional cannot assume that recommendations will be followed just because the patient or client knows what to do.

(xi) Wanting to make changes is a key point to consider in adopting changes in dietary practices. Why should any person change a lifetime of unrestricted eating that may be pleasurable? The person's motivation for change should be examined since people can be expected to resist change.

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(3) Applying " Stage of change"

The processes of change should be integrated into the stages of change so that the treatment intervention matches the client's stage of change. Accordingly, dietary programs and change initiation has to be designed and addressed when the client at the action stage. In counseling and education programs, to assume that everyone is at the action stage may lead one to plan an inappropriate intervention. Furthermore, most of programs and efforts are delivered in earlier stages and as if people are at the action stage.

(i) *precontemplation:* people are unaware or less than fully aware that a problem exists, deny they have a problem, or are not interested in change, and thus have no intention of changing behaviour in the near future. They may have tried a change previously and failed, and be resistant to one's efforts to suggest changes. To identify this stage, one may ask: "Are you seriously intending to change (name the problem behaviour) in the next six months?" For example, with people ignoring the relationship between a high-fat diet and coronary heart disease, one may ask, "Have you thought about eating less fat (or more fruits and vegetables) in the next six months?" .

(ii) *contemplation:* people are aware that a problem exists, such as needing to eat differently or exercise more, but they have no serious thought or commitment to making a change and may remain in this stage for months or years. They may be mentally struggling with the amount of energy, effort, and cost of overcoming a problem or may be discouraged by previous failures. One may ask, ' 'What have you been thinking about making a change?" "What are the pros and cons of doing it?" "How can you change your environment?" For example, "What do you think about eating less fat? What are the barriers to doing it?"

(iii) *preparation:* individuals are more determined to change and intending to take initial action soon, or in about 30 days. They may report small changes in the problem behaviour, such as reading a few food labels or buying fat-free ice cream. One may ask, "What, if any, changes have you planned or made in the past few weeks?"

(iv) *Action:* people overcome the problem by actively modifying their habits, behaviours, environments or experiences. It is important to remember that a majority of clients are not in the action stage when referred for counseling. Considerable commitment of time and energy is required for the action stage when individuals are trying to change. One may ask, "What are you doing differently?"

(v) *Maintenance:* people consolidate and stabilize gains made over several months in order to maintain the new, healthier habits and prevent relapse. For some people, this stage continues for months, years, or a lifetime. One may ask, "How do you handle small lapses?"

(vi) *Termination:* This is the ultimate goal's stage. However, some types of problems, such as eating changes, require a lifetime of maintenance instead. People, for example, tend to become more sedentary and overweight as they age, contributing to continual problems.

1.10 AFFILIATED HEALTH AND THERAPY SYSTEMS

There are several health systems and therapies that include nutritional counseling within their philosophies of healing.

- Alternative/complementary/integrative health system and therapies. A different dimension of health services that has it distinguished preventive and curative approaches based on own philosophy, principles and school of thoughts. Nutrition and dietary modifications are considered as a basic and first intervention in the application of most of its disciplines.
- Naturopathic: The foremost discipline in this type of counseling and adhere to the philosophy that proper food choices will influence the physical, mental, and emotional aspects of the body.
- Oriental Nutrition selects foods according to the philosophies of Traditional Chinese Medicine, concentrating primarily on the principles of yin (cooler) and yang (warmer). This philosophy teaches the idea of opposites, with one unable to exist without the other. Therefore, the human body requires both for healthy function. The diet is also determined according to constitution, personality, health, climate, and physical environment of the individual.
- Macrobiotic Counseling extends the principles of oriental nutrition while keeping the basic principles of yin and yang. The individual will be taught to select seasonal foods grown locally, to prepare them in a specific manner, and to cook them according to macrobiotic principles. It is the belief that illness is the result of poor nutrition, bad eating habits, and a sedentary lifestyle. Individuals will be encouraged in an exercise program as well as diet.
- Food Allergy: Counseling helps individuals become "food detectives" to sort through and eliminate those foods causing hypersensitivities, allergies, and intolerances. Foods that are eliminated may be introduced carefully one at a time at a later date. A food allergist will ultimately design a specific diet for the individual.

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- Clinical Nutrition tends to stress whole natural foods and nutritional supplements for health maintenance and treatment of disorders. Natural foods include organically grown, as well as those not processed, refined, or stored for any length of time.
- Western Nutrition (formal): Teaches the scientific knowledge behind the nutritional function of foods, as well as the daily nutritional requirements for optimal health. By understanding the chemical components of foods, individuals are taught to make healthier choices in their selections. Nutritionists in this direction will point out the risks involved in consuming processed, refined, and chemically treated foodstuffs and will recommend changes in eating habits to promote a healthier lifestyle.

Case Application

Changes in dietary fat intake were less affected by messages based on dietary guidelines than on those individually tailored to a person's stage of change. The stages of change model was used in defining the energy level of dietary fat in people's diets and was found to be effective in characterizing people by relative fat intake. Those in the stages of precontemplation to preparation may be expected to have higher intakes of fat than those in the action or maintenance stages. It is expected, also, that those on a higher fat intake will be in the earlier stages (precontemplation, contemplation, and preparation) and those consuming less fat and more fiber would be in the later stages (action and maintenance).

A woman with diabetes, for example, should believe that she has diabetes and is susceptible to serious complications, such as retinopathy; that adherence to the dietary regimen will reduce the likelihood of serious complications; that she has the ability to comply; and that the benefits of adherence outweigh the costs.

A man diagnosed with a high serum cholesterol level and made aware of its relationship to coronary heart disease may or may not decide to reduce the amount of fat and cholesterol in his diet, based on his health beliefs. Many individuals use denial as a defense mechanism and do not acknowledge that these consequences can happen to them.

General Considerations

Group sessions are often used for nutrition education and for employee communication and training. The effect of support groups on change can be either positive or negative. Face-to-face communication with someone who has successfully altered a behaviour can be effective in promoting the adoption of changes by others. While increasing knowledge and growing awareness may be developed in such groups, the actual change occurs through an individual's decision making.

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If the individual views taking action as time consuming, difficult, unpleasant, expensive, inconvenient, destroying quality of life, or upsetting, denial and avoidance motives may serve as barriers to change. If there are destructive beliefs, it is necessary to understand them and to facilitate more constructive ones. Best results are obtained when readiness to act is high and when physical, psychological, financial, and other barriers are low.

The pleasure of eating, perceived quality of life, cost considerations, accessibility, and social and cultural practices are examples of other factors affecting food choices. One needs to ask, "What does the individual value?" "Does the individual believe that personal actions can modify the threat?" "Does the individual feel capable of carrying out the recommended actions?"

Effective change in participants' food behaviours depended on the ability to recognize proper foods with low scores and substitute them for foods with higher scores, a process providing awareness and insight. Individual counseling encouraged achievement of a specific guideline score. The scores became an objective means of assessing dietary adherence and understanding.

In summary, no one intervention is successful all of the time in promoting dietary adherence. Feelings of personal control, self-efficacy, social support, self-monitoring, perceived threat of a disease, perceived benefits from changing behaviours, and other factors should be included in one's intervention strategies as well as into the stages of change. Goal setting, relapse prevention, stimulus control, and cognitive restructuring may be helpful. A comprehensive approach by the professional considering many of these factors is more likely to be successful.

Box 1. Overall Nutrition Counseling Description

Counseling Task

- work In a group setting.
- work In an individual setting.
- Acquiring information.
- Delivering nutrition education.
- Motivational Interviewing.
- Facilitated Discussion Groups.
- Communicate risk.
- Provide information.
- Initiate a behaviour change sequence.
- Identify client's goals and readiness.
- Present a menu of choices.
- Relationship building skills.

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- Helping skills.
- Ability to gain collaboration and empower the client.
- Sensitivity to multicultural and other client-specific uniqueness.
- Ability to sustain a long-term counseling relationship.
- Ability to assess and teach developmental skills.
- Measuring Success :
  - Treatment Goal followed.
  - Documenting in client record.
  - Evaluate client's willingness to comply.
  - Progress toward final treatment goal.
  - Develop a relationship – use helping skills.
  - Determine patient's "Stage of Change".
  - Empower the patient to set his/her own goals.
- Empowering Clients :
  - Identify diet related problems and issues.
  - Identify associated thoughts and feelings.
  - Identify health-related attitudes and beliefs and establish self-care goals.
  - Help patient develop and commit to a plan for achieving the goal.
  - Patients are active participants.
  - Does not dwell on failures of past.
  - Engage in problem solving as partners.
- Effective Communication :
  - Ability to use language appropriate to patient's level of understanding.
  - Giving enough knowledge but not overwhelming.
  - Ability to develop a relationship between themselves and their patients.
  - Ability to talk to them in a way that relieves anxiety.
  - Ability to communicate in a way that assures their being able to recall information.
- Building Assumptions :
  - No two clients or situations are alike.
  - No person or situation is static.
  - Client is expert on own problems.
  - No one best approach in dealing with each problem.

- Building Behavioral :
  - Commitment.
  - Belief that change is possible.
  - Be encouraging.
  - Learning healthier behaviors.
  - Brainstorming.
  - Care support, acceptance and feedback.
- Using the Model :
  - Know a patient's stage of change.
  - Design programs for action stage.
  - Consciousness raising techniques.
  - Appropriate strategic models.

Executing Tasks

- Assessing dietary habits.
- Identifying changes needed.
- Making dietary change is a gradual process.
- Identifying barriers to change.
- Setting goals
- Finding support
- Maintaining changes
- Applying stage of change theory.

REMARK

Nutrition counseling is a prime health subject that must be invested in for the benefit of people's health. It requires adequate qualifications, updated knowledge and skillfulness. All nutrition specialists and dietetics practitioners need to be confident of their abilities in patient/client and employee interactions. In addition to their scientific and technical competence, professionals are expected to be skilled in interpersonal relations. With patients and clients, a comprehensive approach considering psychological, cultural, environmental, and behavioral factors is needed in influencing dietary changes for the betterment of the individual's health. Clearly, information about proper diet is insufficient in changing dietary behaviors. The professional must be able to use a number of intervention strategies, including the helping process model, the health belief model, and stages of change in promoting adherence.

1.11 NUTRITION AND MENTAL HEALTH

NOTES

A person's food intake affects mood, behaviour, and brain function. A hungry person may feel irritable and restless, whereas a person who has just eaten a meal may feel calm and satisfied. A sleepy person may feel more productive after a cup of coffee and a light snack. A person who has consistently eaten less food or energy than needed over a long period of time may be apathetic and moody.

The human brain has high energy and nutrient needs. Changes in energy or nutrient intake can alter both brain chemistry and the functioning of nerves in the brain. Intake of energy and several different nutrients affect levels of chemicals in the brain called neurotransmitters . Neurotransmitters transmit nerve impulses from one nerve cell to another, and they influence mood, sleep patterns, and thinking. Deficiencies or excesses of certain vitamins or minerals can damage nerves in the brain, causing changes in memory, limiting problem-solving ability, and impairing brain function.

Several nutritional factors can influence mental health, including: overall energy intake, intake of the energy-containing nutrients (proteins, carbohydrates, and fats), alcohol intake, and intake of vitamins and minerals. Often deficiencies of multiple nutrients rather than a single nutrient are responsible for changes in brain functioning.

In the United States and other developed countries, alcoholism is often responsible for nutritional deficiencies that affect mental functioning. Diseases can also cause nutritional deficiencies by affecting absorption of nutrients into the body or increasing nutritional requirements. Poverty, ignorance, and fad diets also contribute to nutritional deficiencies.

Energy Intake and Mental Health

Energy, often referred to as the calorie content of a food, is derived from the carbohydrate, protein, fat, and alcohol found in foods and beverages. Although vitamins and minerals are essential to the body, they provide no energy. The human brain is metabolically very active and uses about 20 to 30% of a person's energy intake at rest. Individuals who do not eat adequate calories from food to meet their energy requirements will experience changes in mental functioning. Simply skipping breakfast is associated with lower fluency and problem-solving ability, especially in individuals who are already slightly malnourished. A hungry person may also experience lack of energy or motivation.

Chronic hunger and energy deprivation profoundly affects mood and responsiveness. The body responds to energy deprivation by shutting or slowing down nonessential functions, altering activity levels, hormonal levels, oxygen and nutrient transport, the body's ability to fight infection, and many other bodily functions that directly or indirectly affect brain function. People with a consistently low energy intake often feel apathetic, sad, or hopeless.

Developing fetuses and young infants are particularly susceptible to brain damage from malnutrition. The extent of the damage depends on the timing of the energy deprivation in relation to stage of development. Malnutrition early in life has been associated with below-normal intelligence, and functional and cognitive defects.

Carbohydrates and Mental Health

Carbohydrates include starches, naturally occurring and refined sugars, and dietary fiber. Foods rich in starches and dietary fiber include grain products like breads, rice, pasta and cereals, especially whole-grain products; fruits; and vegetables, especially starchy vegetables like potatoes. Foods rich in refined sugars include cakes, cookies, desserts, candy, and soft drinks.

Carbohydrates significantly affect mood and behavior. Eating a meal high in carbohydrates triggers release of a hormone called insulin in the body. Insulin helps let blood sugar into cells where it can be used for energy, but insulin also has other effects in the body. As insulin levels rise, more tryptophan enters the brain. Tryptophan is an amino acid, or a building block of protein, that affects levels of neurotransmitters in the brain. As more tryptophan enters the brain, more of the neurotransmitter serotonin is produced. Higher serotonin levels in the brain enhance mood and have a sedating effect, promoting sleepiness. This effect is partly responsible for the drowsiness some people experience after a large meal.

Some researchers claim that a high sugar intake causes hyperactivity in children. Although carefully controlled studies do not support this conclusion, high sugar intake is associated with dental problems. Further, foods high in refined sugars are often low in other nutrients, making it prudent to limit their use.

Proteins and Mental Health

Proteins are made up of amino acids linked together in various sequences and amounts. The human body can manufacture some of the amino acids, but there are eight essential amino acids that must be supplied in the diet. A complete or high-quality protein contains all eight of the essential amino acids in the amounts needed by the body. Foods rich in high-quality protein include meats, milk and other dairy products, and eggs. Dried beans and peas, grains, and nuts

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and seeds also contain protein, although the protein in these plant foods may be low in one or more essential amino acid. Generally, combining any two types of plant protein foods together will yield a complete, high-quality protein. For example, a peanut butter and jelly sandwich combines grain protein from the bread with nut protein from the peanut butter to yield a complete protein. A bean rice hot dish combines bean and grain protein for another complete protein combination.

Protein intake and intake of individual amino acids can affect brain functioning and mental health. Many of the neurotransmitters in the brain are made from amino acids. The neurotransmitter dopamine is made from the amino acid tyrosine. The neurotransmitter serotonin is made from the amino acid tryptophan. If the needed amino acid is not available, levels of that particular neurotransmitter in the brain will fall, and brain functioning and mood will be affected. For example, if there is a lack of tryptophan in the body, not enough serotonin will be produced, and low brain levels of serotonin are associated with low mood and even aggression in some individuals. Likewise, some diseases can cause a buildup of certain amino acids in the blood, leading to brain damage and mental defects. For example, a buildup of the amino acid phenylalanine in individuals with a disease called pheylketonuria can cause brain damage and mental retardation.

Fats and Mental Health

Dietary intake of fats may also play a role in regulating mood and brain function. Dietary fats are found in both animal and plant foods. Meats, regular-fat dairy products, butter, margarine, and plant oils are high in fats.

Although numerous studies clearly document the benefits of a cholesterol-lowering diet for the reduction of heart disease risk, some studies suggest that reducing fat and cholesterol in the diet may deplete brain serotonin levels, causing mood changes, anger, and aggressive behavior.

Other studies have looked at the effects of a particular kind of fat, the omega-3 fatty acids found in fish oils, and brain functioning. Although a few studies suggest omega-3 fatty acids are helpful with bipolar affective disorder and stress, results are inconclusive.

High levels of fat and cholesterol in the diet contribute to atherosclerosis, or clogging of the arteries. Atherosclerosis can decrease blood flow to the brain, impairing brain functioning. If blood flow to the brain is blocked, a stroke occurs.

Alcohol and Mental Health

A high alcohol intake can interfere with normal sleep patterns, and thus can affect mood. Alcoholism is one of the most common causes of nutritional

deficiencies in developed countries. Alcoholic beverages provide energy but virtually no vitamins or minerals. A person who consumes large amounts of alcohol will meet their energy needs but not their vitamin and mineral needs. In addition, extra amounts of certain vitamins are needed to break down alcohol in the body, further contributing to nutrient deficiencies.

Vitamins and Mental Health

Thiamin

Thiamin is a B vitamin found in enriched grain products, pork, legumes, nuts, seeds, and organ meats. Thiamin is intricately involved with metabolizing glucose, or blood sugar, in the body. Glucose is the brain's primary energy source. Thiamin is also needed to make several neurotransmitters.

Alcoholism is often associated with thiamin deficiency. Alcohol interferes with thiamin metabolism in the body, and diets high in alcohol are often deficient in vitamins and minerals. Individuals with a thiamin deficiency can develop Wernicke-Korsakoff syndrome, which is characterized by confusion, mental changes, abnormal eye movements, and unsteadiness that can progress to severe memory loss.

Vitamin B-12

Vitamin B-12 is found only in foods of animal origin like milk, meat, or eggs. Strict vegans who consume no animal-based foods need to supplement their diet with vitamin B-12 to meet the body's need for this nutrient.

Vitamin B-12 is needed to maintain the outer coating, called the myelin sheath, on nerve cells. Inadequate myelin results in nerve damage and impaired brain function. Vitamin B-12 deficiency can go undetected in individuals for years, but it eventually causes low blood iron, irreversible nerve damage, dementia , and brain atrophy.

Folic Acid

Folic acid is another B vitamin found in foods such as liver, yeast, asparagus, fried beans and peas, wheat, broccoli, and some nuts. Many grain products are also fortified with folic acid. In the United States, alcoholism is a common cause of folic acid deficiency.

Folic acid is involved in protein metabolism in the body and in the metabolism of some amino acids, particularly the amino acid methionine. When folic acid levels in the body are low, methionine cannot be metabolized properly and levels of another chemical, homocysteine, build up in the blood. High blood homocysteine levels increase risk of heart disease and stroke.

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Even modest folic acid deficiency in women causes an increased risk of neural tube defects, such as spina bifida, in developing fetuses. Folic acid deficiency also increases risk of stroke. Some studies suggest that folic acid deficiency leads to a range of mental disorders, including depression, but this concept remains controversial. Folic acid deficiency can lower levels of serotonin in the brain.

Niacin

The B vitamin niacin is found in enriched grains, meat, fish, wheat bran, asparagus, and peanuts. The body can also make niacin from the essential amino acid tryptophan, which is found in high-quality animal protein foods like meat and milk. Niacin deficiency used to be common in the southern United States but is now common only in developing countries such as India and China.

Niacin is involved in releasing energy in the body from carbohydrates, proteins, and fats. A deficiency of niacin produces many mental symptoms such as irritability, headaches, loss of memory, inability to sleep, and emotional instability. Severe niacin deficiency progresses to a condition called pellagra, which is characterized by the four D's: dermatitis (a rash resembling a sunburn), diarrhoea, dementia, and ultimately, death. The mental symptoms in pellagra can progress to psychosis , delirium , coma, and death.

ESSENTIAL VITAMINS AND THEIR EFFECTS

Vitamin	What It Does For The Body
Vitamin A (Beta Carotene)	Promotes growth and repair of body tissues; reduces susceptibility to infections; aids in bone and teeth formation; maintains smooth skin
Vitamin B-1 (Thiamin)	Promotes growth and muscle tone; aids in the proper functioning of the muscles, heart, and nervous system; assists in digestion of carbohydrates
Vitamin B-2 (Riboflavin)	Maintains good vision and healthy skin, hair, and nails; assists in formation of antibodies and red blood cells; aids in carbohydrate, fat, and protein metabolism
Vitamin B-3 (Niacinamide)	Reduces cholesterol levels in the blood; maintains healthy skin, tongue, and digestive system; improves blood circulation; increases energy
Vitamin B-5	Fortifies white blood cells; helps the body's resistance to stress; builds cells

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Vitamin B-6 (Pyridoxine)	Aids in the synthesis and breakdown of amino acids and the metabolism of fats and carbohydrates; supports the central nervous system; maintains healthy skin
Vitamin B-12 (Cobalamin)	Promotes growth in children; prevents anemia by regenerating red blood cells; aids in the metabolism of carbohydrates, fats, and proteins; maintains healthy nervous system
Biotin	Aids in the metabolism of proteins and fats; promotes healthy skin
Choline	Helps the liver eliminate toxins
Folic Acid (Folate, Folacin)	Promotes the growth and reproduction of body cells; aids in the formation of red blood cells and bone marrow
Vitamin C (Ascorbic Acid)	One of the major antioxidants; essential for healthy teeth, gums, and bones; helps to heal wounds, fractures, and scar tissue; builds resistance to infections; assists in the prevention and treatment of the common cold; prevents scurvy
Vitamin D	Improves the absorption of calcium and phosphorous (essential in the formation of healthy bones and teeth) maintains nervous system
Vitamin E	A major antioxidant; supplies oxygen to blood; provides nourishment to cells; prevents blood clots; slows cellular aging
Vitamin K (Menadione)	Prevents internal bleeding; reduces heavy menstrual flow

Vitamin B-6

Vitamin B-6, also known as pyridoxine, is found in many plant and animal foods, including chicken, fish, pork, whole wheat products, brown rice, and some fruits and vegetables. In healthy individuals, deficiency of vitamin B-6 is rare, but certain drugs, including some antidepressant drugs, can induce vitamin B-6 deficiency. Vitamin B-6 is needed by the body to produce most of the brain's neurotransmitters. It is also involved in hormone production. Although rare, vitamin B-6 deficiency is characterized by mental changes such as fatigue , nervousness, irritability, depression, insomnia , dizziness, and nerve changes. These

mental changes are related to the body's decreased ability to manufacture neurotransmitters with vitamin B-6 deficiency.

Just as vitamin B-6 deficiency causes mental changes, so does excess of vitamin B-6. Vitamin B-6 supplements are used by many individuals for a variety of conditions, including carpal tunnel syndrome, premenstrual syndrome, and fibrocystic breast disease. Doses of 500 mg per day or more can cause nerve damage, dizziness, sensory loss, and numbness.

Vitamin E

Vitamin E is a fat-soluble vitamin that is plentiful in the diet, particularly in plant oils, green leafy vegetables, and fortified breakfast cereals. Vitamin E deficiency is very rare, except in disorders that impair absorption of fat-soluble vitamins into the body, such as cystic fibrosis, and liver diseases.

Vitamin E deficiency causes changes in red blood cells and nerve tissues. It progresses to dizziness, vision changes, muscle weakness, and sensory changes. If left untreated, the nerve damage from vitamin E deficiency can be irreversible. Because it is an antioxidant, vitamin E has also been studied for treatment of neurological conditions such as Parkinson's and Alzheimer's disease . Although results are inconclusive, vitamin E shows some promise in slowing the progression of Parkinson's disease.

Vitamin A

Vitamin A is a fat-soluble vitamin found in meats, fish and eggs. A form of vitamin A, beta-carotene, is found in orange and green leafy vegetables such as carrots, yellow squash, and spinach. Headache and increased pressure in the head is associated with both deficient and excess vitamin A intake. Among other effects, excess vitamin A intake can cause fatigue, irritability, and loss of appetite. Generally, doses must exceed 25,000 international units of vitamin A over several months to develop such symptoms.

Minerals and Mental Health

Iron

Iron is a trace mineral that is essential for formation of hemoglobin, the substance that carries oxygen to cells throughout the body. Iron is found in meat, poultry, and fish. Another form of iron that is not as well absorbed as the form in animal foods is found in whole or enriched grains, green leafy vegetables, dried beans and peas, and dried fruits. Consuming a food rich in vitamin C, such as orange juice, at the same time as an iron-containing plant food will enhance iron absorption from the food.

Iron deficiency eventually leads to anemia, with insufficient oxygen reaching the brain. The anemia can cause fatigue and impair mental functioning. Iron deficiency during the first two years of life can lead to permanent brain damage.

Magnesium

The mineral magnesium is found in green leafy vegetables, whole grains, nuts, seeds, and bananas. In areas with hard water, the water may provide a significant amount of magnesium. In addition to its involvement in bone structure, magnesium aids in the transmission of nerve impulses.

Magnesium deficiency can cause restlessness, nervousness, muscular twitching, and unsteadiness. Acute magnesium deficiency can progress to apathy, delirium, convulsions, coma, and death.

Manganese

Manganese is a trace mineral found in whole grains and nuts, and to a lesser extent, fruits and vegetables. Manganese is involved in carbohydrate metabolism and brain functioning. Although very rare, manganese deficiency can cause abnormalities in brain function. Miners of manganese in South America have developed manganese toxicity called manganese madness, with neurological symptoms similar to Parkinson's disease.

Copper

The richest sources of the trace mineral copper in the diet are organ meats, seafood, nuts, seeds, whole grain breads and cereals, and chocolate. In addition to other functions, copper is involved in iron metabolism in the body and in brain function. Deficiency of copper causes anemia, with inadequate oxygen delivery to the brain and other organs. Copper deficiency also impairs brain functioning and immune system response, including changes in certain chemical receptors in the brain and lowered levels of neurotransmitters.

Zinc

The trace mineral zinc is found in red meats, liver, eggs, dairy products, vegetables, and some seafoods. Among other functions, zinc is involved in maintaining cell membranes and protecting cells from damage. Zinc deficiency can cause neurological impairment, influencing appetite, taste, smell, and vision. It has also been associated with apathy, irritability, jitteriness, and fatigue.

Selenium

Good sources of the trace mineral selenium include seafood, liver, and eggs. Grains and seeds can also be good sources of selenium depending on the selenium

content of the soil they are grown in. Selenium is needed for the synthesis of some hormones and helps protect cell membranes from damage.

Although selenium deficiency is very rare, selenium toxicity has occurred in regions of the world with high selenium soil content, such as China. Selenium toxicity causes nervous system changes, fatigue, and irritability.

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1.12 DIET THERAPY

Special diets are designed to help individuals make changes in their usual eating habits or food selection. Some special diets involve changes in the overall diet, such as diets for people needing to gain or lose weight or eat more healthfully. Other special diets are designed to help a person limit or avoid certain foods or dietary components that could interfere with the activity of a medication. Still other special diets are designed to counter nutritional effects of certain medications.

Purpose

Special diets are used in the treatment of persons with certain mental disorders to:

- identify and correct disordered eating patterns
- prevent or correct nutritional deficiencies or excesses
- prevent interactions between foods or nutrients and medications

Special types of diets or changes in eating habits have been suggested for persons with certain mental disorders. In some disorders, such as eating disorders or substance abuse, dietary changes are an integral part of therapy. In other disorders, such as attention-deficit/hyperactivity disorder, various proposed diets have questionable therapeutic value.

Many medications for mental disorders can affect a person's appetite or nutrition-related functions such as saliva production, ability to swallow, bowel function, and activity level. Changes in diet or food choices may be required to help prevent negative effects of medications.

Finally, interactions can occur between some medications used to treat persons with mental disorders and certain foods or nutritional components of the diet. For example, grapefruit and apple juice can interact with some specific psychotropic drugs (medications taken for psychiatric conditions) and should be avoided by individuals taking those medicines. Tyramine, a natural substance found in aged or fermented foods, can interfere with the functioning of monoamine oxidase inhibitors and must be restricted in individuals using these types of medications. A person's pre-existing medical condition and nutritional needs should be taken into account when designing any special diet.

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Eating Disorders

The two main types of eating disorders are anorexia nervosa and bulimia nervosa. Individuals with anorexia nervosa starve themselves, while individuals with bulimia nervosa usually have a normal or slightly above normal body weight but engage in binge eating followed by purging with laxatives, vomiting, or exercise.

Special diets for individuals with eating disorders focus on restoration of a normal body weight and control of bingeing and purging. These diets are usually carried out under the supervision of a multidisciplinary team, including a physician, psychologist , and dietitian.

The overall dietary goal for individuals with anorexia nervosa is to restore a healthy body weight. An initial goal might be to stop weight loss and improve food choices. Energy intake is then increased gradually until normal weight is restored. Because individuals with anorexia nervosa have an intense fear of gaining weight and becoming fat, quantities of foods eaten are increased very slowly so that the patient will continue treatments and therapy.

The overall dietary goal for individuals with bulimia nervosa is to gain control over eating behaviour and to achieve a healthy body weight. An initial goal is to stabilize weight and eating patterns to help the individual gain control over the binge-purge cycle. Meals and snacks are eaten at regular intervals to lessen the possibility that hunger and fasting will trigger a binge. Once eating behaviours have been stabilized, energy intake can be gradually adjusted to allow the individual to reach a normal body weight healthfully.

For individuals with either anorexia nervosa and bulimia, continued follow-up and support are required even after normal weight and eating behaviors are restored, particularly since the rate of relapse is quite high. (Relapse occurs when a patient returns to the old behaviours that he or she was trying to change or eliminate.) In addition to dietary changes, psychotherapy is an essential part of the treatment of eating disorders and helps the individual deal with fears and misconceptions about body weight and eating behaviour.

Attention-Deficit/Hyperactivity Disorder

Attention-Deficit/Hyperactivity Disorder (ADHD) accounts for a substantial portion of referrals to child mental health services. Children with ADHD are inappropriately active, easily frustrated or distracted, impulsive, and have difficulty sustaining concentration. Usual treatment of ADHD involves medication, behavioural management, and education.

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Many dietary factors have been proposed as causes of ADHD, including sugar, food additives, and food allergies. In the 1970s the Feingold diet became popular for treatment of ADHD. The Feingold diet excludes artificial colorings and flavorings, natural sources of chemicals called salicylates (found in fruits), and preservatives called BHT and BHA. Although scientific evidence does not support the effectiveness of the Feingold diet, a modified Feingold diet including fruits has been shown to be nutritionally balanced and should not be harmful as long as the child continues to receive conventional ADHD treatment also.

A high intake of sugar and sugary foods has also been implicated as a cause of ADHD. Although carefully controlled studies have shown no association between sugar and ADHD, diets high in sugar should be discouraged because they are often low in other nutrients and can contribute to dental problems.

Food allergies have also been implicated as a cause of ADHD, and some groups have suggested using elimination diets to treat ADHD. Elimination diets omit foods that most commonly cause allergies in children, such as eggs, milk, peanuts, or shellfish. Although research does not support the value of elimination diets for all children with ADHD, children with specific food allergies can become irritable and restless. A child with a suspected food allergy should be evaluated by an allergist.

Stimulant medications used to treat ADHD, such as methylphenidate (Ritalin), can cause appetite loss (anorexia) and retard growth, although recent research suggests that a child's ultimate height appears not to be affected by stimulant medications. As a precaution, children on such medicines should receive close monitoring of growth patterns, and parents should carefully observe their child's appetite and interest in meals and snacks. Providing regular meals and snacks, even when the child is not hungry, can help to assure adequate growth.

Mood Disorders

Mood disorders include both depression (unipolar disorder) and episodes of mania followed by depression (bipolar disorder). Both types of disorders can affect appetite and eating behaviour.

Although some depressed individuals eat more than usual and gain weight, depression more often causes loss of appetite and weight loss. As depressed individuals lose interest in eating and social relationships, they often skip meals and ignore feelings of hunger. Unintentional weight losses of up to 15% of body mass can occur.

Treatment with antidepressant medications often reverses weight loss and restores appetite and interest in eating. If the individual has lost a significant amount of weight, he or she may need to follow a high-calorie diet to restore weight to normal levels and replaced nutritional deficiencies. High-calorie diets

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usually include three balanced meals from all the food groups and several smaller snacks throughout the day. A protein/calorie supplement may also be necessary for some individuals.

Depression is sometimes treated with medications called monoamine oxidase inhibitors. Individuals on these medications will need to follow a tyramine-restricted diet (see below under monoamine oxidase inhibitors).

Individuals with mania are often treated with lithium. Sodium and caffeine intake can affect lithium levels in the blood, and intake of these should not suddenly be increased or decreased. Weight gain can occur in response to some antidepressant medications and lithium.

Schizophrenia

Individuals with schizophrenia can have hallucinations, delusional thinking, and bizarre behaviour. These distorted behaviours and thought processes can also be extended to delusions and hallucinations about food and diet, making people with schizophrenia at risk for poor nutrition.

Individuals with schizophrenia may believe that certain foods are poisonous or have special properties. They may think they hear voices telling them not to eat. Some may eat huge quantities of food thinking that it gives them special powers. Individuals with untreated schizophrenia may lose a significant amount of weight. Delusional beliefs and thinking about food and eating usually improve once the individual is started on medication to treat schizophrenia.

Substance Abuse

Substance abuse can include abuse of alcohol, cigarettes, marijuana, cocaine, or other drugs. Individuals abusing any of these substances are at risk for nutritional problems. Many of these substances can reduce appetite, decrease absorption of nutrients into the body, and cause the individual to make poor food choices.

Special diets used for withdrawal from substance abuse are designed to correct any nutritional deficiencies that have developed, aid in the withdrawal of the substance, and prevent the individual from making unhealthful food substitutions as the addictive substance is withdrawn. For example, some individuals may compulsively overeat when they stop smoking, leading to weight gain. Others may substitute caffeine-containing beverages such as soda or coffee for an addictive drug. Such harmful substitutions should be discouraged, emphasizing well-balanced eating combined with adequate rest, stress management, and regular exercise. Small, frequent meals and snacks that are rich in vitamins and minerals from healthful foods should be provided. Fluid intake should be generous, but caffeine-containing beverages should be limited.

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Individuals withdrawing from alcohol may need extra thiamin supplementation, either intravenously or through a multivitamin supplement because alcohol metabolism in the body requires extra thiamin. Individuals taking drugs to help them avoid alcohol will need to avoid foods with even small amounts of alcohol (see below).

Common withdrawal symptoms and dietary suggestions for coping with these symptoms include:

- *Appetite loss:* eat small, frequent meals and snacks; limit caffeine; use nutritional supplements if necessary.
- *Appetite increase:* eat regular meals; eat a variety of foods; limit sweets and caffeine.
- *Diarrhea:* eat moderate amounts of fresh fruits, vegetables, concentrated sugars, juices, and milk; increase intake of cereal fiber.
- *Constipation:* drink plenty of fluids; increase fiber in the diet; increase physical activity.
- *Fatigue:* eat regular meals; limit sweets and caffeine; drink plenty of fluid.

Dietary Considerations and Medications

Medications that Affect Body Weight

Many medications used to treat mental disorders promote weight gain, including:

- anticonvulsants (divalproex)
- certain types of antidepressants (amitriptyline)
- antipsychotic medications (clozapine , olanzapine , quetiapine , and risperidone)

Dietary treatments for individuals taking these medications should focus on a balanced, low-fat diet coupled with an increase in physical activity to counter the side effects of these medications. Nutrient-rich foods such as fruits, vegetables, and whole grain products should be emphasized in the diet, whereas sweets, fats, and other foods high in energy but low in nutrients should be limited. Regular physical activity can help limit weight gain caused by these medications.

Some medications can cause loss of appetite, restlessness, and weight loss. Individuals on such medications should eat three balanced meals and several smaller snacks of protein and calorie-rich foods throughout the day. Eating on a regular schedule rather than depending on appetite can help prevent weight loss associated with loss of appetite.

Medications that Affect Gastrointestinal Function

Many psychiatric medications can affect gastrointestinal functioning. Some drugs can cause dry mouth, difficulty swallowing, constipation, altered taste, heartburn, diarrhoea, or nausea. Consuming frequent smaller meals, drinking adequate fluids, modifying texture of foods if necessary, and increasing fiber content of foods can help counter gastrointestinal effects of medications.

Monoamine Oxidase Inhibitors

Individuals being treated with monoamine oxidase inhibitors (MAOIs) such as tranylcypromine , phenelzine , and isocarboxazid, must carefully follow a tyramine-restricted diet. Tyramine, a nitrogen-containing substance normally present in certain foods, is usually broken down in the body by oxidases. However, in individuals taking MAOIs, tyramine is not adequately broken down and builds up in the blood, causing the blood vessels to constrict and increasing blood pressure.

Tyramine is normally found in many foods, especially protein-rich foods that have been aged or fermented, pickled, or bacterially contaminated. Cheese is especially high in tyramine. A tyramine intake of less than 5 milligrams daily is recommended. A diet that includes even just 6 milligrams of tyramine can increase blood pressure; a diet that provides 25 milligrams of tyramine can cause life-threatening increases in blood pressure.

Tyramine-Restricted Diet. Tyramine is found in aged, fermented and spoiled food products. The tyramine content of a specific food can vary greatly depending on storage conditions, ripeness, or contamination. Reaction to tyramine-containing foods in individuals taking MAOIs can also vary greatly depending on what other foods are eaten with the tyramine-containing food, the length of time between MAOI dose and eating the food, and individual characteristics such as weight, age, etc.

Foods to avoid on a tyramine-controlled diet include:

- all aged and mature cheeses or cheese spreads, including foods made with these cheeses, such as salad dressings, casseroles, or certain breads
- any outdated or nonpasteurized dairy products
- dry fermented sausages such as summer sausage, pepperoni, salami, or pastrami
- smoked or pickled fish
- non-fresh meat or poultry
- leftover foods containing meat or poultry
- tofu and soy products

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- overripe, spoiled, or fermented fruits or vegetables
- sauerkraut
- fava or broad beans
- soups containing meat extracts or cheese
- gravies containing meat extracts or nonfresh meats
- tap beer
- nonalcoholic beer
- yeast extracts
- soy sauce
- liquid powdered protein supplements

Perishable refrigerated items such as milk, meat, or fruit should be eaten within 48 hours of purchase. Any spoiled food and food stored in questionable conditions should not be eaten.

Lithium

Lithium is often used to treat individuals with mania. Lithium can cause nausea, vomiting, anorexia, diarrhoea, and weight gain. Almost one-half of individuals taking lithium gain weight.

Individuals taking lithium should maintain a fairly constant intake of sodium (found in table salt and other food additives) and caffeine in their diet. If an individual restricts sodium intake, less lithium is excreted in the urine and blood lithium levels rise. If an individual increases caffeine intake, more lithium is excreted in the urine and blood levels of lithium fall.

Anticonvulsant Medications

Sodium caseinate and calcium caseinate can interfere with the action and effectiveness of some anticonvulsants. Individuals taking these anticonvulsants should read labels carefully to avoid foods containing these additives.

Psychotropic Medications

Some psychotropic medications, such as amitriptyline, can decrease absorption of the vitamin riboflavin from food. Good food sources of riboflavin include milk and milk products, liver, red meat, poultry, fish, and whole grain, and enriched breads and cereals. Riboflavin supplements may also be needed.

Other psychotropic drugs, such as fluvoxamine , sertraline , fesasodone, alprazolam , triazolam , midazolam, carbamazepine , and clonazepam , interact with grapefruit juice, so individuals taking these drugs must take care to avoid

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grapefruit juice. In some cases, apple juice must be avoided, as well. Patients should discuss potential drug interactions with their doctor or pharmacist.

Caffeine-Restricted Diet

Caffeine is a stimulant and can interfere with the actions of certain medications. As stated, people taking lithium and people recovering from addictions may be asked by their treatment team to monitor (and, in the case of addictions, restrict) their caffeine intake. Foods and beverages high in caffeine include:

- chocolate
- cocoa mix and powder
- chocolate ice cream, milk, and pudding
- coffee
- cola beverages
- tea

Alcohol-Restricted Diet

Alcohol interacts with some medications used to treat mental disorders. In the case of alcoholism recovery, the negative interaction resulting from the combination of one medication (disulfiram or Antabuse) and alcohol consumption is actually part of treatment for some people. (The medication causes an extremely unpleasant reaction to any alcohol consumed, reinforcing or rewarding the avoidance of alcohol.)

When individuals are taking medication that requires that they avoid alcohol, foods containing alcohol must be avoided as well as beverage alcohol. The following foods contain small amounts of alcohol:

- flavor extracts, such as vanilla, almond, or rum flavorings
- cooking wines
- candies or cakes prepared or filled with liqueur
- apple cider
- cider and wine vinegar
- commercial eggnog
- bernaise or bordelaise sauces
- desserts such as crepes suzette or cherries jubilee
- teriyaki sauce
- fondues

1.13 NUTRITIONAL SUPPORT IN HOSPITAL

NOTES

A high level of malnutrition has been reported in hospital and is linked to poor clinical outcome. Oral, enteral or parenteral nutrition support, alone or in combination, should be considered for all people who are either malnourished or at risk of malnutrition. Potential swallowing problems should be taken into account.

Identifying Patients at Risk

- All hospital inpatients on admission and all outpatients at their first clinic appointment should be screened. Screening should be repeated weekly for inpatients and when there is clinical concern for outpatients.
- Nutrition support should be considered in people who are malnourished, as defined by any of the following:
  - A body mass index (BMI) of less than 18.5 kg/m^2.
  - Unintentional weight loss greater than 10% within the last 3–6 months.
  - A BMI of less than 20 kg/m^2 and unintentional weight loss greater than 5% within the last 3–6 months.
- Nutrition support should be considered in people at risk of malnutrition, defined as those who have:
  - Eaten little or nothing for more than 5 days and/or are likely to eat little or nothing for 5 days or longer.
  - A poor absorptive capacity.
  - High nutrient losses.
  - Increased nutritional needs from causes such as catabolism.

Improving Nutrition

- Anti-emetics for nausea.
- Sloppy or liquid meals for patients with dysphagia from oesophageal stricture.
- Swallowing disorders from neurological causes benefit from more viscous liquids.
- Adequate pain relief may improve appetite.
- Dedicating time to feeding by nurses, healthcare assistants or relatives can help with the weak and elderly.
- When these measures are inadequate, oral supplements may be added.
- If these fail, enteral or parenteral nutrition may be required, *e.g.*, for patients with prolonged unconsciousness, inability to swallow or intestinal failure.

May also be needed following major GI surgery, in aggressive chemotherapy with severe inflammation of the mouth.

