CLINICAL NUTRITION AND DIETETICS

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

Specialization-I: Clinical Nutrition and Dietetics

Study Material Prepared by:

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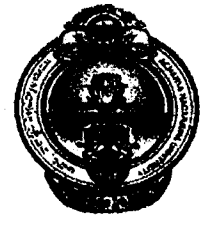

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FOREWORD

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

SYLLABUS

M.Sc (Course Code-139)

Paper - III: CLINICAL NUTRITION AND DIETETICS

UNIT - I

- Indian dietetics association: History of dietetics–origin of the association, membership and registration board.
- Dietician: Definition, Qualification, classification, responsibilities, Code of Ethics and obligations.
- Dietetics: Classification of foods and preparation of normal diets, Principles in formulation of therapeutics diets and factors to be considered for therapeutic diets.

UNIT - II

- Obesity and under weight: Etiology, physiology metabolic changes, complications, dietary modifications – grades of obesity and under weight.

UNIT - III

- Food allergy and intolerance: Types of allergens, symptoms, metabolic changes, diagnostic tests and dietary management.

UNIT - IV

- Gastro intestinal disorders: Dyspepsia, gastritis, ulcer, malabsorption syndrome, sprue and diverticular disorders.

UNIT - V

- Liver and gall bladder and biliary track disorders: Jaundice, hepatitis, cirrhosis and cholilithiasis, pancreatitis.

CONTENTS

UNIT—I

INDIAN DIETETICS ASSOCIATION, DIETICIAN AND DIETETICS

OBJECTIVES

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

- understand the composition, functioning, juridiction etc., of Indian Dietetics Association;
- define and classify the dietician;
- state the responsibilities of dietician;
- discuss the code of ethics and obligations of dietician;
- classify the foods as well as know the concept of preparation of normal diet;
- state the principles used in the formulation of therapeutical diets.

STRUCTURE

1.1 INTRODUCTION

Dietetics (from the Greek diaita, meaning "mode of life") has been implicated in the cause, cure and prevention of disease from earliest recorded history. The profession of dietetics, which is based on the philosophy that optimal nutrition is essential for the health and well-being of every person, is concerned with the science of human nutritional care; the practice of dietetics involves the application of knowledge about nutrition. Dieticians may specialize in various areas, including general practice, administration of dietetic services, community nutrition, clinical nutrition and nutrition education.

1.2 HISTORY OF DIETETICS

In the history of dietetics we discuss three narrative overviews of dietetics from its beginnings until after the end of the mediaeval and then Renaissance periods in Europe; of nutrition science in its first phase from its beginnings in the mid-nineteenth century until the middle of the twentieth century, with reasons for its rise; and of nutrition science in its second phase in the second half of the twentieth century, with reasons for its decline.

4000 BCE–1850 CE. The Fulfilled Life

The first known teachings on nutrition and health are African. The Egyptian Imhotep gave accounts of the use of food as medicine about 6000 years ago. Traditional Chinese teachings from those of the 'Yellow' Emperor Huang Ti around 2500 BCE, and of the Indian Ayurvedic tradition, also stress the importance of specified dietary patterns, foods and drinks, and plants with medicinal qualities, to prevent and treat disease and also as ways to a spiritually, morally, emotionally and mentally enlightened life.

From the beginnings of recorded history and in Europe up to and beyond the mediaeval era, teaching and practice on food, nutrition and health have been deep and broad. In what is now Europe, Pythagoras, Heraclitus, Alcmaeon, Hippocrates, Celsus, Dioscorides, Plotinus, Pliny the Elder, Plutarch and Porphyry, as well as other Greek, Roman and other philosophers, physicians and teachers who laid foundations for Western science and medicine, developed inductive and deductive systems of thinking about food and health between 600 BCE and 300 CE. The flowering of Arab culture between the eighth and the twelfth centuries CE included comparable teachings of Rhazes, Ibn Botlan, Ibn Sina Abu Ali Al Husain (Avicenna) and Moses Maimonides, Jewish physician to Salah al-Din (Saladin); these also became synthesised in 'The Regime of Health' treatise of the first major medical school in Europe at Salerno, published as from 1100 CE and one of the first books to be printed.

Originally ancient philosophies of the fulfilled life, of which teachings on diet and nutrition are an integral part, persisted in Europe through the Renaissance period and up to the period of the 'Enlightenment' of the eighteenth century. Indeed, they still persist in many parts of the world, as do those of oral societies that have developed systematised concepts of food and nutrition within natural environments. The Greek term diaita means 'way of life' or 'way of being', and the term 'diet' was used in this sense in treatises and handbooks until recent times in Europe. Human health and welfare are seen ecologically, in the context of the whole living and physical world, the 'great chain of being'. Their written records resonate with some of the writings now found in the 'healing and nutrition' sections of bookstores and 'wholefood' emporiums.

1850–1950 CE. The Bigger the Better

In post-mediaeval and Renaissance Europe, and then in the USA and other technologically developing countries, human beings and all other living things became identified as marvellous machines, by analogy with clocks, pumps, trains, or other forms of engineering. Study of life itself, and of consciousness and vitality, became seen as metaphysical. Aspects of humanity other than the physical were excluded by the rising sciences, within the context of a dominant ideology based on principles of political and economic power and growth.

Science was used to master nature. Massive machines and men have been better able to create material wealth, exploit the living and physical world, defeat other powerful nations, and dominate less technically developed peoples. Anything that moves faster and grows bigger was identified for this reason as better – and healthier. Scientists interested in the ways of nature and in what it means to be alive were dismissed as 'vitalists' and 'nature philosophers' and such terms became phrases of abuse.

Nutrition science in its modern formdates from the early to mid-nineteenth century; it had the effect of creating dietetics as a separate paramedical profession. The first generations of physiologists, biochemists and physicians who created nutrition science along the lines of the disciplines in which they were trained, believed they could change the world. So they did, once governments and industry endorsed their ideas. The dimensions of nutrition narrowed but its scope widened. It became less a philosophy of life, more an instrument of state.

Chemistry and Revolution

The German chemist Justus von Liebig, building on the work of Antoine Lavoisier, Franc,ois Magendie, Jons Berzelius, William Prout, Gerrit Mulder and others, who worked most of his productive life in Giessen, was a founder of biochemistry, and was the founder of nutrition as a biochemical science. His stamp

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on public affairs is comparable with that of Louis Pasteur in the field of microbiology. Both men possessed astounding energy, both courted the ruling classes; both smashed the reputations of fellow scientists whose views were holistic and ecological; and both facilitated the supremacy of current conventional science and practice. Emerged from its alchemical origins, chemistry became the rising science, and nutrition a biochemical discipline, because von Liebig and his followers realised that physiological chemistry (as it was at first termed) could be used to harness and master nature and to engineer the food systems of industrialising countries. Von Liebig grasped the significance of protein as the chemical compound that accelerates the early growth of plants, animals and humans. Once protein was isolated and identified as the primary or master nutrient and so the nutritional expression of the dominant European ideology, food systems engineered to emphasise animal protein had the power to change the world, as they have done.

The science of nutrition in its first period, roughly between 1850 and 1950, was harnessed by governments of the great European powers and the USA to increase the yield of food from plants and animals, and to build up their human resources, when more and more factory workers and foot soldiers were needed to increase national advantage and to service industrialisation and imperialism. In the most powerful European countries, philanthropists and politicians were united in their interest in nutrition. Both were preoccupied with the condition of the poor, partly for fear of uprisings of enraged ideologues and under-classes. 'Many have died of starvation, where long-continued want of proper nourishment has called forth fatal illness, when it has produced such debility that causes which might otherwise have remained inoperative brought on severe illness and death. The English working class men call this "social murder". This is Friedrich Engels writing in the mid-1840s; in 1848 he and Karl Marx were in Germany fomenting the revolutions that swept through continental Europe.