- Where possible, oral or enteral nutrition should be preferred to parenteral because it is cheaper, simpler and has other benefits, *e.g.*, maintains integrity of gut barrier.

Specific Indications for Nutrition

- Complete mechanical intestinal obstruction
- Ileus or intestinal hypomotility
- Severe uncontrollable diarrhoea
- Severe acute pancreatitis
- High output fistulae
- Shock

In patients who require immediate support but are expected to improve within 1–2 weeks, can use peripheral vein nutritional support via standard intravenous lines. Nutritional support needs to include lipid, dextrose with amino acids.

Nutritional Requirements

- Water:
  - For most patients allow 1,500 ml for the first 20 kg body weight plus 20 ml for every kg after this, and replace additional losses as they occur.
  - In average sized adult, approx. needs are 30–35 ml/kg or 1ml/kcal of energy.
- Energy:
  - Can be estimated by multiplying body weight in kg by 30–35 kcal (in obese patients use ideal body weights).
- Protein:
  - If adequate calories, most patients need 0.8–1.2g of protein/kg/day.
  - In moderate to severe stress up to 1.5 g/kg/day is required.
  - Use ideal weight for patients with significant obesity.
- Electrolytes and minerals:
  - Vary widely but most patients need 45–145 meq/day.
  - Also need adequate vitamins and trace minerals usually supplied by premixed enteral solutions (lower quantities are needed in parenteral nutrition).
- Essential fatty acids:
  - 2.4% of total calories should be given as linoleic acid.
  - In parenteral nutrition, give at least 250 ml 20% intravenous fat 2–3 x weekly.

STUDENT ACTIVITY

NOTES

1. Discuss the primary purpose of diet therapy.

2. Outline the importance of nutritional support in hospital.

1.14 SUMMARY

- Diet counseling is an ongoing process in which a health professional, usually a registered dietitian, works with an individual to assess his or her usual dietary intake and identify areas where change is needed.
- The goal of nutrition counseling is to help a person make and maintain dietary changes. For a person with a mental disorder, dietary change may be needed to promote healthier eating, to adopt a therapeutic diet, or to avoid nutrient-drug interactions.
- Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake.

- The need for nutrition counseling has grown with the increase in chronic conditions such as Cancer, Diabetes, Eating Disorders, Excess Weight Gain, Food Allergies, Gastrointestinal Disorders etc.
- A person's food intake affects mood, behaviour, and brain function. A hungry person may feel irritable and restless, whereas a person who has just eaten a meal may feel calm and satisfied.

1.15 GLOSSARY

- **Diet Counseling:** It is an ongoing process in which a health professional, usually a registered dietitian, works with an individual to assess his or her usual dietary intake and recommend changes.
- **The AIDS Risk Reduction Model (ARRM):** It is a three-stage model, introduced in 1990, provides a framework for explaining and predicting the behaviour change.
- **The TRA Theory:** It a theory which explain and predict a variety of human behaviours based on the premise that humans are rational and that the behaviours being explored are under volitional control.
- **The Health Belief Model (HBM):** It is a psychological model that attempts to explain and predict health behaviours by focusing on the attitudes and beliefs of individuals.
- **The Stages of Change Theory (Trans-theoretical Model):** It is a psychological one theory which includes five components of the Stages in a cyclical process that varies for each individual.

1.16 REVIEW QUESTIONS

1. What is diet counseling?
2. What are the goals of diet counseling? Discuss.
3. What are the main principles of diet counseling?
4. Discuss the role of counselor.
5. How is diet counseling done? Discuss.
6. Discuss the purposes of diet therapy.
7. Write a short note on "Nutritional counseling strategy".

1.17 FURTHER READINGS

- Narayana Rao, S., *Counselling and guidance*, 2nd edition, Tata Mc. Graw Hill Publishing Co., Ltd., New Delhi, 1991.

UNIT—II

TECHNIQUES OF DIET COUNSELING AND FEBRILE CONDITIONS

OBJECTIVES

After going through this unit, students will be able to:

- state the techniques of diet counseling;
- specify the diet counseling for obese people;
- discuss the types and etiology of febrile conditions;
- explain the metabolic changes and diet counseling for febrile conditions.

STRUCTURE

2.1 INTRODUCTION

The definition of a balanced diet is a diet that, when taken regularly, will provide in quantity and quality sufficient macro and micro nutrients and other non-nutrient elements (fibres, vitamins, antioxidants and other bioactive substances) to maintain the optimal functions of the body and therefore optimal health in the general context of a healthy lifestyle (physical activity and mental status).

The amount of food needed everyday depends upon age, body size, level of physical activity, gender, and, in females, pregnancy or breast-feeding. Food habits depend on ethnicity, nationality and culture, but national trends in the composition of a healthy diet may not be dramatically different.

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The adapted recommendations must ensure that the nutrition needs of the population are met, thus reducing the risk of chronic diseases.

A good and healthy diet plan involves eating a variety of foods to get the required nutrients while, at the same time, providing the right amount of calories to maintain a healthy weight.

Counseling in general is a term describing a process that assists people to learn about themselves, their environment and methods of handling problems. Nutrition counseling is an ongoing process in which a health professional, usually a nutritionist works with an individual to assess his or her usual dietary intake and identify areas where change is needed.

All healthy balanced diets should be always complete and incorporate the seven essential nutrients: protein (mostly vegetal), fats, carbohydrates, fibre, water, vitamins and minerals.

2.2 COUNSELING AND TECHNIQUES

Counseling for a healthy diet should form part of the everyday activities in general practice/family medicine. Such counseling may also be given by nurses and, like other health promotion activities, should be given regularly. The dietician could be a key figure in cases where complicated nutritional advice needs to be given.The population must be aware of the advantages and characteristics of a healthy diet, must enjoy following it, and should accept that diet be part of a normal lifestyle.

Primary care professionals should be trained in the practice of dietary counseling in order to transmit to the population up-to-date knowledge about food in a comprehensible way. This should be done through systematic counseling so that the concept of a healthy diet is integrated into the population's way of life.

Counseling must be, first and foremost, understandable as most patients do not know the correct characteristics of a healthy diet. Counseling may be given on an individual basis or in groups. Busy healthcare professionals who do not have enough time to counsel patients individually can organise counseling sessions for 8–12 people in order to facilitate participation. Such sessions may be led by a nurse, dietician or doctor (or a combination of these),may last for one hour and may need to be repeated.

Parameters for Effective Counseling

For such counseling to be effective, one needs to consider the following parameters:

Organisation

1. Arrange for an appointment (individual or group) to have enough time and to take place in a quiet environment.

2. Remember that the patient's confidence in the GP and nurses, and a close relationship between them, are essential for success. Primary care is therefore the right place for providing counseling on a healthy diet.
3. If the support of the dietician is needed, use it! Be careful that both messages are the same.
4. It is very important that a healthy lifestyle is made popular. Spread the message using publicity pamphlets, web pages,TV / radio / press campaigns, and speeches in the media. Trial recipes must accompany healthy diet messages. On a regular basis, provide different recipes, messages and posters to the public in waiting rooms in health centres.
5. Arrange meetings where patients discuss healthy food with medical teams and with other patients who can share their experiences of the change towards a healthy diet, and where they can be shown healthy recipes.

Method

1. Assess the person's capacity for understanding the message correctly. Use words appropriate to the person's educational level.
2. Talk to the person quietly. Do not judge or preach. Ask the person about his/her doubts, troubles and fears.
3. Explore the person's knowledge about a healthy diet.
4. Explore the person's eating behaviours and traditional food habits.
5. Do not make a person feel anxious or threatened by setting a time limit for observing results.
6. When closing the counseling session, answer questions and clarify any mistaken concepts about the topic.
7. Do reinforce positive behaviour in successive visits, in order to achieve:
   - An increase in the person's self-esteem.
   - Maintenance of the changed behaviour.
   - Credibility in the eyes of the person.
   - The proposed objective.

Concepts

1. Communicate to persons the concept that, when they have changed their dietary habits, they are going to feel better both physically and mentally and enjoy a healthy way of life.
2. Try to convert persons to the concept of a healthy diet so that they feel rewarded.
3. Try to convey the idea that a healthy diet is not an obligation but a way to increase health and well-being.

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4. Use the word 'food' instead of 'diet'. The word 'diet' may convey the impression of a ban on food.
5. An inflexible ban on some foods can lead to frustration and result in a person not keeping a healthy diet.
6. The regular consumption of healthy food must be voluntary and done willingly.
7. In order to obtain a change in lifestyle, encourage people to think about its "pros" rather than "cons", like: healthy food is inexpensive, easy to find and to prepare.
8. In adolescents, do not convey the simple message: "the thinner the better", because there are a lot of problems with anorexia. Young people need more caution with counseling and special care.

THE TECHNIQUES

Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake and records the estimated amounts of all the foods and beverages the person consumed. The 24-hour food recall can be used to provide an estimate of energy and nutrient intake. As people tend to over- or underestimate intake of certain foods, and food intake on one day may not accurately represent typical food intake. The food frequency questionnaire can sometimes provide a more accurate picture of a person's typical eating patterns.

The nutrition counselor may ask the client how often he or she consumes certain food groups or the consumed servings of dairy products, fruits, vegetables, grains and cereals, meats, or fats he or she consumes in a typical day, week, or month. Daily food records can be useful in assessing food intake as individual keeps a written record of the amounts of all foods and beverages consumed over a given period of time. Also the three-day food records kept over two weekdays and one weekend day are often used. The nutrition counselor can then use the food records to analyze actual energy and nutrient intake.

Identifying Changes Needed

The initial dietary assessment and interview provide the basis for identifying behaviours that need to be changed. Sometimes a person already has a good idea of what dietary changes are needed, but may require help in making the changes. Other times the nutrition counselor can help educate a person on the health effects of different dietary choices. The nutrition counselor and client work together to identify areas where change is needed prioritize changes and problem-solve as to how to make the changes.

Setting Goals

The nutrition counselor and client set behaviour-oriented goals together. Goals should focus on the behaviours needed to achieve the desired dietary change, not on an absolute value, such as achieving a certain body weight. For a person working to prevent weight gain associated with certain medications, for example, his or her goals might be to increase the amount of fruits, vegetables, and whole grains consumed each day. Such changes would help prevent weight gain while placing the emphasis on needed behaviours rather than on actual weight.

Making Dietary Change

In making dietary changes, each individual's situation, background and the factors that affect food decisions must be carefully considered. An individual, in gradual process, may start with one or two easier dietary changes the first few weeks and gradually make additional or more difficult changes over several weeks or months.

Identifying Barriers to Change

Once the needed changes have been identified, the client and nutrition counselor think through potential problems that may arise. For example, changing eating behaviours may mean involving others, purchasing different foods, planning ahead for social events, or bringing special foods to work. Some common barriers to changing eating habits include: inconvenience, social gatherings, food preferences, lack of knowledge or time, cost etc.

Finding Support

Family members are encouraged to attend nutrition counseling sessions with the client, especially if they share responsibility for food selection and preparation. Although the individual must make food choices and take responsibility for dietary changes, having the support and understanding of family and friends makes success more likely.

Maintaining Changes

The challenge for the nutrition client lies not in making the initial dietary changes, but in maintaining them over the long term. Self-monitoring, realistic expectations, and continued follow-up can help a person maintain dietary changes. Self-monitoring involves regularly checking eating habits against desired goals and keeping track of eating behaviours. Keeping a food diary on a daily or periodic basis helps the individual be more aware of his or her eating behaviors and provides a ready tool to analyze eating habits. Sometimes a simplified checklist to assure adequate intake of different food groups may be used. Individuals and nutrition counselors should not expect perfect dietary compliance—slips inevitably occur.

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The goal is to keep small slips, such as eating a few extra cookies, from becoming big slips, like total abandonment of dietary change. The counselor can help the client identify situations that may lead to relapse and plan ways to handle the situations ahead of time.

Responsibility Sharing

(i) The relationship is a key to the effectiveness of problem solving.

(ii) The interaction between the helper and the individual is a goal-oriented process through which change occurs in the form of learning new information, knowledge, or skills; gaining new insights and perspectives; modifying feelings; changing behaviours; and developing new resources as decisions are made and problems resolved. This can be imitated through an environment of respect, trust, concern, understanding, careful listening and practicing nonjudgmental responses.

(iii) Clients and patients should not be perceived as passive recipients of services. They are active participants in their treatment, working with the professional to restore or optimize their health. Ultimately, clients or patients are responsible for managing their own nutrition and health. The acceptance of help is voluntary, and the aim of the professional is to make people self-sufficient so that eventually they can manage on their own, solving future problems alone.

(iv) Those being counseled should be given an opportunity to discuss changes and to ask questions, because the more they internalize the new ideas and solutions, the greater is the likelihood of their being committed to them.

(v) Some people are more resistant than others to making changes in lifestyles, and such resistance to modifying their old patterns of behaviour is normal.

(vi) Change upsets the established ways of doing things, creates uncertainty and anxiety, and forces the need for adjustments. Because attitudes are thought to be the predisposing agents of practice, they should be explored as well.

(vii) Every dietary problem has two aspects—what the client thinks about it and how he or she feels about it. A client may think, for example, that a dietary regimen would be beneficial, but feel that it would be too difficult to follow. Both thoughts and feelings must be considered and dealt with for problem solving and change to occur.

(viii) The client's priorities take precedence over those of the professional. Although helpers may see exactly what needs changing, they should bear in mind that the client is the one who decides which changes to make and who ensures that they continue.

(ix) When an individual deals with the necessity of change in food patterns and behaviours, knowledge and education are not in themselves sufficient to motivate change. Many people already know what they should eat! But they do not always act on their knowledge.

(x) The health professional cannot assume that recommendations will be followed just because the patient or client knows what to do.

(xi) Wanting to make changes is a key point to consider in adopting changes in dietary practices. Why should any person change a lifetime of unrestricted eating that may be pleasurable? The person's motivation for change should be examined since people can be expected to resist change.

Applying " Stage of Change"

The processes of change should be integrated into the stages of change so that the treatment intervention matches the client's stage of change. Accordingly, dietary programs and change initiation has to be designed and addressed when the client at the action stage. In counseling and education programs, to assume that everyone is at the action stage may lead one to plan an inappropriate intervention. Furthermore, most of programs and efforts are delivered in earlier stages and as if people are at the action stage.

(i) *precontemplation:* people are unaware or less than fully aware that a problem exists, deny they have a problem, or are not interested in change, and thus have no intention of changing behaviour in the near future. They may have tried a change previously and failed, and be resistant to one's efforts to suggest changes. To identify this stage, one may ask: "Are you seriously intending to change (name the problem behaviour) in the next six months?" For example, with people ignoring the relationship between a high fat diet and coronary heart disease, one may ask, "Have you thought about eating less fat (or more fruits and vegetables) in the next six months?".

(ii) *contemplation:* people are aware that a problem exists, such as needing to eat differently or exercise more, but they have no serious thought or commitment to making a change and may remain in this stage for months or years. They may be mentally struggling with the amount of energy, effort, and cost of overcoming a problem or may be discouraged by previous failures. One may ask, ' 'What have you been thinking about making a change?" "What are the pros and cons of doing it?" "How can you change your environment?" For example, "What do you think about eating less fat? What are the barriers to doing it?"

(iii) *preparation:* individuals are more determined to change and intending to take initial action soon, or in about 30 days. They may report small changes

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in the problem behaviour, such as reading a few food labels or buying fat-free ice cream. One may ask, "What, if any, changes have you planned or made in the past few weeks?"

(iv) *Action:* people overcome the problem by actively modifying their habits, behaviours, environments or experiences. It is important to remember that a majority of clients are not in the action stage when referred for counseling. Considerable commitment of time and energy is required for the action stage when individuals are trying to change. One may ask, "What are you doing differently?"

(v) *Maintenance:* people consolidate and stabilize gains made over several months in order to maintain the new, healthier habits and prevent relapse. For some people, this stage continues for months, years, or a lifetime. One may ask, "How do you handle small lapses?"

(vi) *Termination:* This is the ultimate goal's stage. However, some types of problems, such as eating changes, require a lifetime of maintenance instead. People, for example, tend to become more sedentary and overweight as they age, contributing to continual problems.

2.3 DIET COUNSELING FOR OBESES PEOPLE

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy. Body Mass Index (BMI), which compares weight and height, is used to define a person as overweight (pre-obese) when their BMI is between 25 kg/m^2 and 30 kg/m^2 and obese when it is greater than 30 kg/m^2.

Obesity is associated with many diseases, particularly heart disease, type 2 diabetes, breathing difficulties during sleep, certain types of cancer, and osteoarthritis. Obesity is most commonly caused by a combination of excessive dietary calories, lack of physical activity, and genetic susceptibility, though a limited number of cases are due solely to genetics, medical reasons or psychiatric illness.

The primary treatment for obesity is dieting and physical exercise. If this fails, anti-obesity drugs may be taken to reduce appetite or inhibit fat absorption. In severe cases, surgery is performed or an intragastric balloon is placed to reduce stomach volume and or bowel length, leading to earlier satiation and reduced ability to absorb nutrients from food.

Obesity is a leading preventable cause of death worldwide, with increasing prevalence in adults and children, and authorities view it as one of the most serious public health problems of the 21st century. Obesity is stigmatized in the modern Western world, though it has been perceived as a symbol of wealth and fertility at other times in history, and still is in many parts of Africa.

Management

The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the short term, but keeping this weight off can be a problem and often requires making exercise and a lower calorie diet a permanent part of a person's lifestyle. Success rates of long-term weight loss maintenance are low and range from 2–20%. In a more structured setting, however, 67% of people who lost greater than 10% of their body mass maintained or continued to lose weight one year later. An average maintained weight loss of more than 3 kg (6.6 lb) or 3% of total body mass could be sustained for five years. Some studies have found significant benefits in mortality in certain populations. In a prospective study of obese women with weight related diseases, intentional weight loss of any amount was associated with a 20% reduction in mortality. In obese women without obesity related illnesses a weight loss of greater than 9 kg (20 lb) was associated with a 25% reduction in mortality. A recent review however concluded that "benefits of weight loss on all cause mortality for the overweight and obese is meagre." Benefits of weight loss for certain subgroups however is well supported by evidence such as in people with type 2 diabetes, women, and those with severe obesity.

The most effective, but also most risky treatment for obesity is bariatric surgery. Due to its cost and risk of complications, researchers are fervently searching for new obesity treatments.

Diet Therapy

Diets to promote weight loss are generally divided into four categories: low-fat, low-carbohydrate, low-calorie, and very low calorie. A meta-analysis of six randomized controlled trials found no difference between the main diet types (low calorie, low carbohydrate, and low fat), with a 2–4 kilogram (4.4–8.8 lb) weight loss in all studies. At two years, all diet methods resulted in similar weight loss irrespective of the macronutrients emphasized.

Low-fat diets

Low-fat diets involve the reduction of the percentage of fat in one's diet. Calorie consumption is reduced but not purposely so. Diets of this type include NCEP Step I and II. A meta-analysis of 16 trials of 2–12 months' duration found that low-fat diets resulted in weight loss of 3.2 kg (7.1 lb) over eating as normal.

Low-carbohydrate diets

Low carbohydrate diets such as Atkins and Protein Power are relatively high in fat and protein. They are very popular in the press but are not recommended by the American Heart Association. A review of 107 studies did not find that low-carbohydrate diets cause weigh loss, except when calorie intake was restricted. No adverse effects from low carbohydrate diets were detected.

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Low-calorie diets

Low-calorie diets usually produce an energy deficit of 500–1,000 calories per day, which can result in a 0.5 kilogram (1.1 lb) weight loss per week. They include the DASH diet and Weight Watchers among others. The National Institutes of Health reviewed 34 randomized controlled trials to determine the effectiveness of low-calorie diets. They found that these diets lowered total body mass by 8% over 3–12 months.

Very low-calorie diets

Very low calorie diets provide 200–800 kcal/day, maintaining protein intake but limiting calories from both fat and carbohydrates. They subject the body to starvation and produce an average weekly weight loss of 1.5–2.5 kilograms (3.3–5.5 lb). These diets are not recommended for general use as they are associated with adverse side effects such as loss of lean muscle mass, increased risks of gout, and electrolyte imbalances. People attempting these diets must be monitored closely by a physician to prevent complications.

Exercise

With use, muscles consume energy derived from both fat and glycogen. Due to the large size of leg muscles, walking, running, and cycling are the most effective means of exercise to reduce body fat. Exercise affects macronutrient balance. During moderate exercise, there is a shift to greater use of fat as a fuel.

A meta-analysis of 43 randomized controlled trials by the Cochrane Collaboration found that exercising alone led to limited weight loss. In combination with diet, however, it resulted in a 1 kilogram weight loss over dieting alone. A 1.5 kilogram (3.3 lb) loss was observed with a greater degree of exercise. Even though exercise as carried out in the general population has only modest effects, a dose response curve is found, and very intense exercise can lead to substantial weight loss. During 20 weeks of basic military training with no dietary restriction, obese military recruits lost 12.5 kg (27.6 lb). High levels of physical activity seem to be necessary to maintain weight loss.

A systematic review found that people who use pedometers, during on average an 18-week period, increased their physical activity by 27% and subsequently decreased their BMI by 0.38.

2.4 FEBRILE CONDITIONS

A febrile seizure, also known as a fever fit or febrile convulsion is a convulsion triggered by a rise in body temperature. They most commonly occur in children between the ages of 3 months and 5 years and are twice as common in boys than girls. The direct cause of a febrile seizure is not known; however, it is normally precipitated by a recent upper respiratory infection or gastroenteritis. A febrile

seizure is the effect of a sudden rise in temperature (over 39°C or 102°F), rather than a fever that has been present for a prolonged length of time.

Febrile seizures represent the meeting point between a low seizure threshold (genetically and age determined) - some children have a greater tendency to have a seizure under certain circumstances - and a trigger: fever. The genetic causes of febrile seizures are still being researched. Some mutations that cause a neuronal hyperexcitability and could be responsible for febrile seizures have already been discovered.

Diagnosis

The diagnosis is one that must be arrived at by eliminating more serious causes of seizure and fever: in particular, meningitis and encephalitis must be ruled out. Therefore, a doctor's opinion should be sought and in many cases the child would be admitted to hospital overnight for observation and/or tests. As a general rule, if the child returns to a normal state of health soon after the seizure, a nervous system infection is unlikely. Even in cases where the diagnosis is febrile seizure, doctors will try to identify and treat the source of fever.

Types

There are two types of febrile seizures. A simple febrile seizure is one in which the seizure lasts less than 15 minutes, does not recur in the next 24 hours, and involves the entire body (classically a generalized tonic-clonic seizure). A complex febrile seizure is characterized by longer duration, recurrence, or focus on only part of the body. The simple seizure represents the majority of cases and is considered to be less of a cause for concern than the complex.

Simple febrile seizures generally do not cause permanent brain injury; do not tend to recur frequently, as children tend to 'out-grow' them; and do not make the development of adult epilepsy significantly more likely (less than 3-5% which is similar to that of the general public). Children with febrile convulsions are more likely to suffer from afebrile epileptic attacks in the future if they have a complex febrile seizure, a family history of afebrile convulsions in first degree relatives (a parent or sibling), or a pre-convulsion history of abnormal neurological signs or developmental delay. Similarly, the prognosis after a simple febrile seizure is excellent, whereas an increased risk of death has been shown for complex febrile seizures (partly related to underlying conditions).

Treatment

When anticonvulsant therapy is judged by a doctor to be indicated anticonvulsants can be prescribed. Sodium valproate or clonazepam are active against febrile seizures with sodium valproate showing superiority over clonazepam.

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SYMPTOMS

During simple febrile seizures, the body will become stiff and the arms and legs will begin twitching. The patient may lose consciousness and possibly wet or soil themselves. They may also vomit and foam at the mouth. The seizure normally lasts for less than five minutes.

DIET MANAGEMENT

A specially formulated diet, known as the ketogenic diet, has been used as a treatment for children with some types of seizures that do not respond to antiepileptic drugs (AEDs). The diet consists of high fat, relatively low carbohydrate and nutritionally adequate protein. The nutritional content of all meals must be calculated and each food item weighed. For infants who require formula, there is a commercially available product that comes as a powder and is mixed with water. AEDs are usually slowly discontinued during dietary treatment if there is a significant improvement in seizure control.

The high fat and low carbohydrate content of the ketogenic diet causes persistent ketosis. Ketosis occurs when the body uses fat for energy rather than sugar (from carbohydrates), which appears to have a direct effect that prevents seizures. The level of ketosis is monitored daily with urine test strips, and intermittently with blood tests. The diet is not intended to "cure" the seizures, but it lowers seizure frequency by at least 50 percent in approximately 40 percent of patients, particularly those between one and 10 years of age.

Parents should not attempt to start their child on a ketogenic diet alone; it should be supervised by a well-trained dietitian in an Epilepsy Center with experience managing the diet. The diet is usually started in a hospital setting, although some experts are able to manage patients at home. The child must be monitored to ensure he or she is growing and getting all the necessary nutrients. The diet is often continued for at least two years in children who improve significantly.

The restriction on eating carbohydrates may be difficult for some children, especially those who are reluctant to try new foods. Parents, teachers, relatives, and friends need to understand that even one bite or taste of a restricted food can lead to seizure recurrence. The diet may significantly change experiences like birthday parties and holidays, which frequently include high carbohydrate foods. Talking to other parents of children who have used the diet may be helpful in deciding whether to attempt it.

Complications— Several complications can occur in children who follow a ketogenic diet, although most complications are temporary and can be managed without stopping the diet. The parents, healthcare provider, and child should review the risks, benefits, and guidelines for the diet before it is started; it may not be an acceptable option in some cases.

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Surgery options— Most children achieve reasonably good seizure control with anticonvulsant medication(s). However, up to 30 to 40 percent of children continue to have seizures despite numerous medications, and are not candidates for or do not improve with the ketogenic diet. Surgical treatments may be considered for children who have persistent, frequent seizures that are not controlled after a trial of at least three appropriate medications.

Surgeries for epilepsy may be minimally invasive (*e.g.*, vagus nerve stimulation) or invasive (brain surgery), which removes that abnormal brain tissue that is causing the seizure. The procedure selected depends upon the type of seizure and area of the brain that is involved.

STUDENT ACTIVITY

1. Discuss the paramaters of effective counseling.

2. Outline the required diets for obeses people.

2.5 SUMMARY

- A balanced diet is a diet that, when taken regularly, will provide in quantity and quality sufficient macro and micro nutrients and other non-nutrient elements (fibres, vitamins, antioxidants and other bioactive substances).
- The amount of food needed every day depends upon age, body size, level of physical activity, gender, and, in females, pregnancy or breast-feeding.
- Counseling in general is a term describing a process that assists people to learn about themselves, their environment and methods of handling problems. Nutrition counseling is an ongoing process in which a health professional, usually a nutritionist works with an individual to assess his or her usual dietary intake and identify areas where change is needed.

- Nutrition counseling usually begins with an interview in which the counselor asks questions about a person's typical food intake. Nutrition counselors use different methods to assess typical food intake and records the estimated amounts of all the foods and beverages the person consumed.
- The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the short term, but keeping this weight off can be a problem and often requires making exercise and a lower calorie diet a permanent part of a person's lifestyle.
- A febrile seizure, also known as a fever fit or febrile convulsion is a convulsion triggered by a rise in body temperature. They most commonly occur in children between the ages of 3 months and 5 years and are twice as common in boys than girls.

2.6 GLOSSARY

- **Balanced diet:** A diet that, when taken regularly, will provide in quantity and quality sufficient macro and micro nutrients and other non-nutrient elements.
- **Nutrition counselor:** A person who is qualified dietician or nutrients expert, assess and advice people.
- **Obesity:** A medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health.
- **Febrile seizure:** Also known as a fever fit or febrile convulsion is a convulsion triggered by a rise in body temperature.

2.7 REVIEW QUESTIONS

1. What is the essential techniques of diet counseling?
2. How is diet managed for obesesity?
3. What is febrile seizure?
4. How is febrile seizure treated?
5. What is the importance of diet management in febrile seizure?

2.8 FURTHER READINGS

- Rani Reddy, *Dental Anthropology Applications and Methods*, Inter India Publications, 1985.

UNIT—III

DIABETES MELLITUS

NOTES

OBJECTIVES

After going through this unit, students will be able to:

- discuss the etiology and classification of diabetes mellitus;
- describe the diagnosis and complications of diabetes;
- state the diet management and diet counseling for diabetes.

STRUCTURE

3.1 INTRODUCTION

Diabetes mellitus often referred to simply as diabetes—is a condition in which the body either does not produce enough, or does not properly respond to, insulin, a hormone produced in the pancreas. Insulin enables cells to absorb glucose in order to turn it into energy. In diabetes, the body either fails to properly respond to its own insulin, does not make enough insulin, or both. This causes glucose to accumulate in the blood, often leading to various complications.

Many types of diabetes are recognize. The principal three are:

- **Type 1:** Results from the body's failure to produce insulin. It is estimated that 5—10% of Americans who are diagnosed with diabetes have type 1

diabetes. Presently almost all persons with type 1 diabetes must take insulin injections.

- **Type 2:** Results from Insulin resistance, a condition in which cells fail to use insulin properly, sometimes combined with relative insulin deficiency. Most Americans who are diagnosed with diabetes have type 2 diabetes. Many people destined to develop type 2 diabetes spend many years in a state of Pre-diabetes: Termed "America's largest healthcare epidemic," pre-diabetes indicates a condition that occurs when a person's blood glucose levels are higher than normal but not high enough for a diagnosis of type 2 diabetes. As of 2009 there are 57 million Americans who have pre-diabetes.
- **Gestational diabetes:** Pregnant women who have never had diabetes before but who have high blood sugar (glucose) levels during pregnancy are said to have gestational diabetes. Gestational diabetes affects about 4% of all pregnant women. It may precede development of type 2 (or rarely type 1).

  Many other forms of diabetes mellitus are categorized separately from these. Examples include congenital diabetes due to genetic defects of insulin secretion, cystic fibrosis-related diabetes, steroid diabetes induced by high doses of glucocorticoids, and several forms of monogenic diabetes.

3.2 CLASSIFICATION AND ETIOLOGY

All forms of diabetes have been treatable since insulin became medically available in 1921, but there is no cure for the common types except a pancreas transplant, although gestational diabetes usually resolves after delivery. Diabetes and its treatments can cause many complications. Acute complications including hypoglycemia, diabetic ketoacidosis, or nonketotic hyperosmolar coma may occur if the disease is not adequately controlled. Serious long-term complications include cardiovascular disease, chronic renal failure, retinal damage, which can lead to blindness, several types of nerve damage, and microvascular damage, which may cause erectile dysfunction and poor wound healing. Poor healing of wounds, particularly of the feet, can lead to gangrene, and possibly to amputation.

Adequate treatment of diabetes, as well as increased emphasis on blood pressure control and lifestyle factors such as not smoking and maintaining a healthy body weight, may improve the risk profile of most of the chronic complications. In the developed world, diabetes is the most significant cause of adult blindness in the non-elderly and the leading cause of non-traumatic amputation in adults, and diabetic nephropathy is the main illness requiring renal dialysis in the United States.

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The term diabetes, without qualification, usually refers to diabetes mellitus, which is associated with excessive sweet urine (known as "glycosuria") but there are several rarer conditions also named diabetes. The most common of these is diabetes insipidus in which the urine is not sweet (insipidus meaning "without taste" in Latin); it can be caused either by kidney (nephrogenic DI) or pituitary ntral DI) damage. It is a noninfectious disease. Among the body systems ed are the nerve, digestive, circulatory, endocrine and urinary systems.

The term "type 1 diabetes" has universally replaced several former terms including childhood-onset diabetes, juvenile di diabetes mellitus (IDDM) several former and non-insu ther ndard nome clature. Various have defined ong others, ges nal diabetes. in ulin-resistant type 1 or double diabetes"), type 2 di to require injected insulin, and latent autoimmune di diabetes.)

Type 1 Diabetes

acterized by insulin-produc he pancreas leading to a deficiency of liabetes d as immune-mediated or idi pe 1 di of the immune-mediated nature, where beta cell loss is a ted autoimmune re is no known preventive measure which can approximately 10% of diabetes mellitus cases in North America (though this varies by geographical location). Most affected people are otherwise healthy and of a healthy weight when onset occurs. Sensitivity and responsiveness to insulin are usually normal, especially in the early stages. Type 1 diabetes can affect or adults but was traditionally termed "juvenile diabetes" because iority of the diabetes cases in children.

The p pe 1 diabetes, even in its earliest stages, is the delivery of artin ion combined with careful monitoring of blood glucose levels using bl g monitors. Without insulin, diabetic ketoacidosis often develops which a or death. Treatment emphasis is now also placed on lifestyle atments ercise) though these cannot reverse the progress of the disease. Ap mmon subcutaneous injections, it is also possible to deliver insulin by allows continuous infusion of insulin 24 hours a day at preset levels, and the ability to program doses (a bolus) of insulin as needed at meal times.

Type 1 treatment must be continued indefinitely in essentially all cases. The longest surviving Type I diabetes patient is Gladys Dull, who has lived with the condition for over 83 years. Treatment need not significantly impair normal activities, if sufficient patient training, awareness, appropriate care, discipline in testing and dosing of insulin is taken. However, treatment is burdensome for patients; insulin is replaced in a non-physiological manner, and this approach is therefore far from ideal. The average glucose level for the type 1 patient should be as close to normal (80–120 mg/dl, 4–6 mmol/L) as is safely possible. Some physicians suggest up to 140–150 mg/dl (7–7.5 mmol/L) for those having trouble with lower values, such as frequent hypoglycemic events. Values above 400 mg/dl (20 mmol/L) are sometimes accompanied by discomfort and frequent urination leading to dehydration. Values above 600 mg/dl (30 mmol/L) usually require medical treatment and may lead to ketoacidosis, although they are not immediately life-threatening. However, low levels of blood glucose, called hypoglycemia, may lead to seizures or episodes of unconsciousness and absolutely must be treated immediately, via emergency high-glucose gel placed in the patient's mouth, intravenous administration of dextrose, or an injection of glucagon.

Type 2 Diabetes

Type 2 diabetes mellitus is characterized differently and is due to insulin resistance or reduced insulin sensitivity, combined with relatively reduced insulin secretion which in some cases becomes absolute. The defective responsiveness of body tissues to insulin almost certainly involves the insulin receptor in cell membranes. However, the specific defects are not known. Diabetes mellitus due to a known specific defect are classified separately. Type 2 diabetes is the most common type.

In the early stage of type 2 diabetes, the predominant abnormality is reduced insulin sensitivity, characterized by elevated levels of insulin in the blood. At this stage hyperglycemia can be reversed by a variety of measures and medications that improve insulin sensitivity or reduce glucose production by the liver. As the disease progresses, the impairment of insulin secretion worsens, and therapeutic replacement of insulin often becomes necessary.

There are numerous theories as to the exact cause and mechanism in type 2 diabetes. Central obesity (fat concentrated around the waist in relation to abdominal organs, but not subcutaneous fat) is known to predispose individuals to insulin resistance. Abdominal fat is especially active hormonally, secreting a group of hormones called adipokines that may possibly impair glucose tolerance. Obesity is found in approximately 55% of patients diagnosed with type 2 diabetes. Other factors include aging (about 20% of elderly patients in North America have diabetes) and family history (type 2 is much more common in those with close

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relatives who have had it). In the last decade, type 2 diabetes has increasingly begun to affect children and adolescents, probably in connection with the increased prevalence of childhood obesity seen in recent decades in some places. Environmental exposures may contribute to recent increases in the rate of type 2 diabetes. A positive correlation has been found between the concentration in the urine of bisphenol A, a constituent of polycarbonate plastic from some producers, and the incidence of type 2 diabetes.

Type 2 diabetes may go unnoticed for years because visible symptoms are typically mild, non-existent or sporadic, and usually there are no ketoacidotic episodes. However, severe long-term complications can result from unnoticed type 2 diabetes, including renal failure due to diabetic nephropathy, vascular disease (including coronary artery disease), vision damage due to diabetic retinopathy, loss of sensation or pain due to diabetic neuropathy, liver damage from non-alcoholic steatohepatitis and heart failure from diabetic cardiomyopathy.

Type 2 diabetes is usually first treated by increasing physical activity, decreasing carbohydrate intake, and losing weight. These can restore insulin sensitivity even when the weight loss is modest, for example around 5 kg (10 to 15 lb), most especially when it is in abdominal fat deposits. It is sometimes possible to achieve long-term, satisfactory glucose control with these measures alone. However, the underlying tendency to insulin resistance is not lost, and so attention to diet, exercise, and weight loss must continue. The usual next step, if necessary, is treatment with oral antidiabetic drugs. Insulin production is initially only moderately impaired in type 2 diabetes, so oral medication (often used in various combinations) can be used to improve insulin production (*e.g.*, sulfonylureas), to regulate inappropriate release of glucose by the liver and attenuate insulin resistance to some extent (*e.g.*, metformin), and to substantially attenuate insulin resistance (*e.g.*, thiazolidinediones). According to one study, overweight patients treated with metformin compared with diet alone, had relative risk reductions of 32% for any diabetes endpoint, 42% for diabetes related death and 36% for all cause mortality and stroke. Oral medication may eventually fail due to further impairment of beta cell insulin secretion. At this point, insulin therapy is necessary to maintain normal or near normal glucose levels.

Gestational Diabetes

Gestational Diabetes Mellitus (GDM) resembles type 2 diabetes in several respects, involving a combination of relatively inadequate insulin secretion and responsiveness. It occurs in about 2%–5% of all pregnancies and may improve or disappear after delivery. Gestational diabetes is fully treatable but requires careful medical supervision throughout the pregnancy. About 20%–50% of affected women develop type 2 diabetes later in life.

Even though it may be transient, untreated gestational diabetes can damage the health of the fetus or mother. Risks to the baby include macrosomia (high birth weight), congenital cardiac and central nervous system anomalies, and skeletal muscle malformations. Increased fetal insulin may inhibit fetal surfactant production and cause respiratory distress syndrome. Hyperbilirubinemia may result from red blood cell destruction. In severe cases, perinatal death may occur, most commonly as a result of poor placental perfusion due to vascular impairment. Induction may be indicated with decreased placental function. A cesarean section may be performed if there is marked fetal distress or an increased risk of injury associated with macrosomia, such as shoulder dystocia.

Other Types

Most cases of diabetes mellitus fall into the two broad etiologic categories of type 1 or type 2 diabetes. However, many types of diabetes mellitus have more specific known causes, and thus fall into more specific categories. As more research is done into diabetes, many patients who were previously diagnosed as type 1 or type 2 diabetes will have their condition reclassified.

Some cases of diabetes are caused by the body's tissue receptors not responding to insulin (even when insulin levels are normal, which is what separates it from type 2 diabetes); this form is very uncommon. Genetic mutations (autosomal or mitochondrial) can lead to defects in beta cell function. Abnormal insulin action may also have been genetically determined in some cases. Any disease that causes extensive damage to the pancreas may lead to diabetes (for example, chronic pancreatitis and cystic fibrosis). Diseases associated with excessive secretion of insulin-antagonistic hormones can cause diabetes (which is typically resolved once the hormone excess is removed). Many drugs impair insulin secretion and some toxins damage pancreatic beta cells. The ICD-10 (1992) diagnostic entity, malnutrition-related diabetes mellitus (MRDM or MMDM, ICD-10 code E12), was deprecated by the World Health Organization when the current taxonomy was introduced in 1999.

3.3 SIGNS AND SYMPTOMS

The classical symptoms are polyuria and polydipsia which are, respectively, frequent urination and increased thirst and consequent increased fluid intake. Symptoms may develop quite rapidly (weeks or months) in type 1 diabetes, particularly in children. However, in type 2 diabetes symptoms usually develop much more slowly and may be subtle or completely absent. Type 1 diabetes may also cause a rapid yet significant weight loss (despite normal or even increased eating) and irreducible mental fatigue. All of these symptoms except weight loss can also manifest in type 2 diabetes in patients whose diabetes is poorly controlled,

although unexplained weight loss may be experienced at the onset of the disease. Final diagnosis is made by measuring the blood glucose concentration.

When the glucose concentration in the blood is raised beyond its renal threshold, reabsorption of glucose in the proximal renal tubuli is incomplete, and part of the glucose remains in the urine (glycosuria). This increases the osmotic pressure of the urine and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume will be replaced osmotically from water held in body cells and other body compartments, causing dehydration and increased thirst.

Prolonged high blood glucose causes glucose absorption, which leads to changes in the shape of the lenses of the eyes, resulting in vision changes; sustained sensible glucose control usually returns the lens to its original shape. Blurred vision is a common complaint leading to a diabetes diagnosis; type 1 should always be suspected in cases of rapid vision change, whereas with type 2 change is generally more gradual, but should still be suspected.

Patients (usually with type 1 diabetes) may also initially present with diabetic ketoacidosis (DKA), an extreme state of metabolic dysregulation characterized by the smell of acetone on the patient's breath; a rapid, deep breathing known as Kussmaul breathing; polyuria; nausea; vomiting and abdominal pain; and any of many altered states of consciousness or arousal (such as hostility and mania or, ...ally, confusion and lethargy). In severe DKA, coma may follow, progressing t... h. Diabetic ketoacidosis is a medical emergency and requires immediate hosp... i ...tion.

A rarer but equally severe possibility is hyperosmolar nonketotic state, which is more common in type 2 diabetes and is mainly the result of dehydration due to loss of body water. Often, the patient has been drinking extreme amounts of sugar-containing drinks, leading to a vicious circle in regard to the water loss.

3.4 GENETICS

Both type 1 and type 2 diabetes are at least partly inherited. Type 1 diabetes appears to be triggered by some (mainly viral) infections, or less commonly, by stress or environmental exposure (such as exposure to certain chemicals or drugs). There is a genetic element in individual susceptibility to some of these triggers which has been traced to particular HLA genotypes (i.e., the genetic "self" identifiers relied upon by the immune system). However, even in those who have inherited the susceptibility, type 1 diabetes mellitus seems to require an environmental trigger. There is also maturity onset diabetes of the young (MODY) which is a group of several single gene (monogenic) disorders with strong heritability patterns which present as type 2 diabetes early in life, usually before

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30 years, and sometimes in childhood. In some cases diabetes can be brought out by some viruses like the chicken pox.