> "Nutrition scientists have taken a deep or broad view of their work and its implications since the beginnings of the discipline in the early nineteenth century; as have their predecessors, whose teachings and writings laid the foundations of dietetics as an empirical discipline. In the first years of the twenty-first century, the original vision and scope of nutrition science and of food and nutrition policy are being revived."

It was then that von Liebig and his followers throughout Europe and then the USA blazoned chemistry as the solution for plant, animal and human breeding, and even as containing the secrets of life itself. This was the time when the priorities of chemical nutrition ceased to be conceptual and experimental, and became dictated by social, economic and political factors. Its prescription was protein of

animal origin. 'A vastly more important question than even the victualling of the navy. . .is that of victualling of the masses at home wrote a British commentator. 'What is at the moment deteriorating the lower stratum of the population? – the want of a sufficient supply of nitrogenous food. . .why should we not have meat too?'

Food Security and War

Nutrition science was in the big time. Its teaching and practice were at first dominated by Britain and Germany, and later by the USA and Britain. Half the economic growth in the UK and other Western European countries between 1790 and 1980 is attributed to improvements in population nutrition, together with other public health measures such as proper sanitation. Its impetus continued, with early twentieth century experiments identifying a series of diseases whose usual fundamental cause is deprivation and destitution as vitamin-deficiency diseases. In the USA, following Wilbur Atwater's work on energy and protein, Elmer McCollum and others established 'the newer knowledge of nutrition'. In Britain, the prescriptions of John Boyd Orr, Jack Drummond, Hugh Sinclair and others were adopted by government as an essential part of the 1939–1945 war effort, and the national food system was engineered so as to become more nourishing.

Boyd Orr, the most eminent founder of public health nutrition, was the first director-general of the Food and Agriculture Organization of the United Nations (UN) and to date the last nutrition scientist to be a Nobel laureate – a Peace Prize for his work advocating equity of world food supplies. While trained as a physician and physiologist, he embraced the environmental, social, economic, political, ethical and human rights dimensions of nutrition, and pressed its importance on policy-makers and in the media spotlight.

Boyd Orr had an impact on national public policy in Britain in the 1930s and 1940s comparable with that of von Liebig in the previous century. His proposals were built into programmes celebrated by Peter Medawar as 'the best single example known to me of synergy between science and government'18, which controlled and shaped the British food supply during the 1939–1945 war, and were a factor in its outcome.

Thus, between the mid-nineteenth and the mid-twentieth centuries, nutrition in its biochemical aspect became applied as part of central government policies in Europe and North America. The overall objectives of successive governments were internal social security, competitive advantage over other industrialised nations, and world domination. Consequent food and nutrition policies included legal, fiscal, regulatory and other methods affecting price, availability and quality.

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1950–2000 CE. DECLINE AND FALL

In the second half of the twentieth century the genie of nutrition was put back in the bottle. External forces – some global, others with special effect in the USA, the UK and their spheres of influence – made the theory and practice of nutrition less significant and more specialist, and nutrition scientists less confident and more defensive. The thrill had gone.

These forces and others explain the paradoxical waning importance of nutrition in a period when it became evident that food and nutrition modifies the risk of those chronic diseases that are now the major immediate causes of premature disability and death throughout the world. Many of these forces do not apply only to the science of nutrition and to nutrition and food policy. They can be identified under the headings of complacency, oligarchy, cacophony, technocracy and ideology.

Complacency

1. 'Conquest' of nutritional deficiencies

After the 1939–1945 World War it was generally agreed that human nutrition was no longer a subject worth serious scientific attention. Jack Drummond wrote: 'There is no problem of nutrition in Britain today. . . The position is perfectly clear-cut'. Donald Acheson, who in the 1980s as Chief Medical Officer controlled UK official nutrition policy committees, said when he was a medical student in the late 1940s: 'There were no remaining problems in human nutrition. . . All that was necessary was to eat a good mixed diet. . .avoid obesity and all would be well'. Problems of food insecurity and nutritional deficiency remained front-rank public health priorities for much of the world, but these were seen mostly as mundane issues of food supply, emergency aid or clinical intervention.

2. 'Conquest' of infectious diseases

Coincidentally, antibacterial drugs were first manufactured on a mass scale in the 1939–1945 war. In the 1950s and 1960s the first generations of antibiotics proved successful treatment for many transmissible diseases, and so seemed to prove the 'germtheory' of Louis Pasteur, Paul Ehrlich and others: not only that most (if not all) diseases are caused by microbes, but also that they can be cured by antimicrobial drugs. In the professional and lay mind diseases have become confused with external agents, needing aggressive intervention with pharmaceuticals administered by physicians. The discovery of the therapeutic power of antimicrobials, and thus the spectacular growth of the pharmaceutical industry, has led to the ascendancy of modern medicine and therefore to the decline of public health and of social and environmental aspects of nutrition.

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Oligarchy

3. *Maintenance of government control*

Between 1939 and 1945 democracy was modified or suspended in those countries fighting a war of national survival. National food supplies were shaped by government-appointed committees of civil servants, industrialists and scientists whose main task was to increase production and ensure food and nutrition security. Having got into the habit of secretive policy-making, the UK government maintained this closed committee system into the 1970s; and perhaps because of the freak chance that Margaret Thatcher was an industrial food chemist by training (having devised cake fillings before she entered politics) also throughout the 1980s. Many senior nutrition scientists advised government, subject to the Official Secrets Act. If they attempted to question any aspect of the national food system, their findings were ignored, overturned, suppressed or denounced.

4. *International agency unaccountability*

The victorious nations who controlled the newly formed UN specified that its policies on food and nutrition should focus on increased industrialisation and production of food in rich countries; and programmes whose stated purpose was to achieve food security and to control and treat nutritional deficiencies in poor countries. Until the 1990s the UN system was unresponsive to the evidence causally linking inappropriate food and nutrition to major chronic diseases. Remoteness of the UN agencies is as nothing compared with that of the World Bank, the International Monetary Fund and the World Trade Organization, whose policies and programmes, which increasingly shape global food systems, are commonly seen as an extension of the foreign and trade policies of the most powerful nations.

Cacophony

5. *Unexplained policy U-turns*

In the 1950s and 1960s it was said that fat should 'provide at least 25 per cent of the calorie value of the diet' and 'it is appropriate to increase this proportion to about 35 per cent'. From the 1960s the recommendation went from more to less: consumption of fat should be cut to 30% or 30–35% of calories, and from 1990 on a global basis to 15–30% of calories19,20,31. The explanation, that the shift reflected changed priorities from prevention of deficiencies to prevention of obesity and heart disease, was never successfully communicated to politicians or the public. Also demoralising to the profession was the 'great protein fiasco': the massive recalculation of human protein requirements in the 1970s which 'at the stroke of a pen' closed the 'protein gap' and destroyed the theory of pandemic 'protein malnutrition'.

6. Marketing and advertising babble

The spend on marketing and advertising of the largest food and drink manufacturers, most of whose lead products have nutritional profiles generally agreed to be unhealthy, continues to increase exponentially. The global spend of Coca-Cola and also of McDonald's was recently calculated to be $US 1.4 billion a year. Many manufacturers' messages conflict with others, and also with those of food retailers, and most conflict with the findings of nutrition science. Nutrition advice has been distorted by trade groups protecting the interests of milk, baked goods, soft drinks, sugar and salt, with massive budgets spent on lobbying legislators; and also by national and global nutrition foundations controlled or influenced by those sectors of industry whose products are energydense, high in fat, sugar and/or salt.

Technocracy

7. Accelerating specialism

From postgraduate to Nobel laureate, scientists are expected to undertake and publish original work. What this means is an exponential increase of more and more specialist research and journals. Nutrition scientists are not trained to see much of the context of their work and few can find the time to do so. Much research is minutely detailed, and a considerable fraction, especially in sensitive areas, is funded defensively by interested parties such as sectors of the food industry.