There is a stronger inheritance pattern for type 2 diabetes. Those with first-degree relatives with type 2 have a much higher risk of developing type 2, increasing with the number of those relatives. Concordance among monozygotic twins is close to 100%, and about 25% of those with the disease have a family history of diabetes. Genes significantly associated with developing type 2 diabetes, include TCF7L2, PPARG, FTO, KCNJ11, NOTCH2, WFS1, CDKAL1, IGF2BP2, SLC30A8, JAZF1, and HHEX. KCNJ11 (potassium inwardly rectifying channel, subfamily J, member 11), encodes the islet ATP-sensitive potassium channel Kir6.2, and TCF7L2 (transcription factor 7–like 2) regulates proglucagon gene expression and thus the production of glucagon-like peptide-1. Moreover, obesity (which is an independent risk factor for type 2 diabetes) is strongly inherited.

Monogenic forms, *e.g.*, MODY, constitute 1—5 % of all cases.

Various hereditary conditions may feature diabetes, for example myotonic dystrophy and Friedreich's ataxia. Wolfram's syndrome is an autosomal recessive neurodegenerative disorder that first becomes evident in childhood. It consists of diabetes insipidus, diabetes mellitus, optic atrophy, and deafness, hence the acronym DIDMOAD.

3.5 PATHOPHYSIOLOGY

Insulin is the principal hormone that regulates uptake of glucose from the blood into most cells (primarily muscle and fat cells, but not central nervous system cells). Therefore, deficiency of insulin or the insensitivity of its receptors plays a central role in all forms of diabetes mellitus.

Most of the carbohydrates in food are converted within a few hours to the monosaccharide glucose, the principal carbohydrate found in blood and used by the body as fuel. The most significant exceptions are fructose, most disaccharides (except sucrose and in some people lactose), and all more complex polysaccharides, with the outstanding exception of starch. Insulin is released into the blood by beta cells (â-cells), found in the Islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from the blood for use as fuel, for conversion to other needed molecules, or for storage.

Insulin is also the principal control signal for conversion of glucose to glycogen for internal storage in liver and muscle cells. Lowered glucose levels result both in the reduced release of insulin from the beta cells and in the reverse conversion of glycogen to glucose when glucose levels fall. This is mainly controlled by the hormone glucagon which acts in an opposite manner to insulin. Glucose

thus recovered by the liver re-enters the bloodstream; muscle cells lack the necessary export mechanism.

Higher insulin levels increase some anabolic ("building up") processes such as cell growth and duplication, protein synthesis, and fat storage. Insulin (or its lack) is the principal signal in converting many of the bidirectional processes of metabolism from a catabolic to an anabolic direction, and vice versa. In particular, a low insulin level is the trigger for entering or leaving ketosis (the fat burning metabolic phase).

If the amount of insulin available is insufficient, if cells respond poorly to the effects of insulin (insulin insensitivity or resistance), or if the insulin itself is defective, then glucose will not be absorbed properly by those body cells that require it nor will it be stored appropriately in the liver and muscles. The net effect is persistent high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as acidosis.

3.6 DIAGNOSIS

The diagnosis of type 1 diabetes, and many cases of type 2, is usually prompted by recent-onset symptoms of excessive urination (polyuria) and excessive thirst (polydipsia), often accompanied by weight loss. These symptoms typically worsen over days to weeks; about a quarter of people with new type 1 diabetes have developed some degree of diabetic ketoacidosis(Ketoacidosis is a type of metabolic acidosis which is caused by high concentrations of ketone bodies, formed by the breakdown of fatty acids and the deamination of amino acids.) by the time the diabetes is recognized. The diagnosis of other types of diabetes is usually made in other ways. These include ordinary health screening; detection of hyperglycemia during other medical investigations; and secondary symptoms such as vision changes or unexplainable fatigue. Diabetes is often detected when a person suffers a problem that is frequently caused by diabetes, such as a heart attack, stroke, neuropathy, poor wound healing or a foot ulcer, certain eye problems, certain fungal infections, or delivering a baby with macrosomia or hypoglycemia.

Diabetes mellitus is characterized by recurrent or persistent hyperglycemia, and is diagnosed by demonstrating any one of the following:

- Fasting plasma glucose level at or above 126 mg/dL (7.0 mmol/L).
- Plasma glucose at or above 200 mg/dL (11.1 mmol/L) two hours after a 75 g oral glucose load as in a glucose tolerance test.
- Symptoms of hyperglycemia and casual plasma glucose at or above 200 mg/dL (11.1 mmol/L).

A positive result, in the absence of unequivocal hyperglycemia, should be confirmed by a repeat of any of the above-listed methods on a different day. Most physicians prefer to measure a fasting glucose level because of the ease of measurement and the considerable time commitment of formal glucose tolerance testing, which takes two hours to complete and offers no prognostic advantage over the fasting test. According to the current definition, two fasting glucose measurements above 126 mg/dL (7.0 mmol/L) is considered diagnostic for diabetes mellitus.

Patients with fasting glucose levels from 100 to 125 mg/dL (6.1 and 7.0 mmol/L) are considered to have impaired fasting glucose. Patients with plasma glucose at or above 140 mg/dL or 7.8 mmol/L, but not over 200, two hours after a 75 g oral glucose load are considered to have impaired glucose tolerance. Of these two pre-diabetic states, the latter in particular is a major risk factor for progression to full-blown diabetes mellitus as well as cardiovascular disease.

While not used for diagnosis, an elevated level of glucose irreversibly bound to hemoglobin (termed glycated hemoglobin or HbA1c) of 6.0% or higher (the 2003 revised U.S. standard) is considered abnormal by most labs; HbA1c is primarily used as a treatment-tracking test reflecting average blood glucose levels over the preceding 90 days (approximately) which is the average lifetime of red blood cells which contain hemoglobin in most patients. However, some physicians may order this test at the time of diagnosis to track changes over time. The current recommended goal for HbA1c in patients with diabetes is 6.5%.

Screening

Diabetes screening is recommended for many people at various stages of life, and for those with any of several risk factors. The screening test varies according to circumstances and local policy, and may be a random blood glucose test, a fasting blood glucose test, a blood glucose test two hours after 75 g of glucose, or an even more formal glucose tolerance test. Many healthcare providers recommend universal screening for adults at age 40 or 50, and often periodically thereafter. Earlier screening is typically recommended for those with risk factors such as obesity, family history of diabetes, high-risk ethnicity (Hispanic, Native American, Afro-Caribbean, Pacific Islander).

Many medical conditions are associated with diabetes and warrant screening. A partial list includes: high blood pressure, elevated cholesterol levels, coronary artery disease, past gestational diabetes, polycystic ovary syndrome, chronic pancreatitis, fatty liver, hemochromatosis, cystic fibrosis, several mitochondrial neuropathies and myopathies, myotonic dystrophy, Friedreich's ataxia, some of the inherited forms of neonatal hyperinsulinism. The risk of diabetes is higher with chronic use of several medications, including high-dose

glucocorticoids, some chemotherapy agents (especially L-asparaginase), as well as some of the antipsychotics and mood stabilizers (especially phenothiazines and some atypical antipsychotics).

People with a confirmed diagnosis of diabetes are tested routinely for complications. This includes yearly urine testing for microalbuminuria and examination of the retina of the eye for retinopathy.

3.7 PREVENTION

Type 1 diabetes risk is known to depend upon a genetic predisposition based on HLA types (particularly types DR3 and DR4), an unknown environmental trigger (suspected to be an infection, although none has proven definitive in all cases), and an uncontrolled autoimmune response that attacks the insulin producing beta cells. Some research has suggested that breastfeeding decreased the risk in later life; various other nutritional risk factors are being studied, but no firm evidence has been found. Giving children 2000 IU of Vitamin D during their first year of life is associated with reduced risk of type 1 diabetes, though the causal relationship is obscure.

Children with antibodies to beta cell proteins (*i.e.*, at early stages of an immune reaction to them) but no overt diabetes, and treated with vitamin B-3 (niacin), had less than half the diabetes onset incidence in a 7-year time span as did the general population, and an even lower incidence relative to those with antibodies as above, but who received no vitamin B3.

Type 2 diabetes risk can be reduced in many cases by making changes in diet and increasing physical activity.

There is inadequate evidence that eating foods of low glycemic index is clinically helpful despite recommendations and suggested diets emphasizing this approach.

Diets that are very low in saturated fats reduce the risk of becoming insulin resistant and diabetic. Study group participants whose "physical activity level and dietary, smoking, and alcohol habits were all in the low-risk group had an 82% lower incidence of diabetes." In another study of dietary practice and incidence of diabetes, "foods rich in vegetable oils, including non-hydrogenated margarines, nuts, and seeds, should replace foods rich in saturated fats from meats and fat-rich dairy products. Consumption of partially hydrogenated fats should be minimized."

There are numerous studies which suggest connections between some aspects of Type II diabetes with ingestion of certain foods or with some drugs. Some studies have shown delayed progression to diabetes in predisposed patients through prophylactic use of metformin, rosiglitazone, or valsartan. In patients

on hydroxychloroquine for rheumatoid arthritis, incidence of diabetes was reduced by 77% though causal mechanisms are unclear. Breastfeeding may also be associated with the prevention of type 2 of the disease in mothers. Clear evidence for these and any of many other connections between foods and supplements and diabetes is sparse to date; none, despite secondary claims for (or against), is sufficiently well established to justify as a standard clinical approach.

3.8 TREATMENT AND MANAGEMENT

Diabetes mellitus is currently a chronic disease with no cure. Medical emphasis must necessarily be on managing/avoiding possible short-term as well as long-term diabetes-related problems. There is an exceptionally important role for patient education, dietetic support, sensible exercise, self-monitoring of blood glucose, with the goal of keeping both short-term and long-term blood glucose levels within acceptable bounds. Careful control is needed to reduce the risk of long-term complications. This is theoretically achievable with combinations of diet, exercise and weight loss (type 2), various oral diabetic drugs (type 2 only), and insulin use (type 1 and for type 2 not responding to oral medications, mostly those with extended duration diabetes). In addition, given the associated higher risks of cardiovascular disease, lifestyle modifications should be undertaken to control blood pressure and cholesterol by exercising more, smoking less or ideally not at all, consuming an appropriate diet, wearing diabetic socks, wearing diabetic shoes, and if necessary, taking any of several drugs to reduce blood pressure. Many type 1 treatments include combination use of regular or NPH insulin, and/or synthetic insulin analogs (*e.g.*, Humalog, Novolog or Apidra) in combinations such as Lantus/Levemir and Humalog, Novolog or Apidra. Another type 1 treatment option is the use of the insulin pump (*e.g.*, from Deltec Cozmo, Animas, Medtronic Minimed, Insulet Omnipod, or ACCU-CHEK). A blood lancet is used to pierce the skin (typically of a finger), in order to draw blood to test it for sugar levels.

In countries using a general practitioner system, such as the United Kingdom, care may take place mainly outside hospitals, with hospital-based specialist care used only in case of complications, difficult blood sugar control, or research projects. In other circumstances, general practitioners and specialists share care of a patient in a team approach. Optometrists, podiatrists/chiropodists, dietitians, physiotherapists, nursing specialists (*e.g.*, DSNs (Diabetic Specialist Nurse)), nurse practitioners, or Certified Diabetes Educators, may jointly provide multidisciplinary expertise. In countries where patients must provide for their own health care (*e.g.*, in the US, and in much of the undeveloped world), the impact of out-of-pocket costs of adequate diabetic care can be very high. In addition to the medications and supplies needed, patients are often advised to

receive regular consultation from a physician (*e.g.*, at least every three to six months).

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Oral administration of aloe vera might be a useful adjunct for lowering blood glucose in diabetic patients as well as for reducing blood lipid levels in patients with hyperlipidaemia. Ten controlled clinical trials were found to reach that conclusion in four independent literature searches. However, caveats reported in each study led the researchers to conclude that aloe vera's clinical effectiveness was not yet sufficiently defined in 1999.

Peer support links people living with diabetes. Within peer support, people with a common illness share knowledge and experience that others, including many health workers, do not have. Peer support is frequent, ongoing, accessible and flexible and can take many forms—phone calls, text messaging, group meetings, home visits, and even grocery shopping. It complements and enhances other health care services by creating the emotional, social and practical assistance necessary for managing disease and staying healthy.

CURE

Cures for Type 1 Diabetes

There is no practical cure, at this time, for type 1 diabetes. The fact that type 1 diabetes is due to the failure of one of the cell types of a single organ with a relatively simple function (*i.e.*, the failure of the beta cells in the Islets of Langerhans) has led to the study of several possible schemes to cure this form of diabetes mostly by replacing the pancreas or just the beta cells. Only those type 1 diabetics who have received either a pancreas or a kidney-pancreas transplant (often when they have developed diabetic kidney disease (*i.e.*, nephropathy) and become insulin-independent) may now be considered "cured" from their diabetes. A simultaneous pancreas-kidney transplant is a promising solution, showing similar or improved survival rates over a kidney transplant alone. Still, they generally remain on long-term immunosuppressive drugs and there is a possibility that the immune system will mount a host versus graft response against the transplanted organ.

Transplants of exogenous beta cells have been performed experimentally in both mice and humans, but this measure is not yet practical in regular clinical practice partly due to the limited number of beta cell donors. Thus far, like any such transplant, it has provoked an immune reaction and long-term immunosuppressive drugs have been needed to protect the transplanted tissue. An alternative technique has been proposed to place transplanted beta cells in a semi-permeable container, isolating and protecting them from the immune system. Stem cell research has also been suggested as a potential avenue for a

cure since it may permit regrowth of Islet cells which are genetically part of the treated individual, thus perhaps eliminating the need for immuno-suppressants. This new method, autologous nonmyeloablative HSTC, was recently developed by a research team composed of scientists from the US and Brazil. This was originally tested in mice and in 2007 there was the first trial with fifteen patients. Recently this trial was continued and 8 more patients were added. In the trial, the researchers implanted diabetes type 1 patients with their own stem cells raised from their own bone marrow. The stem cell transplant led to an appreciable repopulation of functioning insulin-producing beta cells in the pancreas so the patients became insulin free. Most of these patients became insulin independent for a mean period of 18.8 months. At the present time, autologous nonmyeloablative HSCT remains the only treatment capable of reversing type 1 DM in humans.

Microscopic or nanotechnological approaches are under investigation as well, in one proposed case with implanted stores of insulin metered out by a rapid response valve sensitive to blood glucose levels. At least two approaches have been demonstrated in vitro. These are, in some sense, closed-loop insulin pumps.

Cures for Type 2 Diabetes

Type 2 diabetes is usually first treated by increasing physical activity, and eliminating saturated fat and reducing sugar and carbohydrate intake with a goal of losing weight. These can restore insulin sensitivity even when the weight loss is modest, for example around 5 kg (10 to 15 lb), most especially when it is in abdominal fat deposits. Diets that are very low in saturated fats can reverse insulin resistance.

Recently it has been shown that a type of gastric bypass surgery can normalize blood glucose levels in 80-100% of severely obese patients with diabetes. The precise causal mechanisms are being intensively researched; its results are not simply attributable to weight loss, as the improvement in blood sugars precedes any change in body mass. This approach may become a standard treatment for some people with type 2 diabetes in the relatively near future. This surgery has the additional benefit of reducing the death rate from all causes by up to 40% in severely obese people. A small number of normal to moderately obese patients with type 2 diabetes have successfully undergone similar operations.

3.9 COMPLICATIONS AND PROGNOSIS

Patient education, understanding, and participation is vital since the complications of diabetes are far less common and less severe in people who have well-controlled blood sugar levels. Wider health problems accelerate the

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deleterious effects of diabetes. These include smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise. According to one study, women with high blood pressure (hypertension) were three times more likely to develop type 2 diabetes as compared with women with optimal BP after adjusting for various factors such as age, ethnicity, smoking, alcohol intake, body mass index (BMI), exercise, family history of diabetes, etc.

Anecdotal evidence suggests that some of those with type 2 diabetes who exercise regularly, lose weight, and eat healthy diets may be able to keep some of the disease or some of the effects of the disease in 'remission.' Certainly these tips can help prevent people predisposed to type 2 diabetes and those at pre-diabetic stages from actually developing the disorder as it helps restore insulin sensitivity. However, patients should talk to their doctors about this for real expectations before undertaking it (esp. to avoid hypoglycemia or other complications); few people actually seem to go into total 'remission,' but some may find they need less of their insulin medications since the body tends to have lower insulin requirements during and shortly following exercise. Regardless of whether it works that way or not for an individual, there are certainly other benefits to this healthy lifestyle for both diabetics and nondiabetics.

The way diabetes is managed changes with age. Insulin production decreases because of age-related impairment of pancreatic beta cells. Additionally, insulin resistance increases because of the loss of lean tissue and the accumulation of fat, particularly intra-abdominal fat, and the decreased tissue sensitivity to insulin. Glucose tolerance progressively declines with age, leading to a high prevalence of type 2 diabetes and postchallenge hyperglycemia in the older population. Age-related glucose intolerance in humans is often accompanied by insulin resistance, but circulating insulin levels are similar to those of younger people. Treatment goals for older patients with diabetes vary with the individual, and take into account health status, as well as life expectancy, level of dependence, and willingness to adhere to a treatment regimen.

Acute Complications

Diabetic Ketoacidosis

Diabetic ketoacidosis (DKA) is an acute and dangerous complication that is always a medical emergency. Low insulin levels cause the liver to turn to fat for fuel (*i.e.*, ketosis); ketone bodies are intermediate substrates in that metabolic sequence. This is normal when periodic, but can become a serious problem if sustained. Elevated levels of ketone bodies in the blood decrease the blood's pH, leading to DKA. On presentation at hospital, the patient in DKA is typically dehydrated, and breathing rapidly and deeply. Abdominal pain is common and

may be severe. The level of consciousness is typically normal until late in the process, when lethargy may progress to coma. Ketoacidosis can easily become severe enough to cause hypotension, shock, and death. Urine analysis will reveal significant levels of ketone bodies (which have exceeded their renal threshold blood levels to appear in the urine, often before other overt symptoms). Prompt, proper treatment usually results in full recovery, though death can result from inadequate or delayed treatment, or from complications (*e.g.*, brain edema). DKA is always a medical emergency and requires medical attention. Ketoacidosis is much more common in type 1 diabetes than type 2.

Hyperglycemia Hyperosmolar State

Hyperosmolar nonketotic state (HNS) is an acute complication sharing many symptoms with DKA, but an entirely different origin and different treatment. A person with very high (usually considered to be above 300 mg/dl (16 mmol/L)) blood glucose levels, water is osmotically drawn out of cells into the blood and the kidneys eventually begin to dump glucose into the urine. This results in loss of water and an increase in blood osmolarity. If fluid is not replaced (by mouth or intravenously), the osmotic effect of high glucose levels, combined with the loss of water, will eventually lead to dehydration. The body's cells become progressively dehydrated as water is taken from them and excreted. Electrolyte imbalances are also common and are always dangerous. As with DKA, urgent medical treatment is necessary, commonly beginning with fluid volume replacement. Lethargy may ultimately progress to a coma, though this is more common in type 2 diabetes than type 1.

Hypoglycemia

Hypoglycemia, or abnormally low blood glucose, is an acute complication of several diabetes treatments. It is rare otherwise, either in diabetic or non-diabetic patients. The patient may become agitated, sweaty, weak, and have many symptoms of sympathetic activation of the autonomic nervous system resulting in feelings akin to dread and immobilized panic. Consciousness can be altered or even lost in extreme cases, leading to coma, seizures, or even brain damage and death. In patients with diabetes, this may be caused by several factors, such as too much or incorrectly timed insulin, too much or incorrectly timed exercise (exercise decreases insulin requirements) or not enough food (specifically glucose containing carbohydrates). The variety of interactions makes cause identification difficult in many instances.

It is more accurate to note that iatrogenic hypoglycemia is typically the result of the interplay of absolute (or relative) insulin excess and compromised glucose counterregulation in type 1 and advanced type 2 diabetes. Decrements

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in insulin, increments in glucagon, and, absent the latter, increments in epinephrine are the primary glucose counterregulatory factors that normally prevent or (more or less rapidly) correct hypoglycemia. In insulin-deficient diabetes (exogenous) insulin levels do not decrease as glucose levels fall, and the combination of deficient glucagon and epinephrine responses causes defective glucose counterregulation.

Furthermore, reduced sympathoadrenal responses can cause hypoglycemia unawareness. The concept of hypoglycemia-associated autonomic failure (HAAF) in diabetes posits that recent incidents of hypoglycemia causes both defective glucose counterregulation and hypoglycemia unawareness. By shifting glycemic thresholds for the sympathoadrenal (including epinephrine) and the resulting neurogenic responses to lower plasma glucose concentrations, antecedent hypoglycemia leads to a vicious cycle of recurrent hypoglycemia and further impairment of glucose counterregulation. In many cases (but not all), short-term avoidance of hypoglycemia reverses hypoglycemia unawareness in affected patients, although this is easier in theory than in clinical experience.

In most cases, hypoglycemia is treated with sugary drinks or food. In severe cases, an injection of glucagon (a hormone with effects largely opposite to those of insulin) or an intravenous infusion of dextrose is used for treatment, but usually only if the person is unconscious. In any given incident, glucagon will only work once as it uses stored liver glycogen as a glucose source; in the absence of such stores, glucagon is largely ineffective. In hospitals, intravenous dextrose is often used.

Respiratory Infections

The immune response is impaired in individuals with diabetes mellitus. Cellular studies have shown that hyperglycemia both reduces the function of immune cells and increases inflammation. The vascular effects of diabetes also tend to alter lung function, all of which leads to an increase in susceptibility to respiratory infections such as pneumonia and influenza among individuals with diabetes. Several studies also show diabetes associated with a worse disease course and slower recovery from respiratory infections.

Chronic Complications

Vascular Disease

Chronic elevation of blood glucose level leads to damage of blood vessels (angiopathy). The endothelial cells lining the blood vessels take in more glucose than normal, since they don't depend on insulin. They then form more surface glycoproteins than normal, and cause the basement membrane to grow thicker and weaker. In diabetes, the resulting problems are grouped under "microvascular

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disease" (due to damage to small blood vessels) and "macrovascular disease" (due to damage to the arteries).

However, some research challenges the theory of hyperglycemia as the cause of diabetic complications. The fact that 40% of diabetics who carefully control their blood sugar nevertheless develop neuropathy, and that some of those with good blood sugar control still develop nephropathy, requires explanation. It has been discovered that the serum of diabetics with neuropathy is toxic to nerves even if its blood sugar content is normal. Recent research suggests that in type 1 diabetics, the continuing autoimmune disease which initially destroyed the beta cells of the pancreas may also cause retinopathy, neuropathy, and nephropathy. One researcher has even suggested that retinopathy may be better treated by drugs to suppress the abnormal immune system of diabetics than by blood sugar control. The familial clustering of the degree and type of diabetic complications indicates that genetics may also play a role in causing complications such as diabetic retinopathy and nephropathy. Non-diabetic offspring of type 2 diabetics have been found to have increased arterial stiffness and neuropathy despite normal blood glucose levels, and elevated enzyme levels associated with diabetic renal disease have been found in non-diabetic first-degree relatives of diabetics. Even rapid tightening of blood glucose levels has been shown to worsen rather than improve diabetic complications, though it has usually been held that complications would improve over time with more normal blood sugar, provided this could be maintained. However, one study continued for 41 months found that the initial worsening of complications from improved glucose control was not followed by the expected improvement in the complications.

The damage to small blood vessels leads to a microangiopathy, which can cause one or more of the following:

- Diabetic retinopathy, growth of friable and poor-quality new blood vessels in the retina as well as macular edema (swelling of the macula), which can lead to severe vision loss or blindness. Retinal damage (from microangiopathy) makes it the most common cause of blindness among non-elderly adults in the US.
- Diabetic neuropathy, abnormal and decreased sensation, usually in a 'glove and stocking' distribution starting with the feet but potentially in other nerves, later often fingers and hands. When combined with damaged blood vessels this can lead to diabetic foot (see below). Other forms of diabetic neuropathy may present as mononeuritis or autonomic neuropathy. Diabetic amyotrophy is muscle weakness due to neuropathy.
- Diabetic nephropathy, damage to the kidney which can lead to chronic renal failure, eventually requiring dialysis. Diabetes mellitus is the most common cause of adult kidney failure worldwide in the developed world.

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- Diabetic cardiomyopathy, damage to the heart, leading to diastolic dysfunction and eventually heart failure.

Macrovascular disease leads to cardiovascular disease, to which accelerated atherosclerosis is a contributor:

- Coronary artery disease, leading to angina or myocardial infarction ("heart attack")
- Stroke (mainly the ischemic type)
- Peripheral vascular disease, which contributes to intermittent claudication (exertion-related leg and foot pain) as well as diabetic foot.
- Diabetic myonecrosis ('muscle wasting')

Diabetic foot, often due to a combination of sensory neuropathy (numbness or insensitivity) and vascular damage, increases rates of skin ulcers and infection and, in serious cases, necrosis and gangrene. It is why diabetics are prone to leg and foot infections and why it takes longer for them to heal from leg and foot wounds. It is the most common cause of non-traumatic adult amputation, usually of toes and or feet, in the developed world.

Carotid artery stenosis does not occur more often in diabetes, and there appears to be a lower prevalence of abdominal aortic aneurysm. However, diabetes does cause higher morbidity, mortality and operative risks with these conditions.

Diabetic encephalopathy is the increased cognitive decline and risk of dementia observed in diabetes. Various mechanisms are proposed, including alterations to the vascular supply of the brain and the interaction of insulin with the brain itself.

3.10 DIET MANAGEMENT FOR DIABETICS

Popular misconceptions about nutrition and diabetes include the idea that a "diabetic diet" is a "sugar free diet"; or that refined sugar is "bad" and "natural sweeteners" are "good". Can "non-sugar" foods be eaten in any amounts? Can a person with diabetes "cheat" every once in a while? What is a "diabetic diet"? Because so many questions and misunderstandings exist, it is important for a person with diabetes to be able to understand the fundamentals of nutrition, one of several essential elements of successful diabetes management.

There is actually no such thing as a single "diabetic diet". The diet that a person with diabetes follows to help manage his or her blood sugar levels is based on the same nutrition principles that any healthy person, with or without diabetes, should follow for good health. When a person with diabetes sees a Registered Dietitian for nutrition counseling, the goal is to create a nutrition plan. This will help the person manage his or her blood sugar levels, reduce the risk of heart disease and other diet-related conditions, maintain a healthy weight, as well as meet the person's nutritional, lifestyle, social, and cultural needs.

The energy that we get from foods, measured in calories, comes from three types of nutrients: fats, proteins, and carbohydrates. Any food that provides calories will raise blood sugar. When foods are digested, they are broken down into the body's basic fuel— glucose, a type of sugar. The glucose is absorbed by the bloodstream, and is then known as blood glucose or blood sugar. In a person without diabetes, insulin is released by the pancreas after a meal or snack to allow the glucose in the blood to get into the body's cells, where it is burned for energy. This brings the level of glucose in the blood back down to the normal range. If insulin is not produced or is not working properly, the glucose cannot enter the cells to be used, and it builds up in the bloodstream. This results in high blood sugar, and this condition is known as diabetes.

Although all foods that provide calories are converted into glucose by the body, certain nutrients have a more direct effect on the blood's glucose level. Fats in foods are eventually digested and converted into glucose, but this can take up to 6 to 8 or more hours after a meal, and the release of glucose into the blood is very slow. Protein in foods (such as meats, poultry, fish, eggs, soy and other beans, and milk) takes about 3 to 4 hours after a meal to "show up" as blood glucose.

Carbohydrates, on the other hand, take only about half an hour to an hour after a meal to be turned into blood glucose. The word "carbohydrate" actually means "sugars and starches." Chemically, a starchy food is just a "chain" of glucose molecules. In fact, if a starchy food like a soda cracker is held in the mouth for a few minutes, it will start to taste sweet as the digestive enzymes in the saliva begin to break the starch down into its glucose parts.

Any food that is high in any type of carbohydrate will raise blood glucose levels soon after a meal. Whether a food contains one ounce of sugar (natural or refined) or one ounce of starch, it will raise blood glucose the same amount, because the total amount of carbohydrate is the same. Although a glass of fruit juice and the same amount of sugary soda may seem like a "good" versus "bad" choice, each will raise blood glucose about the same amount. This information regarding the amount of carbohydrate in different foods is the center of a nutrition management tool for people with diabetes called Carbohydrate Counting. Foods high in carbohydrates include starches such as rice, pasta, breads, cereals, and similar foods; fruits and juices; vegetables; milk and milk products; and anything made with added sugars, such as candies, cookies, cakes, and pies.

The goal of a diabetes nutrition plan is to provide a mixture of fats, carbohydrates, and proteins at each meal at an appropriate calorie level to both provide essential nutrients as well as create an even release of glucose into the blood from meal to meal and from day to day. A Registered Dietitian assesses the nutritional needs of a person with diabetes and calculates the amounts of fat, protein, carbohydrate, and total calories needed per day, and then converts this information into recommendations for amounts and types of foods to include in

the daily diet. The total number of meals and snacks and their timing throughout the day can differ for each person, based on his or her nutritional needs, lifestyle, and the action and timing of medications.

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Overall, a nutrition plan for a person with diabetes includes 10 to 20 percent of calories from protein, no more than 30 percent of calories from fats (with no more than 10 percent from saturated fats), and the remaining 50 to 60 percent from carbohydrates. Carbohydrate foods that contain dietary fiber are encouraged, as a high fiber diet has been associated with decreased risks of colon and other cancers. For people with high blood cholesterol levels, lower total fat and saturated fat contents may be recommended. Sodium intake of no more than 3000 mg per day is suggested; for people with high blood pressure, sodium should be limited to 2400 mg per day or as advised by a physician.

One "diabetic diet" definitely does not fit all. In fact, any food can fit into the diet of someone with diabetes, with the help and guidance of a Registered Dietitian. Managing blood glucose levels does not have to mean giving up favorite foods, sweets, or restaurants and fast foods. Each person with diabetes has very different nutritional and personal needs, making ongoing assessment and counseling with a Registered Dietitian an essential element of successful diabetes management.

STUDENT ACTIVITY

1. Discuss the features of gestational diabetes.

2. Discuss the important measures to prevent diabetes.

3. Outline the complications of diabetes.

3.11 SUMMARY

- Diabetes mellitus often referred to simply as diabetes—is a condition in which the body either does not produce enough, or does not properly respond to, insulin, a hormone produced in the pancreas.
- All forms of diabetes have been treatable since insulin became medically available in 1921, but there is no cure for the common types except a pancreas transplant.
- The classical symptoms are polyuria and polydipsia which are, respectively, frequent urination and increased thirst and consequent increased fluid intake.
- Diabetes screening is recommended for many people at various stages of life, and for those with any of several risk factors.

3.12 GLOSSARY

- **Diabetes:** It is a condition in which the body either does not produce enough, or does not properly respond to, insulin, a hormone produced in the pancreas.
- **Gestational diabetes mellitus (GDM):** A diabetes which involve a combination of relatively inadequate insulin secretion and responsiveness.
- **Insulin:** A principal hormone that regulates uptake of glucose from the blood into most cells.
- **Hypoglycemia:** This is a condition of diabetes in which abnormally low blood glucose is noticed, is an acute complication of several diabetes treatments.

3.13 REVIEW QUESTIONS

1. What are the signs and symptoms of diabetes? Classify it.
2. How is diabetes treated?
3. Discuss the diagnosis of diabetes.
4. What are the complications of diabetes?
5. Write a short note on "diet management for diabetics".

3.14 FURTHER READINGS

- Rhodes, P.M., *Genetics*, Tata Mc Graw Hill Publishing Pvt. Ltd., 5th edition 1985.

UNIT—IV

CARDIOVASCULAR DISEASES (ISCHEMIC HEART DISEASES)

NOTES

OBJECTIVES

After going through this unit, students will be able to:

- state about cardiovascular diseases such as hypertension, atheroseclerosis, myocardial infraction etc.;
- explain the consecutive cardiac failure;
- discuss the diet counseling and required diets for cardiovascular diseases.

STRUCTURE

4.1 INTRODUCTION

Cardiovascular diseases refers to the class of diseases that involve the heart or blood vessels (arteries and veins). While the term technically refers to any disease that affects the cardiovascular system, it is usually used to refer to those related to atherosclerosis (arterial disease). These conditions have similar causes, mechanisms, and treatments. In practice, cardiovascular disease is treated by cardiologists, thoracic surgeons, vascular surgeons, neurologists, and interventional

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radiologists, depending on the organ system that is being treated. There is considerable overlap in the specialties, and it is common for certain procedures to be performed by different types of specialists in the same hospital.

Most countries face high and increasing rates of cardiovascular disease. Each year, heart disease kills more Americans than cancer.

It is the number one cause of death and disability in the United States and most European countries (data available through 2005). A large histological study (PDAY) showed vascular injury accumulates from adolescence, making primary prevention efforts necessary from childhood.

By the time that heart problems are detected, the underlying cause (atherosclerosis) is usually quite advanced, having progressed for decades. There is therefore increased emphasis on preventing atherosclerosis by modifying risk factors, such as healthy eating, exercise and avoidance of smoking.

Biomarkers

Some biomarkers are thought to offer a more detailed risk of cardiovascular disease. However, the clinical value of these biomarkers is questionable. Currently, biomarkers which may reflect a higher risk of cardiovascular disease include:

- Higher fibrinogen and PAI-1 blood concentrations
- Elevated homocysteine, or even upper half of normal
- Elevated blood levels of asymmetric dimethylarginine
- High inflammation as measured by C-reactive protein
- Elevated blood levels of brain natriuretic peptide (also known as B-type) (BNP)

Awareness

Despite the success of the Dean Ornish studies in the eighties and nineties, most people and doctors are still unaware of the extensive research showing that stress reduction and diet can prevent, eliminate, and reduce nearly all forms of heart disease. At the same time many people rely on medications (such as aspirin) which may decrease certain risk factors such as heart attacks, but increase others such as hemorrhaging, and in the end often have no impact on long-term morbidity.

Dietary Factors

In his book "The Heart Healthy Program", the cardiologist Dr. Richard M. Fleming has identified several key dietary factors that can lower the risk of heart disease, including:

- Lowering of LDL cholesterol by reducing saturated fat intake.
- Lowering of Triglyceride levels by reducing consumption of sugary and processed foods.
- Reduction of Homocysteine levels by supplementation with Vitamins B6 and B12, and folic acid.
- Increased antioxidant activity by higher consumption of fruits and vegetables.
- Lowering of fibrinogen and growth factors by cutting back on foods such as red meat, dairy products, poultry and eggs.

TREATMENT

Unlike many other chronic medical conditions, Cardiovascular disease is treatable and reversible, even after a long history of disease. Treatment is primarily focused on diet and stress reduction.

4.2 HYPERTENSION

Hypertension is a chronic medical condition in which the blood pressure is elevated. It is also referred to as high blood pressure or shortened to HT, HTN or HPN. The word "hypertension", by itself, normally refers to systemic, arterial hypertension.

Hypertension can be classified as either essential (primary) or secondary. Essential or primary hypertension means that no medical cause can be found to explain the raised blood pressure. It is common. About 90-95% of hypertension is essential hypertension. Secondary hypertension indicates that the high blood pressure is a result of (*i.e.*, secondary to) another condition, such as kidney disease or tumours (adrenal adenoma or pheochromocytoma).

Persistent hypertension is one of the risk factors for strokes, heart attacks, heart failure and arterial aneurysm, and is a leading cause of chronic renal failure. Even moderate elevation of arterial blood pressure leads to shortened life expectancy. At severely high pressures, defined as mean arterial pressures 50% or more above average, a person can expect to live no more than a few years unless appropriately treated. Beginning at a systolic pressure (which is peak pressure in the arteries, which occurs near the end of the cardiac cycle when the ventricles are contracting) of 115 mmHg and diastolic pressure (which is minimum pressure in the arteries, which occurs near the beginning of the cardiac cycle when the ventricles are filled with blood) of 75 mmHg (commonly written as 115/75 mmHg), cardiovascular disease (CVD) risk doubles for each increment of 20/10 mmHg.

CLASSIFICATION

The variation in pressure in the left ventricle (blue line) and the aorta (red line) over two cardiac cycles ("heart beats"), showing the definitions of systolic and diastolic pressure.

A recent classification recommends blood pressure criteria for defining normal blood pressure, prehypertension, hypertension (stages I and II), and isolated systolic hypertension, which is a common occurrence among the elderly. These readings are based on the average of seated blood pressure readings that were properly measured during 2 or more office visits. In individuals older than 50 years, hypertension is considered to be present when a person's blood pressure is consistently at least 140 mmHg systolic or 90 mmHg diastolic. Patients with blood pressures over 130/80 mmHg along with Type 1 or Type 2 diabetes, or kidney disease require further treatment.

Classification	Systolic Pressure		Diastolic Pressure	
	mmHg	*kPa (kN/m²)*	*mmHg*	*kPa (kN/m²)*
Normal	90–119	12–15.9	60–79	8.0–10.5
Prehypertension	120–139	16.0–18.5	80–89	10.7–11.9
Stage 1	140 -159	18.7–21.2	90–99	12.0–13.2
Stage 2	>160	>21.3	>100	>13.3
Isolated systolichypertension	>140	>18.7	<90	<12.0

Source: American Heart Association (2003).

Excessive elevation in blood pressure during exercise is called exercise hypertension. The upper normal systolic values during exercise reach levels between 200 and 230 mm Hg. Exercise hypertension may be regarded as a precursor to established hypertension at rest.

SIGNS AND SYMPTOMS

Mild to moderate essential hypertension is greatly asymptomatic. Accelerated hypertension is associated with headache, somnolence, confusion, visual disturbances, and nausea and vomiting (hypertensive encephalopathy). Retinas are affected with narrowing of arterial diameter to less than 50% of venous diameter, copper or silver wire appearance, exudates, hemorrhages, or papilledema. Some signs and symptoms are especially important in infants and neonates such as failure to thrive, seizure, irritability or lethargy, and respiratory distress. While in children hypertension may cause headache, fatigue, blurred vision, epistaxis, and bell palsy.

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Some signs and symptoms are especially important in suggesting a secondary medical cause of chronic hypertension, such as centripetal obesity, "buffalo hump," and/or wide purple abdominal striae and may be a recent onset of diabetes suggest glucocorticoid excess either due to Cushing's syndrome or other causes. Hypertension due to other secondary endocrine diseases such as hyperthyroidism, hypothyroidism, or growth hormone excess show symptoms specific to these disease such as in hyperthyrodism there may be weight loss, tremor, tachycardia or atrial arrhythmia, palmar erythema and sweating. Signs and symptoms associated with growth hormone excess such as coarsening of facial features, prognathism, macroglossia, hypertrichosis, hyperpigmentation, and hyperhidrosis may occur in these patients. Other endocrine causes such as hyperaldosteronism may cause less specific symptoms such as numbness, polyuria, polydipsia, hypernatraemia, and metabolic alkalosis. A systolic bruit heard over the abdomen or in the flanks suggests renal artery stenosis. Also radiofemoral delay or diminished pulses in lower versus upper extremities suggests coarctation of the aorta. Hypertension in patients with pheochromocytomas is usually sustained but may be episodic. The typical attack lasts from minutes to hours and is associated with headache, anxiety, palpitation, profuse perspiration, pallor, tremor, and nausea and vomiting. Blood pressure is markedly elevated, and angina or acute pulmonary edema may occur. In primary aldosteronism, patients may have muscular weakness, polyuria, and nocturia due to hypokalemia. Chronic hypertension often leads to left ventricular hypertrophy, which can present with exertional and paroxysmal nocturnal dyspnea. Cerebral involvement causes stroke due to thrombosis or hemorrhage from microaneurysms of small penetrating intracranial arteries. Hypertensive encephalopathy is probably caused by acute capillary congestion and exudation with cerebral edema, which is reversible.

Signs and symptoms associated with pre-eclampsia and eclampsia, can be proteinuria, edema, and hallmark of eclampsia which is convulsions, Other cerebral signs may precede the convulsion such as nausea, vomiting, headaches, and blindness.

Causes

Essential Hypertension

Hypertension is one of the most common complex disorders. The etiology of hypertension differs widely amongst individuals within a large population. Essential hypertension is the form of hypertension that by definition, has no identifiable cause. It is the more common type and affects 90-95% of hypertensive patients, and even though there are no direct causes, there are many risk factors

such as sedentary lifestyle, obesity (more than 85% of cases occur in those with a body mass index greater than 25), salt (sodium) sensitivity, alcohol intake, and vitamin D deficiency. It is also related to aging and to some inherited genetic mutations. Family history increases the risk of developing hypertension. Renin elevation is another risk factor, Renin is an enzyme secreted by the juxtaglomerular apparatus of the kidney and linked with aldosterone in a negative feedback loop. Also sympathetic overactivity is implicated. Insulin resistance which is a component of syndrome X, or the metabolic syndrome is also thought to cause hypertension. Recently low birth weight has been questioned as a risk factor for adult essential hypertension.

Secondary Hypertension

On the other hand, secondary hypertension by definition results from an identifiable cause. This type is important to recognize since its treated differently than essential type by treating the underlying cause.

Many secondary cause can cause hypertension, some are common and well recognized secondary causes such as Cushing's syndrome, which is a condition where both adrenal glands can overproduce the hormone cortisol. Hypertension results from the interplay of several pathophysiological mechanisms regulating plasma volume, peripheral vascular resistance and cardiac output, all of which may be increased. More than 80% of patients with Cushing's syndrome have hypertension. Another important cause is the congenital abnormality coarctation of the aorta.