The recent drive originating in the USA to give pre-eminent credibility to the results of randomised controlled trials, a type of study whose main use is to test the efficacy of drugs would if generally accepted have the effect of vitiating most epidemiological and experimental studies designed to establish causal relationships between food and nutrition, and health and disease.

8. Corralling of science

Ever since the establishment of 'Big Science' in the USA to serve what President Eisenhower called 'the military–industrial complex', the scientific enterprise has become increasingly interdependent with government and industry, in general and in the biological sciences, including nutrition. Research scientists are now mostly dependent on funding from government and its agencies as well as from industry; and research institutes supported by public money are graded by ability to raise funds from industry. When such funding comes with expectations of a result favourable to the funder's policies, science is degraded and scientists demoralised. Scientists respond to such systematic pressure either by becoming detached from policy, or else by choosing to court government and industry funding.

Ideology

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9. Let the consumer beware

The laissez-faire ideology dominant since the 1980s is hostile to legal, fiscal and regulatory intervention in the public interest and to admission that food, nutrition and disease patterns have social, economic and political causes. Governments have withdrawn from public health, resist interventions designed to improve food systems, and mostly confine food and nutrition policies to information and education on prudent 'lifestyle'. Industry now lists some chemical constituents of processed food on standardised 'nutrition' labels. The approach generally adopted by governments is strikingly different from those for control of smoking of tobacco and drinking of alcohol, whose supply and demand are modified by taxation of price and regulation of advertising and marketing.

10. Band aid

UN agency programmes designed to eliminate food insecurity and deficiency diseases and achieve 'health for all' can be developed, maintained and sustained only if dominant nations are genuinely determined to address the fundamental causes of deprivation and disease. Instead, as dedicated officials within the UN system know and sometimes acknowledge, inequalities between and within high- and low-income countries are widening and the wealthiest nations continue to use food trade and aid in order to create increased dependency and indebtedness in the South. Aid agencies distribute, sell and promote food of minimum nutritional standard as famine relief, and together with UN agencies distribute and promote nutritional supplements and foods 'fortified' with vitamins and minerals in poor countries.

Concluding Remark

Nutrition scientists have taken a deep or broad view of the scope of their work and its implications since the beginnings of the discipline in modern form in the early nineteenth century; as have their predecessors whose teachings and writings laid the foundations of dietetics as an empirical discipline, and hence nutrition as a modern science.

The scope of conventional nutrition in its second phase in the second half of the twentieth century has been reduced and confined. This decline should be seen as historical, albeit not yet defunct.

1.3 INDIAN DIETETICS ASSOCIATION

In 1963, a band of nutritionists, dietitians and workers in the allied health fields resolved to form a scientific body to highlight the importance of dietetics and nutrition in the maintenance of health, and in the prevention and treatment

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of diseases. Thus, the Indian Dietetic Association was founded, with Prof. Kalyan Bagchi as Secretary and Dr. C. Gopalan as President. The association was affiliated to the International congress of Dietetics in 1975.

AIMS AND OBJECTIVES OF THE ASSOCIATION

- To promote the cause of science by encouraging the spirit of active pursuit of knowledge and original scientific research particularly in the field of Nutrition and Dietetics.
- To facilitate social, scientific and cultural fellowship and cultivation of goodwill among its members.
- To promote close contact and interaction between persons following different branches and thus facilitate the development of a wider outlook and the integration and application of available scientific knowledge for the welfare of society.
- To safeguard the interests of scientists generally and its members in particular and work for their welfare.

1.4 THE INDIAN DIETETIC ASSOCIATION CONSTITUTION

ARTICLE – I

The name of the Association is the "Indian Dietetic Association" (IDA), hereafter called the "Association".