Adrenal

A variety of adrenal cortical abnormalities can cause hypertension, In primary aldosteronism there is a clear relationship between the aldosterone-induced sodium retention and the hypertension. Another related disorder that causes hypertension is apparent mineralocorticoid excess syndrome which is an autosomal recessive disorder results from mutations in gene encoding (*i.e.*, hydroxysteroid dehydrogenase) which normal patient inactivates circulating cortisol to the less-active metabolite cortisone. Cortisol at high concentrations can cross-react and activate the mineralocorticoid receptor, leading to aldosterone-like effects in the kidney, causing hypertension. This effect can also be produced by prolonged ingestion of liquorice(which can be of potent strength in liquorice candy), can result in inhibition of the hydroxysteroid dehydrogenase enzyme and cause secondary apparent mineralocorticoid excess syndrome. Frequently, if liquorice is the cause of the high blood pressure, a low blood level of potassium will also be present. Yet another related disorder causing hypertension is glucocorticoid remediable aldosteronism, which is an autosomal dominant

disorder in which the increase in aldosterone secretion produced by ACTH is no longer transient, causing of primary hyperaldosteronism, the Gene mutated will result in an aldosterone synthase that is ACTH-sensitive, which is normally not. GRA appears to be the most common monogenic form of human hypertension. Compare these effects to those seen in Conn's disease, an adrenocortical tumor which causes excess release of aldosterone, that leads to hypertension.

Another adrenal related cause is Cushing's syndrome which is a disorder caused by high levels of cortisol. Cortisol is a hormone secreted by the cortex of the adrenal glands. Cushing's syndrome can be caused by taking glucocorticoid drugs, or by tumors that produce cortisol or adrenocorticotropic hormone (ACTH). More than 80% of patients with Cushing's syndrome develop hypertension., which is accompanied by distinct symptoms of the syndrome, such as central obesity, buffalo hump, moon face, sweating, hirsutism and anxiety.

Kidney

Other well known causes include diseases of the kidney. This includes diseases such as polycystic kidney disease which is a cystic genetic disorder of the kidneys, PKD is characterized by the presence of multiple cysts (hence, "polycystic") in both kidneys, can also damage the liver, pancreas, and rarely, the heart and brain. It can be autosomal dominant or autosomal recessive, with the autosomal dominant form being more common and characterized by progressive cyst development and bilaterally enlarged kidneys with multiple cysts, with concurrent development of hypertension, renal insufficiency and renal pain. Or chronic glomerulonephritis which is a disease characterized by inflammation of the glomeruli, or small blood vessels in the kidneys. Hypertension can also be produced by diseases of the renal arteries supplying the kidney. This is known as renovascular hypertension; it is thought that decreased perfusion of renal tissue due to stenosis of a main or branch renal artery activates the renin-angiotensin system also some renal tumors can cause hypertension. The differential diagnosis of a renal tumor in a young patient with hypertension includes Juxtaglomerular cell tumor, Wilms' tumor, and renal cell carcinoma, all of which may produce renin.

Neuroendocrine tumors are also a well known cause of secondary hypertension. Pheochromocytoma (most often located in the adrenal medulla) increases secretion of catecholamines such as epinephrine and norepinephrine, causing excessive stimulation of adrenergic receptors, which results in peripheral vasoconstriction and cardiac stimulation. This diagnosis is confirmed by demonstrating increased urinary excretion of epinephrine and norepinephrine and/or their metabolites (vanillylmandelic acid).

Medications

Certain medications, especially NSAIDs (Motrin/Ibuprofen) and steroids can cause hypertension. High blood pressure that is associated with the sudden withdrawal of various antihypertensive medications is called Rebound Hypertension. The increases in blood pressure may result in blood pressures greater than when the medication was initiated. Depending on the severity of the increase in blood pressure, rebound hypertension may result in a hypertensive emergency. Rebound hypertension is avoided by gradually reducing the dose (also known as "dose tapering"), thereby giving the body enough time to adjust to reduction in dose. Medications commonly associated with rebound hypertension include centrally-acting antihypertensive agents, such as clonidine and beta-blockers.

Pregnancy

Few women of childbearing age have high blood pressure, up to 11% develop hypertension of pregnancy. While generally benign, it may herald three complications of pregnancy: pre-eclampsia, HELLP syndrome and eclampsia. Follow-up and control with medication is therefore often necessary.

Sleep Disturbances

Another common and under-recognized cause is sleep apnea, which is often best treated with nocturnal nasal continuous positive airway pressure, but other approaches include the Mandibular advancement splint (MAS), UPPP, tonsilectomy, adenoidectomy, septoplasty, or weight loss. Another cause is an exceptionally rare neurological disease called Binswanger's disease, causing dementia; it is a rare form of multi-infarct dementia, and is one of the neurological syndromes associated with hypertension.

Diagnosis

Initial assessment of the hypertensive patient should include a complete history and physical examination to confirm a diagnosis of hypertension. Most patients with hypertension have no specific symptoms referable to their blood pressure elevation.

Although popularly considered a symptom of elevated arterial pressure, headache generally occurs only in patients with severe hypertension. Characteristically, a "hypertensive headache" occurs in the morning and is localized to the occipital region. Other nonspecific symptoms that may be related to elevated blood pressure include dizziness, palpitations, easy fatigability, and impotence.

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Measuring Blood Pressure

Diagnosis of hypertension is generally on the basis of a persistently high blood pressure. Usually this requires three separate measurements at least one week apart. Exceptionally, if the elevation is extreme, or end-organ damage is present then the diagnosis may be applied and treatment commenced immediately.

Obtaining reliable blood pressure measurements relies on following several rules and understanding the many factors that influence blood pressure reading.

For instance, measurements in control of hypertension should be at least 1 hour after caffeine, 30 minutes after smoking or strenuous exercise and without any stress. Cuff size is also important. The bladder should encircle and cover two-thirds of the length of the (upper) arm. The patient should be sitting upright in a chair with both feet flat on the floor for a minimum of five minutes prior to taking a reading. The patient should not be on any adrenergic stimulants, such as those found in many cold medications.

When taking manual measurements, the person taking the measurement should be careful to inflate the cuff suitably above anticipated systolic pressure. The person should inflate the cuff to 200 mmHg and then slowly release the air while palpating the radial pulse. After one minute, the cuff should be reinflated to 30 mmHg higher than the pressure at which the radial pulse was no longer palpable. A stethoscope should be placed lightly over the brachial artery. The cuff should be at the level of the heart and the cuff should be deflated at a rate of 2 to 3 mmHg/s. Systolic pressure is the pressure reading at the onset of the sounds described by Korotkoff (Phase one). Diastolic pressure is then recorded as the pressure at which the sounds disappear (K5) or sometimes the K4 point, where the sound is abruptly muffled. Two measurements should be made at least 5 minutes apart, and, if there is a discrepancy of more than 5 mmHg, a third reading should be done. The readings should then be averaged. An initial measurement should include both arms. In elderly patients who particularly when treated may show orthostatic hypotension, measuring lying sitting and standing BP may be useful. The BP should at some time have been measured in each arm, and the higher pressure arm preferred for subsequent measurements.

BP varies with time of day, as may the effectiveness of treatment, and archetypes used to record the data should include the time taken. Analysis of this is rare at present.

Automated machines are commonly used and reduce the variability in manually collected readings. Routine measurements done in medical offices of patients with known hypertension may incorrectly diagnose 20% of patients with uncontrolled hypertension.

Home blood pressure monitoring can provide a measurement of a person's blood pressure at different times throughout the day and in different environments, such as at home and at work. Home monitoring may assist in the diagnosis of high or low blood pressure. It may also be used to monitor the effects of medication or lifestyle changes taken to lower or regulate blood pressure levels. Home monitoring of blood pressure can also assist in the diagnosis of white coat hypertension. The American Heart Association states, "You may have what's called 'white coat hypertension'; that means your blood pressure goes up when you're at the doctor's office. Monitoring at home will help you measure your true blood pressure and can provide your doctor with a log of blood pressure measurements over time. This is helpful in diagnosing and preventing potential health problems."

Some home blood pressure monitoring devices also make use of blood pressure charting software. These charting methods provide printouts for the patient's physician and reminders to take a blood pressure reading. However, a simple and cheap way is simply to manually record values with pen and paper, which can then be inspected by a doctor.

Systolic hypertension is defined as an elevated systolic blood pressure. If systolic blood pressure is elevated with a normal diastolic blood pressure, it is called isolated systolic hypertension. Systolic hypertension may be due to reduced compliance of the aorta with increasing age.

Prevention

The degree to which hypertension can be prevented depends on a number of features including: current blood pressure level, changes in end/target organs (retina, kidney, heart – among others), risk factors for cardiovascular diseases and the age at presentation. Unless the presenting patient has very severe hypertension, there should be a relatively prolonged assessment period within which repeated measurements of blood pressure should be taken. Following this, lifestyle advice and non-pharmacological options should be offered to the patient, before any initiation of drug therapy.

The process of managing hypertension according the guidelines of the British Hypertension Society suggest that non-pharmacological options should be explored in all patients who are hypertensive or pre-hypertensive. These measures include:

- Weight reduction and regular aerobic exercise (*e.g.*, walking) are recommended as the first steps in treating mild to moderate hypertension. Regular exercise improves blood flow and helps to reduce resting heart rate and blood pressure. Several studies indicate that low intensity exercise may be more effective in lowering blood pressure than higher intensity exercise. These steps are highly effective in reducing blood pressure,

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although drug therapy is still necessary for many patients with moderate or severe hypertension to bring their blood pressure down to a safe level.

- Reducing dietary sugar intake.
- Reducing sodium (salt) in the diet may be effective: It decreases blood pressure in about 33% of people (see above). Many people use a salt substitute to reduce their salt intake.
- Additional dietary changes beneficial to reducing blood pressure includes the DASH diet (dietary approaches to stop hypertension), which is rich in fruits and vegetables and low-fat or fat-free dairy foods. This diet has been shown to be effective based on research sponsored by the National Heart, Lung, and Blood Institute. In addition, an increase in daily calcium intake has the benefit of increasing dietary potassium, which theoretically can offset the effect of sodium and act on the kidney to decrease blood pressure. This has also been shown to be highly effective in reducing blood pressure.
- Discontinuing tobacco use and alcohol consumption has been shown to lower blood pressure. The exact mechanisms are not fully understood, but blood pressure (especially systolic) always transiently increases following alcohol or nicotine consumption. Besides, abstention from cigarette smoking is important for people with hypertension because it reduces the risk of many dangerous outcomes of hypertension, such as stroke and heart attack. Note that coffee drinking (caffeine ingestion) also increases blood pressure transiently but does not produce chronic hypertension.
- Reducing stress, for example with relaxation therapy, such as meditation and other mindbody relaxation techniques, by reducing environmental stress such as high sound levels and over-illumination can be an additional method of ameliorating hypertension. Jacobson's Progressive Muscle Relaxation and biofeedback are also used, particularly, device-guided paced breathing, although meta-analysis suggests it is not effective unless combined with other relaxation techniques.

Treatment

Lifestyle Modifications

Unless hypertension is severe, lifestyle changes such as those discussed in the preceding section are strongly recommended before initiation of drug therapy. Adoption of the DASH diet is one example of lifestyle change repeatedly shown to effectively lower mildly-elevated blood pressure. If hypertension is high enough to justify immediate use of medications, lifestyle changes are initiated concomitantly.

Biofeedback

Biofeedback devices can be used alone or in conjunction with lifestyle changes or medications to monitor and possibly reduce hypertension. One example is Resperate, a portable, battery-operated personal therapeutic medical device, sold over the counter (OTC) in the United States.

Medications

There are many classes of medications for treating hypertension, together called antihypertensives, which -- by varying means — act by lowering blood pressure. Evidence suggests that reduction of the blood pressure by 5–6 mmHg can decrease the risk of stroke by 40%, of coronary heart disease by 15–20%, and reduces the likelihood of dementia, heart failure, and mortality from vascular disease.

The aim of treatment should be blood pressure control to <140/90 mmHg for most patients, and lower in certain contexts such as diabetes or kidney disease (some medical professionals recommend keeping levels below 120/80 mmHg). Each added drug may reduce the systolic blood pressure by 5–10 mmHg, so often multiple drugs are often necessary to achieve blood pressure control.

Commonly used drugs include the typical groups of:

- ACE inhibitors such as captopril, enalapril, fosinopril (Monopril), lisinopril (Zestril), quinapril, ramipril (Altace)
- Angiotensin II receptor antagonists may be used where ACE inhibitors are not tolerated: *e.g.*, telmisartan (Micardis, Pritor), irbesartan (Avapro), losartan (Cozaar), valsartan (Diovan), candesartan (Amias)
- Calcium channel blockers such as nifedipine (Adalat) amlodipine (Norvasc), diltiazem, verapamil
- Diuretics: *e.g.*, bendroflumethiazide, chlortalidone, hydrochlorothiazide (also called HCTZ)

Other additionally used groups include:

- Additional diuretics such a furosemide or low-dosages of spironolactone
- Alpha blockers such as prazosin, or terazosin. Doxazosin has been shown to increase risk of heart failure, and to be less effective than a simple diuretic.
- Beta blockers such as atenolol, labetalol, metoprolol (Lopressor, Toprol-XL), propranolol. Whilst once first line agents, now less directly used for this in the United Kingdom due to the risk of diabetes.
- Direct renin inhibitors such as aliskiren (Tekturna)

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Finally several agents may be given simultaneously:

- Combination products (which usually contain HCTZ and one other drug). The advantage of fixed combinations resides in the fact that they increase compliance with treatment by reducing the number of pills taken by the patients. A fixed combination of the ACE inhibitor perindopril and the calcium channel blocker amlodipine, recently been proved to be very effective even in patients with additional impaired glucose tolerance and in patients with the metabolic syndrome.

Choice of Initial Medication

Unless the blood pressure is severely elevated, consensus guidelines call for medically-supervised lifestyle changes and observation before recommending initiation of drug therapy. All drug treatments have side effects, and while the evidence of benefit at higher blood pressures is overwhelming, drug trials to lower moderately-elevated blood pressure have failed to reduce overall death rates.

If lifestyle changes are ineffective or the presenting blood pressure is critical, then drug therapy is initiated, often requiring more than one agent to effectively lower hypertension. Which type of many medications should be used initially to treat hypertension has been the subject of several large studies and various national guidelines.

The Antihypertensive and Lipid-Lowering Treatment to Prevent Heart Attack Trial (ALLHAT) study showed better cost-effectiveness and slightly better outcomes for the thiazide diuretic chlorthalidone compared with a calcium channel blocker and an ACE inhibitor in a 33,357-member ethnically mixed study group. The 1993 consensus recommendation for use of thiazide diuretics as initial treatment stems in part from the ALLHAT study results, which concluded in 2002 that "Thiazide-type diuretics are superior in preventing 1 or more major forms of CVD and are less expensive. They should be preferred for first-step antihypertensive therapy."

A subsequent smaller study (ANBP2) did not show the slight advantages in thiazide diuretic outcomes observed in the ALLHAT study, and actually showed slightly better outcomes for ACE-inhibitors in older white male patients.

Thiazide diuretics are effective, recommended as the best first-line drug for hypertension by many experts, and are much more affordable than other therapies, yet they are not prescribed as often as some newer drugs. Hydrochlorothiazide is perhaps the safest and most inexpensive agent commonly used in this class and is very frequently combined with other agents in a single pill. Doses in excess of 25 milligrams per day of this agent incur an unacceptable risk of low potassium or Hypokalemia. Patients with an exaggerated hypokalemic

response to a low dose of a thiazide diuretic should be suspected to have Hyperaldosteronism, a common cause of secondary hypertension.

The consensus recommendations of thiazide diuretics as first-line therapy for hypertension stand against the backdrop that all blood pressure treatments have side-effects. Potentially serious side effects of the thiazide diuretics include hypercholesterolemia, and impaired glucose tolerance with consequent increased risk of developing Diabetes mellitus type 2. The thiazide diuretics also deplete circulating potassium unless combined with a potassium-sparing diuretic or supplemental potassium. On this basis, the consensus recommendations to prefer use of thiazides as first line treatment for essential hypertension have been repeatedly and strongly questioned. However as the Merck Manual of Geriatrics notes, "thiazide-type diuretics are especially safe and effective in the elderly."

Current UK guidelines suggest starting patients over the age of 55 years and all those of African/Afrocaribbean ethnicity firstly on calcium channel blockers or thiazide diuretics, whilst younger patients of other ethnic groups should be started on ACE-inhibitors. Subsequently if dual therapy is required to use ACE-inhibitor in combination with either a calcium channel blocker or a (thiazide) diuretic. Triple therapy is then of all three groups and should the need arise then to add in a fourth agent, to consider either a further diuretic (*e.g.*, spironolactone or furosemide), an alpha-blocker or a beta-blocker. Prior to the demotion of beta-blockers as first line agents, the UK sequence of combination therapy used the first letter of the drug classes and was known as the "ABCD rule".

COMPLICATIONS

Hypertension is a risk factor for all clinical manifestations of atherosclerosis since it is a risk factor for atherosclerosis itself. It is an independent predisposing factor for heart failure, coronary artery disease, stroke, renal disease, and peripheral arterial disease. It is the most important risk factor for cardiovascular morbidity and mortality, in industrialized countries. The risk is increased for:

- Cerebrovascular accident (CVAs or strokes)
- Myocardial infarction (heart attack)
- Hypertensive cardiomyopathy (heart failure due to chronically high blood pressure)
- Left ventricular hypertrophy - thickening of the myocardium (muscle) of the left ventricle of the heart.
- Hypertensive retinopathy - damage to the retina
- Hypertensive nephropathy- chronic renal failure due to chronically high blood pressure "benign nephrosclerosis".

- Hypertensive encephalopathy - confusion, headahe, convulsion due to vasogenic edema in brain due to high blood pressure.

4.3 ATHEROSCLEROSIS

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Atherosclerosis (also known as Arteriosclerotic Vascular Disease or ASVD) is the condition in which an artery wall thickens as the result of a build-up of fatty materials such as cholesterol. It is a syndrome affecting arterial blood vessels, a chronic inflammatory response in the walls of arteries, in large part due to the accumulation of macrophage white blood cells and promoted by low density (especially small particle) lipoproteins (plasma proteins that carry cholesterol and triglycerides) without adequate removal of fats and cholesterol from the macrophages by functional high density lipoproteins (HDL). It is commonly referred to as a hardening or furring of the arteries. It is caused by the formation of multiple plaques within the arteries.

The atheromatous plaque is divided into three distinct components:

1. The atheroma ("lump of wax", from Athera, wax in Greek,), which is the nodular accumulation of a soft, flaky, yellowish material at the center of large plaques, composed of macrophages nearest the lumen of the artery
2. Underlying areas of cholesterol crystals
3. Calcification at the outer base of older/more advanced lesions.

The following terms are similar, yet distinct, in both spelling and meaning, and can be easily confused: arteriosclerosis, arteriolosclerosis, and atherosclerosis. Arteriosclerosis is a general term describing any hardening (and loss of elasticity) of medium or large arteries (from the Greek Arterio, meaning artery, and sclerosis, meaning hardening); arteriolosclerosis is any hardening (and loss of elasticity) of arterioles (small arteries); atherosclerosis is a hardening of an artery specifically due to an atheromatous plaque. Therefore, atherosclerosis is a form of arteriosclerosis.

Atherosclerosis, though typically asymptomatic for decades, eventually produces two main problems: First, the atheromatous plaques, though long compensated for by artery enlargement, eventually lead to plaque ruptures and clots inside the artery lumen over the ruptures. The clots heal and usually shrink but leave behind stenosis (narrowing) of the artery (both locally and in smaller downstream branches), or worse, complete closure, and, therefore, an insufficient blood supply to the tissues and organ it feeds. Second, if the compensating artery enlargement process is excessive, then a net aneurysm results.

These complications of advanced atherosclerosis are chronic, slowly progressive and cumulative. Most commonly, soft plaque suddenly ruptures (see vulnerable plaque), causing the formation of a thrombus that will rapidly slow or

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stop blood flow, leading to death of the tissues fed by the artery in approximately 5 minutes. This catastrophic event is called an infarction. One of the most common recognized scenarios is called coronary thrombosis of a coronary artery, causing myocardial infarction (a heart attack). Even worse is the same process in an artery to the brain, commonly called stroke. Another common scenario in very advanced disease is claudication from insufficient blood supply to the legs, typically due to a combination of both stenosis and aneurysmal segments narrowed with clots. Since atherosclerosis is a body-wide process, similar events occur also in the arteries to the brain, intestines, kidneys, legs, etc.

Yet, many infarctions involve only very small amounts of tissue and are termed clinically silent, because the person having the infarction does not notice the problem, does not seek medical help or when they do, physicians do not recognize what has happened.

Causes

Atherosclerosis develops from low-density lipoprotein molecules (LDL) becoming oxidized (ldl-ox) by free radicals, particularly oxygen free radicals (ROS). Blood in arteries contains plenty of oxygen and is where atherosclerosis develops. Blood in veins contains little oxygen where atherosclerosis rarely develops. When oxidized LDL comes in contact with an artery wall, a series of reactions occur to repair the damage to the artery wall caused by oxidized LDL. The LDL molecule is globular shaped with a hollow core to carry cholesterol throughout the body to generate brain tissues, vitamin D, and so on. Cholesterol does not dissolve in water. Blood is 70% water. Cholesterol can move in the bloodstream only by being transported by LDL.

The body's immune system responds to the damage to the artery wall caused by oxidized—LDL by sending specialized white blood cells (macro-phages and T-lymphocytes) to absorb the oxidized—LDL forming specialized foam cells. Unfortunately, these white blood cells are not able to process the oxidized-LDL, and ultimately grow then rupture, depositing a greater amount of oxidized cholesterol into the artery wall. This triggers more white blood cells, continuing the cycle.

Eventually, the artery becomes inflamed. The cholesterol plaque causes the muscle cells to enlarge and form a hard cover over the affected area. This hard cover is what causes a narrowing of the artery, reduces the blood flow and increases blood pressure.

Some researchers believe that atherosclerosis may be caused by an infection of the vascular smooth muscle cells. Chickens, for example, develop atherosclerosis when infected with the Marek's disease herpesvirus. Herpesvirus infection of arterial smooth muscle cells has been shown to cause cholesteryl

ester (CE) accumulation. Cholesteryl ester accumulation is associated with atherosclerosis.

Also, cytomegalovirus (CMV) infection is associated with cardiovascular diseases.

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Symptoms

Atherosclerosis typically begins in early adolescence, and is usually found in most major arteries, yet is asymptomatic and not detected by most diagnostic methods during life. Atheroma in arm, or more often in leg arteries, which produces decreased blood flow is called peripheral artery occlusive disease (PAOD).

According to United States data for the year 2004, for about 65% of men and 47% of women, the first symptom of atherosclerotic cardiovascular disease is heart attack or sudden cardiac death (death within one hour of onset of the symptom).

Most artery flow disrupting events occur at locations with less than 50% lumen narrowing (~20% stenosis is average). [The reader might reflect that the illustration above, like most illustrations of arterial disease, overemphasizes lumen narrowing, as opposed to compensatory external diameter enlargement (at least within smaller arteries, *e.g.*, heart arteries) typical of the atherosclerosis process as it progresses. The relative geometry error within the illustration is common to [illegible]der illustrations, an error slowly being more commonly recognized within the [illegible]ade.]

Cardiac stress testing, traditionally the most commonly performed non-invasive testing method for blood flow limitations, in general, detects only lumen narrowing of ~75% or greater, although some physicians claim that nuclear stress methods can detect as little as 50%.

Atherogenesis

Atherogenesis is the developmental process of atheromatous plaques. It is characterized by a remodeling of arteries involving the concomitant accumulation of fatty substances called plaques. One recent theory suggests that, for unknown reasons, leukocytes, such as monocytes or basophils, begin to attack the endothelium of the artery lumen in cardiac muscle. The ensuing inflammation leads to formation of atheromatous plaques in the arterial tunica intima, a region of the vessel wall located between the endothelium and the tunica media. The bulk of these lesions is made of excess fat, collagen, and elastin. At first, as the plaques grow, only wall thickening occurs without any narrowing, stenosis of the artery opening, called the lumen; stenosis is a late event, which may never

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occur and is often the result of repeated plaque rupture and healing responses, not just the atherosclerosis process by itself.

Cellular

The first step of atherogenesis is the development of so called "fatty streak"s, which are small sub-endothelial deposits of monocyte-derived macrophages. The primary documented driver of this process is oxidized Lipoprotein particles within the wall, beneath the endothelial cells, though upper normal or elevated concentrations of blood glucose also plays a major role and not all factors are fully understood. Fatty streaks may appear and disappear.

Low Density Lipoprotein particles in blood plasma, when they invade the endothelium and become oxidized creates a risk for cardiovascular disease. A complex set of biochemical reactions regulates the oxidation of LDL, chiefly stimulated by presence of enzymes, *e.g.*, Lp-LpA2 and free radicals in the endothelium or blood vessel lining.

The initial damage to the blood vessel wall results in a "call for help," an inflammatory response. Monocytes (a type of white blood cell) enter the artery wall from the bloodstream, with platelets adhering to the area of insult. This may be promoted by redox signaling induction of factors such as VCAM-1, which recruit circulating monocytes. The monocytes differentiate macrophages, which ingest oxidized LDL, slowly turning into large "foam cells" – so-described because of their changed appearance resulting from the numerous internal cytoplasmic vesicles and resulting high lipid content. Under the microscope, the lesion now appears as a fatty streak. Foam cells eventually die, and further propagate the inflammatory process. There is also smooth muscle proliferation and migration from tunica media to intima responding to cytokines secreted by damaged endothelial cells. This would cause the formation of a fibrous capsule covering the fatty streak.

Calcification and Lipids

Intracellular microcalcifications form within vascular smooth muscle cells of the surrounding muscular layer, specifically in the muscle cells adjacent to the atheromas. In time, as cells die, this leads to extracellular calcium deposits between the muscular wall and outer portion of the atheromatous plaques. A similar form of an intramural calcification, presenting the picture of an early phase of arteriosclerosis, appears to be induced by a number of drugs that have an antiproliferative mechanism of action (Rainer Liedtke 2008).

Cholesterol is delivered into the vessel wall by cholesterol-containing low-density lipoprotein (LDL) particles. To attract and stimulate macrophages, the cholesterol must be released from the LDL particles and oxidized, a key step in

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the ongoing inflammatory process. The process is worsened if there is insufficient high-density lipoprotein (HDL), the lipoprotein particle that removes cholesterol from tissues and carries it back to the liver.

The foam cells and platelets encourage the migration and proliferation of smooth muscle cells, which in turn ingest lipids, become replaced by collagen and transform into foam cells themselves. A protective fibrous cap normally forms between the fatty deposits and the artery lining (the intima).

These capped fatty deposits (now called 'atheromas') produce enzymes that cause the artery to enlarge over time. As long as the artery enlarges sufficiently to compensate for the extra thickness of the atheroma, then no narrowing ("stenosis") of the opening ("lumen") occurs. The artery becomes expanded with an egg-shaped cross-section, still with a circular opening. If the enlargement is beyond proportion to the atheroma thickness, then an aneurysm is created.

Visible Features

Although arteries are not typically studied microscopically, two plaque types can be distinguished:

1. The fibro-lipid (fibro-fatty) plaque is characterized by an accumulation of lipid-laden cells underneath the intima of the arteries, typically without narrowing the lumen due to compensatory expansion of the bounding muscular layer of the artery wall. Beneath the endothelium there is a "fibrous cap" covering the atheromatous "core" of the plaque. The core consists of lipid-laden cells (macrophages and smooth muscle cells) with elevated tissue cholesterol and cholesterol ester content, fibrin, proteoglycans, collagen, elastin, and cellular debris. In advanced plaques, the central core of the plaque usually contains extracellular cholesterol deposits (released from dead cells), which form areas of cholesterol crystals with empty, needle—like clefts. At the periphery of the plaque are younger "foamy" cells and capillaries. These plaques usually produce the most damage to the individual when they rupture.
2. The fibrous plaque is also localized under the intima, within the wall of the artery resulting in thickening and expansion of the wall and, sometimes, spotty localized narrowing of the lumen with some atrophy of the muscular layer. The fibrous plaque contains collagen fibers (eosinophilic), precipitates of calcium (hematoxylinophilic) and, rarely, lipid-laden cells.

In effect, the muscular portion of the artery wall forms small aneurysms just large enough to hold the atheroma that are present. The muscular portion of artery walls usually remain strong, even after they have remodeled to compensate for the atheromatous plaques.

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However, atheromas within the vessel wall are soft and fragile with little elasticity. Arteries constantly expand and contract with each heartbeat, *i.e.*, the pulse. In addition, the calcification deposits between the outer portion of the atheroma and the muscular wall, as they progress, lead to a loss of elasticity and stiffening of the artery as a whole.

The calcification deposits, after they have become sufficiently advanced, are partially visible on coronary artery computed tomography or electron beam tomography (EBT) as rings of increased radiographic density, forming halos around the outer edges of the atheromatous plaques, within the artery wall. On CT, >130 units on the Hounsfield scale (some argue for 90 units) has been the radiographic density usually accepted as clearly representing tissue calcification within arteries. These deposits demonstrate unequivocal evidence of the disease, relatively advanced, even though the lumen of the artery is often still normal by angiographic or intravascular ultrasound.

Rupture and Stenosis

Although the disease process tends to be slowly progressive over decades, it usually remains asymptomatic until an atheroma ulcerates which leads to immediate blood clotting at the site of atheroma ulcer. This triggers a cascade of events that leads to clot enlargement which may quickly obstruct the lumen (opening) of the artery itself. A complete blockage leads to ischemia of the myocardial (heart) muscle and damage. This process is the myocardial infarction or "heart attack."

If the heart attack is not fatal, fibrous organization of the clot within the lumen ensues, covering the rupture but also producing stenosis or closure of the lumen, or over time and after repeated ruptures, resulting in a persistent, usually localized stenosis or blockage of the artery lumen. Stenoses can be slowly progressive, whereas plaque ulceration is a sudden event that occurs specifically in atheromas with thinner/weaker fibrous caps that have become "unstable."

Repeated plaque ruptures, ones not resulting in total lumen closure, combined with the clot patch over the rupture and healing response to stabilize the clot, is the process that produces most stenoses over time. The stenotic areas tend to become more stable, despite increased flow velocities at these narrowings. Most major blood-flow-stopping events occur at large plaques, which, prior to their rupture, produced very little if any stenosis.

From clinical trials, 20% is the average stenosis at plaques that subsequently rupture with resulting complete artery closure. Most severe clinical events do not occur at plaques that produce high-grade stenosis. From clinical trials, only 14% of heart attacks occur from artery closure at plaques producing a 75% or greater stenosis prior to the vessel closing.

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If the fibrous cap separating a soft atheroma from the bloodstream within the artery ruptures, tissue fragments are exposed and released, and blood enters the atheroma within the wall and sometimes results in a sudden expansion of the atheroma size. Tissue fragments are very clot-promoting, containing collagen and tissue factor; they activate platelets and activate the system of coagulation. The result is the formation of a thrombus (blood clot) overlying the atheroma, which obstructs blood flow acutely. With the obstruction of blood flow, downstream tissues are starved of oxygen and nutrients. If this is the myocardium (heart muscle), angina (cardiac chest pain) or myocardial infarction (heart attack) develops.

Diagnosis of Plaque-Related Disease

Areas of severe narrowing, stenosis, detectable by angiography, and to a lesser extent "stress testing" have long been the focus of human diagnostic techniques for cardiovascular disease, in general. However, these methods focus on detecting only severe narrowing, not the underlying atherosclerosis disease. As demonstrated by human clinical studies, most severe events occur in locations with heavy plaque, yet little or no lumen narrowing present before debilitating events suddenly occur. Plaque rupture can lead to artery lumen occlusion within seconds to minutes, and potential permanent debility and sometimes sudden death.

Plaques that have ruptured are called complicated plaques. The lipid matrix breaks through the thinning collagen gap and when the lipids come in contact with the blood, clotting occurs. After rupture the platelet adhesion causes the clotting cascade to contact with the lipid pool causing a thrombus to form. This thrombus will eventually grow and travel throughout the body. The thrombus will travel through different arteries and veins and eventually become lodged in an area that narrows. Once the area is blocked, blood and oxygen will not be able to supply the vessels and will cause death of cells and lead to necrosis and poisoning. Serious complicated plaques can cause death of organ tissues, causing serious complications to that organ system.

Greater than 75% lumen stenosis used to be considered by cardiologists as the hallmark of clinically significant disease because it is typically only at this severity of narrowing of the larger heart arteries that recurring episodes of angina and detectable abnormalities by stress testing methods are seen. However, clinical trials have shown that only about 14% of clinically-debilitating events occur at locations with this, or greater severity of narrowing. The majority of events occur due to atheroma plaque rupture at areas without narrowing sufficient enough to produce any angina or stress test abnormalities. Thus, since the later-1990s, greater attention is being focused on the "vulnerable plaque."

Though any artery in the body can be involved, usually only severe narrowing or obstruction of some arteries, those that supply more critically-important organs are recognized. Obstruction of arteries supplying the heart muscle result in a heart attack. Obstruction of arteries supplying the brain result in a stroke. These events are life-changing, and often result in irreversible loss of function because lost heart muscle and brain cells do not grow back to any significant extent, typically less than 2%.

Over the last couple of decades, methods other than angiography and stress-testing have been increasingly developed as ways to better detect atherosclerotic disease before it becomes symptomatic. These have included both *(a)* anatomic detection methods and *(b)* physiologic measurement methods.

Examples of anatomic methods include: (1) coronary calcium scoring by CT, (2) carotid IMT (intimal media thickness) measurement by ultrasound, and (3) IVUS.

Examples of physiologic methods include: (1) lipoprotein subclass analysis, (2) HbA1c, (3) hs-CRP, and (4) homocysteine.

The example of the metabolic syndrome combines both anatomic (abdominal girth) and physiologic (blood pressure, elevated blood glucose) methods.

Advantages of these two approaches: The anatomic methods directly measure some aspect of the actual atherosclerotic disease process itself, thus offer potential for earlier detection, including before symptoms start, disease staging and tracking of disease progression. The physiologic methods are often less expensive and safer and changing them for the better may slow disease progression, in some cases with marked improvement.

Disadvantages of these two approaches: The anatomic methods are generally more expensive and several are invasive, such as IVUS. The physiologic methods do not quantify the current state of the disease or directly track progression. For both, clinicians and third party payers have been slow to accept the usefulness of these newer approaches.

Physiologic Factors that Increase Risk

Various anatomic, physiological and behavioural risk factors for atherosclerosis are known. These can be divided into various categories: congenital vs acquired, modifiable or not, classical or non-classical. The points labelled '+' in the following list form the core components of "metabolic syndrome".

Factors add to each other multiplicatively, with two factors increasing the risk of atherosclerosis fourfold. Hyperlipidemia, hypertension and cigarette smoking together increases the risk seven times.

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Modifiable

- Having diabetes or Impaired glucose tolerance (IGT) +
- Dyslipoproteinemia (unhealthy patterns of serum proteins carrying fats and cholesterol):
  - High serum concentration of low-density lipoprotein (LDL, "bad if elevated concentrations and small"), and / or very low density lipoprotein (VLDL) particles, *i.e.*, "lipoprotein subclass analysis"
  - Low serum concentration of functioning high density lipoprotein (HDL "protective if large and high enough" particles), *i.e.*, "lipoprotein subclass analysis"
  - An LDL:HDL ratio greater than 3:1
- Tobacco smoking, increases risk by 200% after several pack years
- Having high blood pressure +, on its own increasing risk by 60%
- Elevated serum C-reactive protein concentrations

Nonmodifiable

- Advanced age
- Male sex
- Having close relatives who have had some complication of atherosclerosis (*e.g.*, coronary heart disease or stroke)
- Genetic abnormalities, *e.g.*, familial hypercholesterolemia

Lesser or Uncertain

The following factors are of relatively lesser importance, are uncertain or nonquantitated:

- Being obese (in particular central obesity, also referred to as abdominal or male-type obesity)
- A sedentary lifestyle
- Postmenopausal estrogen deficiency
- High carbohydrate intake
- Intake of trans fat
- Elevated serum levels of triglycerides
- Elevated serum levels of homocysteine
- Elevated serum levels of uric acid (also responsible for gout)
- Elevated serum fibrinogen concentrations
- Elevated serum lipoprotein(a) concentrations

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- Chronic systemic inflammation as reflected by upper normal WBC concentrations, elevated hs-CRP and many other blood chemistry markers, most only research level at present, not clinically done.
- Stress or symptoms of clinical depression
- Hyperthyroidism (an over-active thyroid)
- Elevated serum insulin levels
- Short sleep duration
- Chlamydia pneumoniae infection

Dietary Risk Factors

The relation between dietary fat and atherosclerosis is a contentious field. Most of the agencies, in their food pyramid, promote a low-fat diet, based largely on its view that fat in the diet is atherogenic. The American Heart Association, the American Diabetes Association and the National Cholesterol Education Program make similar recommendations. In contrast, Prof Walter Willett (Harvard School of Public Health, PI of the second Nurses' Health Study) recommends much higher levels, especially of monounsaturated and polyunsaturated fat. Writing in Science, Gary Taubes detailed that political considerations played into the recommendations of government bodies. These differing views reach a consensus, though, against consumption of trans fats.

The role of dietary oxidized fats / lipid peroxidation (rancid fats) in humans is not clear. Laboratory animals fed rancid fats develop atherosclerosis. Rats fed DHA-containing oils experienced marked disruptions to their antioxidant systems, as well as accumulated significant amounts of peroxide in their blood, livers and kidneys. In another study, rabbits fed atherogenic diets containing various oils were found to undergo the greatest amount of oxidative susceptibility of LDL via polyunsaturated oils. In a study involving rabbits fed heated soybean oil, "grossly induced atherosclerosis and marked liver damage were histologically and clinically demonstrated".

Rancid fats and oils taste very bad even in small amounts; people avoid eating them. It is very difficult to measure or estimate the actual human consumption of these substances. In addition, the majority of oils consumed in the United States are refined, bleached, deodorized and degummed by manufacturers. The resultant oils are colourless, odorless, tasteless and have a longer shelf life than their unrefined counterparts. This extensive processing serves to make peroxidated, rancid oils much more elusive to detection via the various human senses than the unprocessed alternatives.

The French paradox is the observation that despite having a diet similar to those United States in terms of fat intake, rates of heart disease are lower in

France. There is evidence to suggest the French paradox is due to underestimation of the rates of heart disease in France.

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TREATMENT

If atherosclerosis leads to symptoms, some symptoms such as angina pectoris can be treated. Non-pharmaceutical means are usually the first method of treatment, such as cessation of smoking and practicing regular exercise. If these methods do not work, medicines are usually the next step in treating cardiovascular diseases, and, with improvements, have increasingly become the most effective method over the long-term. However, medicines are criticized for their expense, patented control and occasional undesired effects.

Statins

In general, the group of medications referred to as statins has been the most popular and are widely prescribed for treating atherosclerosis. They have relatively few short-term or longer-term undesirable side-effects, and multiple comparative treatment/placebo trials have fairly consistently shown strong effects in reducing atherosclerotic disease 'events' and generally ~25% comparative mortality reduction in clinical trials, although one study design, ALLHAT, was less strongly favourable.

The newest statin, rosuvastatin, has been the first to demonstrate regression of atherosclerotic plaque within the coronary arteries by IVUS (intravascular ultrasound evaluation). The study was set up to demonstrate effect primarily on atherosclerosis volume within a 2 year time-frame in people with active/ symptomatic disease (angina frequency also declined markedly) but not global clinical outcomes, which was expected to require longer trial time periods; these longer trials remain in progress.

However, for most people, changing their physiologic behaviours, from the usual high risk to greatly reduced risk, requires a combination of several compounds, taken on a daily basis and indefinitely. More and more human treatment trials have been done and are ongoing that demonstrate improved outcome for those people using more-complex and effective treatment regimens that change physiologic behaviour patterns to more closely resemble those that humans exhibit in childhood at a time before fatty streaks begin forming.

The statins, and some other medications, have been shown to have antioxidant effects, possibly part of their basis for some of their therapeutic success in reducing cardiac 'events'.

The success of statin drugs in clinical trials is based on some reductions in mortality rates, however by trial design biased toward men and middle-age, the data is as, as yet, less strongly clear for women and people over the age of 70. For

example, in the Scandinavian Simvastatin Survival Study (4S), the first large placebo controlled, randomized clinical trial of a statin in people with advanced disease who had already suffered a heart attack, the overall mortality rate reduction for those taking the statin, vs. placebo, was 30%. For the subgroup of people in the trial that had Diabetes Mellitus, the mortality rate reduction between statin and placebo was 54%. 4S was a 5.4-year trial that started in 1989 and was published in 1995 after completion. There were 3 more dead women at trial's end on statin than in the group on placebo drug whether chance or some relation to the statin remains unclear.

Primary and Secondary Prevention

Combinations of statins, niacin, intestinal cholesterol absorption-inhibiting supplements (ezetimibe and others, and to a much lesser extent fibrates) have been the most successful in changing common but sub-optimal lipoprotein patterns and group outcomes. In the many secondary prevention and several primary prevention trials, several classes of lipoprotein expression (less correctly termed "cholesterol-lowering") altering agents have consistently reduced not only heart attack, stroke and hospitalization but also all-cause mortality rates.

DIET AND DIETARY SUPPLEMENTS

Vitamin B3, AKA niacin, in pharmacologic doses, (generally 1,000 to 3,000 mg/day), sold in many OTC and prescription formulations, tends to improve (a) HDL levels, size and function, (b) shift LDL particle distribution to larger particle size and (c) lower lipoprotein(d), an atherosclerosis promoting genetic variant of LDL. Additionally, individual responses to daily niacin, while mostly evident after a month at effective doses, tends to continue to slowly improve further over time. (However, careful patient understanding of how to achieve this without nuisance symptoms is needed, though not often achieved.) Research work on increasing HDL particle concentration and function, beyond the usual niacin effect/response, even more important, is slowly advancing.

Dietary changes to achieve benefit have been more controversial, generally far less effective and less widely adhered to with success. One key reason for this is that most cholesterol, typically 80-90%, within the body is created and controlled by internal production by all cells in the body (true of all animals), with typically slightly greater relative production by hepatic/liver cells. (Cell structure relies on fat membranes to separate and organize intracellular water, proteins and nucleic acids and cholesterol is one of the components of all animal cell membranes.)