ARTICLE – II

The Registered Office of the Association shall be, for the present, situated at AE – 645, Salt Lake City, Kolkata – 700 064 in the State of West Bengal. (How did the change of address take place from that mentioned in the Original Constitution? Please confirm which is your Original Constitution and Bye –laws, as have been filed with the Registrar of Societies, West Bengal. I am not touching this Document *i.e.*, the alleged 'Constitution' till you confirm, as changing the Constitution may require more than just your Association's approval. I am therefore only changing the Bye-laws.)

Awards:

Any new Award to be instituted carrier a minimum of Rs.25,000/- as corpus fund and the award will be give from the interest accured.

Appointment of Consultations:

Auditors can be appointed by NEC for IDA Tax Consultants and Lawyers can be appointed as and when the necessity arises.

ARTICLE – III

The aims and objectives of the Association shall be :

(a) To promote the cause of science by encouraging the spirit of active pursuit of knowledge and original scientific research in the field of nutrition and dietetics.

(b) To facilitate social, scientific and cultural fellowship and cultivation of goodwill among the Association members.

(c) To promote close contact between persons practicing different specializations of dietetics and thus facilitate the development of a wider outlook and the integration and application of available scientific knowledge for the welfare of society.

(d) To uphold dietetics as a profession in terms of academic and clinical training and research.

(e) To disseminate dietetics information through its official journal and other publications.

(f) To conduct conventions, seminars, symposia, workshop, etc., for the promotion of the science of Dietetics.

(g) To do and perform any or all other acts, matters and things as are conducive to, or incidental to, or necessary for the above objects.

ARTICLE – IV

The Membership of the Association shall be open to all those persons who qualify under the eligibility criteria of the Byelaws.

ARTICLE – V

The Association shall have the following statutory bodies :

(1) The General Body (GB)

(2) The National Council (NC)

(3) The National Executive Committee (NEC)

(4) Officers of the Association

The composition and functions of these statutory bodies shall be provided for in the Byelaws.

ARTICLE – VI

Regular meetings of the Association and its statutory bodies shall be held at such times and places as determined by the Executive Committee.

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ARTICLE – VII

Amendments to the Constitution may be made according to the provision of the Registrations of Societies Act of 1980 or any act, which may supersede it.

ARTICLE – VIII

There shall be awards and prizes instituted by the Association and/or other donors.

ARTICLE – IX

The Association may be dissolved through a specific resolution by a three-fourths majority of the voting members present at a regular Council meeting or one called specially for the purpose after giving the Council members sufficient due notice.

BYE-LAWS

Rules and Regulations as amended in _____

SECTION – I

Headquarters

The Headquarters of the Association shall presently be situated at AE – 645, Salt Lake City, Kolkata – 700 064 in the State of West Bengal. However, it can be moved to another place by a two-thirds majority vote of the Council Members present and voting at a general or special Council Meeting.

SECTION – II

2.1 Membership

There shall be the following classes of Memberships: Patron Members, Honorary Members, Ordinary Members, Associate Members, Life Members, Associate Life Members, Student Members, Corporate Members and Institutional Members..

1. Patron (P) :

Patron Membership may be conferred on a person :

(a) Of outstanding merit with national stature who has made significant contributions to the field of dietetics and/or rendered valuable services to the cause of the Association.

A Patron Membership shall be unanimously conferred by the National Executive Committee (NEC) of the Association.

2. Honorary Member (HM) :

Any person interested in the field of Dietetics and who believes and supports the aims and objectives of the Association is eligible for Honorary Membership

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on a one-time donation of more than Rs. 10,000/- provided she is nominated by unanimous vote of the NEC. The name of the donor shall be duly proposed and seconded for honorary membership by members of the Executive.

3. *Ordinary Member (OM) :*

A person is eligible for ordinary membership if she/he holds a degree in foods and nutritional sciences or their specializations and food technology from a recognized Indian University or its equivalent in other countries, and is willing to pay the Annual Membership Fee.