Caldwell B Esselstyn Jr. MD has had an article published in Preventive Cardiology 2001;4: 171-177 in which he has published angiograms showing

regression of atherosclerosis brought about by a very low fat vegan diet in some cases with cholesterol lowering medications.

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While the absolute production quantities vary with the individual, group averages for total human body content of cholesterol within the U.S. population commonly run about ~35,000 mg (assuming lean build; varies with body weight and build) and ~1,000 mg/day ongoing production. Dietary intake plays a smaller role, 200-300 mg/day being common values; for pure vegetarians, essentially 0 mg/day, but this typically does not change the situation very much because internal production increases to largely compensate for the reduced intake. For many, especially those with greater than optimal body mass and increased glucose levels, reducing carbohydrate (especially simple forms) intake, not fats or cholesterol, is often more effective for improving lipoprotein expression patterns, weight and blood glucose values. For this reason, medical authorities much less frequently promote the low dietary fat concepts than was commonly the case prior to about year 2005. However, evidence has increased that processed, particularly industrial non-enzymatic hydrogenation produced trans fats, as opposed to the natural *cis*-configured fats, which living cells primarily produce, is a significant health hazard.

Dietary supplements of Omega-3 oils, especially those from the muscle of some deep salt water living fish species, also have clinical evidence of significant protective effects as confirmed by 6 double blind placebo controlled human clinical trials.

There is also a variety of evidence, though less robust, that homocysteine and uric acid levels, including within the normal range promote atherosclerosis and that lowering these levels is helpful, up to a point.

In animals Vitamin C deficiency has been confirmed as an important role in development of hypercholesterolemia and atherosclerosis, but due to ethical reasons placebo-controlled human studies are impossible to do. Vitamin C acts as an antioxidant in vessels and inhibits inflammatory process. It has therapeutic properties on high blood pressure and its fluctuation, and arterial stiffness in diabetes. Vitamin C is also a natural regulator of cholesterol and higher doses (over 150 mg/kg daily) may confer significant protection against atherosclerosis even in the situation of elevated cholesterol levels.

The scale of vitamin C benefits on cardiovascular system led several authors to the theory, that vitamin C deficiency is the primary cause of cardiovascular diseases. The theory was unified by twice Nobel prize winner Linus Pauling and Matthias Rath. They suggest, that clinical manifestations of cardiovascular diseases are merely overshoot of body defense mechanisms, that are involved in stabilisation of vascular wall, after it is weakened by the vitamin C deficiency and the

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subsequent collagen degradation. They discuss several metabolic and genetic predispositions and their pathomechanism.

Trials on Vitamin E have been done, but they have failed to find a beneficial effect, for various reasons, but for some patients at high risk for atherosclerosis there may be some benefits.

Menaquinone (Vitamin K2), but not phylloquinone (Vitamin K1), intake is associated with reduced risk of CHD mortality, all-cause mortality and severe aortic calcification.

It has been suggested that excess iron may be involved in development of atherosclerosis, but one study found reducing body iron stores in patients with symptomatic peripheral artery disease through phlebotomy did not significantly decrease all-cause mortality or death plus nonfatal myocardial infarction and stroke. Further studies may be warranted.

STUDENT ACTIVITY

1. Point out the dietary factors responsible for cardiovascular diseases.

2. Discuss the two most important causes of hypertension.

3. Discuss the symptoms of Atherosclerosis.

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4.4 MYOCARDIAL INFARCTION

Myocardial infarction (MI) commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing some heart cells to die. This is most commonly due to occlusion (blockage) of a coronary artery following the rupture of a vulnerable atherosclerotic plaque, which is an unstable collection of lipids (like cholesterol) and white blood cells (especially macrophages) in the wall of an artery. The resulting ischemia (restriction in blood supply) and oxygen shortage, if left untreated for a sufficient period of time, can cause damage or death (infarction) of heart muscle tissue (myocardium).

Classical symptoms of acute myocardial infarction include sudden chest pain (typically radiating to the left arm or left side of the neck), shortness of breath, nausea, vomiting, palpitations, sweating, and anxiety (often described as a sense of impending doom). Women may experience fewer typical symptoms than men, most commonly shortness of breath, weakness, a feeling of indigestion, and fatigue. Approximately one quarter of all myocardial infarctions are silent, without chest pain or other symptoms. A heart attack is a medical emergency, and people experiencing chest pain are advised to alert their emergency medical services because prompt protection with an external defibrillator can save your life from primary ventricular fibrillation which occurs unexpectedly in 10% of all myocardial infarctions especially during the first hours of symptoms. Contemporary treatment of many myocardial infarctions can result in survival and even good outcomes. While it is true that certain less amenable cases are very massive and rapidly fatal "widowmakers", it is also true that in small attacks with limited damage and optimal treatment the heart muscle can be salvaged.

Heart attacks are the leading cause of death for both men and women all over the world. Important risk factors are previous cardiovascular disease (such as angina, a previous heart attack or stroke), older age (especially men over 40 and women over 50), tobacco smoking, high blood levels of certain lipids (triglycerides, low-density lipoprotein or "bad cholesterol") and low levels of high density lipoprotein (HDL, "good cholesterol"), diabetes, high blood pressure, obesity, chronic kidney disease, heart failure, excessive alcohol consumption, the abuse of certain drugs (such as cocaine and methamphetamine), and chronic high stress levels.

Immediate treatment for suspected acute myocardial infarction includes oxygen, aspirin, and sublingual glyceryl trinitrate (colloquially referred to as nitroglycerin and abbreviated as NTG or GTN). Pain relief is also often given, classically morphine sulfate. However, a 2009 review about the use of high flow oxygen for treating myocardial infarction found its administration increased mortality and infarct size, calling into question the recommendation for its routine use.

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The patient will receive a number of diagnostic tests, such as an electrocardiogram (ECG, EKG), a chest X-ray and blood tests to detect elevations in cardiac markers (blood tests to detect heart muscle damage). The most often used markers are the creatine kinase-MB (CK-MB) fraction and the troponin I (TnI) or troponin T (TnT) levels. On the basis of the ECG, a distinction is made between ST elevation MI (STEMI) or non-ST elevation MI (NSTEMI). Most cases of STEMI are treated with thrombolysis or if possible with percutaneous coronary intervention (PCI, angioplasty and stent insertion), provided the hospital has facilities for coronary angiography. NSTEMI is managed with medication, although PCI is often performed during hospital admission. In patients who have multiple blockages and who are relatively stable, or in a few extraordinary emergency cases, bypass surgery of the blocked coronary artery is an option.

The phrase "heart attack" is sometimes used incorrectly to describe sudden cardiac death, which may or may not be the result of acute myocardial infarction. A heart attack is different from, but can be the cause of cardiac arrest, which is the stopping of the heartbeat, and cardiac arrhythmia, an abnormal heartbeat. It is also distinct from heart failure, in which the pumping action of the heart is impaired; severe myocardial infarction may lead to heart failure, but not necessarily.

Classification

There are two basic types of acute myocardial infarction. (1) transmural MI- IS associated with atherosclerosis involving major coronary artery. It can be subclassified into anterior,posterior or inferior. (2) subendocardial MI- involves small area, in the subendocardial wall of the left ventricle,ventricular septum,papillary muscles.

Clinically, myocardial infarction is further subclassified into ST elevation MI versus non ST elevation MI based on ECG changes.

Signs and Symptoms

The onset of symptoms in myocardial infarction (MI) is usually gradual, over several minutes, and rarely instantaneous. Chest pain is the most common symptom of acute myocardial infarction and is often described as a sensation of tightness, pressure, or squeezing. Chest pain due to ischemia (a lack of blood and hence oxygen supply) of the heart muscle is termed angina pectoris. Pain radiates most often to the left arm, but may also radiate to the lower jaw, neck, right arm, back, and epigastrium, where it may mimic heartburn. Levine's sign, in which the patient localizes the chest pain by clenching their fist over the sternum, has classically been thought to be predictive of cardiac chest pain, although a prospective observational study showed that it had a poor positive predictive value.

Shortness of breath (dyspnea) occurs when the damage to the heart limits the output of the left ventricle, causing left ventricular failure and consequent pulmonary edema. Other symptoms include diaphoresis (an excessive form of sweating), weakness, light-headedness, nausea, vomiting, and palpitations. These symptoms are likely induced by a massive surge of catecholamines from the sympathetic nervous system which occurs in response to pain and the hemodynamic abnormalities that result from cardiac dysfunction. Loss of consciousness (due to inadequate cerebral perfusion and cardiogenic shock) and even sudden death (frequently due to the development of ventricular fibrillation) can occur in myocardial infarctions.

Women and older patients experience a typical symptoms more frequently than their male and younger counterparts. Women also have more symptoms compared to men (2.6 on average vs 1.8 symptoms in men). The most common symptoms of MI in women include dyspnea, weakness, and fatigue. Fatigue, sleep disturbances, and dyspnea have been reported as frequently occurring symptoms which may manifest as long as one month before the actual clinically manifested ischemic event. In women, chest pain may be less predictive of coronary ischemia than in men.

Approximately half of all MI patients have experienced warning symptoms such as chest pain prior to the infarction.

Approximately one fourth of all myocardial infarctions are silent, without chest pain or other symptoms. These cases can be discovered later on electrocardiograms or at autopsy without a prior history of related complaints. A silent course is more common in the elderly, in patients with diabetes mellitus and after heart transplantation, probably because the donor heart is not connected to nerves of the host. In diabetics, differences in pain threshold, autonomic neuropathy, and psychological factors have been cited as possible explanations for the lack of symptoms.

Any group of symptoms compatible with a sudden interruption of the blood flow to the heart are called an acute coronary syndrome.

The differential diagnosis includes other catastrophic causes of chest pain, such as pulmonary embolism, aortic dissection, pericardial effusion causing cardiac tamponade, tension pneumothorax, and esophageal rupture.

Causes and Risk Factors

Heart attack rates are higher in association with intense exertion, be it psychological stress or physical exertion, especially if the exertion is more intense than the individual usually performs. Quantitatively, the period of intense exercise and subsequent recovery is associated with about a 6-fold higher myocardial

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infarction rate (compared with other more relaxed time frames) for people who are physically very fit. For those in poor physical condition, the rate differential is over 35-fold higher. One observed mechanism for this phenomenon is the increased arterial pulse pressure stretching and relaxation of arteries with each heart beat which, as has been observed with intravascular ultrasound, increases mechanical "shear stress" on atheromas and the likelihood of plaque rupture.

Acute severe infection, such as pneumonia, can trigger myocardial infarction. A more controversial link is that between Chlamydophila pneumoniae infection and atherosclerosis. While this intracellular organism has been demonstrated in atherosclerotic plaques, evidence is inconclusive as to whether it can be considered a causative factor. Treatment with antibiotics in patients with proven atherosclerosis has not demonstrated a decreased risk of heart attacks or other coronary vascular diseases.

Risk Factors

Risk factors for atherosclerosis are generally risk factors for myocardial infarction:

- *Diabetes.* (with or without insulin resistance) - the single most important risk factor for ischaemic heart disease (IHD)
- *Tobacco smoking*
- *Hypercholesterolemia* (more accurately hyperlipoproteinemia, especially high low density lipoprotein and low high density lipoprotein)
- *High blood pressure*
- Family history of ischaemic heart disease (IHD)
- *Obesity* (defined by a body mass index of more than 30 kg/m^2, or alternatively by waist circumference or waist-hip ratio).
- *Age.* Men acquire an independent risk factor at age 45, Women acquire an independent risk factor at age 55; in addition individuals acquire another independent risk factor if they have a first-degree male relative (brother,father)who suffered a coronary vascular event at or before age 55. Another independent risk factor is acquired if one has a first-degree female relative (mother,sister) who suffered a coronary vascular event at age 65 or younger.
- *Hyperhomocysteinemia* (high homocysteine, a toxic blood amino acid that is elevated when intakes of vitamins B2, B6, B12 and folic acid are insufficient)
- *Stress* (occupations with high stress index are known to have susceptibility for atherosclerosis)

- *Alcohol.* Studies show that prolonged exposure to high quantities of alcohol can increase the risk of heart attack

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Males are more at risk than females

Many of these risk factors are modifiable, so many heart attacks can be prevented by maintaining a healthier lifestyle. Physical activity, for example, is associated with a lower risk profile. Non-modifiable risk factors include age, sex, and family history of an early heart attack (before the age of 60), which is thought of as reflecting a genetic predisposition.

Socio-economic factors such as a shorter education and lower income (particularly in women), and unmarried cohabitation may also contribute to the risk of MI. To understand epidemiological study results, it's important to note that many factors associated with MI mediate their risk via other factors. For example, the effect of education is partially based on its effect on income and marital status.

Women who use combined oral contraceptive pills have a modestly increased risk of myocardial infarction, especially in the presence of other risk factors, such as smoking.

Inflammation is known to be an important step in the process of atherosclerotic plaque formation. C-reactive protein (CRP) is a sensitive but non-specific marker for inflammation. Elevated CRP blood levels, especially measured with high sensitivity assays, can predict the risk of MI, as well as stroke and development of diabetes. Moreover, some drugs for MI might also reduce CRP levels. The use of high sensitivity CRP assays as a means of screening the general population is advised against, but it may be used optionally at the physician's discretion, in patients who already present with other risk factors or known coronary artery disease. Whether CRP plays a direct role in atherosclerosis remains uncertain.

Inflammation in periodontal disease may be linked coronary heart disease, and since periodontitis is very common, this could have great consequences for public health. Serological studies measuring antibody levels against typical periodontitis-causing bacteria found that such antibodies were more present in subjects with coronary heart disease. Periodontitis tends to increase blood levels of CRP, fibrinogen and cytokines; thus, periodontitis may mediate its effect on MI risk via other risk factors. Preclinical research suggests that periodontal bacteria can promote aggregation of platelets and promote the formation of foam cells. A role for specific periodontal bacteria has been suggested but remains to be established. There is some evidence that influenza may trigger a acute myocardial infarction.

Baldness, hair greying, a diagonal earlobe crease (Frank's sign) and possibly other skin features have been suggested as independent risk factors for MI. Their role remains controversial; a common denominator of these signs and the risk of MI is supposed, possibly genetic.

Calcium deposition is another part of atherosclerotic plaque formation. Calcium deposits in the coronary arteries can be detected with CT scans. Several studies have shown that coronary calcium can provide predictive information beyond that of classical risk factors.

Diagnosis

The diagnosis of myocardial infarction is made by integrating the history of the presenting illness and physical examination with electrocardiogram findings and cardiac markers (blood tests for heart muscle cell damage). A coronary angiogram allows visualization of narrowings or obstructions on the heart vessels, and therapeutic measures can follow immediately. At autopsy, a pathologist can diagnose a myocardial infarction based on anatomopathological findings.

A chest radiograph and routine blood tests may indicate complications or precipitating causes and are often performed upon arrival to an emergency department. New regional wall motion abnormalities on an echocardiogram are also suggestive of a myocardial infarction. Echo may be performed in equivocal cases by the on-call cardiologist. In stable patients whose symptoms have resolved by the time of evaluation, technetium-99m 2-methoxyisobutylisonitrile (Tc99m MIBI) or thallium-201 chloride can be used in nuclear medicine to visualize areas of reduced blood flow in conjunction with physiologic or pharmocologic stress. Thallium may also be used to determine viability of tissue, distinguishing whether non-functional myocardium is actually dead or merely in a state of hibernation or of being stunned.

Diagnostic Criteria

WHO criteria formulated in 1979 have classically been used to diagnose MI; a patient is diagnosed with myocardial infarction if two (probable) or three (definite) of the following criteria are satisfied:

1. Clinical history of ischaemic type chest pain lasting for more than 20 minutes
2. Changes in serial ECG tracings
3. Rise and fall of serum cardiac biomarkers such as creatine kinase-MB fraction and troponin

The WHO criteria were refined in 2000 to give more prominence to cardiac biomarkers. According to the new guidelines, a cardiac troponin rise accompanied

by either typical symptoms, pathological Q waves, ST elevation or depression or coronary intervention are diagnostic of MI.

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Physical Examination

The general appearance of patients may vary according to the experienced symptoms; the patient may be comfortable, or restless and in severe distress with an increased respiratory rate. A cool and pale skin is common and points to vasoconstriction. Some patients have low-grade fever (38–39 °C). Blood pressure may be elevated or decreased, and the pulse can be become irregular.

If heart failure ensues, elevated jugular venous pressure and hepatojugular reflux, or swelling of the legs due to peripheral edema may be found on inspection. Rarely, a cardiac bulge with a pace different from the pulse rhythm can be felt on precordial examination. Various abnormalities can be found on auscultation, such as a third and fourth heart sound, systolic murmurs, paradoxical splitting of the second heart sound, a pericardial friction rub and rales over the lung.

Prevention

The risk of a recurrent myocardial infarction decreases with strict blood pressure management and lifestyle changes, chiefly smoking cessation, regular exercise, a sensible diet for patients with heart disease, and limitation of alcohol intake.

Patients are usually commenced on several long-term medications post-MI, with the aim of preventing secondary cardiovascular events such as further myocardial infarctions, congestive heart failure or cerebrovascular accident (CVA). Unless contraindicated, such medications may include:

- Antiplatelet drug therapy such as aspirin and/or clopidogrel should be continued to reduce the risk of plaque rupture and recurrent myocardial infarction. Aspirin is first-line, owing to its low cost and comparable efficacy, with clopidogrel reserved for patients intolerant of aspirin. The combination of clopidogrel and aspirin may further reduce risk of cardiovascular events, however the risk of hemorrhage is increased.
- Beta blocker therapy such as metoprolol or carvedilol should be commenced. These have been particularly beneficial in high-risk patients such as those with left ventricular dysfunction and/or continuing cardiac ischaemia. â-Blockers decrease mortality and morbidity. They also improve symptoms of cardiac ischemia in NSTEMI.
- ACE inhibitor therapy should be commenced 24–48 hours post-MI in hemodynamically-stable patients, particularly in patients with a history of MI, diabetes mellitus, hypertension, anterior location of infarct (as assessed by ECG), and/or evidence of left ventricular dysfunction. ACE

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inhibitors reduce mortality, the development of heart failure, and decrease ventricular remodelling post-MI.

- Statin therapy has been shown to reduce mortality and morbidity post-MI. The effects of statins may be more than their LDL lowering effects. The general consensus is that statins have plaque stabilization and multiple other ("pleiotropic") effects that may prevent myocardial infarction in addition to their effects on blood lipids.
- The aldosterone antagonist agent eplerenone has been shown to further reduce risk of cardiovascular death post-MI in patients with heart failure and left ventricular dysfunction, when used in conjunction with standard therapies above.
- Omega-3 fatty acids, commonly found in fish, have been shown to reduce mortality post-MI. While the mechanism by which these fatty acids decrease mortality is unknown, it has been postulated that the survival benefit is due to electrical stabilization and the prevention of ventricular fibrillation. However, further studies in a high-risk subset have not shown a clear-cut decrease in potentially fatal arrhythmias due to omega-3 fatty acids.

Complications

Complications may occur immediately following the heart attack (in the acute phase), or may need time to develop (a chronic problem). After an infarction, an obvious complication is a second infarction, which may occur in the domain of another atherosclerotic coronary artery, or in the same zone if there are any live cells left in the infarct.

Congestive Heart Failure

A myocardial infarction may compromise the function of the heart as a pump for the circulation, a state called heart failure. There are different types of heart failure; left- or right-sided (or bilateral) heart failure may occur depending on the affected part of the heart, and it is a low-output type of failure. If one of the heart valves is affected, this may cause dysfunction, such as mitral regurgitation in the case of left-sided coronary occlusion that disrupts the blood supply of the papillary muscles. The incidence of heart failure is particularly high in patients with diabetes and requires special management strategies.

Myocardial Rupture

Myocardial rupture is most common three to five days after myocardial infarction, commonly of small degree, but may occur one day to three weeks later. In the modern era of early revascularization and intensive pharmacotherapy

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as treatment for MI, the incidence of myocardial rupture is about 1% of all MIs. This may occur in the free walls of the ventricles, the septum between them, the papillary muscles, or less commonly the atria. Rupture occurs because of increased pressure against the weakened walls of the heart chambers due to heart muscle that cannot pump blood out effectively. The weakness may also lead to ventricular aneurysm, a localized dilation or ballooning of the heart chamber.

Risk factors for myocardial rupture include completion of infarction (no revascularization performed), female sex, advanced age, and a lack of a previous history of myocardial infarction. In addition, the risk of rupture is higher in individuals who are revascularized with a thrombolytic agent than with PCI. The shear stress between the infarcted segment and the surrounding normal myocardium (which may be hypercontractile in the post-infarction period) makes it a nidus for rupture.

Rupture is usually a catastrophic event that may result a life-threatening process known as cardiac tamponade, in which blood accumulates within the pericardium or heart sac, and compresses the heart to the point where it cannot pump effectively. Rupture of the intraventricular septum (the muscle separating the left and right ventricles) causes a ventricular septal defect with shunting of blood through the defect from the left side of the heart to the right side of the heart, which can lead to right ventricular failure as well as pulmonary overcirculation. Rupture of the papillary muscle may also lead to acute mitral regurgitation and subsequent pulmonary edema and possibly even cardiogenic shock.

Life-Threatening Arrhythmia

Since the electrical characteristics of the infarcted tissue change (see pathophysiology section), arrhythmias are a frequent complication. The re-entry phenomenon may cause rapid heart rates (ventricular tachycardia and even ventricular fibrillation), and ischemia in the electrical conduction system of the heart may cause a complete heart block (when the impulse from the sinoatrial node, the normal cardiac pacemaker, does not reach the heart chambers).

Pericarditis

As a reaction to the damage of the heart muscle, inflammatory cells are attracted. The inflammation may reach out and affect the heart sac. This is called pericarditis. In Dressler's syndrome, this occurs several weeks after the initial event.

Cardiogenic Shock

A complication that may occur in the acute setting soon after a myocardial infarction or in the weeks following it is cardiogenic shock. Cardiogenic shock is

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defined as a hemodynamic state in which the heart cannot produce enough of a cardiac output to supply an adequate amount of oxygenated blood to the tissues of the body.

While the data on performing interventions on individuals with cardiogenic shock is sparse, trial data suggests a long-term mortality benefit in undergoing revascularization if the individual is less than 75 years old and if the onset of the acute myocardial infarction is less than 36 hours and the onset of cardiogenic shock is less than 18 hours. If the patient with cardiogenic shock is not going to be revascularized, aggressive hemodynamic support is warranted, with insertion of an intra-aortic balloon pump if not contraindicated. If diagnostic coronary angiography does not reveal a culprit blockage that is the cause of the cardiogenic shock, the prognosis is poor.

4.5 CONSECUTIVE CARDIAC FAILURE (HEART FAILURE)

Cardiac failure is a condition in which a problem with the structure or function of the heart impairs its ability to supply sufficient blood flow to meet the body's needs. Common causes of heart failure include myocardial infarction and other forms of ischemic heart disease, hypertension, valvular heart disease and cardiomyopathy. Heart failure can cause a large variety of symptoms such as shortness of breath (typically worse when lying flat, which is called orthopnea), coughing, ankle swelling and reduced exercise capacity. Heart failure is often undiagnosed due to a lack of a universally agreed definition and challenges in definitive diagnosis. Treatment commonly consists of lifestyle measures (such as decreased salt intake) and medications, and sometimes devices or even surgery.

Heart failure is a common, costly, disabling and deadly condition. In developing countries, around 2% of adults suffer from heart failure, but in those over the age of 65, this increases to 6–10%. Mostly due to costs of hospitalization, it is associated with a high health expenditure; costs have been estimated to amount to 2% of the total budget of the National Health Service in the United Kingdom, and more than $35 billion in the United States. Heart failure is associated with significantly reduced physical and mental health, resulting in a markedly decreased quality of life. With the exception of heart failure caused by reversible conditions, the condition usually worsens with time. Although some patients survive many years, progressive disease is associated with an overall annual mortality rate of 10%.

Heart failure is a global term for the physiological state in which cardiac output is insufficient for the body's needs.

This occurs most commonly when the cardiac output is low (often termed "congestive heart failure").

In contrast, it may also occur when the body's requirements for oxygen and nutrients are increased, and demand outstrips what the heart can provide, (termed "high output cardiac failure"). This can occur in the context of severe anemia, Gram negative septicaemia, beriberi (vitamin B_1/thiamine deficiency), thyrotoxicosis, Paget's disease, arteriovenous fistulae or arteriovenous malformations.

Fluid overload is a common problem for people with heart failure, but is not synonymous with it. Patients with treated heart failure will often be euvolaemic (a term for normal fluid status), or more rarely, dehydrated.

Doctors use the words "acute" to mean of rapid onset, and "chronic" of long duration. Chronic heart failure is therefore a long-term situation, usually with stable treated symptomatology.

Acute decompensated heart failure, which should just describe sudden onset HF, is also used to describe exacerbated or decompensated heart failure, referring to episodes in which a patient with known chronic heart failure abruptly develops symptoms.

There are several terms which are closely related to heart failure, and may be the cause of heart failure, but should not be confused with it:

- Cardiac arrest, and asystole both refer to situations in which there is no cardiac output at all. Without urgent treatment, these result in sudden death.
- Heart attack refers to a blockage in a coronary (heart) artery resulting in heart muscle damage.
- Cardiomyopathy refers specifically to problems within the heart muscle, and these problems usually result in heart failure. Ischemic cardiomyopathy implies that the cause of muscle damage is coronary artery disease. Dilated cardiomyopathy implies that the muscle damage has resulted in enlargement of the heart. Hypertrophic cardiomyopathy involves enlargement and thickening of the heart muscle.

Classification

There are many different ways to categorize heart failure, including:

- the side of the heart involved, (left heart failure versus right heart failure)
- whether the abnormality is due to contraction or relaxation of the heart (systolic dysfunction vs. diastolic dysfunction)
- whether the problem is primarily increased venous back pressure (behind) the heart, or failure to supply adequate arterial perfusion (in front of) the heart (backward vs. forward failure)

- whether the abnormality is due to low cardiac output with high systemic vascular resistance or high cardiac output with low vascular resistance (low-output heart failure vs. high-output heart failure)
- the degree of functional impairment conferred by the abnormality (as in the NYHA functional classification)

In its 2001 guidelines, the American College of Cardiology/American Heart Association working group introduced four stages of heart failure:

- **Stage A:** Patients at high risk for developing HF in the future but no functional or structural heart disorder;
- **Stage B:** a structural heart disorder but no symptoms at any stage;
- **Stage C:** previous or current symptoms of heart failure in the context of an underlying structural heart problem, but managed with medical treatment;
- **Stage D:** advanced disease requiring hospital-based support, a heart transplant or palliative care.

Symptoms and Signs

Heart failure symptoms are traditionally and somewhat arbitrarily divided into "left" and "right" sided, recognizing that the left and right ventricles of the heart supply different portions of the circulation. However, heart failure is not exclusively backward failure (in the part of the circulation which drains to the ventricle).

There are several other exceptions to a simple left-right division of heart failure symptoms. Left sided forward failure overlaps with right sided backward failure. Additionally, the most common cause of right-sided heart failure is left-sided heart failure. The result is that patients commonly present with both sets of signs and symptoms.

Left-Sided Failure

Forward failure of the left ventricle causes congestion of the pulmonary vasculature, and so the symptoms are predominantly respiratory in nature. Forward failure can be subdivided into failure of the left atrium, the left ventricle or both within the left circuit. The patient will have dyspnea (shortness of breath) on exertion (dyspnée d'effort) and in severe cases, dyspnea at rest. Increasing breathlessness on lying flat, called orthopnea, occurs. It is often measured in the number of pillows required to lie comfortably, and in severe cases, the patient may resort to sleeping while sitting up. Another symptom of heart failure is paroxysmal nocturnal dyspnea also known as "cardiac asthma", a sudden nighttime attack of severe breathlessness, usually several hours after going to

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sleep. Easy fatigueability and exercise intolerance are also common complaints related to respiratory compromise.

Compromise of left ventricular forward function may result in symptoms of poor systemic circulation such as dizziness, confusion and cool extremities at rest.

Right-Sided Failure

Backward failure of the right ventricle leads to congestion of systemic capillaries. This helps to generate excess fluid accumulation in the body. This causes swelling under the skin (termed peripheral edema or anasarca) and usually affects the dependent parts of the body first (causing foot and ankle swelling in people who are standing up, and sacral edema in people who are predominantly lying down). Nocturia (frequent nighttime urination) may occur when fluid from the legs is returned to the bloodstream while lying down at night. In progressively severe cases, ascites (fluid accumulation in the abdominal cavity causing swelling) and hepatomegaly (enlargement of the liver) may develop. Significant liver congestion may result in impaired liver function, and jaundice and even coagulopathy (problems of decreased blood clotting) may occur.

Signs

Left-Sided Failure

Common respiratory signs are tachypnea (increased rate of breathing) and increased work of breathing (non-specific signs of respiratory distress). Rales or crackles, heard initially in the lung bases, and when severe, throughout the lung fields suggest the development of pulmonary edema (fluid in the alveoli). Dullness of the lung fields to finger percussion and reduced breath sounds at the bases of the lung may suggest the development of a pleural effusion (fluid collection in between the lung and the chest wall). Cyanosis which suggests severe hypoxemia, is a late sign of extremely severe pulmonary edema.

Additional signs indicating left ventricular failure include a laterally displaced apex beat (which occurs if the heart is enlarged) and a gallop rhythm (additional heart sounds) may be heard as a marker of increased blood flow, or increased intra-cardiac pressure. Heart murmurs may indicate the presence of valvular heart disease, either as a cause (*e.g.*, aortic stenosis) or as a result (*e.g.*, mitral regurgitation) of the heart failure.

Right-Sided Failure

Physical examination can reveal pitting peripheral edema, ascites, and hepatomegaly. Jugular venous pressure is frequently assessed as a marker of fluid status, which can be accentuated by the hepatojugular reflux. If the right ventriclar

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pressure is increased, a parasternal heave may be present, signifying the compensatory increase in contraction strength.

CAUSES

Chronic Heart Failure

The predominance of causes of heart failure are difficult to analyze due to challenges in diagnosis, differences in populations, and changing prevalence of causes with age.

A 19 year study of 13000 healthy adults in the United States (the National Health and Nutrition Examination Survey (NHANES I) found the following causes ranked by Population Attributable Risk score:

1. Ischaemic Heart Disease 62%
2. Cigarette Smoking 16%
3. Hypertension (high blood pressure)10%
4. Obesity 8%
5. Diabetes 3%
6. Valvular heart disease 2% (much higher in older populations)

An Italian registry of over 6200 patients with heart failure showed the following underlying causes:

1. Ischaemic Heart Disease 40%
2. Dilated Cardiomyopathy 32%
3. Valvular Heart Disease 12%
4. Hypertension 11%
5. Other 5%

Rarer causes of heart failure include:

- Viral Myocarditis (an infection of the heart muscle)
- Infiltrations of the muscle such as amyloidosis
- HIV cardiomyopathy. (caused by Human Immunodeficiency Virus)
- Connective Tissue Diseases such as Systemic lupus erythematosus
- Abuse of drugs such as alcohol
- Pharmaceutical drugs such as chemotherapeutic agents.
- Arrhythmias

Obstructive Sleep Apnea a condition of sleep disordered breathing overlaps with obesity, hypertension and diabetes and is regarded as an independent cause of heart failure.

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Acute Decompensated Heart Failure

Chronic stable heart failure may easily decompensate. This most commonly results from an intercurrent illness (such as pneumonia), myocardial infarction (a heart attack), arrhythmias, uncontrolled hypertension, or a patient's failure to maintain a fluid restriction, diet or medication. Other well recognised precipitating factors include anaemia and hyperthyroidism which place additional strain on the heart muscle. Excessive fluid or salt intake, and medication that causes fluid retention such as NSAIDs and thiazolidinediones, may also precipitate decompensation.

Pathophysiology

Heart failure is caused by any condition which reduces the efficiency of the myocardium, or heart muscle, through damage or overloading. As such, it can be caused by as diverse an array of conditions as myocardial infarction (in which the heart muscle is starved of oxygen and dies), hypertension (which increases the force of contraction needed to pump blood) and amyloidosis (in which protein is deposited in the heart muscle, causing it to stiffen). Over time these increases in workload will produce changes to the heart itself:

- Reduced contractility, or force of contraction, due to overloading of the ventricle. In health, increased filling of the ventricle results in increased contractility (by the Frank-Starling law of the heart) and thus a rise in cardiac output. In heart failure this mechanism fails, as the ventricle is loaded with blood to the point where heart muscle contraction becomes less efficient. This is due to reduced ability to cross-link actin and myosin filaments in over-stretched heart muscle.
- A reduced stroke volume, as a result of a failure of systole, diastole or both. Increased end systolic volume is usually caused by reduced contractility. Decreased end diastolic volume results from impaired ventricular filling – as occurs when the compliance of the ventricle falls (*i.e.*, when the walls stiffen).
- Reduced spare capacity. As the heart works harder to meet normal metabolic demands, the amount cardiac output can increase in times of increased oxygen demand (*e.g.*, exercise) is reduced. This contributes to the exercise intolerance commonly seen in heart failure. This translates to the loss of one's cardiac reserve. The cardiac reserve refers to the ability of the heart to work harder during exercise or strenuous activity. Since the heart has to work harder to meet the normal metabolic demands, it is incapable of meeting the metabolic demands of the body during exercise.
- Increased heart rate, stimulated by increased sympathetic activity in order to maintain cardiac output. Initially, this helps compensate for heart failure

by maintaining blood pressure and perfusion, but places further strain on the myocardium, increasing coronary perfusion requirements, which can lead to worsening of ischemic heart disease. Sympathetic activity may also cause potentially fatal arrhythmias.

- Hypertrophy (an increase in physical size) of the myocardium, caused by the terminally differentiated heart muscle fibres increasing in size in an attempt to improve contractility. This may contribute to the increased stiffness and decreased ability to relax during diastole.
- Enlargement of the ventricles, contributing to the enlargement and spherical shape of the failing heart. The increase in ventricular volume also causes a reduction in stroke volume due to mechanical and contractile inefficiency.

The general effect is one of reduced cardiac output and increased strain on the heart. This increases the risk of cardiac arrest (specifically due to ventricular dysrhythmias), and reduces blood supply to the rest of the body. In chronic disease the reduced cardiac output causes a number of changes in the rest of the body, some of which are physiological compensations, some of which are part of the disease process:

- Arterial blood pressure falls. This destimulates baroreceptors in the carotid sinus and aortic arch which link to the nucleus tractus solitarius. This center in the brain increases sympathetic activity, releasing catecholamines into the blood stream. Binding to alpha-1 receptors results in systemic arterial vasoconstriction. This helps restore blood pressure but also increases the total peripheral resistance, increasing the workload of the heart. Binding to beta-1 receptors in the myocardium increases the heart rate and make contractions more forceful, in an attempt to increase cardiac output. This also, however, increases the amount of work the heart has to perform.
- Increased sympathetic stimulation also causes the hypothalamus to secrete vasopressin (also known as antidiuretic hormone or ADH), which causes fluid retention at the kidneys. This increases the blood volume and blood pressure.
- Reduced perfusion (blood flow) to the kidneys stimulates the release of renin – an enzyme which catalyses the production of the potent vasopressor angiotensin. Angiotensin and its metabolites cause further vasocontriction, and stimulate increased secretion of the steroid aldosterone from the adrenal glands. This promotes salt and fluid retention at the kidneys, also increasing the blood volume.

- The chronically high levels of circulating neuroendocrine hormones such as catecholamines, renin, angiotensin, and aldosterone affects the myocardium directly, causing structural remodelling of the heart over the long-term. Many of these remodelling effects seem to be mediated by transforming growth factor beta (TGF-beta), which is a common downstream target of the signal transduction cascade initiated by catecholamines and angiotensin II, and also by epidermal growth factor (EGF), which is a target of the signaling pathway activated by aldosterone.
- Reduced perfusion of skeletal muscle causes atrophy of the muscle fibres. This can result in weakness, increased fatigueability and decreased peak strength—all contributing to exercise intolerance.

The increased peripheral resistance and greater blood volume place further strain on the heart and accelerates the process of damage to the myocardium. Vasoconstriction and fluid retention produce an increased hydrostatic pressure in the capillaries. This shifts of the balance of forces in favour of interstitial fluid formation as the increased pressure forces additional fluid out of the blood, into the tissue. This results in edema (fluid build-up) in the tissues. In right-sided heart failure this commonly starts in the ankles where venous pressure is high due to the effects of gravity (although if the patient is bed-ridden, fluid accumulation may begin in the sacral region.) It may also occur in the abdominal cavity, where the fluid build-up is called ascites. In left-sided heart failure edema can occur in the lungs - this is called cardiogenic pulmonary oedema. This reduces spare capacity for ventilation, causes stiffening of the lungs and reduces the efficiency of gas exchange by increasing the distance between the air and the blood. The consequences of this are shortness of breath, orthopnoea and paroxysmal nocturnal dyspnea.

The symptoms of heart failure are largely determined by which side of the heart fails. The left side pumps blood into the systemic circulation, whilst the right side pumps blood into the pulmonary circulation. Whilst left-sided heart failure will reduce cardiac output to the systemic circulation, the initial symptoms often manifest due to effects on the pulmonary circulation. In systolic dysfunction, the ejection fraction is decreased, leaving an abnormally elevated volume of blood in the left ventricle. In diastolic dysfunction, end-diastolic ventricular pressure will be high. This increase in volume or pressure backs up to the left atrium and then to the pulmonary veins. Increased volume or pressure in the pulmonary veins impairs the normal drainage of the alveoli and favours the flow of fluid from the capillaries to the lung parenchyma, causing pulmonary edema. This impairs gas exchange. Thus, left-sided heart failure often presents with respiratory symptoms: shortness of breath, orthopnea and paroxysmal nocturnal dyspnea.

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In severe cardiomyopathy, the effects of decreased cardiac output and poor perfusion become more apparent, and patients will manifest with cold and clammy extremities, cyanosis, claudication, generalized weakness, dizziness, and syncope.

The resultant hypoxia caused by pulmonary edema causes vasoconstriction in the pulmonary circulation, which results in pulmonary hypertension. Since the right ventricle generates far lower pressures than the left ventricle (approximately 20 mmHg versus around 120 mmHg, respectively, in the healthy individual) but nonetheless generates cardiac output exactly equal to the left ventricle, this means that a small increase in pulmonary vascular resistance causes a large increase in amount of work the right ventricle must perform. However, the main mechanism by which left-sided heart failure causes right-sided heart failure is actually not well understood. Some theories invoke mechanisms that are mediated by neurohormonal activation. Mechanical effects may also contribute. As the left ventricle distends, the intraventricular septum bows into the right ventricle, decreasing the capacity of the right ventricle.

Systolic Dysfunction

Heart failure caused by systolic dysfunction is more readily recognized. It can be simplistically described as failure of the pump function of the heart. It is characterized by a decreased ejection fraction (less than 45%). The strength of ventricular contraction is attenuated and inadequate for creating an adequate stroke volume, resulting in inadequate cardiac output. In general, this is caused by dysfunction or destruction of cardiac myocytes or their molecular components. In congenital diseases such as Duchenne muscular dystrophy, the molecular structure of individual myocytes is affected. Myocytes and their components can be damaged by inflammation (such as in myocarditis) or by infiltration (such as in amyloidosis). Toxins and pharmacological agents (such as ethanol, cocaine, and amphetamines) cause intracellular damage and oxidative stress. The most common mechanism of damage is ischemia causing infarction and scar formation. After myocardial infarction, dead myocytes are replaced by scar tissue, deleteriously affecting the function of the myocardium. On echocardiogram, this is manifest by abnormal or absent wall motion.

Because the ventricle is inadequately emptied, ventricular end-diastolic pressure and volumes increase. This is transmitted to the atrium. On the left side of the heart, the increased pressure is transmitted to the pulmonary vasculature, and the resultant hydrostatic pressure favours extravassation of fluid into the lung parenchyma, causing pulmonary edema. On the right side of the heart, the increased pressure is transmitted to the systemic venous circulation and systemic capillary beds, favouring extravassation of fluid into the tissues of target organs and extremities, resulting in dependent peripheral edema.

Diastolic Dysfunction

Heart failure caused by diastolic dysfunction is generally described as the failure of the ventricle to adequately relax and typically denotes a stiffer ventricular wall. This causes inadequate filling of the ventricle, and therefore results in an inadequate stroke volume. The failure of ventricular relaxation also results in elevated end-diastolic pressures, and the end result is identical to the case of systolic dysfunction (pulmonary edema in left heart failure, peripheral edema in right heart failure.)

Diastolic dysfunction can be caused by processes similar to those that cause systolic dysfunction, particularly causes that affect cardiac remodeling.

Diastolic dysfunction may not manifest itself except in physiologic extremes if systolic function is preserved. The patient may be completely asymptomatic at rest. However, they are exquisitely sensitive to increases in heart rate, and sudden bouts of tachycardia (which can be caused simply by physiological responses to exertion, fever, or dehydration, or by pathological tachyarrhythmias such as atrial fibrillation with rapid ventricular response) may result in flash pulmonary edema. Adequate rate control (usually with a pharmacological agent that slows down AV conduction such as a calcium channel blocker or a beta-blocker) is therefore key to preventing decompensation.

Left ventricular diastolic function can be determined through echocardiography by measurement of various parameters such as the E/A ratio (early-to-atrial left ventricular filling ratio), the E (early left ventricular filling) deceleration time, and the isovolumic relaxation time.

4.6 DIETARY REQUIREMENTS AND COUNSELING FOR CARDIOVASCULAR DISEASES

A growing body of scientific research is beginning to provide important clues about how diet choices affect health. In some areas, the relationship between specific foods or dietary supplements and particular health outcomes is fairly clear; in other cases, more research is needed.

Although the vast majority of research studies have focused on individual nutrients and foods, it is well recognised that multiple dietary factors influence the risk of developing cardiovascular disease (CVD).

Diet and lifestyle goals for cardiovascular disease risk reduction:

- Consume an overall healthy diet.
- Aim for a healthy body weight. Obesity is an independent risk factor for CVD.
- A healthy body weight is currently defined as a body mass index (BMI) of 18.5 to 24.9 kg/m^2. Overweight is a BMI between 25 and 29.9 kg/m^2, and obesity is a BMI greater than 30 kg/m^2.

- Aim for recommended levels of low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides.
- Aim for a normal blood pressure.
- A normal blood pressure is a systolic BP less than 120 mm Hg and a diastolic BP less than 80 mm Hg. BP is a strong risk factor for cardiovascular- renal disease.
- Aim for a normal blood glucose level.
- A normal fasting glucose level is 5-7 mmol/l, whereas diabetes is defined by a fasting glucose level of greater than 7 mmol/l. Hyperglycaemia and the often associated insulin resistance are related to numerous cardiovascular complications, including coronary heart disease, stroke, peripheral vascular disease, cardiomyopathy, and heart failure.
- Be physically active.
- Avoid use of and exposure to tobacco products.
- Balance energy intake and physical activity to achieve or maintain a healthy body weight.
- Consume a diet rich in vegetables and fruit.

Fruits and Vegetables

A number of studies have demonstrated the health benefits of eating fruits and vegetables. These foods decrease the risk of cardiovascular diseases, including coronary heart disease (CHD) and stroke; intake of up to six servings a day appears to provide the most benefit. Cruciferous vegetables (broccoli, cabbage, cauliflower, brussels sprouts), green leafy vegetables, citrus fruits, and vitamin C-rich fruit and vegetables may lower the risk of cardiovascular disease to the greatest extent.

Fibre

A high fibre intake is associated with a 40-50% reduction in the risk of coronary heart disease and stroke compared with low intake. Cereal fibres in particular are most strongly associated with a decreased risk of heart attack. Eating fibre also protects against type 2 diabetes, and eating soluble fibre (such as that found in vegetables, fruits, and especially legumes) may help people who already have diabetes to control their blood sugar.

The recommended amount of dietary fibre is 20-35g a day. Many breakfast cereals are excellent sources of dietary fibre. By reading the product information panel on the side of the package, it is possible to determine the number of grams of fibre in a serving.

Fish

Eat fish, especially oily fish, at least twice a week. It is rich in the very long-chain omega-3 polyunsaturated fatty acids EPA and DHA. The consumption of

two servings a week of fish high in EPA and DHA is associated with a reduced risk of both sudden death and death from CHD in adults.

Fat

Limit your intake of saturated and trans fat and cholesterol. Blood cholesterol levels have been clearly linked to an increased risk of coronary heart disease. Eating foods lower in certain types of fat, in addition to cutting back on foods that contain cholesterol, can lower cholesterol levels and reduce the risk of coronary heart disease.

The type of fat consumed appears to be more important than the amount of total fat. In particular, saturated fats and trans fats must be avoided. Trans fats are those that are solid at room temperature, and are found in many margarines and in other fats labelled "partially hydrogenated". Another major source is oils that are maintained at high temperature for a long period, such as those in fast food restaurants. Saturated fats come mainly from animal products, such as cheese, butter, and red meat.

When considering a low-fat diet, it is important not to simply replace fat with carbohydrates, a common practice of low-fat food manufacturers. Increases in carbohydrate intake may lower levels of high density lipoprotein (HDL) cholesterol (good cholesterol), and actually increases the risk of coronary heart disease.

Limit your intake of saturated fat to less than 7% of energy, trans fat to less than 1% and cholesterol to less than 300mg a day. These goals can be achieved by:

- Choosing lean meats and vegetable alternatives;
- Selecting fat-free (skim), 1%-fat, and low-fat dairy products; and
- Minimising intake of partially hydrogenated fats.

Sugar

Minimise your intake of beverages and foods with added sugar.

Salt

Choose and prepare foods with little or no salt. An achievable recommendation for sodium (salt) is 2-3g a day.

Alcohol

If you consume alcohol, do so in moderation. Moderate alcohol intake is associated with reduced heart disease, including a reduced risk of death from cardiovascular causes. However, it is not clear what amount of alcohol is best.

There are some risks associated with alcohol use, including breast cancer in women; cancers of the mouth, oesophagus, throat, larynx, and liver; other illnesses

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such as cirrhosis and alcoholism; and injuries and other trauma-related problems, particularly in men.

Dietary guidelines recommend alcohol intake in moderation, if at all. This means no more than one drink a day for women and up to two drinks a day for men. Drinking is discouraged for those under 40 years who are at low risk of cardiovascular disease because the risks are likely to outweigh the benefits in this group.

Fluids

Watch your fluid intake. For severe heart failure you may need to limit the fluids (liquids) you drink throughout the day. If your doctor advises you to limit your fluid intake, you will need to watch how much fluid is in the foods that you consume (water, tea, coffee, soup, jelly, fruit juice, etc). Your doctor will tell you how much fluid you are allowed. Practical hints to control fluid intake:

- Divide fluid allowance evenly throughout the day.
- Avoid drinking sugary fluids.
- Take medication with a meal (unless instructed not to). Some tablets require little or no fluid to swallow if taken with food.
- Rinse your mouth with water and gargle if necessary, but do not swallow.
- Stimulate the production of saliva by sucking a lemon wedge or sweets like sherbet or chewing gum.
- Cool off by wiping your face, neck and underarms with a wet towel.

Weight

Weigh yourself on the same scale each morning, after urinating and before eating. Write down your weight every day in a diary. Bring this with you to your doctor's visit. Call your doctor sooner if you gain 1kg in one day or 2.5kg in one week. Remember, one litre of fluid weighs one kilogram.

Dietary Factors with Unproven or Uncertain Effects on CVD Risk

Folate

Folate is a type of B vitamin that is important in the production of red blood cells. Low levels of folate in pregnant women have been linked to a group of birth defects called neural tube defects. Vitamins containing folate and breakfast cereals fortified with folate are recommended as the best ways to ensure adequate folate intake.

However, supplements containing folate (called folic acid) are no longer recommended to reduce the risk of colon cancer or heart disease.

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Antioxidants

The antioxidant vitamins include vitamins A, C, E, and beta carotene. Many other foods, especially fruits and vegetables, also have antioxidant properties. A number of studies have examined the theory that antioxidants prevent cancer and cardiovascular disease by helping the body dispose of substances called "toxic free radicals". The results of these studies are mixed.

Studies have not clearly shown that antioxidant vitamins prevent cancer, and some studies show they may actually cause harm.

No recommendations can yet be made regarding the use of vitamin C to prevent coronary heart disease (CHD). Vitamin E supplements, either alone or in combination with other antioxidant vitamins, are of no benefit in the prevention of CHD. Studies have also failed to show that supplements of vitamins E and C decrease the risk of stroke.

Soy Protein

Evidence of a direct cardiovascular health benefit from consuming soy protein products instead of dairy or other proteins or of isoflavone supplements is minimal. No meaningful benefit of soy consumption is evident with regard to HDL cholesterol, triglycerides, or lipoprotein *(a)*.

Consumption of foods rich in soy protein may indirectly reduce CVD risk if they replace animal and dairy products that contain saturated fat and cholesterol.

Phytochemicals

Flavonoids and sulphur-containing compounds are classes of compounds found in fruits and vegetables that may be important in reducing the risk of atherosclerosis.

A diet consistent with the AHA recommendations will provide sufficient amounts of macroand micronutrients, as well as associated bioactive compounds, until further research.

Practical Dietary Guidelines to Prevent and Manage Heart Disease

Eat More

- Fruit and vegetables: five portions a day – apples, pears, bananas, oranges, pumpkin, spinach, tomatoes, cabbage, carrots, beetroot, green beans
- Wholegrain starches and high-fibre foods – oats, maltabella, All Bran, samp and beans, brown rice, brown or wholewheat bread, lentils, dried beans, split peas, etc
- Meat and chicken (no fat, no skin) – grilled, roast, boiled, steamed
- Fish – grilled or steamed, especially sardines and pilchards (in tomato sauce)

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- Dairy – low-fat milk, yoghurt, cheese
- In moderation: monounsaturated fats and oils— olive oil, canola oil (for stir-fry, salad dressings, etc.), nuts, peanut butter
- In moderation: poly-unsaturated fats and oils— sunflower oil and soft tub margarine
- Herbs and spices instead of salt to flavour food
- Drink more water – at least one or two litres a day

Eat Less

- Fried food – deep-fried potato chips, chicken, pizza, hamburgers, pies
- Processed food – sausages, russians, polony, viennas, bacon
- Salted snacks – potato crisps, salted peanuts, savoury biscuits, biltong
- Soya sauce, Worcester sauce, stock cubes, packet soup, Aromat
- Don't add extra salt to food after food has been cooked
- Food with added sugar– cakes, biscuits, sweets, chocolate
- Drink less cooldrink (sweetened)
- Drink less alcohol

Eat Less Fat

- Keep overall fat intake low (1-2 teaspoons a day of margarine or olive or canola oil)
- Avoid animal fats (also avoid coconut and palm kernel oil)
- Keep intake of foods high in cholesterol minimal, *i.e.*, use a maximum of 2-3 eggs a week and have shellfish and organ meats no more than once a month
- Use small quantities of monounsaturated fats daily (avocado, olive or canola oil, olives, small quantities of nuts – not cashews or brazils)
- Use essential fatty acids (like salmon omega-3: 1 000mg a day)
- Avoid trans fatty acids, often found in "hydrogenated" or "partially hydrogenated" oils (like hard brick margarines) and in commercial bakery products (to enhance the stability and shelflife of these products)

To limit saturated fats and trans fat and cholesterol (bad fat) altogether

- Use skim milk, preferably (or mix it with 2% fat milk)
- Use low-fat cheeses and fatfree cottage cheese
- Use fat-free yoghurt
- Use soft, tub margarines (Floro Lite, Ole, canola)
- Avoid tropical oils (coconut and palm kernel oil, used in coffee creamers and in commercially baked cakes and pastries) – these are extremely high in saturated fats

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- Avoid products whose labels read "hydrogenated" (hard, brick margarines)
- Avoid fatty red meat – remove all visible fat before cooking
- Use red meat no more than three times a week, and small portions (about 60—90g cooked)
- Grill meat, fish and poultry – at least to the point where it is "medium done"
- Avoid frying foods
- Avoid processed meats (viennas, sausages, bacon etc.)
- Eat plenty of fish (giving you the good type of fatty acids)

Eat More Fibre

Some good sources of soluble fibre:

- Oat bran
- Bokomo, NNB, Woolworths oats porridge
- High fibre bran cereal
- High fibre breads, *e.g.*, heavy rye bread, seed loaf, "health" bread
- High fibre grains, such as brown rice, pearl barley, durum wheat or wholewheat pasta
- Legumes: beans, lentils and split peas
- Potato with the skin on, sweet potato
- Fresh fruit and vegetables

Extra Notes on Legumes, Including Beans and Lentils

Legumes include beans of every variety (green beans, baked beans, kidney beans, pinto beans, butter beans, sugar beans); also chickpeas, lentils and split peas.

Legumes are:

- Rich in protein, and are therefore ideal in vegetarian dishes
- Rich in fibre
- Very low in fat
- Contain some B vitamins, copper and zinc
- Cheap

legumes's health advantages:

- Due to their high fibre content, legumes help prevent or reduce colon-disorder symptoms such as irritable bowel syndrome and constipation
- Legumes also help protect against heart disease

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- Legumes help lower fasting levels of blood glucose
- Legumes added to a meal help sustain energy levels after that meal is eaten
- Legumes leave one feeling fuller and more satisfied after a meal (great in weight reducing diets)
- Legumes help stimulate the immune system in the colon

STUDENT ACTIVITY

1. Point out the causes and risk factors of Myocardial infarction.

2. Discuss the complications of heart failure.

3. Discuss the causes of chronic heart failure.

4.7 SUMMARY

- Cardiovascular diseases refers to the class of diseases that involve the heart or blood vessels (arteries and veins). While the term technically refers to any disease that affects the cardiovascular system, it is usually used to refer to those related to atherosclerosis (arterial disease).
- Hypertension is a chronic medical condition in which the blood pressure is elevated. It is also referred to as high blood pressure or shortened to HT, HTN or HPN. The word "hypertension", by itself, normally refers to systemic, arterial hypertension.
- The degree to which hypertension can be prevented depends on a number of features including: current blood pressure level, changes in end/target organs (retina, kidney, heart – among others), risk factors for cardiovascular diseases and the age at presentation.
- Atherosclerosis (also known as Arteriosclerotic Vascular Disease or ASVD) is the condition in which an artery wall thickens as the result of a build-up of fatty materials such as cholesterol.
- Myocardial infarction (MI) commonly known as a heart attack, is the interruption of blood supply to part of the heart, causing some heart cells to die.
- Cardiac failure is a condition in which a problem with the structure or function of the heart impairs its ability to supply sufficient blood flow to meet the body's needs.
- Folate is a type of B vitamin that is important in the production of red blood cells.

4.8 GLOSSARY

- **Cardiovascular diseases:** The class of diseases that involve the heart or blood vessels (arteries and veins).
- **Hypertension:** A chronic medical condition in which the blood pressure is elevated.
- **Atherosclerosis:** It is the condition in which an artery wall thickens as the result of a build-up of fatty materials such as cholesterol.
- **Myocardial infarction:** It is the interruption of blood supply to part of the heart, causing some heart cells to die.
- **Cardiac failure:** A condition in which a problem with the structure or function of the heart impairs its ability to supply sufficient blood flow to meet the body's needs.

4.9 REVIEW QUESTIONS

1. What are the fundamental causes of hypertension?
2. How is hypertension prevented?
3. What are the symptoms of Atherosclerosis?
4. Focus on the dietary risk factors of Atherosclerosis.
5. What are the complications of Myocardial infarction? How is it prevented?
6. Discuss signs and symptoms of Cardiac failure.
7. Prepare a diet chart suited for a person suffering from cardiovascular diseases.

4.10 FURTHER READINGS

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UNIT—V

RENAL DISORDERS AND OTHER DISEASES

OBJECTIVES

After going through this unit, students will be able to:

- describe the Renal disorders such as Nephritis, Nephrotic syndome and Renal calculi;
- state the diagnosis, role of diet and diet counseling for renal disorders;
- discuss the causes, sign and symptoms of HIV/AIDS and Cancer deseases;
- explain the dietary requirements and counseling for HIV/AIDS and Cancer.

STRUCTURE

5.1 INTRODUCTION

The kidneys are paired organs, which have the production of urine as their primary function. Kidneys are seen in many types of animals, including vertebrates and some invertebrates. They are part of the urinary system, but

have several secondary functions concerned with homeostatic functions. These include the regulation of electrolytes, acid-base balance, and blood pressure. In producing urine, the kidneys excrete wastes such as urea and ammonium; the kidneys also are responsible for the reabsorption of glucose and amino acids. Finally, the kidneys are important in the production of hormones including vitamin D, renin and erythropoietin.

Located behind the abdominal cavity in the retroperitoneum, the kidneys receive blood from the paired renal arteries, and drain into the paired renal veins. Each kidney excretes urine into a ureter, itself a paired structure that empties into the urinary bladder.

Renal physiology is the study of kidney function, while nephrology is the medical specialty concerned with diseases of the kidney. Diseases of the kidney are diverse, but individuals with kidney disease frequently display characteristic clinical features. Common clinical presentations include the nephritic and nephrotic syndromes, acute kidney failure, chronic kidney disease, urinary tract infection, nephrolithiasis, and urinary tract obstruction.

The kidneys receive blood from the renal arteries, left and right, which branch directly from the abdominal aorta. Despite their relatively small size, the kidneys receive approximately 20% of the cardiac output.

Each renal artery branches into segmental arteries, dividing further into interlobar arteries which penetrate the renal capsule and extend through the renal columns between the renal pyramids. The interlobar arteries then supply blood to the arcuate arteries that run through the boundary of the cortex and the medulla. Each arcuate artery supplies several interlobular arteries that feed into the afferent arterioles that supply the glomeruli.

After filtration occurs the blood moves through a small network of venules that converge into interlobular veins. As with the arteriole distribution the veins follow the same pattern, the interlobular provide blood to the arcuate veins then back to the interlobar veins which come to form the renal vein exiting the kidney for transfusion for blood.

Generally, humans can live normally with just one kidney, as one has more functioning renal tissue than is needed to survive. Only when the amount of functioning kidney tissue is greatly diminished will chronic kidney disease develop.

Renal replacement therapy, in the form of dialysis or kidney transplantation, is indicated when the glomerular filtration rate has fallen very low or if the renal dysfunction leads to severe symptoms.

5.2 NEPHROLOGY

Nephrology (from Greek nephros, "kidney", combined with the suffix -logy, "the study of") is a branch of internal medicine and pediatrics dealing with the study of the function and diseases of the kidney.

Nephrology concerns itself with the diagnosis and treatment of kidney diseases, including electrolyte disturbances and hypertension, and the care of those requiring renal replacement therapy, including dialysis and renal transplant patients. Many diseases affecting the kidney are systemic disorders not limited to the organ itself, and may require special treatment. Examples include acquired conditions such as systemic vasculitides (*e.g.*, ANCA vasculitis) and autoimmune diseases (*e.g.*, lupus), as well as congenital or genetic conditions such as polycystic kidney disease.

Patients are referred to nephrology specialists for various reasons, such as:

- Acute renal failure, a sudden loss of renal function
- Chronic kidney disease, declining renal function, usually with an inexorable rise in creatinine.
- Hematuria, blood loss in the urine
- Proteinuria, the loss of protein especially albumin in the urine
- Kidney stones, usually only recurrent stone formers.
- Chronic or recurrent urinary tract infections
- Hypertension that has failed to respond to multiple forms of anti-hypertensive medication or could have a secondary cause
- Electrolyte disorders or acid/base imbalance

Urologists are surgical specialists of the urinary tract (see urology). They are involved in renal diseases that might be amenable to surgery:

Diseases of the Bladder and prostate such as malignancy, stones, or obstruction of the urinary tract.

Diagnosis

As with the rest of medicine, important clues as to the cause of any symptom are gained in the history and physical examination.

Laboratory tests are almost always aimed at: urea, creatinine, electrolytes, and urinalysis, which is frequently the key test in suggesting a diagnosis.

More specialized tests can be ordered to discover or link certain systemic diseases to kidney failure such as hepatitis b or hepatitis c, lupus serologies, paraproteinemias such as amyloidosis or multiple myeloma or various other systemic diseases that lead to kidney failure. Collection of a 24-hour sample of

urine can give valuable information on the filtering capacity of the kidney and the amount of protein loss in some forms of kidney disease. However, 24-hour urine samples have recently, in the setting of chronic renal disease, been replaced by spot urine ratio of protein and creatinine.

Other tests often performed by nephrologists are:

- Renal biopsy, to obtain a tissue diagnosis of a disorder when the exact nature or stage remains uncertain;
- Ultrasound scanning of the urinary tract and occasionally examining the renal blood vessels;
- CT scanning when mass lesions are suspected or to help diagnosis nephrolithiasis;
- Scintigraphy (nuclear medicine) for accurate measurement of renal function (rarely done), and MAG3 scans for diagnosis of renal artery disease or 'split function' of each kidney;
- Angiography or Magnetic resonance imaging angiography when the blood vessels might be affected.

Therapy

Many kidney diseases are treated with medication, such as steroids, DMARDs (disease-modifying antirheumatic drugs), antihypertensives (many kidney diseases feature hypertension). Often erythropoietin and vitamin D treatment is required to replace these two hormones, the production of which stagnates in chronic kidney disease.

When chronic kidney disease progresses to stage five, dialysis or transplant is required. Please refer to the main articles dialysis and renal transplant for a comprehensive account of these treatments.

Sub-specialties within nephrology include interventional nephrologists who focus on access placement and maintenance, a dialytician who focus upon ordering dialysis for patients, and transplant nephrologists who focus on the acute or sub-acute monitoring of immunosuppression in the transplant patient.

If patients proceed to transplant, nephrologists will continue to follow patients to monitor the immunosuppressive regimen and watch for the infection that can occur post.

5.3 NEPHROTIC SYNDROME

Nephrotic syndrome is a nonspecific disorder in which the kidneys are damaged, causing them to leak large amounts of protein (proteinuria at least 3.5 grams per day per 1.73m^2 body surface area) from the blood into the urine.

Kidneys affected by nephrotic syndrome have small pores in the podocytes, large enough to permit proteinuria (and subsequently hypoalbuminemia, because some of the protein albumin has gone from the blood to the urine) but not large enough to allow cells through (hence no hematuria). By contrast, in nephritic syndrome, RBCs pass through the pores, causing hematuria.

It is characterized by proteinuria (>3.5g/day), hypoalbuminemia, hyperlipidemia and edema. A few other characteristics are :

- The most common sign is excess fluid in the body. This may take several forms:
  - Puffiness around the eyes, characteristically in the morning.
  - Edema over the legs which is pitting (*i.e.*, leaves a little pit when the fluid is pressed out, which resolves over a few seconds).
  - Fluid in the pleural cavity causing pleural effusion. More commonly associated with excess fluid is pulmonary edema.
  - Fluid in the peritoneal cavity causing ascites.
- Hypertension (rarely)
- Some patients may notice foamy urine, due to a lowering of the surface tension by the severe proteinuria. Actual urinary complaints such as hematuria or oliguria are uncommon, and are seen commonly in nephritic syndrome.
- May have features of the underlying cause, such as the rash associated with Systemic Lupus Erythematosus, or the neuropathy associated with diabetes.
- Examination should also exclude other causes of gross edema—especially the cardiovascular and hepatic system.

INVESTIGATIONS

The following are baseline, essential investigations :

- Urine sample shows proteinuria (>3.5g per 1.73 m^2 per 24 hour). It is also examined for urinary casts; which is more a feature of active nephritis.
- Comprehensive metabolic panel (CMP) shows Hypoalbuminemia: albumin level d"2.5g/dL (normal=3.5-5g/dL).
- High levels of cholesterol (hypercholesterolemia), specifically elevated LDL, usually with concomitantly elevated VLDL
- Electrolytes, urea and creatinine (EUCs): to evaluate renal function

Further investigations are indicated if the cause is not clear

- Biopsy of kidney

- Auto-immune markers (ANA, ASOT, C3, cryoglobulins, serum electrophoresis)

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CAUSES

Nephrotic syndrome has many causes and may either be the result of a disease limited to the kidney, called primary nephrotic syndrome, or a condition that affects the kidney and other parts of the body, called secondary nephrotic syndrome.

Primary Causes

Primary causes of nephrotic syndrome are usually described by the histology, *i.e.*, minimal change disease (MCD), focal segmental glomerulosclerosis (FSGS) and membranous nephropathy (MN).

They are considered to be "diagnoses of exclusion", *i.e.*, they are diagnosed only after secondary causes have been excluded.

Secondary Causes

Secondary causes of nephrotic syndrome have the same histologic patterns as the primary causes, though may exhibit some differences suggesting a secondary cause, such as inclusion bodies.

They are usually described by the underlying cause.

Secondary causes by histologic pattern

Membranous nephropathy (MN)

- Hepatitis B
- Sjogren's syndrome
- Systemic lupus erythematosus (SLE)
- Diabetes mellitus
- Sarcoidosis
- Syphilis
- Drugs
- Malignancy (cancer)

Focal segmental glomerulosclerosis (FSGS)

- Hypertensive Nephrosclerosis
- Human immunodeficiency virus (HIV)
- Diabetes mellitus
- Obesity
- Kidney loss

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Minimal change disease (MCD)

- Drugs
- Malignancy, especially Hodgkin's lymphoma

Diagnosis

Diagnosis is based on blood and urine tests and sometimes imaging of the kidneys, a biopsy of the kidneys, or both.

Differential Diagnosis of Gross Edema

When someone presents with generalized edema, the following causes should be excluded:

1. *Heart failure:* The patient is older, with a history of heart disease. Jugular venous pressure is elevated on examination, might hear heart murmurs. An echocardiogram is the gold standard investigation.
2. *Liver failure: History suggestive of hepatitis/cirrhosis:* alcoholic, IV drug user, some hereditary causes.

   Stigmata of liver disease are seen: jaundice (yellow skin and eyes), dilated veins over umbilicus (caput medusae), scratch marks (due to widespread itching, known as "pruritus"), enlarged spleen, spider angiomata, encephalopathy, bruising, nodular liver.
3. *Acute fluid overload in someone with kidney failure:* These people are known to have kidney failure, and have either drunk too much or missed their dialysis.
4. *Metastatic cancer:* When cancer seeds the lungs or abdomen it causes effusions and fluid accumulation due to obstruction of lymphatics and veins as well as serous exudation.

Treatment

Treatment includes:

General measures (supportive)

- Monitoring and maintaining euvolemia (the correct amount of fluid in the body):
  - monitoring urine output, BP regularly
  - fluid restrict to 1L
  - diuretics (IV furosemide)
- Monitoring kidney function:
  - do EUCs daily and calculating GFR

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- Prevent and treat any complications
- Albumin infusions are generally not used because their effect lasts only transiently.

Prophylactic anticoagulation may be appropriate in some circumstances.

Specific treatment of underlying cause

- Immunosuppression for the glomerulonephritides (corticosteroids, ciclosporin).
- Standard ISKDC regime for first episode: prednisolone -60 mg/m²/day in 3 divided doses for 4 weeks followed by 40 mg/m²/day in a single dose on every alternate day for 4 weeks.
- Relapses by prednisolone 2 mg/kg/day till urine becomes negative for protein. Then, 1.5 mg/kg/day for 4 weeks.
- Frequent relapses treated by: cyclophosphamide or nitrogen mustard or ciclosporin or levamisole.
- Achieving stricter blood glucose control if diabetic.
- *Blood pressure control* : ACE inhibitors are the drug of choice. Independent of their blood pressure lowering effect, they have been shown to decrease protein loss.

DIETARY RECOMMENDATIONS

Reduce sodium intake to 1000-2000 milligrams daily. Foods high in sodium include salt used in cooking and at the table, seasoning blends (garlic salt, Adobo, season salt, etc.) canned soups, canned vegetables containing salt, luncheon meats including turkey, ham, bologna, and salami, prepared foods, fast foods, soy sauce, ketchup, and salad dressings. On food labels, compare milligrams of sodium to calories per serving. Sodium should be less than or equal to calories per serving.

Eat a moderate amount of high protein animal food: 3–5 oz per meal (preferably lean cuts of meat, fish, and poultry)

Avoid saturated fats such as butter, cheese, fried foods, fatty cuts of red meat, egg yolks, and poultry skin. Increase unsaturated fat intake, including olive oil, canola oil, peanut butter, avocadoes, fish and nuts. Eat low-fat desserts.

Increase intake of fruits and vegetables. There is no potassium or phosphorus restriction necessary.

Monitor fluid intake, which includes all fluids and foods that are liquid at room temperature. Fluid management in nephrotic syndrome is tenuous, especially during an acute flare.

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COMPLICATIONS

- *Venous thrombosis:* due to leak of anti-thrombin 3, which helps prevent thrombosis. This often occurs in the renal veins. Treatment is with oral anticoagulants (not heparin as heparin acts via anti-thrombin 3 which is lost in the proteinuria so it will be ineffective.)
- *Infection:* due to leakage of immunoglobulins, encapsulated bacteria such as Haemophilus influenzae and Streptococcus pneumonia can cause infection.
- *Acute renal failure is due to hypovolemia:* Despite the excess of fluid in the tissues, there is less fluid in the vasculature. Decreased blood flow to the kidneys causes them to shutdown. Thus it is a tricky task to get rid of excess fluid in the body while maintaining circulatory euvolemia.
- *Pulmonary edema:* again due to fluid leak, sometimes it leaks into lungs causing hypoxia and dyspnoea.
- *Growth retardation:* does not occur in MCNS.It occurs in cases of relapses or resistance to therapy. Causes of growth retardation are protein deficiency from the loss of protein in urine, anorexia (reduced protein intake), and steroid therapy (catabolism).
- *Vitamin D deficiency can occur:* Thyroxine is reduced due to decreased thyroid binding globulin.
- *Microcytic hypochromic anaemia is typical.* It is iron-therapy resistant.
- Hypocalcemia can occur as a result of Nephrotic Syndrome. It may be significant enough to cause Tetany.

5.4 NEPHRITIS

Nephritis is inflammation of the kidney. The word comes from the Greek nephro- meaning "of the kidney" and it is meaning "inflammation". Nephritis is often caused by infections, toxins, and auto-immune diseases.

SUBTYPES

- glomerulonephritis is inflammation of the glomeruli. (Often when the term "nephritis" is used without qualification, this is the condition meant.)
- interstitial nephritis or tubulo-interstitial nephritis is inflammation of the spaces between renal tubules.
- pyelonephritis is when a urinary tract infection has reached the pyelum (pelvis) of the kidney.
- Lupus nephritis is an inflammation of the kidney caused by systemic lupus erythematosus (SLE), a disease of the immune system.

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Nephritis is the most common cause of glomerular injury. It is a disturbance of the glomerular structure with inflammatory cell proliferation. This can lead to: reduced glomerular blood flow leading to reduced urine output (oliguria) and retention of waste products (uremia). As a result, there can also be leakage of red blood cells from damaged glomerulus (hematuria). Low renal blood flow activates the renin-angiotensin-aldosterone system (RAAS), which therefore causes fluid retention and mild hypertension.

Kidney diseases that involve structures in the kidney outside the glomerulus are broadly referred to as tubulointerstitial. These diseases generally involve tubules and/or the interstitium of the kidney and spare the glomeruli. Although primary glomerular diseases are often associated with prominent tubulointerstitial changes, the clinical presentation is dominated by the consequences of glomerular injury; hence, they are not considered in this article.

Tubulointerstitial diseases of the kidney encompass diverse etiologies and pathophysiologic processes, and the patient can present with acute or chronic conditions.

Many forms of tubulointerstitial injury involve exposure to drugs or other nephrotoxic agents such as heavy metals and, rarely, infection. By far the most common form of tubulointerstitial inflammation is immunologic.

Nephritis is inflammation of the kidneys. It may be caused by a bacterial infection of the kidneys (pyelonephritis) or exposure to a toxin. However, it more commonly develops from an abnormal immune reaction, which can occur in two ways:

1. An antibody can attack either the kidney itself or a substance that stimulates an immune reaction (antigen) attached to kidney cells, or
2. An antigen and antibody can combine somewhere else in the body, forming an immune complex, and then attach to cells in the kidney.

Some types of nephritis involve infiltration of kidney tissues by white blood cells and deposits of antibodies. In other types of nephritis, inflammation may consist of tissue swelling or scarring without white blood cells or antibodies. Nephritis can occur anywhere in the kidneys.

Symptoms of Nephritis

Less commonly, nephritis involves the tubules and the tissues that surround them (tubulointerstitial tissues). Such inflammation is called tubulointerstitial nephritis. A kidney tubule is a microscopic tube that carries fluid and substances filtered from the blood in the glomerulus to the duct that drains urine into the pelvis of the kidney. Tubulointerstitial tissues surround each of the tubules and separate one tubule from another.

When inflammation damages the tubules and the tubulointerstitial tissues, the kidneys may become unable to concentrate urine, eliminate (excrete) metabolic waste products from the body, or balance the excretion of sodium and other electrolytes, such as potassium. When the tubules and tubulointerstitial tissues are damaged, kidney failure often develops.

Prognosis of Nephritis

Prognosis for most cases of glomerulonephritis is generally good. Ninety percent of children recover without complications. With proper medical treatment, symptoms usually subside within a few weeks, or at the most, a few months.

Pyelonephritis in the acute form offers a good prognosis if diagnosed and treated early. Follow-up urinalysis studies will determine if the patient remains bacteria-free. If the infection is not cured or continues to recur, it can lead to serious complications such as bacteremia (bacterial invasion of the bloodstream), hypertension, chronic pyelonephritis and even permanent kidney damage.

5.5 RENAL CALCULI

Renal calculi are small chunks of crystallized material which become trapped in the kidneys, bladder, or urinary tract. A renal calculus can be extremely painful, and the patient may not be able to pass it without assistance. A variety of treatments are available to deal with renal calculi, also known as kidney stones, and analgesics are also available to help people cope with the pain involved.

Around one in 10 people experiences a kidney stone at some point during life. Several things appear to increase the risk of developing renal calculi, including dehydration, diet, and certain medical conditions, like gout, diabetes, high blood pressure, and irritable bowel disease. In all cases, the calculus is formed when minerals which would normally naturally precipitate out conglomerate to form a crystallized mass which can vary in size.

A small renal calculus may pass relatively successfully, sometimes with the patient only becoming aware of the issue as the stone moves down the ureter, into the bladder, and out the urethra. In other instances, such as the large staghorn stones which can get quite big, the kidney stone may cause extreme pain, but it will not move on its own. Big renal calculi need to be removed with medical assistance, whether the assistance take the form of a stent inserted to allow the stone to drop out naturally, or surgery to take the calculus out by hand.

Humans have been dealing with renal calculi for a very long time, and lithotomy, the removal of renal calculi, is one of the oldest known surgical procedures. The pain would have been considerable in eras before anesthesia, and complicated by an imperfect understanding of internal anatomy, but often

the pain from the calculus itself was so severe that patients were willing to take the risk. With the 20th century came the development of a number of painless and low-pain techniques, including things like extracorporeal shock wave lithotripsy, in which the stones are broken up with ultrasound so that they will be passed by the body.

People usually know when a renal calculus is present, because it causes a distinctive colicky pain and cramping, especially if it moves out of the kidney and into the urinary tract. Difficulty urinating, blood in the urine, and extreme pain in the lower abdomen are common symptoms. The stone can be diagnosed by a doctor with the assistance of medical imaging, and the doctor can make treatment recommendations based on the size and position of the calculus.

Renal calculi (from Latin ren, renes, "kidney" and calculi, "pebbles") are solid concretions or calculi (crystal aggregations) formed in the kidneys from dissolved urinary minerals. Nephrolithiasis refers to the condition of having kidney stones. Urolithiasis refers to the condition of having calculi in the urinary tract (which also includes the kidneys), which may form or pass into the urinary bladder. Ureterolithiasis is the condition of having a calculus in the ureter, the tube connecting the kidneys and the bladder. The term bladder stones usually applies to urolithiasis of the bladder in non-human animals such as dogs and cats.

Kidney stones typically leave the body by passage in the urine stream, and many stones are formed and passed without causing symptoms. If stones grow to sufficient size before passage—on the order of at least 2-3 millimeters—they can cause obstruction of the ureter. The resulting obstruction causes dilation or stretching of the upper ureter and renal pelvis (the part of the kidney where the urine collects before entering the ureter) as well as muscle spasm of the ureter, trying to move the stone. This leads to pain, most commonly felt in the flank, lower abdomen and groin (a condition called renal colic). Renal colic can be associated with nausea and vomiting. There can be blood in the urine, visible with the naked eye or under the microscope (macroscopic or microscopic hematuria) due to damage to the lining of the urinary tract.

There are several types of kidney stones based on the type of crystals of which they consist. The majority are calcium oxalate stones, followed by calcium phosphate stones. More rarely, struvite stones are produced by urea-splitting bacteria in people with urinary tract infections, and people with certain metabolic abnormalities may produce uric acid stones or cystine stones.

The diagnosis of a kidney stone can be confirmed by radiological studies or ultrasound examination; urine tests and blood tests are also commonly performed. When a stone causes no symptoms, watchful waiting is a valid option. In other

cases, pain control is the first measure, using for example, non-steroidal anti-inflammatory drugs or opioids. Using soundwaves, some stones can be shattered into smaller fragments (this is called extracorporeal shock wave lithotripsy). Sometimes a procedure is required, which can be through a tube into the urethra, bladder and ureter (ureteroscopy), or a keyhole or open surgical approach from the kidney's side. Sometimes, a tube may be left in the ureter (a ureteric stent) to prevent the recurrence of pain. Preventive measures are often advised such as drinking sufficient amounts of water, although the effect of many dietary interventions has not been rigorously studied.

Causes

Kidney stones can be due to underlying metabolic conditions, such as renal tubular acidosis, Dent's disease, hyperparathyroidism and medullary sponge kidney. Many health facilities will screen for such problems in patients with recurrent kidney stones. This is typically done with a 24 hour urine collection that is chemically analyzed for deficiencies and excesses that promote stone formation. Kidney stones are also more common in patients with Crohn's disease.

There has been some evidence that water fluoridation may increase the risk of kidney stone formation. In one study, patients with symptoms of skeletal fluorosis were 4.6 times as likely to develop kidney stones. However, fluoride may also be an inhibitor of urinary stone formation.

A 1998 paper in the Archives of Internal Medicine examined the sources of a widely-held belief in the medical community that vitamin C can cause kidney stones, and found it to be based on several circular references, ultimately attributing the belief to a wider pattern of skepticism regarding efficacy of vitamin supplements. A more recent study suggested a causal relationship may exist, but it was not conclusive.

The American Urological Association has projected that increasing global temperatures will lead to greater future prevalence of kidney stones, notably by expanding the "kidney stone belt" of the southern United States. Astronauts seem to show a higher risk of developing kidney stones during or after long duration space flights.

Calcium Oxalate Stones

The most common type of kidney stone is composed of calcium oxalate crystals, occurring in about 80% of cases, and the factors that promote the precipitation of crystals in the urine are associated with the development of these stones.

Common sense has long held that consumption of too much calcium could promote the development of calcium kidney stones. However, current evidence

suggests that the consumption of low-calcium diets is actually associated with a higher overall risk for the development of kidney stones. This is perhaps related to the role of calcium in binding ingested oxalate in the gastrointestinal tract. As the amount of calcium intake decreases, the amount of oxalate available for absorption into the bloodstream increases; this oxalate is then excreted in greater amounts into the urine by the kidneys. In the urine, oxalate is a very strong promoter of calcium oxalate precipitation, about 15 times stronger than calcium.

Uric Acid (Urate)

Multiple kidney stones composed of uric acid and a small amount of calcium oxalate.

About 5–10% of all stones are formed from uric acid. Uric acid stones form in association with conditions that cause hyperuricosuria with or without high blood serum uric acid levels (hyperuricemia); and with acid/base metabolism disorders where the urine is excessively acidic (low pH) resulting in uric acid precipitation. A diagnosis of uric acid nephrolithiasis is supported if there is a radiolucent stone, a persistent unque urine acidity, and uric acid crystals in fresh urine samples.

Other Types

Other types of kidney stones are composed of struvite (magnesium, ammonium and phosphate); calcium phosphate; and cystine.

The formation of struvite stones is associated with the presence of urea-splitting bacteria, most commonly Proteus mirabilis (but also Klebsiella, Serratia, Providencia species). These organisms are capable of splitting urea into ammonia, decreasing the acidity of the urine and resulting in favourable conditions for the formation of struvite stones. Struvite stones are always associated with urinary tract infections.

The formation of calcium phosphate stones is associated with conditions such as hyperparathyroidism and renal tubular acidosis. Formation of cystine stones is uniquely associated with people suffering from cystinuria, who accumulate cystine in their urine. Cystinuria can be caused by Fanconi's syndrome.

Urolithiasis has also been noted to occur in the setting of therapeutic drug use, with crystals of drug forming within the renal tract in some patients currently being treated with Indinavir, Sulfadiazine or Triamterene.

Symptoms

Symptoms of kidney stones include:

- Colicky pain: "loin to groin". Often described as "the worst pain ever experienced".

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- Hematuria: blood in the urine, due to minor damage to inside wall of kidney, ureter and/or urethra.
- Pyuria: pus in the urine.
- Dysuria: burning on urination when passing stones (rare). More typical of infection.
- Oliguria: reduced urinary volume caused by obstruction of the bladder or urethra by stone, or extremely rarely, simultaneous obstruction of both ureters by a stone.
- Abdominal distension.
- Nausea/vomiting: embryological link with intestine – stimulates the vomiting center.
- Fever and chills.
- Hydronephrosis.
- Postrenal azotemia: when kidney stone blocks ureter.
- Frequency in micturition: Defined as an increase in number of voids per day (>than 5 times), but not polyuria, an increase of total urine output per day (2500 ml).
- Loss of appetite.
- Loss of weight.

Diagnosis

Clinical diagnosis is usually made on the basis of the location and severity of the pain, which is typically colicky in nature (comes and goes in spasmodic waves). Pain in the back occurs when calculi produce an obstruction in the kidney.

Imaging is used to confirm the diagnosis and a number of other tests can be undertaken to help establish both the possible cause and consequences of the stone.

X-rays

The relatively dense calcium renders these stones radio-opaque and they can be detected by a traditional X-ray of the abdomen that includes the Kidneys, Ureters and Bladder – KUB. This may be followed by an IVP (Intravenous Pyelogram; (IntraVenous Urogram (IVU) is the same test by another name)) which requires about 50 ml of a special dye to be injected into the bloodstream that is excreted by the kidneys and by its density helps outline any stone on a repeated X-ray. These can also be detected by a Retrograde pyelogram where similar "dye" is injected directly into the ureteral opening in the bladder by a surgeon, usually a urologist.

About 10% of stones do not have enough calcium to be seen on standard *x-rays* (radiolucent stones).

Computed Tomography

Computed tomography without contrast is considered the gold-standard diagnostic test for the detection of kidney stones. All stones are detectable by CT except very rare stones composed of certain drug residues in the urine. If positive for stones, a single standard x-ray of the abdomen (KUB) is recommended. This gives a clearer idea of the exact size and shape of the stone as well as its surgical orientation. Further, it makes it simple to follow the progress of the stone by doing another x-ray in the future.

Draw back of CT scans include radiation exposure and cost.

Ultrasound

Ultrasound imaging is useful as it gives details about the presence of hydronephrosis (swelling of the kidney—suggesting the stone is blocking the outflow of urine). It can also be used to detect stones during pregnancy when *x-rays* or CT are discouraged. Radiolucent stones may show up on ultrasound however they are also typically seen on CT scans.

Some recommend that US be used as the primary diagnostic technique with CT being reserved for those with negative US result and continued suspicion of a kidney stone. This is due to its lesser cost and lack of radiation exposure.