4. *Associate Member (AM) :*

A person is eligible for Associate membership if she :

(a) Is interested in dietetics and believes and supports the aims and objectives of the Association; or

(b) Has a degree in a field or discipline related/allied to dietetics and nutrition; or,

(c) Is engaged in teaching/instructing students studying in institutions or departments of dietetics and/or other nutritional sciences.

(d) And is willing to pay the Annual Associate Membership Fee.

5. *Life Member (LM) :*

Any individual who is eligible for ordinary membership as laid down by the Byelaws of the Association may become a Life Member by paying the One-time Life Membership fee.

6. *Associate Life Member (ALM) :*

Any individual who is eligible for associate membership may become an Associate Life Member by paying the One-time Associate Life Membership fee.

7. *Student Member (SM) :*

Any student of a diploma or degree course in dietetics or nutritional sciences and is willing to pay the Annual Student Membership Fee is eligible for student membership of the Association.

8. *Institutional Membership (IM) :*

Any non-governmental organization (NGO) or institution teaching or disseminating education in dietetics or foods and nutrition and which the Association recognizes and which is willing to pay the Institutional Membership Fee is eligible for institutional membership.

An Institutional Member is entitled to send not more than Four Representatives to attend any meeting of the General Body of the Association.

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However, the Institutional Member or its Representatives shall not stand for elections to any post or be nominated to any post or have any voting rights on any matters pertaining to or related to the Association or its affairs.

9. Corporate Member (CM) :

Any body entity interested in the field of dietetics and nutrition and promotes the aims and objectives of the Association may be permitted to become a Corporate Member by paying a one-time entry fee to Headquarters and thereafter pay to its local chapter the annual fee as decided by the NEC.

A corporate Member is entitled to send not more than Four Representatives to attend any meeting of the General Body of the Association. However; the Institutional Member or its Representatives shall not stand for elections to any post or be nominated to any post or have any voting rights on any matters pertaining to or related to the Association or its affairs.

2.2 Admission to Membership

A person or individual desiring membership shall be proposed for membership by an existing member who has become eligible to vote. Persons seeking membership other than the Patron or Honorary Membership. Shall apply to the respective Chapter President or the General Secretary of the Association in writing in the prescribed form with copies of their bio-data, certificates and credentials accompanied by the Proposal Letter. Persons seeking Patron or Honorary Membership shall apply to the General Secretary of the Association In writing in the prescribed form with copies of their bio data, certificate, accompanied by the Proposal Letter.

The registration and membership fees shall be enclosed with the application.

The application shall be placed before the Chapter or National EC for consideration. An applicant being admitted to membership will be deemed to have agreed to abide by the rules and regulations of the Association that may be in force from time to time. The EC shall rule on whether the applicant meets the criteria for membership satisfactorily.

Updated directory of IDA Member list should be Published every 2 years.

2.3 Fees :

The executive committee shall propose all dues for different categories of members. These decisions will be implemented with the concurrence of the general body. Membership shall be counted from 1st January to 31st December for purposes of annual subscriptions.

The Membership Fee Structure shall be announced by the Association on its Official Website or in such manner as is normally done.

Members who have not paid their subscription by 31st December shall be sent a registered acknowledgement due notice by the Treasurer of the Society giving them till 31st March to clear all outstanding dues. Non-payment of subscription by 31st March would result in removal of the member's name from the rolls.

Members who have not paid their dues by 31st March, are not eligible to vote in that calendar year.

Members whose names have been erased from the rolls of the Society may be re-admitted to the status of their original Membership after receiving the prescribed application form and re-admission fee.

2.4. *Registration and Other Fees :*

Patrons and Honorary members are exempted from registration fees.

A registration fee will be charged for all other types of new members.

2.5 *Migration Fee :*

Life Members or Associate Life Members who wish to transfer their membership from one chapter to another should first inform in writing the parent chapter of their intention to migrate. They are then required to pay a one-time Migration Fee to the chapter they seek an affiliation to together with a copy of the letter written to their Parent chapter.