Other

Other investigations typically carried out include:

- Microscopic study of urine, which may show proteins, red blood cells, bacteria, cellular casts and crystals.
- Culture of a urine sample to exclude urine infection (either as a differential cause of the patient's pain, or secondary to the presence of a stone)
- Blood tests: Full blood count for the presence of a raised white cell count (Neutrophilia) suggestive of infection, a check of renal function and to look for abnormally high blood calcium blood levels (hypercalcaemia).
- 24 hour urine collection to measure total daily urinary volume, magnesium, sodium, uric acid, calcium, citrate, oxalate and phosphate.
- Catching of passed stones at home (usually by urinating through a tea strainer or stonescreen) for later examination and evaluation by a doctor.

Treatment

Temporizing

About 90% of stones 4 mm or less in size usually will pass spontaneously, however 99% of stones larger than 6 mm will require some form of intervention.

There are various measures that can be used to encourage the passage of a stone. These can include increased hydration, medication for treating infection and reducing pain, and diuretics to encourage urine flow and prevent further stone formation. Caution should be exercised in eating certain foods, such as starfruit, with high concentrations of oxalate which may precipitate acute renal failure in patients with chronic renal disease.

In most cases, a smaller stone that is not symptomatic is often given up to four weeks to move or pass before consideration is given to any surgical intervention as it has been found that waiting longer tends to lead to additional complications. Immediate surgery may be required in certain situations such as in people with only one working kidney, intractable pain or in the presence of an infected kidney blocked by a stone which can rapidly cause severe sepsis and toxic shock. Taking a warm bath helps with minor kidney stones pain.

Analgesia

Management of pain from kidney stones varies from country to country and even from physician to physician, but usually requires intravenous administration of opioids in an emergency room setting for acute situations. Similarly, drugs may be reasonably effective orally in an outpatient setting for less severe discomfort where nonsteroidal anti-inflammatories or opioids such as codeine or hydrocodone can be prescribed. Some doctors will give patients with recurring passing of small stones a small supply prescription for hydrocodone to avoid a future visit to the ER when the next episode occurs. Taken at the first sign of pain, hydrocodone can eliminate much of the acute pain, nausea and vomiting which necessitates the hospital visit and still facilitate stone passage, although a follow-up with a physician is still necessary.

Patients who are to be treated non-surgically, may also be started on an alpha adrenergic blocking agent (such as Flomax, Uroxatral, terazosin or doxazosin), which acts to reduce the muscle tone of the ureter and facilitate stone passage. For smaller stones near the bladder, this type of medical treatment can increase the spontaneous stone passage rate by about 30%.

After treatment, the pain may return if the stone moves but re-obstructs in another location. Patients are encouraged to strain their urine so they can collect the stone when it eventually passes and send it for chemical composition analysis which will be used along with a 24 hour urine chemical analysis test to establish preventative options.

Urologic Interventions

Most kidney stones do not require surgery and will pass on their own. Surgery is necessary when the pain is persistent and severe, in renal failure and

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when there is a kidney infection. It may also be advisable if the stone fails to pass or move after 30 days. Finding a significant stone before it passes into the ureter allows physicians to fragment it surgically before it causes any severe problems. In most of these cases, non-invasive extracorporeal shock wave lithotripsy (ESWL) will be used. Otherwise some form of invasive procedure is required: with approaches including ureteroscopic fragmentation (or simple basket extraction if feasible) using laser, ultrasonic or mechanical (pneumatic, shock-wave) forms of energy to fragment the larger stones. Percutaneous nephrolithotomy or rarely open surgery may ultimately be necessary for large or complicated stones or stones which fail other less invasive attempts at treatment.

A single retrospective study in the USA, at the Mayo Clinic, has suggested that lithotripsy may increase subsequent incidence of diabetes and hypertension, but it has not been felt warranted to change clinical practice at the clinic. The study reflects early experience with the original lithotripsy machine which had a very large blast path, much larger than what is used on modern machines. Further study is believed necessary to determine how much risk this treatment actually has using modern machines and treatment regimens.

More common complications related to ESWL are bleeding, pain related to passage of stone fragments, failure to fragment the stone, and the possible requirement for additional or alternative interventions.

Ureteral (double-J) stents

Three-dimensional reconstructed CT scan image of a ureteral stent in the left kidney (indicated by yellow arrow). There is a kidney stone in the pyelum of the lower pole of the kidney (highest red arrow) and one in the ureter beside the stent (lower red arrow).

One modern medical technique uses a ureteral stent (a small tube between the bladder and the inside of the kidney) to provide immediate relief of a blocked kidney. This is especially useful in saving a failing kidney due to swelling and infection from the stone. Ureteral stents vary in length and width but most have the same shape usually called a "double-J" or "double pigtail", because of the curl at both ends. They are designed to allow urine to drain around any stone or obstruction. They can be retained for some length of time as infections recede and as stones are dissolved or fragmented with ESWL or other treatment. The stents will gently dilate or stretch the ureters which can facilitate instrumentation and they will also provide a clear landmark to help surgeons see the stones on x-ray. Most stents can be removed easily during a final office visit. Discomfort levels from stents typically range from minimal associated pain to moderate discomfort. However, it isn't uncommon for patients to experience severe discomfort too, especially upon removal of said stent.

PREVENTION

Preventive strategies include dietary modifications and sometimes also taking drugs with the goal of reducing excretory load on the kidneys:

- Drinking enough water to make 2 to 2.5 liters of urine per day.
- A diet low in protein, nitrogen and sodium intake.
- Restriction of oxalate-rich foods, such as chocolate, nuts, soybeans, rhubarb and spinach, plus maintenance of an adequate intake of dietary calcium. There is equivocal evidence that calcium supplements increase the risk of stone formation, though calcium citrate appears to carry the lowest, if any, risk.
- Taking drugs such as thiazides, potassium citrate, magnesium citrate and allopurinol, depending on the cause of stone formation.
- Some fruit juices, such as orange, blackcurrant, and cranberry, may be useful for lowering the risk factors for specific types of stones. Orange juice may help prevent calcium oxalate stone formation, black currant may help prevent uric acid stones, and cranberry may help with UTI-caused stones.
- Avoidance of cola beverages.
- Avoiding large doses of vitamin C.

For those patients interested in optimizing their kidney stone prevention options, a 24 hour urine test can be a useful diagnostic.

Restricting Oxalate Consumption

Calcium plays a vital role in body chemistry so limiting calcium may be unhealthy. Since calcium in the intestinal tract will bind with available oxalate, thereby preventing its absorption into the blood stream, some nephrologists and urologists recommend chewing calcium tablets during meals containing oxalate foods. However, a more reliable approach is to restrict the intake of food that is high in oxalate. This is only helpful in those patients who are absorbing excess oxalate which is a minority of patients as most oxalate excreted in the urine is actually made by the liver.

Diuretics

Although it has been claimed that the diuretic effects of alcohol can result in dehydration, which is important for kidney stone sufferers to avoid, there are no conclusive data demonstrating any cause and effect regarding kidney stones. However, some have theorized that frequent and binge drinkers create situations that set up dehydration: alcohol consumption, hangovers, and poor sleep and

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stress habits. In this view, it is not the alcohol that creates a kidney stone but it is the alcohol drinker's associated behavior that sets it up.

One of the recognized medical therapies for prevention of stones is thiazides, a class of drugs usually thought of as diuretics. These drugs prevent calcium stones through an effect independent of their diuretic properties: they reduce urinary calcium excretion. Nonetheless, their diuretic property does not preclude their efficacy as stone preventive. Sodium restriction is necessary for clinical effect of thiazides, as sodium excess promotes calcium excretion. Thiazides work best for renal leak hypercalciuria—a condition in which the high urinary calcium levels are from a primary kidney defect. They work well initially for absorptive hypercalciuria—a condition in which high urinary calcium is a result of excess absorption from the GI tract. With this condition they lose effectiveness over time, typically around 2 years, and patients need a period off treatment to regain effectiveness. Thiazides will cause hypokalemia and reduced urinary citrate levels so should be given with supplements for each, usually as a potassium citrate preparation.

Allopurinol

Allopurinol (Zyloprim) is another drug with proven benefits in some calcium kidney stone formers. Allopurinol interferes with the liver's production of uric acid. Hyperuricosuria, too much uric acid in the urine, is a risk factor for calcium stones. Allopurinol reduces calcium stone formation in such patients. The drug is also used in patients with gout or hyperuricemia. However, hyperuricemia is not the critical feature of uric acid stones, which can occur in the presence of hypouricemia. Uric acid stones are more often caused by a combination of high urine uric acid and low urine pH. Even relatively high uric acid excretion will not be associated with uric acid stone formation if the urine pH is alkaline. Therefore prevention of uric acid stones relies on alkalinization of the urine with citrate (in the form of Sholh's solution, sodium bicarbonate, or acetazolamide, a carbonic anhydrase inhibitor).

Allopurinol is reserved for patients in whom alkalinization is difficult. For patients with increased uric acid levels and calcium stones, allopurinol is one of the few treatments that has been shown in double-blinded placebo controlled studies to actually reduce kidney stone recurrences. Dosage is adjusted to maintain a reduced urinary excretion of uric acid. Serumuric acid level at or below 6 mg/dL is often the goal of the drug's use in patients with gout or hyperuricemia.

Decreased Protein Diet

A high protein diet might be partially to blame. Protein from meat and other animal products is broken down into acids, including uric acid. The most

available alkaline base to balance the acid from protein is calcium phosphate (hydroxyapatite) from the bones (buffering). The kidney filters the liberated calcium which may then form insoluble crystals (*i.e.*, stones) in urine with available oxalate (partly from metabolic processes, partly from diet) or phosphate ions, depending on conditions. High protein intake is therefore associated with decreased bone density as well as stones. The acid load is associated with decreased urinary citrate excretion; citrate competes with oxalate for calcium and can thereby prevent stones.

In addition to increased fluid intake, one of the simplest fixes is to moderate animal protein consumption. However, despite epidemiologic data showing that greater protein intake is associated with more stones, randomized controlled trials of protein restriction have not shown reduced stone prevalence. In this regard, it is not just dietary calcium per se that may cause stone formation, but rather the leaching of bone calcium. Some diseases (*e.g.*, distal renal tubular acidosis) which cause a chronically acidic state also decrease urinary citrate levels; since citrates are normally present as potent inhibitors of stone formation, these patients are predisposed to stone formation.

Medications

Potassium citrate is also used in kidney stone prevention. This is available as both a tablet and liquid preparation. The medication increases urinary pH (makes it more alkaline), as well as increases the urinary citrate level, which helps reduce calcium oxalate crystal aggregation. Optimal 24 hour urine levels of citrate are over 320 mg/liter of urine or over 600 mg per day. There are urinary dipsticks that allow patients to monitor and measure urinary pH so patients can optimize their urinary citrate level.

Though caffeine does acutely increase urinary calcium excretion, several independent epidemiologic studies have shown that coffee intake overall is protective against the formation of stones.

Measurements of food oxalate content have been difficult and issues remain about the proportion of oxalate that is bio-available, versus a proportion that is not absorbed by the intestine. Oxalate-rich foods are usually restricted to some degree, particularly in patients with high urinary oxalate levels, but no randomized controlled trial of oxalate restriction has been performed to test that hypotheses.

Calgranulin

Crystallization of calcium oxalate (CaOx) appears to be reduced by molecules in the urine that retard the formation, growth, aggregation, and renal cell adherence of calcium oxalate. By purifying urine using salt precipitation, preparative isoelectric focusing, and sizing chromatography, some researchers

have found that the molecule calgranulin is able to inhibit calcium oxalate crystal growth. Calgranulin is a protein formed in the kidney. Given the large amounts of calcium oxalate in the urine, and considering its potency, calgranulin could become an important contribution to the normal urinary inhibition of crystal growth and aggregation. If so, it will be an important tool in the renal defense against kidney stones.

5.6 ROLE OF DIET AND DIET COUNSELING FOR RENAL DISORDERS

When someone has chronic kidney disease, diet is an important part of his/her treatment plan. the recommended diet may change over time if kidney disease gets worse. A number of tests should be done to "keep tabs" on overall nutritional health. Doctors can also refer to a registered dietitian who will help to plan meals to get the right foods in the right amounts. Some things that are important to diet including:

- getting the right amount of calories and protein
- other important nutrients in your diet
  - sodium
  - phosphorus
  - calcium
  - potassium
  - fluids
  - vitamins and minerals
- staying at a healthy body weight
- handling special diet needs
  - diabetes
  - vegetarian diets
- how your nutritional health is checked
- other resources that can help.

Changing Diet Preferences

As a patient with chronic kidney disease, the person's recommended diet may change over time, depending on how much kidney function he/she has. His/her glomerular filtration rate, or GFR, is the best way to track level of kidney function. Doctors can estimate GFR from the results of a simple blood test for creatinine along with age, sex and body size. If the person's kidney disease progresses, and GFR continues to decrease, the amount of protein, calories and other nutrients in diet will be adjusted to meet your changing needs. If dialysis or a kidney transplant is needed eventually, diet will be based on the treatment

option he or she chooses. Doctors can refer to a registered dietitian who will explain the changes required to make in diet and help to choose the right foods.

Getting the Right Amount of Calories

Getting enough calories is important to overall health and wellbeing. Calories are found in all the foods we eat. They are important because they:

- give body energy
- help stay at a healthy weight
- help body use protein for building muscles and tissues.

Because recommended diet may limit protein, he/she may also be cutting down on an important source of calories. As a result, one may need to get extra calories from other foods. Doctor can refer the person to a registered dietitian who will help to plan the meals to get the right amount of calories each day. The dietitian may recommend using more simple carbohydrates like sugar, jam, jelly, hard candy, honey and syrup. Other good sources of calories come from fats such as soft (tub) margarine, and oils like canola or olive oil, which are low in saturated fat and have no cholesterol. One should talk with his/her dietitian about the best way to get the right amount of calories and keep blood sugar in control if he/she has diabetes.

Maintaining a healthy weight is also important. People who have chronic kidney disease often need to gain weight or remain at their current weight. If one need to lose weight, dietitian will teach how to lose it slowly and carefully without risking health.

Steps to Take

- Ask doctor about a referral to a registered dietitian who specializes in diets for kidney patients.
- Ask dietitian to help to plan meals with the right amount of calories.
- Keep a diary of what to eat each day. Show this to dietitian on a regular basis.
- Ask doctor and dietitian what ideal body weight should be, and weigh each day in the morning.
- If someone is losing too much body weight, ask dietitian how to add extra calories to diet.
- If someone is slowly gaining too much weight, ask dietitian for suggestions on how to safely reduce daily calorie intake and increase activity level.
- If someone gains weight rapidly, speak to doctor. A sudden increase in weight, along with swelling, shortness of breath and a rise in blood pressure may be a sign that he/she have too much fluid in his/her body.

Getting the Right Amount of Protein

Getting the right amount of protein is important to overall health and how well the person feel. Body needs the right amount of protein for:

- building muscles
- repairing tissue
- fighting infections.

Doctor may recommend that the cencerned person follow a diet that has controlled amounts of protein. This may help decrease the amount of wastes in blood and may help kidneys to work longer.

Protein comes from two sources. A person will need to get some protein each day from both of these sources:

- Animal sources, such as eggs, fish, chicken, red meats, milk products and cheese.
- Plant sources, such as vegetables and grains.

Steps to Take

- Ask dietitian how much protein to eat each day.
- Show daily food diary to dietitian, and ask if you are eating the right amount of protein.

Other Important Nutrients in Diet

The dietitian help to plan the meals to get the right amounts of each.

Sodium

Kidney disease, high blood pressure and sodium are often related. Therefore, one may need to limit the amount of sodium in his/her diet. Doctor will let the person know if he/she need to cut back on sodium. If he/she does, the dietitian can teach how to select foods that are lower in sodium. Sodium is a mineral found naturally in foods. It is found in large amounts in table salt and in foods that have added table salt such as:

- seasonings like soy sauce, teriyaki sauce and garlic or onion salt
- most canned foods and some frozen foods
- processed meats like ham, bacon, sausage and cold cuts
- salted snack foods like chips and crackers
- most restaurant and take-out foods
- canned or dehydrated soups (like packaged noodle soup).

Phosphorus

Kidneys may not be able to remove enough phosphorus from blood. This causes the level of phosphorus in blood to become too high. A high blood

phosphorus level may cause skin to itch and the loss of calcium from bones. Bones can become weak and may break easily. Eating fewer foods that are high in phosphorus will help lower the amount of phosphorus in blood. The dietitian will help to choose foods that are lower in phosphorus.

Phosphorus is found in large amounts in the following:

- dairy products such as milk, cheese, pudding, yogurt and ice cream
- dried beans and peas such as kidney beans, split peas and lentils
- nuts and peanut butter
- beverages such as hot chocolate, beer and dark cola drinks.

Calcium

Calcium is a mineral that is important for building strong bones. However, foods that are good sources of calcium are also high in phosphorus. To keep calcium and phosphorus levels in balance and to prevent the loss of calcium from bones, there may need to follow a diet that limits phosphorus-rich foods and take phosphate binders. Doctor may also ask to take calcium supplements and a special prescription form of vitamin D. Take only the supplements or medications recommended by doctor.

Potassium

Potassium is an important mineral in the blood that helps muscles and heart work properly. Too much or too little potassium in the blood can be dangerous. Whether or not one need to change the amount of high-potassium foods in his/her diet depends on stage of kidney disease and whether he/she are taking any medications that change the level of potassium in blood.

Fluid

In general, the person do not need to limit the amount of fluids he/she drinks in the earlier stages of kidney disease. If disease gets worse, doctor will let to know when he/she need to limit fluids and how much fluid is okay for you each day.

Vitamins and Minerals

Vitamins and minerals come from a variety of foods we eat each day. If our diet is limited, we may need to take special vitamins or minerals. Take only the vitamins and minerals doctor recommends. Certain vitamins and minerals may be harmful to people with chronic kidney disease.

Method to Check Nutritional Health

There are several different ways for doctor and dietitian to know if the person is eating the right amount of calories or protein. This section will explain

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these tests and methods. If his/her kidney disease gets worse, and/or his/her nutritional health changes, he/she may need to have these tests more often.

Dietary Interviews and Food Diaries

Dietitian will speak to the person at times about his/her diet. The dietitian may also ask to keep a record of what he/she eat each day. If he/she is not eating the right amount of protein, calories and other nutrients, the dietitian will give ideas about food choices that will help to improve the diet.

Lab Tests for Protein Balance

Serum Albumin

Albumin is a type of protein found in blood. Albumin level will be checked by a blood test. If the level is too low, it may mean the person is not eating enough protein and/or calories. If albumin level continues to be low, the person a greater chance of getting an infection, being hospitalized and generally not feeling well.

nPNA (normalized protein nitrogen appearance)

This is another way to determine if the person are not eating the right amount of protein. The *nPNA* result comes from lab studies that include urine collection and blood work. The *nPNA* helps to check the protein balance in body.

Physical Nutrition Exam

Dietitian may use a method called Subjective Global Assessment (SGA) to check body for signs of nutrition problems. This involves asking the person questions about his/her food intake and looking at the fat and muscle stores in his/her body.

The dietitian will consider:

- changes in weight
- changes in the tissues around face, arms, hands, shoulders and legs
- The food intake
- Activity and energy levels
- problems that might interfere with eating.

STUDENT ACTIVITY

1. Discuss the characteristics of Nephrotic syndrome .

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2. Point out the complications of Renal calculi.

3. Prepare a suitable diet chart for the patient of kidney diseases.

5.7 HIV/AIDS

Acquired immune deficiency syndrome or acquired immunodeficiency syndrome (AIDS) is a disease of the human immune system caused by the human immunodeficiency virus (HIV).

This condition progressively reduces the effectiveness of the immune system and leaves individuals susceptible to opportunistic infections and tumors. HIV is transmitted through direct contact of a mucous membrane or the bloodstream with a bodily fluid containing HIV, such as blood, semen, vaginal fluid, preseminal fluid, and breast milk.

This transmission can involve anal, vaginal or oral sex, blood transfusion, contaminated hypodermic needles, exchange between mother and baby during pregnancy, childbirth, breastfeeding or other exposure to one of the above bodily fluids.

AIDS is now a pandemic. In 2007, it was estimated that 33.2 million people lived with the disease worldwide, and that AIDS killed an estimated 2.1 million people, including 330,000 children. Over three-quarters of these deaths occurred in sub-Saharan Africa, retarding economic growth and destroying human capital.

Genetic research indicates that HIV originated in west-central Africa during the late nineteenth or early twentieth century. AIDS was first recognized by the U.S. Centers for Disease Control and Prevention in 1981 and its cause, HIV, identified in the early 1980s.

Although treatments for AIDS and HIV can slow the course of the disease, there is currently no vaccine or cure. Antiretroviral treatment reduces both the mortality and the morbidity of HIV infection, but these drugs are expensive and routine access to antiretroviral medication is not available in all countries. Due to the difficulty in treating HIV infection, preventing infection is a key aim in controlling the AIDS pandemic, with health organizations promoting safe sex and needle-exchange programmes in attempts to slow the spread of the virus.

Symptoms

A generalized graph of the relationship between HIV copies (viral load) and CD4 counts over the average course of untreated HIV infection; any particular individual's disease course may vary considerably.

The symptoms of AIDS are primarily the result of conditions that do not normally develop in individuals with healthy immune systems. Most of these conditions are infections caused by bacteria, viruses, fungi and parasites that are normally controlled by the elements of the immune system that HIV damages.

Opportunistic infections are common in people with AIDS. HIV affects nearly every organ system.

People with AIDS also have an increased risk of developing various cancers such as Kaposi's sarcoma, cervical cancer and cancers of the immune system known as lymphomas. Additionally, people with AIDS often have systemic symptoms of infection like fevers, sweats (particularly at night), swollen glands, chills, weakness, and weight loss. The specific opportunistic infections that AIDS patients develop depend in part on the prevalence of these infections in the geographic area in which the patient lives.

Pulmonary Infections

Pneumocystis pneumonia (originally known as Pneumocystis carinii pneumonia, and still abbreviated as PCP, which now stands for Pneumocystis pneumonia) is relatively rare in healthy, immunocompetent people, but common among HIV-infected individuals. It is caused by Pneumocystis jirovecii.

Before the advent of effective diagnosis, treatment and routine prophylaxis in Western countries, it was a common immediate cause of death. In developing countries, it is still one of the first indications of AIDS in untested individuals, although it does not generally occur unless the CD4 count is less than 200 cells per μL of blood.

Tuberculosis (TB) is unique among infections associated with HIV because it is transmissible to immunocompetent people via the respiratory route, is easily treatable once identified, may occur in early-stage HIV disease, and is preventable with drug therapy. However, multidrug resistance is a potentially serious problem.

Even though its incidence has declined because of the use of directly observed therapy and other improved practices in Western countries, this is not the case in developing countries where HIV is most prevalent. In early-stage HIV infection (CD4 count >300 cells per μL), TB typically presents as a pulmonary disease. In advanced HIV infection, TB often presents a typically with extrapulmonary (systemic) disease a common feature. Symptoms are usually constitutional and are not localized to one particular site, often affecting bone marrow, bone [illegible] nd gastrointestinal tracts, liver, regional lymph nodes, [illegible] system.

Gastrointestinal Infections

Esophagitis is an inflammation of the lining of the lower end of the esophagus (gullet or swallowing tube leading to the stomach). In HIV infected individuals, this is normally due to fungal (candidiasis)or viral (herpes simplex-1 or cytomegalovirus) infections. In rare cases, it could be due to mycobacteria.

Unexplained chronic diarrhoea in HIV infection is due to many possible causes, including common bacterial (Salmonella, Shigella, Listeria or Campylobacter) and parasitic infections; and uncommon opportunistic infections such as cryptosporidiosis, microsporidiosis, Mycobacterium avium complex (MAC) and viruses, astrovirus, adenovirus, rotavirus and cytomegalovirus, (the latter as a course of colitis).

In some cases, diarrhoea may be a side effect of several drugs used to treat HIV, or it may simply accompany HIV infection, particularly during primary HIV infection. It may also be a side effect of antibiotics used to treat bacterial causes of diarrhoea (common for Clostridium difficile). In the later stages of HIV infection, diarrhoea is thought to be a reflection of changes in the way the intestinal tract absorbs nutrients, and may be an important component of HIV-related wasting.

Neurological and Psychiatric Involvement

HIV infection may lead to a variety of neuropsychiatric sequelae, either by infection of the now susceptible nervous system by organisms, or as a direct consequence of the illness itself.

Toxoplasmosis is a disease caused by the single-celled parasite called Toxoplasma gondii; it usually infects the brain, causing toxoplasma encephalitis, but it can also infect and cause disease in the eyes and lungs. Cryptococcal meningitis is an infection of the meninx (the membrane covering the brain and spinal cord) by the fungus Cryptococcus neoformans. It can cause fevers, headache, fatigue, nausea, and vomiting. Patients may also develop seizures and confusion; left untreated, it can be lethal.

Progressive multifocal leukoencephalopathy (PML) is a demyelinating disease, in which the gradual destruction of the myelin sheath covering the axons of nerve cells impairs the transmission of nerve impulses. It is caused by a virus called JC virus which occurs in 70% of the population in latent form, causing disease only when the immune system has been severely weakened, as is the case for AIDS patients. It progresses rapidly, usually causing death within months of diagnosis.

AIDS dementia complex (ADC) is a metabolic encephalopathy induced by HIV infection and fueled by immune activation of HIV infected brain macrophages and microglia. These cells are productively infected by HIV and secrete neurotoxins of both host and viral origin. Specific neurological impairments are manifested by cognitive, behavioural, and motor abnormalities that occur after years of HIV infection and are associated with low $CD4^+$ T cell levels and high plasma viral loads.

Prevalence is 10–20% in Western countries but only 1–2% of HIV infections in India. This difference is possibly due to the HIV subtype in India. AIDS related mania is sometimes seen in patients with advanced HIV illness; it presents with more irritability and cognitive impairment and less euphoria than a manic episode associated with true bipolar disorder. Unlike the latter condition, it may have a more chronic course. This syndrome is less often seen with the advent of multi-drug therapy.

Tumors and Malignancies

Patients with HIV infection have substantially increased incidence of several cancers. This is primarily due to co-infection with an oncogenic DNA virus, especially Epstein-Barr virus (EBV), Kaposi's sarcoma-associated herpesvirus (KSHV) (also known as human herpesvirus-8 [HHV-8]), and human papillomavirus (HPV).

Kaposi's sarcoma (KS) is the most common tumor in HIV-infected patients. The appearance of this tumor in young homosexual men in 1981 was one of the first signals of the AIDS epidemic. Caused by a gammaherpes virus called Kaposi's sarcoma-associated herpes virus (KSHV), it often appears as purplish nodules on the skin, but can affect other organs, especially the mouth, gastrointestinal tract,

and lungs. High-grade B cell lymphomas such as Burkitt's lymphoma, Burkitt's-like lymphoma, diffuse large B-cell lymphoma (DLBCL), and primary central nervous system lymphoma present more often in HIV-infected patients. These particular cancers often foreshadow a poor prognosis. Epstein-Barr virus (EBV) or KSHV cause many of these lymphomas. In HIV-infected patients, lymphoma often arises in extranodal sites such as the gastrointestinal tract. When they occur in an HIV-infected patient, KS and aggressive B cell lymphomas confer a diagnosis of AIDS.

Invasive cervical cancer in HIV-infected women is also considered AIDS-defining. It is caused by human papillomavirus (HPV).

In addition to the AIDS-defining tumors listed above, HIV-infected patients are at increased risk of certain other tumors, notably Hodgkin's disease, anal and rectal carcinomas, hepatocellular carcinomas, head and neck cancers, and lung cancer. Some of these are causes by viruses, such as Hodgkin's disease (EBV), anal/rectal cancers (HPV), head and neck cancers (HPV), and hepatocellular carcinoma (hepatitis B or C). Other contributing factors include exposure to carcinogens (cigarette smoke for lung cancer), or living for years with subtle immune defects.

Interestingly, the incidence of many common tumors, such as breast cancer or colon cancer, does not increase in HIV-infected patients. In areas where HAART is extensively used to treat AIDS, the incidence of many AIDS-related malignancies has decreased, but at the same time malignant cancers overall have become the most common cause of death of HIV-infected patients. In recent years, an increasing proportion of these deaths have been from non-AIDS-defining cancers.

Other Infections

AIDS patients often develop opportunistic infections that present with non-specific symptoms, especially low-grade fevers and weight loss. These include opportunistic infection with Mycobacterium avium-intracellulare and cytomegalovirus (CMV). CMV can cause colitis, as described above, and CMV retinitis can cause blindness.

Penicilliosis due to Penicillium marneffei is now the third most common opportunistic infection (after extrapulmonary tuberculosis and cryptococcosis) in HIV-positive individuals within the endemic area of Southeast Asia.

An infection that often goes unrecognized in AIDS patients is Parvovirus B19. Its main consequence is anemia, which is difficult to distinguish from the effects of antiretroviral drugs used to treat AIDS itself.

Cause

AIDS is the most severe acceleration of infection with HIV. HIV is a retrovirus that primarily infects vital organs of the human immune system such as $CD4^+$ T

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cells (a subset of T cells), macrophages and dendritic cells. It directly and indirectly destroys $CD4^+$ T cells.

Once HIV has killed so many $CD4^+$ T cells that there are fewer than 200 of these cells per microliter (μL) of blood, cellular immunity is lost. Acute HIV infection progresses over time to clinical latent HIV infection and then to early symptomatic HIV infection and later to AIDS, which is identified either on the basis of the amount of $CD4^+$ T cells remaining in the blood, and/or the presence of certain infections, as noted above.

In the absence of antiretroviral therapy, the median time of progression from HIV infection to AIDS is nine to ten years, and the median survival time after developing AIDS is only 9.2 months. However, the rate of clinical disease progression varies widely between individuals, from two weeks up to 20 years.

Many factors affect the rate of progression. These include factors that influence the body's ability to defend against HIV such as the infected person's general immune function. Older people have weaker immune systems, and therefore have a greater risk of rapid disease progression than younger people.

Poor access to health care and the existence of coexisting infections such as tuberculosis also may predispose people to faster disease progression. The infected person's genetic inheritance plays an important role and some people are resistant to certain strains of HIV. An example of this is people with the homozygous CCR5-Ä32 variation are resistant to infection with certain strains of HIV. HIV is genetically variable and exists as different strains, which cause different rates of clinical disease progression.

Sexual Transmission

Sexual transmission occurs with the contact between sexual secretions of one person with the rectal, genital or oral mucous membranes of another. Unprotected receptive sexual acts are riskier than unprotected insertive sexual acts, and the risk for transmitting HIV through unprotected anal intercourse is greater than the risk from vaginal intercourse or oral sex.

However, oral sex is not entirely safe, as HIV can be transmitted through both insertive and receptive oral sex. Sexual assault greatly increases the risk of HIV transmission as condoms are rarely employed and physical trauma to the vagina or rectum occurs frequently, facilitating the transmission of HIV.

Other sexually transmitted infections (STI) increase the risk of HIV transmission and infection, because they cause the disruption of the normal epithelial barrier by genital ulceration and/or microulceration; and by accumulation of pools of HIV-susceptible or HIV-infected cells (lymphocytes and macrophages) in semen and vaginal secretions. Epidemiological studies from sub-

Saharan Africa, Europe and North America suggest that genital ulcers, such as those caused by syphilis and/or chancroid, increase the risk of becoming infected with HIV by about fourfold. There is also a significant although lesser increase in risk from STIs such as gonorrhoea, chlamydia and trichomoniasis, which all cause local accumulations of lymphocytes and macrophages.

Transmission of HIV depends on the infectiousness of the index case and the susceptibility of the uninfected partner. Infectivity seems to vary during the course of illness and is not constant between individuals. An undetectable plasma viral load does not necessarily indicate a low viral load in the seminal liquid or genital secretions.

However, each 10-fold increase in the level of HIV in the blood is associated with an 81% increased rate of HIV transmission. Women are more susceptible to HIV-1 infection due to hormonal changes, vaginal microbial ecology and physiology, and a higher prevalence of sexually transmitted diseases.

People who have been infected with one strain of HIV can still be infected later on in their lives by other, more virulent strains.

Infection is unlikely in a single encounter. High rates of infection have been linked to a pattern of overlapping long-term sexual relationships. This allows the virus to quickly spread to multiple partners who in turn infect their partners. A pattern of serial monogamy or occasional casual encounters is associated with lower rates of infection.

HIV spreads readily through heterosexual sex in Africa, but less so elsewhere. One possibility being researched is that schistosomiasis, which affects up to 50 per cent of women in parts of Africa, damages the lining of the vagina.

Exposure to Blood-Borne Pathogens

This transmission route is particularly relevant to intravenous drug users, hemophiliacs and recipients of blood transfusions and blood products. Sharing and reusing syringes contaminated with HIV-infected blood represents a major risk for infection with HIV.

Needle sharing is the cause of one third of all new HIV-infections in North America, China, and Eastern Europe. The risk of being infected with HIV from a single prick with a needle that has been used on an HIV-infected person is thought to be about 1 in 150. Post-exposure prophylaxis with anti-HIV drugs can further reduce this risk.

This route can also affect people who give and receive tattoos and piercings. Universal precautions are frequently not followed in both sub-Saharan Africa and much of Asia because of both a shortage of supplies and inadequate training.

The WHO estimates that approximately 2.5% of all HIV infections in sub-Saharan Africa are transmitted through unsafe healthcare injections. Because of

this, the United Nations General Assembly has urged the nations of the world to implement precautions to prevent HIV transmission by health workers.

The risk of transmitting HIV to blood transfusion recipients is extremely low in developed countries where improved donor selection and HIV screening is performed. However, according to the WHO, the overwhelming majority of the world's population does not have access to safe blood and between 5% and 10% of the world's HIV infections come from transfusion of infected blood and blood products.

Perinatal Transmission

The transmission of the virus from the mother to the child can occur in utero during the last weeks of pregnancy and at childbirth. In the absence of treatment, the transmission rate between a mother and her child during pregnancy, labour and delivery is 25%.

However, when the mother takes antiretroviral therapy and gives birth by caesarean section, the rate of transmission is just 1%. The risk of infection is influenced by the viral load of the mother at birth, with the higher the viral load, the higher the risk. Breastfeeding also increases the risk of transmission by about 4 %.

Diagnosis

The diagnosis of AIDS in a person infected with HIV is based on the presence of certain signs or symptoms. Since June 5, 1981, many definitions have been developed for epidemiological surveillance such as the Bangui definition and the 1994 expanded World Health Organization AIDS case definition. However, clinical staging of patients was not an intended use for these systems as they are neither sensitive, nor specific. In developing countries, the World Health Organization staging system for HIV infection and disease, using clinical and laboratory data, is used and in developed countries, the Centers for Disease Control (CDC) Classification System is used.

WHO Disease Staging System

In 1990, the World Health Organization (WHO) grouped these infections and conditions together by introducing a staging system for patients infected with HIV-1. An update took place in September 2005. Most of these conditions are opportunistic infections that are easily treatable in healthy people.

- Stage I: HIV infection is asymptomatic and not categorized as AIDS
- Stage II: includes minor mucocutaneous manifestations and recurrent upper respiratory tract infections
- Stage III: includes unexplained chronic diarrhoea for longer than a month, severe bacterial infections and pulmonary tuberculosis

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- Stage IV: includes toxoplasmosis of the brain, candidiasis of the esophagus, trachea, bronchi or lungs and Kaposi's sarcoma; these diseases are indicators of AIDS.

CDC Classification System

There are two main definitions for AIDS, both produced by the Centers for Disease Control and Prevention (CDC). The older definition is to referring to AIDS using the diseases that were associated with it, for example, lymphadenopathy, the disease after which the discoverers of HIV originally named the virus. In 1993, the CDC expanded their definition of AIDS to include all HIV positive people with a $CD4^+$ T cell count below 200 per μL of blood or 14% of all lymphocytes. The majority of new AIDS cases in developed countries use either this definition or the pre-1993 CDC definition. The AIDS diagnosis still stands even if, after treatment, the $CD4^+$ T cell count rises to above 200 per μL of blood or other AIDS-defining illnesses are cured.

HIV Test

Many people are unaware that they are infected with HIV. Less than 1% of the sexually active urban population in Africa has been tested, and this proportion is even lower in rural populations. Furthermore, only 0.5% of pregnant women attending urban health facilities are counseled, tested or receive their test results. Again, this proportion is even lower in rural health facilities. Therefore, donor blood and blood products used in medicine and medical research are screened for HIV.

HIV tests are usually performed on venous blood. Many laboratories use fourth generation screening tests which detect anti-HIV antibody (IgG and IgM) and the HIV p24 antigen. The detection of HIV antibody or antigen in a patient previously known to be negative is evidence of HIV infection. Individuals whose first specimen indicates evidence of HIV infection will have a repeat test on a second blood sample to confirm the results.

The window period (the time between initial infection and the development of detectable antibodies against the infection) can vary since it can take 3–6 months to seroconvert and to test positive. Detection of the virus using polymerase chain reaction (PCR) during the window period is possible, and evidence suggests that an infection may often be detected earlier than when using a fourth generation EIA screening test.

Positive results obtained by PCR are confirmed by antibody tests. Routinely used HIV tests for infection in neonates and infants (*i.e.*, patients younger than 2 years), born to HIV-positive mothers, have no value because of the presence of maternal antibody to HIV in the child's blood. HIV infection can only be diagnosed by PCR, testing for HIV pro-viral DNA in the children's lymphocytes.

NUTRITION FOR HIV/AIDS

In the absence of a cure, it is important to control symptoms, support the immune system, and lower the levels of HIV circulating in the blood. To lower the level of HIV in the blood, patients take a prescribed combination of antiviral drugs. The role nutrition plays will vary along the disease continuum (disease progression over many years), with consideration given to the patient's age, gender, behaviours, current medication, drug history, socio-economic status , and associated health concerns.

In all cases, adequate hydration (fluid intake) and increased calorie and protein intake are necessary to fight the infection. Proper nutrition must begin immediately to support nutritional deficiencies (including vitamin A and E, the B – vitamins , magnesium, and zinc) that occur early in the disease process. These nutritional deficiencies contribute to decreased immunity and disease progression. Ellen Mazo and Keith Berndtson, in The Immune Advantage , suggest that once the patient has been diagnosed with HIV infection, more protein and complex carbohydrates , along with moderate amounts of fats, should be consumed. The diet should include lean meat, fish, beans, seeds and nuts, whole-grain breads and cereals, and fruits and vegetables. Moderate amounts of fat for energy and calories can be acquired through foods such as nuts, avocado dip, peanut butter, and seeds.

The diet should include each of the five major food groups (dairy, vegetable, meat, fruit, and bread). The sixth group (fats and sugars) should be used sparingly. Patients with a poor appetite should eat six or more small meals throughout the day, rather than three large ones. In prolonged cases of appetite depression, a physician may prescribe an appetite stimulant (*e.g.*, megesterol acetate). It is important to keep all foods refrigerated, to avoid eating rare meats, to practice proper hand washing, and to use soap and hot water to clean sinks and utensils. Food-borne illnesses pose serious threats for HIV/AIDS patients.

HIV/AIDS COMPLICATIONS

Some symptoms will require additional attention beyond general nutritional recommendations. For example, diarrhoea will rapidly reduce the water content of the body, causing severe alterations in the body's metabolism and electrolyte balance. Electrolytes may be replaced with products such as Pedialyte or Gatorade. Proteins and calories should be increased to prevent weight loss, and dairy products, alcohol, caffeine, and spicy and fatty foods should be avoided.

A second complication is that of weight loss and wasting. According to Derek Macallan, in Wasting HIV Infection and AIDS, wasting may be either acute (associated with a secondary disease) or chronic (associated with gastrointestinal

disease), and is the result of a variety of processes, including drug use, medications, concurrent disease, and HIV itself. HIV infection causes abnormal protein and fat metabolism. During episodes of acute wasting the patient may require a prescription for steroids, to help support tissue maintenance and tissue development, in combination with optimal protein and calories in the diet.

Contributing to weight loss and wasting is malabsorption (the failure of nutritional substances to be absorbed in the intestines). Malabsorption occurs in advanced cases of HIV infection when gastrointestinal disease is present. Diseases that can cause malabsorption in HIV/AIDS patients include Kaposi's sarcoma, non-Hodgkin's lymphoma, cytomegalovirus, Myco-bacterium avium complex, and cryptosporidiosis. Malabsorption may require an alternative to oral nutrition.

Alternatives to Oral Nutrition

Alternative routes for nutrition must be considered in patients with fungal growth in the oral cavity, inflammation of the gums and oral mucosa , open sores, difficulty in swallowing, and other debilitating diseases of the oral-pharyngeal region and/or gastrointestinal tract. These alternatives include parenteral (PN) and enteral nutrition. PN replaces essential nutritional requirements via intravenous (IV) access. The IV may be placed in a peripheral vein or in a large central vein, depending on the medical condition of the patient and the choice of nutrition replacement therapy. The cost for PN is high, and there is a risk of severe infection; therefore it is not recommended except for brief treatment measures during known episodic cases of acute weight loss and in the absence of gastrointestinal (GI) function.

Enteral nutrition (placing a tube into the stomach or intestine) is preferred in those patients who have difficulty in swallowing, disease of the oral-pharyngeal region, and adequate GI function. The medical risks with enternal nutrition are less than for PN, but may include injury to the GI tract and infection in the absence safe food practices.

Ethical Considerations for Care

It is strongly advised that those suspected with or diagnosed with HIV infection seek professional attention from a qualified physician and a registered dietician. For these caregivers, the development of new antiviral drugs, changes in methods of administration of existing drugs, and new information regarding nutrition require diligent and regular review. It is important for health care workers to keep an open dialogue with the patient, so that they stay aware of the patient's health status and treatment measures. Many issues regarding the amount (drugs and nutrition) and length of care for AIDS patients remain controversial and should be negotiated with the patient.

Finally, when traditional medical wisdom fails, some patients may turn to alternative medicine. There are many questionable products on the market that make extraordinary health claims, and caution is required. These products are often overpriced and marketed with misleading claims, and should therefore be considered carefully before use.

5.8 NUTRITIONAL CANCER

Our modern society is characterized by a lifestyle with low levels of exercise coupled with consumption of foods that are high in calories, fat, sugar, and salt. But your body still responds in the only way it knows — it stores excess food as fat to prepare for times of prolonged starvation. Of course, because prolonged starvation typically does not happen in modern society, this safety mechanism means that we just continue to gain weight and store more fat.

This excess fat, especially the fat around the middle of your body, has been associated with an increased risk of many diseases, including prostate cancer, and particularly aggressive prostate cancer. But you don't have to be 50 pounds overweight to suffer the ill effects of excess body fat. Body fat is actually an organ with functions. It secretes hormones and specialized proteins that can increase inflammation and oxidation in the cells of your body — two natural processes that are strong contributors to the development and progression of prostate cancer.

Effects of Oxidation and Inflammation

Oxygen is essential to life, but the chemistry of oxygen and oxidation drives cancer development. Oxidation is a normal chemical reaction that occurs when free radicals form within the cells of the prostate. Each oxygen atom contains two electrons that cling together. When heat or light breaks apart the atom, the electrons are separated, leaving unpaired oxygen radicals. These radicals are free to roam around and initiate a process of breaking down normal cellular structures, causing damage and promoting the development of cancer.

The more free radicals present, the more cancercausing damage occurs. This process is similar to what happens during the browning of an apple after it is sliced open and the flesh of the apple is exposed to the oxygen in the air. The oxygen atoms in the air interact with the sugar in the apple, forming oxygen radicals. These radicals breakdown the flesh of the apple, or oxidize it, and the apple begins to rot.

As long as the outer peel of the apple protects the inner flesh from oxygen, it is not oxidized. But when protective "antioxidants" are removed, the damage from oxidation is allowed to occur unimpeded. Likewise, our bodies have many sophisticated defenses against oxidation. But when these defenses breakdown, cancerous cells form and are allowed to grow.

One of the most common causes of the loss of protective antioxidants is inflammation, a biochemical process that your body initiates when fighting off an infection. If the body senses invaders, such as bacteria, white blood cells are mobilized to go to the site of the invasion and to release oxygen and nitrogen radicals to help kill the invaders. Unfortunately, if they remain unchecked, these same oxygen radicals can also breakdown normal tissue and promote the development of cancer. Oxygen radicals damage normal DNA, causing errors that allow cancer growth.

In fact, investigators have noted the presence of inflammatory cells in virtually all prostate cancer tissue that is removed surgically, and have found that inflammation leads to the atrophy, or wasting away, of normal prostate tissue adjacent to precancerous and cancerous areas of prostate tissue.

Based on these and other observations, evidence is mounting that inflammation and oxidation play key roles in the development of prostate cancer. Why is this important? Because although other contributory factors such as aging and altered hormone secretions are difficult or impossible to change, nutritional and exercise habits that reduce the development of inflammation and oxidation can be changed.

There are many anti-inflammatory and antioxidant substances found in colourful fruits and vegetables, whole grains, and spices — nearly all of which are absent from the processed foods that rely on sugar, salt, and fat for flavor. By focusing your diet on fresh fruits and vegetables, ocean-caught fish, and whole grains, you can increase the protective antiinflammatory components of your diet and begin to benefit from their effects.

For example, tomato-based products such as soups, pasta, and juices can increase levels of the antioxidant lycopene in the prostate gland. Drinking beverages such as pomegranate juice and green and black tea can increase levels of antioxidant-containing polyphenols. The cruciferous vegetables such as broccoli, Brussels sprouts, bok choy, wasabi mustard, and horseradish all contain substances that may induce protective proteins in your liver and tissues, while vitamins, minerals, extracts of fruits and vegetables, herbs, and spices can all act against both oxidation and inflammation.

Finally, recent research has suggested that regular exercise may be one of the best natural antioxidants. Regular exercise causes many changes in your body that help reduce circulating levels of reactive oxygen inflammation. Beyond burning calories, endurancetype exercises, such as walking, running, cycling, and swimming, are particularly effective at increasing the body's natural levels of antioxidants, eliminating inflammatory molecules that drive cancer.

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THE CONTRIBUTION OF CARCINOGENS

Inflammation and oxidation are two of the body's natural processes, which, when they are allowed to proceed unchecked, can influence the development of prostate cancer. But external substances also play an important role — and can also be thwarted by keeping to a healthy diet.

A carcinogen is a chemical that directly or indirectly causes or leads to more aggressive forms of cancer. Hundreds of chemicals have been definitively linked to cellular changes that lead to cancer development, and hundreds more have been implicated in processes that might be involved. In today's industrial society, it's hard to avoid all exposure to carcinogens. But by focusing on a healthy diet and on healthy eating practices, you can avoid increasing your exposure to carcinogens that contribute to the development of prostate cancer.

For example, overcooking of any type of meat at very high temperatures produces a set of carcinogens called heterocyclic amines, one of which, known as PhIP, has been shown to cause prostate cancer in animal studies. In addition, charbroiling red meat or chicken, with its skin intact, produces yet another set of carcinogens, called polycyclic aromatic hydrocarbons.

The deleterious effects of these two carcinogens are well known when tobacco leaves are burned in cigarettes, heterocyclic amines and polycyclic aromatic hydrocarbons are produced and inhaled, playing a role in the development of lung cancer. Remarkably, by eating overcooked and charbroiled meats, the average American consumes the same amount of carcinogens (as PhIPs) as are inhaled in a pack-and-a-half of cigarettes a day.

Laboratory research findings have suggested that intake of these charred meat carcinogens triggers mutations in prostate cell DNA and leads to a chronic inflammatory response in the prostate. This combination of mutations and inflammation appear to be a key to the development of prostate cancer.

Switching to alternate sources of protein that are not prone to forming carcinogens when cooked, such as soy, is an important first step in minimizing the damage caused by overcooking and charbroiling meats. Also, using alternate methods to cook meat can significantly cut down on the amount of carcinogens produced: choose steaming or baking over charbroiling or pan-frying, marinate the meat, and turn the meat frequently to prevent overcooking. Finally, increase your consumption of cruciferous vegetables, which have unique properties that enable them to help "sponge up" carcinogens and possibly even counteract some of the damage caused by these carcinogens.

EFFECTS OF EXCESS SUGAR

Over the last 20 years, as intake of sugars from processed foods has gone up, obesity rates have skyrocketed, leading many researchers to implicate excess

sugar in the current obesity epidemic. In fact, recent work suggests that highfructose corn syrup — a form of sugar frequently found in processed foods such as soft drinks — is converted to fat much more quickly than is naturally occurring glucose.

Yet, the negative effects of excess sugar begin even before it is stored as fat. Sugar is a prime energy source for many cancers, including prostate cancer. Most normal cells can adapt to an environment low in sugar and use other energy sources — a process developed through evolution when people would go through periods of starvation. However, cancer, which grows faster than normal cells, does not have the same ability to adapt to low sugar environments. Thus, the more excess sugar consumed, the more the tumor is stimulated. Indeed, several animal studies suggest that cutting simple sugar intake can slow prostate cancer growth.

Excess sugar intake is further linked to prostate cancer growth through its interactions with insulin. Upon consumption of sugar, the body produces insulin, which helps to breakdown the sugar, ensuring that the sugar is stored as needed. When too much sugar is consumed and the body constantly produces high levels of insulin to help process it, the cells can become immune to the effects of insulin, resulting in too-high levels of sugar in the blood — a common sign of metabolic syndrome and a strong risk factor for diabetes. In addition, high insulin levels have been linked to an increased risk of diabetes, heart disease, and prostate cancer growth, independent of its interaction with sugar.

Putting this evidence together, research is beginning to suggest that the more processed sugars you eat, the higher your insulin levels, and the more likely it is that your prostate cancer will grow.

Yet, cutting back on sugar intake is only one important step. Studies of animals with prostate cancer have shown that restricting overall caloric intake can slow advancement of their disease. Maintaining a healthy diet and engaging in a regular exercise regimen will not only help you achieve and maintain your goal weight, it might also help slow the growth of cancer. Working with a qualified nutritional counselor will help you identify "good" and "bad" foods, while working with a qualified exercise physiologist will help you develop an exercise regimen to keep you fit.

Putting It All Together

The modern diet and sedentary lifestyle can lead to an accumulation of body fat, which, in turn, can contribute to the development of inflammation and raise insulin levels. At the same time, a highcalorie, high-sugar, pro-inflammatory diet can promote age-related chronic diseases and may prove an important link between nutrition and prostate cancer development and progression. Even modest

changes in body fat composition through changes in diet choices and regular exercise can be beneficial – a 5% weight loss has been shown to decrease markers of inflammation by 30% in obese diabetic patients and to reduce insulin levels by 20% in non-diabetic people.

While excess weight has been associated with more severe and rapidly progressive prostate cancer, up to 60% of men at normal body weight and with an average-sized waist carry excess body fat around their abdomens. Focusing on a diet that is rich in antioxidants, low in proinflammatory and carcinogenic substances, and low in simple sugars – coupled with a regular exercise regimen – can make an important difference in improving the overall health of every prostate cancer thriver.

Key Points to Remember

1. Oxidation and inflammation play important roles in the development of prostate cancer
2. Anti-inflammatory and antioxidant substances found in colourful fruits and vegetables can counteract the damage caused by oxidation and inflammation
3. Carcinogens from charred meat can trigger chronic inflammation in the prostate
4. Using alternate methods to cook meat and increasing cruciferous vegetables can minimize intake of and damage from carcinogens
5. Sugar is a primary energy source for cancer and stimulates production of insulin, which is linked to an increased risk of diabetes, heart disease, and prostate cancer
6. Cutting back on sugar intake, maintaining a healthy diet, and engaging in a regular exercise regimen can help slow the growth of cancer

The Metabolic Syndrome

For many years, doctors noticed that overweight or obese patients often had the following common diseases – high blood pressure or hypertension, increased blood sugar or diabetes, and high levels of blood fats, specifically triglycerides. In addition, these patients were at greater risk of developing insulin resistance and type 2 diabetes.

Because these different conditions are so common, their coexistence seemed to be a simple coincidence – after all, common diseases occur commonly. However, in the last two decades, the coexistence of these conditions, collectively known as metabolic syndrome, has been found to have a very specific underlying cause – increased abdominal fat and insulin resistance.

Metabolic syndrome affects up to 40% of Indians between the ages of 45 and 65. Since an estimated 55% of adults in India are overweight or obese, it is not surprising that metabolic syndrome and the detrimental effects of its component chronic diseases is so common.

Effects of Excess Body Fat

Obesity is the result of an imbalance of food intake and exercise. When you eat more and exercise less, fat accumulates in the body. The first place that fat accumulates in men is in the middle of the body around the belly and abdomen.

Belly fat is specially evolved to store fat quickly and release it quickly. Thus, this is the first place the fat settles when you gain weight and the first place it leaves when you lose weight. Since this fat grows so quickly, it can sometimes outgrow its blood supply, causing the fat cells to die. When this happens, the body's immune system sends out scavenger white blood cells to clean up the debris – which, as we described earlier, sets off a cascade of inflammatory and oxidative events that can ultimately promote heart disease, diabetes, and certain types of cancer, including prostate cancer.

But the effects of belly fat on prostate cancer growth are not limited to its impact on inflammation. Hormones produced by belly fat affect the ability of cells to properly take up insulin. In turn, this resistance to insulin results in the overproduction of insulin and insulin-like growth factor (IGF), both of which are potent stimulants for prostate cancer growth. When applied to human prostate cells in the laboratory, IGF prevents prostate cancer cells from dying and helps them grow. At the same time, the release of special proteins that "sponge up" and reduce the levels of IGF is decreased, thereby exacerbating the effects of the increased circulating IGF. Of note, one of these special binding proteins (IGF binding protein-3) has been shown to directly stimulate prostate cancer cell death—compounding the effects of the loss of IGF binding proteins resulting from insulin resistance and excess belly fat.

> Hormones produced by belly fat lead to an overproduction of insulin and insulin–like growth factor, both of which are potent stimulants for prostate cancer growth.

Beyond the effects of the excess body fat, the consequences of caloric imbalance that leads to fat accumulation can be significant. Cancer cells grow faster than do normal cells and require excess energy for their growth. Thus, excess calories – above your body's need – will help feed tumor growth.

Also, rapidly growing tumors live on the edge of survival due to a lack of an adequate blood supply and low levels of oxygen. Without oxygen, the cells can't breakdown fat but instead rely on sugars and carbohydrates for energy.

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Thus, the higher your sugar intake, the more nutrients you are providing to the tumor.

In human studies, cutting down on carbohydrate intake has been shown to be one of the best ways to lose weight, which, in turn, has dramatic health benefits and can slow tumor growth.

Despite some very promising research, the degree to which cutting all carbohydrates is helpful in the long run remains unknown. But what is not disputed is that eating simple sugars has no real benefit, promotes obesity and possibly tumor growth, and should be avoided.

Effects of Muscle Loss

Muscle plays a number of roles in maintaining health. Not only are muscles critical to posture, balance, and movement, but they maintain healthy bones by putting a physical stress across the bones. Hormonal therapies often used in men with advancing prostate cancer can have a detrimental effect on muscle, leading to muscle atrophy or wasting. Thus, with muscle loss from aging, inactivity, and hormonal therapies, the bones become more brittle and the loss of balance can lead to bone fractures.

In addition, the loss of muscle can undermine a potential mechanism to alleviate insulin resistance. The uptake of sugars into the muscle can occur via a process that is independent of insulin. Therefore, in men with metabolic syndrome, the loss of muscle mass precludes their ability to effectively control blood sugar control and overcome insulin resistance through exercise.

Nutrition at the Molecular Level

It has been only a few years since the human genome was sequenced, and what we learned through this processs has radically altered our understanding of human genetics. As a general rule, scientists thought that each gene contained the information needed to produce one protein, a key element used in nearly every bodily process. And, given the complexity of humans compared with other animals and with plants, it was estimated that the human genome would have 100,000 genes or more that coded for proteins. In fact, the human genome has only about 30,000 genes that code for proteins; at least 98% of the genome does not code for proteins at all.

It turns out that most of our DNA is the software involved in determining how and when 30,000 genes are expressed. Regulation of this expression can be affected by environmental, nutritional, and other factors. These changes to the genome by external factors, called epigenetic changes, can have significant effects on a wide variety of molecular processes.

For example, one of the most important nutritional factors modulating gene expression is folic acid: a lack of folic acid has been linked to an increased risk of heart disease and cancer. Folic acid, which is found in dark green leafy vegetables such as spinach and green lettuce, participates in a pathway leading to the stabilization of DNA. Early man evolved on a rich plant-based diet, so our metabolism came to depend upon a rich supply of folic acid to assist in multiple processes protecting against disease. However, as humans began to migrate out of agricultural lands, the ability to grow enough green plant food to provide adequate folic acid was reduced. As a result, it is believed that changes occurred in the genome of individuals to conserve folic acid for DNA integrity while sacrificing its other roles in metabolism —ultimately contributing to an increased risk of heart disease and cancer.

The Contribution of Antioxidants and Phytochemicals

Early bacteria living in Earth's oxygen-poor atmosphere learned to extract energy from the sun— by combining the energy with carbon dioxide from the air and water from the ocean, they were able to form the glucose they needed for their cells to function. This process, known as photosynthesis, gave these bacteria a huge advantage over competing species, but created a problem: the oxygen they produced as a byproduct of this chemical reaction threatened to destroy them through oxidation of their DNA. So, they developed specialized antioxidants called phytochemicals, which have properties that enable them to absorb the extra electrons found on oxidized chemicals and oxygen radicals. Phytochemicals are truly sponges for oxygen radicals.

In the human body, different types of antioxidants are found in specific locations where they can be most effective. For example, some act only in the oily environment of fat cells while others act in the liquid, water-like environment of muscle cells. This latter adaptation is particularly important, as the primary energy producers within muscle cells, the mitochondria, also leak oxygen radicals in oxygen-poor environments. The ability of antioxidants to mop up these radicals enables them to play an important role in the fight against cell damage and the development of cancer. This is where exercise can be particularly useful —exercise increases the levels of many antioxidants in the muscles, thus reducing the levels of dangerous free radicals.

The Colour System of Antioxidants

The different types of antioxidants can, for the most part, be grouped by colour. For example, the antioxidants found in red tomatoes are identical to those found in red watermelon or pink grapefruit. Although the system is by no means perfect, organizing phytochemicals by color is an easy way to help you differentiate between the different types of antioxidants and learn how to get a variety of phytochemicals and antioxidants into your diet.

The red group, including tomatoes, pink grapefruit, and watermelon, contain lycopene, one of the most well-studied antioxidants in the fight against prostate cancer. Population-based studies that were conducted when prostate cancer was diagnosed at more advanced stages clearly demonstrated that increased blood levels of lycopene and increased intake of lycopene-containing foods were associated with a reduced risk of aggressive prostate cancer. In recent years, as the population of prostate cancer patients has shifted to the identification of cancers at earlier stages, and as the population of patients has changed, some of these associations can no longer be demonstrated.

There are several short-term studies in which tomato paste or lycopene supplements were given to men prior to prostatectomy. Lycopene was identified in the prostate tissue after surgery and there were changes in prostate cells suggesting benefit. Multiple animal studies have also demonstrated the ability of lycopene to reduce tumor growth as well. It is also clear from multiple studies that the benefits of lycopene are more readily available when absorbed from cooked tomato products and juices than from whole tomatoes. In fact, more than 80% of the lycopene in the American diet comes from cooked tomato-based products such as pasta sauce, tomato soup, tomato juice, and ketchup.

Ultimately, studies focused on the ability of lycopene to prevent the initiation and progression of prostate cancer have not yet established definitively the benefits of increasing the intake of lycopene-containing foods or supplements. More research is needed to clarify the potential benefit of this nutritional component.

The red/purple group, including pomegranates, grapes, plums, and assorted berries, all contain anthocyanins, which accounts for the colour of the group. However, different berries in this group have unique properties. For example, pomegranates have ellagitannins, which inhibit inflammation and may have benefits for heart health, cancer prevention, and dementia, while cranberries have proanthocyanidins, which target a bacteria common in urinary tract infections.

The full benefits of blackberries, strawberries, and raspberries are still being studied, but they all have antioxidant power and work together with the other red/purple berries.

The orange group, including carrots, mangoes, apricots, cantaloupes, pumpkin, and sweet potatoes, contain alpha and beta carotenes. Beta-carotene, the more well-studied of the two, is converted by the body into vitamin A, which is important for vision, and works together with the red, green, and yellow/green antioxidants. Note that carrots provide about half the alpha and beta carotene in the average American diet, with significant contributions from tomato-based products.

The orange/yellow group, including oranges, peaches, papaya, and nectarines, contain betacryptoxanthin, a minor carotenoid that accounts for only a minute amount of the daily intake of all carotenoids by the average American. About 87% of cryptoxanthin comes from orange juice, oranges, and tangerines. However, one must be cautious about relying on processed juices as some of the nutrients are removed during production and high amounts of sugars are often added.

The yellow/green group, including spinach, collard, yellow corn, green peas, avocado, and honeydew melon, contain lutein and zeaxanthin. These carotenoids concentrate in the eye and contribute to eye health. Lower intakes have been associated with cataracts and age-related macular degeneration, the primary preventable cause of blindness in America.

The green group, including broccoli, Brussels sprouts, cabbage, bok choy, and kale, contain sulforaphane, isothiocyanates, and indoles. These compounds stimulate genes in the liver to produce enzymes that breakdown carcinogens, including those that are produced when overcooking and/or charbroiling meats.

The white/green group, including garlic, onions, asparagus, leeks, shallots, and chives, contain allyl sulfides, which activate an antioxidant response in cells.

Plant foods that don't fit into the colour system can also have unique benefits. For example, celery has salicylic acid, which is closely related to the active ingredient in aspirin and has been used for centuries to relieve headaches. Mushrooms are a complex group of plant foods with possible effects on the immune system at the level of the intestines.

Also, keep in mind that because the colour of the fruit or vegetable is tied to its chemical properties, foods with deeper, richer colours are typically more nutritious. Compare, for example, a regular storebought tomato with one bought from a local farm. To be able to ship a firm tomato by truck, the tomato is picked while it is still green and is rapidly ripened by being blasted with ethylene gas, a substance normally produced by the plant as a signal to ripen. While ripening, the family of lycopene compounds accumulates, especially in response to heat and light. However, once ripening stops, the accumulation of lycopene stops. Because the ripening process is stilted, the colour of the typical store-bought tomato is often somewhat washed out. By contrast, the tomato that ripens naturally on the vine at the local farm is typically deeper in colour and richer in taste — and thus more nutritious.

Incorporating a variety of both colourful and colourless phytochemicals in fruits and vegetables can help to maximize intake of key chemical elements required to maintain healthy tissues and reduce the risk of disease.

BALANCING ACT OF SUPPLEMENT USE

For more than thirty years, there have been comprehensive multivitamin/multimineral tablets on the market that provide what scientists have agreed are the adequate amounts of vitamins and minerals for prevention of vitamindeficiency diseases. True vitamin-deficiency diseases are also rare in the United States today because of the wide variety of available foods and because so many staples are fortified with additional vitamins to ensure that at least the bare minimum requirements are met. Thus, researchers have identified a new purpose for multivitamins, multiminerals, and other specialized supplements – providing optimum nutrition for the prevention of chronic diseases through improved cellular nutrition.

This is an area of continuing controversy among opinion leaders in nutritional science. In fact, dietary guidelines contain no recommendations for a multivitamin, instead offering an outline of which types of foods contribute which types of nutrients. Nevertheless, the public has embraced the idea of benefit in pill form, as evidenced by the continued purchase of supplements in health food stores, grocery stores, and drug stores.

Avoiding Toxicity and Overdosing

Because some vitamins can be toxic if taken at extremely high doses and/or in the wrong form, learning how they are formed, how they act, and how they are cleared from the body can help in understanding how best to ensure the safe use of supplements. Vitamin A, for example, offers an excellent lesson in how excess intake, particularly in the wrong form, can have toxic consequences.

Vitamin A is actually a hormone, which the body forms from the beta-carotene found in bright orange carrots, pumpkin, or squash. Because the body will only convert the orange pigment betacarotene to vitamin A when it is needed, if you eat too many orange plant foods, your skin might turn orange, but you will not be subject to vitamin A toxicity.

By contrast, vitamin A toxicity is possible when vitamin A or vitamin A containing foods are eaten directly. For example, eskimos, who eat whale liver that is rich in stored vitamin A, could develop health issues from vitamin A poisoning, such as liver disease or bone disease.

Toxicity occurs with intakes of greater than 25,000 international units (IU) of vitamin A, which is only five times the required amount or RDA of 5,000 IU. At one time, multivitamins could contain up to 10,000 IU of vitamin A. However, after reports from Harvard population scientists that nurses taking more than 8,500 IU of vitamin A from foods and supplements had evidence of bone disease, the vitamin industry reduced or eliminated vitamin A from multivitamins and replaced half or all with beta-carotene, which could be safely converted to vitamin A without leading to vitamin A toxicity.

Of note, although beta-carotene is safe when ingested from foods, some have questioned its safety when taken in mega-doses as supplements. After noting a lower incidence of lung cancer in people who had high levels of beta-carotene in their blood, investigators at the National Cancer Institute gave beta carotene supplements to smokers in an effort to prevent the development of lung cancer. The study was stopped prematurely because the subjects actually showed an increased risk of lung cancer. However, the dose was so high that subjects were, in effect, given six times the amount of betacarotene found in a healthy diet. Thus, although the study results led to questions of whether excessive intake of beta-carotene is safe in smokers, the vitamin remains safe when ingested from foods or when taken at standard doses in multivitamins.

Making the Smart Choice

Dietary supplements are just that — a supplement to a healthy diet, not a replacement for one. Taking megadoses of any vitamin is never a good idea. The body's own protective mechanisms have ensured that it's nearly impossible to get to toxic levels when ingesting vitamins and minerals from eating fruits and vegetables. Start with these natural plant sources, the ideal way to receive the right amounts in the most absorbable form. Then, if you wish, supplement with a multivitamin/multimineral and educate yourself on the scientific literature and so you can make an informed choice.

Keep in mind that multivitamin formulations that include a mix of different complexes of vitamins and minerals can offer a reasonable way to get some additional nutritional value into your diet in the most efficient way possible. For example, because high levels of zinc can block absorption of copper and can lead to a copper deficiency anemia, the ratio of these two minerals is controlled in multivitamins.

Be careful not only to avoid taking toxic doses, but also to avoid supplementation of a single vitamin or mineral if there is no specific deficiency. For example, taking a single B vitamin in high doses instead of a B vitamin complex can lead to what is called a conditioned deficiency: the high dose of the single B vitamin causes an increase in the breakdown of the B vitamins that are not being supplemented. In extreme cases, the breakdown of these B vitamins to low enough levels can cause neurologic problems, such as seizures.

Ultimately, it is important to remember that you should not rely on supplements to make up for a poor diet. Fruits and vegetables are rich sources of mixed vitamins, minerals, antioxidants and other specialized substances in combinations that cannot be duplicated in most supplements. A colourful diet, with representation every day from as many color groups as possible, remains the best way to ensure you receive a well balanced set of key nutrients.

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IMPLEMENTING A PLAN FOR SUCCESS

Personalized nutrition advice goes beyond the general dietary guidelines for a population and hones in on the needs of an individual. Body composition determination can provide information on total energy needs, lean body mass, protein requirement, deviation from healthy body fat, and a personalized target weight. The diet can be analyzed at three levels: (1) the overall caloric content and macronutrient profile (*i.e.*, protein, carbohydrate, and fat composition); (2) the vitamin and micronutrient adequacy for prevention of deficiency diseases; and (3) the adequacy of vitamin and mineral intake from a combination of foods and supplements for optimized nutrition.

The science of optimized nutrition and personalized nutritional advice is still evolving. The new field of gene-nutrient interaction drawing from basic science and studies of populations is in its infancy, and efforts to improve the science and its application to personalization of nutrition advice are underway.

In the meantime, it is critical to focus on the benefits of a healthy diet and regular exercise. While the relationships between diet and exercise and cellular processes within the prostate gland are not yet fully established, it is clear that antioxidants found in many foods can play a role in protecting against cancer cell growth while substances that promote inflammation and oxidation can stimulate prostate cancer cell growth. In addition, the overall status of your diet and exercise can affect the development of other diseases that are common among older men with prostate cancer, including heart disease and diabetes.

Building Strong Muscles

Increased protein intake at the upper end of the range recommended by the Institute of Medicine (10% to 35% of total calories) has been shown, in several clinical studies, to reduce hunger and improve lean body mass during weight loss. Exercising for durations of approximately one hour each day is an excellent strategy for weight maintenance, while progressive resistance training has been shown to build muscle, increase resting metabolism, improve glucose tolerance, increase strength, muscle function, and aerobic fitness, contribute to increased bone density, and improve quality of life.

Note also that maintaining muscle mass can have a positive impact on metabolism. A pound of muscle burns 14 calories for every 3 calories burned by a pound of fat. Thus, the number of calories burned per day is at least in part determined by the ratio of muscle to fat.

Avoiding the muscle loss common in aging, inactivity, and hormonal therapies and/or gaining muscle through increased protein intake and exercise can help you achieve and maintain a healthy body weight, giving you more energy and an ability to enjoy active sports more fully.

Restoring a Healthful Caloric Balance

Concentrating on fruits and vegetables can pay dividends immediately. The average vegetable serving is only 50 calories and the average fruit serving is only 70 calories, while the average serving of refined carbohydrates such as potatos, rice, pasta, bread, bagels, or cakes is more than 200 calories.

Considering that you have to run 3 miles in 30 minutes to burn off the calories in a medium potato and bicycle 8 miles in 30 minutes to burn off the calories in a donut, minimizing your caloric intake from these types of refined carbohydrates can help you maximize the effects of regular exercise.

Reviewing the Benefits Stage by Stage

In the majority of cases, when cancer is detected early, primary surgical or radiation treatment is curative and taking additional steps to prevent the growth of the cancer might seem unnecessary. Nevertheless, men at this stage would do well to take an opportunity to inventory and tune-up their nutritional habits. Remember, maintaining a healthy diet and regularly exercising can be important steps toward preventing other diseases that commonly occur with aging, including heart disease and diabetes.

Men with advanced prostate cancer or with disease that has recurred can begin incorporating healthy diet and regular exercise into their regular routines to help slow the progression of disease, while recognizing that there is limited direct proof of the effectiveness of such changes. The key in this phase of the disease is to rationally make dietary and exercise changes in a way that complement any ongoing treatments.

Men who have received hormonal treatments are at particularly increased risk of developing weaker bones and muscles. For these men, endurance training to keep the cardiovascular system strong, and resistance or weight training to keep the muscles strong, coupled with healthy dietary choices focusing on achieving a goal weight, can be integrated into the treatment plan on an ongoing basis.

Finally, in men with metastatic or advanced disease, supportive nutrition is most important. Because weight loss and loss of appetite is common in men at this stage of disease, healthy nutrition and ensuring adequate calories is critical to the success of therapy and to the support of a healthy immune system.

Adopting a Healthy Approach

It is a lot easier than you think to incorporate good nutrition and exercise into your everyday routine. The key is to recognize that implementing dietary changes does not mean giving up things you love and that exercising does not mean spending additional time that you don't have. The key is to see your life in

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a new way — with a new set of lifestyle changes all designed to turn you from a prostate cancer survivor into a prostate cancer thriver.

1. Lose the body fat

Losing fat is a simple equation: eat fewer calories per day than you burn. This can be done by changing your dietary pattern away from high fat foods, sweets, fast foods, and savory snacks and eating more colourful fruits and vegetables, low-fat proteins from poultry, fish, and seafood, and fewer refined carbohydrates. Here are some practical examples of places you can cut calories:

- Substitute white meat of turkey or chicken for high-fat red meats and farmed fish
- Substitute colourful fruits and vegetables for rice, pasta, potato, and breads
- Substitute mixed berries and fruits for ice creams, cakes, pastries, snack chips and highfat, high-sugar desserts
- Substitute non-fat and low-fat dairy and soy products for cheese and full-fat dairy products
- Substitute water for soft drinks
- Reduce the use of added fats, oils, margarine, butter, and salad dressing

2. Maintain muscle mass

In order to maintain muscle mass as you age, it is important to take in adequate amounts of protein and to exercise muscles adequately to maintain them. As you age, the body's metabolism slows down due in large part to the decrease in muscle mass that is seen with inactivity. It is harder to build muscle as you age and it breaks down more quickly with inactivity than in younger individuals. However, it is possible to build and maintain muscle mass well into your 90s by simply eating adequate protein and doing muscle building exercises.

Also, building muscle mass is one of the most effective ways to change your metabolism: build 10 extra pounds of muscle, and you will burn an extra 140 calories per day. However, to maintain that muscle you need to do more than simply provide the extra 140 calories per 10 pounds; you must also supply the right amount of the right kinds of protein to nourish the muscles.

Your lean body mass determines how much protein you need each day. In fact, it's about twice what was recommended by government advisory groups until recently, when the Institute of Medicine broadened its recommendation to 10% to 35% of calorie intake. It takes about 1 gram of protein per pound of lean body mass or 29% of resting energy expenditure, which is the number of calories you burn at rest to build and maintain muscle. For example, a typical man with 150 pounds of lean body mass will burn 2100 calories at rest per day and will need about 150 grams of protein per day.

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Here is a list of some low-fat, high protein choices you can make:

- 7 egg whites: 25 g protein and 115 calories
- Chicken breast (4 oz): 25 g protein and 140 calories
- Ocean-caught fish (4 oz): 25 g protein and 140 calories
- Canned tuna in water (3.5 oz): 25 g protein and 110 calories
- Two veggie burgers: 28 g protein and 180—220 calories
- Soy protein shake with fruit: 25 g protein and 200 calories

3. *Exercise everyday*

Regular exercise will help you lose fat, build muscle, and improve your outlook overall. A combination of cardiofitness and weight lifting will not only help to round out the benefits, but the variety will help make it more interesting. This is key to sticking to a regular routine: choose an exercise you like and/or one that you can do with friends to make it more enjoyable. For example, walk 30 minutes everyday at a comfortable pace and lift weights three times each week, alternating different body parts and allowing a day of rest between weightlifting days. If you work on your chest muscles and triceps one day, switch to your back muscles and biceps on the next, followed by leg muscles and shoulders on the third day.

Most important, obtain professional instruction on how to perform exercises for each of these muscle groups and consult with your doctor before starting the exercise program to be sure it is safe for you. A certified fitness instructor, exercise physiologist, or physical therapist can provide exercise instruction and ensure that you are maximizing the benefit from your workouts.

The Four S's of Success

There are four S's of general behaviours that you can use to make lifestyle change more successful — stress reduction, stimulus control, self-monitoring, and social support.

1. *Stress Reduction*

Express your feelings. If feelings of stress, sadness, or anxiety are causing physical problems, keeping these feelings inside can make you feel worse. It's okay to let your loved ones know when something is bothering you. However, remember that your family and friends may not be able to help you deal with your feelings appropriately. At these times, ask someone outside the situation — such as your family doctor, a counselor, or a religious advisor — for advice and support to help you improve your emotional health.

Live a balanced life. Try not to obsess about the problems at work, school, or home that lead to negative feelings. This doesn't mean you have to pretend to be happy when you feel stressed, anxious, or upset. It's important to deal with

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these negative feelings, but try to focus on the positive things in your life, too. You may want to use a journal to keep track of things that make you feel happy or peaceful. Research has shown that having a positive outlook can improve your quality of life and give your health a boost. You may also need to find ways to let go of some things in your life that make you feel stressed and overwhelmed. Make time for things you enjoy.

Calm your mind and body. Relaxation methods, such as meditation through exercising, stretching or breathing deeply, are useful ways to bring your emotions into balance.

Take care of yourself. To have good emotional health, it's important to take care of your body by having a regular routine for eating healthy meals, getting enough sleep, and exercising to relieve pent-up tension. Avoid overeating and underexercising, and don't abuse drugs or alcohol.

2. *Stimulus Control*

Plan ahead. If you knew that you would be waking up to a cold floor when you got out of bed, it would make sense to prepare by leaving a pair of slippers at the side of your bed. Similarly, if you are going to eat at a restaurant, plan ahead and think about how to ensure you eat healthy foods. If you are going to be stressed, you may need to have a heightened level of determination to stick with your plan. If you can plan your week and make an appointment with yourself for exercise and relaxation at appropriate times, you will be more likely to follow through.

3. *Self-Monitoring*

A conscience is said to be the knowledge that someone is watching you. While implementing change behaviours, be your own conscience and monitor your actions. Set up a food and exercise log to track your progress. Use an established computer-based program or just a paper journal. Try to record the behaviours as they happen in terms of foods, exercise, and lifestyle behaviours and set a time to review the record so that you can chart your performance weekly.

4. *Social Support*

Social support is available from family, friends, relatives, religious groups, hobby groups, or cancer survivor groups. It is important that you maintain a healthy relationship with individuals who understand what you are going through. In particular, cancer support groups can be helpful in finding fellowship among others who are dealing with the same issues you are facing now.

Lifestyle changes carried out in a balanced fashion can lead to optimum health in the physical, mental, and spiritual realms — establishing for you a healthy approach toward thrivership.

5.9 DIET COUNSELLING FOR HIV/AIDS AND CANCER

To manage AIDS-related symptoms and improve diets, nutrition, and health education and counseling should form an integral component of PLHIV treatment, care, and support. Nutrition counseling should be incorporated into treatment protocols.

Nutrition counseling and support should engage clients in dialogue about food and dietary constraints, practices, and preferences, and utilize the understanding gained to help PLHIVs improve their diet.

Counseling and support should provide information on :

- Dietary recommendations : It is also advised to conduct a session with the client that translates the recommendations into a daily or weekly meal/ snack plan involving local foods, sufficient water consumption, and macro- and micronutrient supplements as needed.
- Drug-nutrient interactions for any currently prescribed medications and how to manage them.
- Management of oral and digestive tract problems and other related symptoms that may affect the consumption or utilization of food.
- Traditional therapies, as needed. Traditional approaches are not well documented. Their nutritional effects and how they interact with drugs are mostly unknown. In addition, the evidence of their efficacy is limited, and some have unpleasant side effects.

If lack of food or micronutrients has been identified as a problem, referral to programs that provide food, micronutrients, training, or other appropriate assistance may be needed. Periodic follow-up on specific identified issues and ongoing counseling are advised.

Healthy lifestyle education can help prevent infections and improve the quality of life for PLHIVs. Education should include information on :

- Hygiene, such as food and water safety, sanitation, and personal hygiene.
- Lifestyle habits, such as smoking, alcohol, and drug abuse, and their detrimental effects on food intake, absorption, and use.
- The importance and promotion of adequate amounts of physical activity as it maintains muscle mass, stimulates appetite, and enhances the feeling of well-being. Individuals who are very or overly active may need to reduce their level of physical activity.
- The importance of adequate sleep and extra rest.
- The importance and promotion of safe sex practices in order to avoid transmitting HIV to others and to prevent reinfection.
- Psychosocial support as it decreases depression, stigma, and stress, and improves quality of life, often with positive impacts on appetite and nutritional intake.

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- The recognition and prompt treatment of illnesses, as well as identification of appropriate and accessible sources of care. Since PLHIVs often become sick, with illnesses affecting food intake and nutritional status, any illness should be treated quickly.

Diet Counselling for Cancer

Many factors influence the development of cancer. During the last 20 years, science has shown that diet is one of them. By following a healthy diet and staying physically active, we can boost our own body's capacity to resist cancer.

Study after study shows that a healthful diet - one high in a variety of vegetables, fruits, whole grains and beans, and low in fat- can fight cancer at several stages. Scientists have known for some time that these foods contain nutrients that help defend the body against disease. And now research is revealing that the vitamins, minerals and other protective substances within these foods can do more than this - much more.

Scientists are now identifying a host of naturally occurring compounds in plant foods that can actually defuse potential carcinogens. Some of these nutrients and natural phytochemicals seek out dangerous substances and usher them from the body before they can cause the kind of cellular damage that may lead to cancer. Others make it easier for the body to make cellular repairs, and still others may help to starve new cancer cells before they can reproduce.

Even after a cell begins to become cancerous, diet and lifestyle can still help short-circuit the process. Several nutrients have been shown to delay the progression of cancers from one stage to another; this gives the body more time to defend itself. Maintaining a healthy weight also helps establish a bodily environment that discourages cancer growth.

Scientists have only just begun to investigate how the benefits of a healthy diet may impact cancer survivors. In the meantime, it makes sense to follow a few simple dietary guidelines that can help prevent cancer and possibly guard against its return.

Diet and Counselling for Survivors

In 1997, AICR published the landmark report, Food, Nutrition and the Prevention of Cancer: a global perspective. A panel of expert scientists evaluated over 4,500 studies on diet and cancer. The report includes their conclusions and their recommendations for cancer prevention. AICR summarizes these points in the following guidelines.

Diet and Counselling Guidelines for Cancer Prevention

1. Choose a diet rich in a variety of plant-based foods.
2. Eat plenty of vegetables and fruits.

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3. Maintain a healthy weight and be physically active.
4. Drink alcohol only in moderation, if at all.
5. Select foods low in fat and salt.
6. Prepare and store food safely.

Scientists estimate that these guidelines could help prevent 30 to 40 percent of all cancers. The most exciting news is that even small dietary and lifestyle changes can produce large health benefits. Eating one or two more servings of vegetables and fruits, switching from white bread to whole grain bread or increasing the length of your daily walk will all make a positive impact on your health.

STUDENT ACTIVITY

1. Discuss the dietary requirements of a AIDS patient.

2. Prepare a diet chart suited to a cancer patient.

5.9 SUMMARY

- The kidneys are paired organs, which have the production of urine as their primary function.
- Nephrology (from Greek nephros, "kidney", combined with the suffix -logy, "the study of") is a branch of internal medicine and pediatrics dealing with the study of the function and diseases of the kidney.
- Nephrotic syndrome is a nonspecific disorder in which the kidneys are damaged, causing them to leak large amounts of protein (proteinuria at least 3.5 grams per day per 1.73 m^2 body surface area) from the blood into the urine.
- Nephritis is inflammation of the kidney. The word comes from the Greek nephro- meaning "of the kidney" and it is meaning "inflammation". Nephritis is often caused by infections, toxins, and auto-immune diseases.
- Less commonly, nephritis involves the tubules and the tissues that surround them (tubulointerstitial tissues). Such inflammation is called tubulointerstitial nephritis.
- Renal calculi are small chunks of crystallized material which become trapped in the kidneys, bladder, or urinary tract. A renal calculus can be extremely painful, and the patient may not be able to pass it without assistance.
- The most common type of kidney stone is composed of calcium oxalate crystals, occurring in about 80% of cases, and the factors that promote the precipitation of crystals in the urine are associated with the development of these stones.
- Kidney stones can be due to underlying metabolic conditions, such as renal tubular acidosis, Dent's disease, hyperparathyroidism and medullary sponge kidney.
- Acquired immune deficiency syndrome or acquired immunodeficiency syndrome (AIDS) is a disease of the human immune system caused by the human immunodeficiency virus (HIV).

5.10 GLOSSARY

- **Nephrology:** A branch of internal medicine and pediatrics dealing with the study of the function and diseases of the kidney.
- **Nephrotic syndrome:** A non-specific disorder in which the kidneys are damaged, causing them to leak large amounts of protein.

- **Nephritis:** It is inflammation of the kidney which is often caused by infections, toxins, and auto-immune diseases.
- **Renal calculi:** Small chunks of crystallized material which become trapped in the kidneys, bladder, or urinary tract.
- **Acquired immuno-deficiency syndrome (AIDS):** A-disease of the human immune system caused by the human immunodeficiency virus (HIV).

5.11 REVIEW QUESTIONS

1. What is the importance of Nephrology?
2. What are the principal causes of Nephrotic syndome?
3. How is Renal calculi diagnosed?
4. What are the primary symptoms of HIV/AIDS?
5. How is Renal calculi treated? Point out the dietary requirements of a patient suffering from renal disorder.
6. Discuss the interrelation of nutrition and cancer.

5.12 FURTHER READINGS

- Purohit C. G., *Cell Biology,* 1st edition, K.K. Mittal Prakashan.
- Sylivia medal, *Understanding Human Anatomy and Physiology,* 2nd edition, WMC Brown Publishing, 1991.