Official Communications:

The official mode of communication of all Notices, including Election Notices, Membership Fee structures, *etc.*, shall be the official website of the Association, which at present is *www.idaindia.com.*

All Notices shall be posted on the website for a period of at least Four months continuously.

The member shall check the official website for all notices at least every quarter *i.e.*, on December 31, March 31., June 30 and September 30. The Association or its Statutory Bodies shall not be responsible for the ignorance of a Member due to not accessing the Official Website at least every quarterly.

The Association discourage the postal Method of sending Notices to individual Member in order to save costs and the environment.

The President shall be the custodian of the Official Website.

(Code of Conduct of Members (Rights, Responsibilities and Obligations)

(1) A member should regularly attend the Annual general meetings of the Association, as also other local Chapter/Council events such as seminars, workshops, lectures, discussions etc.

(2) A member should not lend/couple her/his name with any merchandise so that it appears that she/he is promoting or advertising a brand.

(3) Members should abide by the Professional Code of Ethics laid down by the Association and use their best efforts to promote the objectives of the Association.

(4) Members shall have the duty to pay membership fees on time.

(5) Only those members who have been awarded the R.D. (Registered Dietician) by the IDA are officially permitted to affix R.D. after their names.

2.6 Cessation of Membership

Membership shall cease :

On receipt of a written resignation by the member;

1. If fees are in arrears for more than twelve months after the last date they are due, *i.e.*, December 31st. However, the NEC or CEC, on the request from the defaulting member may grant an extension of time. If the person gets admitted to the Association again after cessation of Membership, such admission shall be a new admission and the member shall not get the privileges of her earlier membership.
2. If any member is found working against the interest of the Association or is convicted in a criminal offence she/he may be removed from membership by the Executive Committee after giving the member a chance to defend his/her case before the Disciplinary Committee.
3. If any member hampers or attempts to hamper the working of the Association or brings or attempts to bring the working of the association to a standstill, with or without legitimate cause, she may be removed from membership by the Executive Committee after giving the member a chance to defend his/ her case before the Disciplinary Committee. The members acknowledge that whatever differences may arise within the Association, including differences between the members or any member and the management or between chapters, such grievances shall be addressed only through the Grievance Redressal Mechanism mentioned in these Bye-laws.

SECTION – III

3.1 Structure of the Association

The Association shall facilitate the organization of its Members into Chapters. Such Chapters shall not have a separate legal status and are organized only for the purposes of administrative convenience and efficient functioning.

The Chapters shall have its organization similar to the Association as stated under these bye-laws. The Chapters shall not have separate Bye-laws.

A Chapter can be formed if 50 Life Members residing within a geographical area apply to the National EC for recognition as a chapter of the Association.

The Application should include a detailed list of the number and type of members, together with the affiliation fees. To form a chapter members may be new or existing ones. In case of new members, their filled in membership forms together with their entire membership and registration fees should be submitted with the application.

The Executive Committee will rule on whether the applying group/ organization satisfactorily meets the criteria for recognition as a Chapter.

If the Chapter President is not able to attend any general and/or special meeting of the Association, she may depute before the said meeting, in writing, the name of the officer/member who will substitute her as official representative at the said meeting. However, such official representative shall not exercise the voting rights of the Chapter President.

The chapter shall appoint an auditor every year at the beginning of every year and such information shall be conveyed to the President for forwarding the same to the NEC.

Each chapter shall help the HQs in collecting all the dues from the members belonging to their chapter and shall take turns in hosting the Annual Convention of the Association. They shall also remit 25% only from the Registration fees collected during the convention to the accounts of the HQ and the ICDA; *i.e.*,' to each one alternately.

Each chapter shall remit 50% of the membership fees collected from life members enrolled in the current calendar year to HQ. which in any case, shall not be less than the amount equivalent to 50% of the membership fees of Five life members and such amount shall be paid by the chapter even if it unable to enroll new Life Members.

Each Chapter shall send the updated membership list as on March 31st of every year to the HQ.

At every Annual General meeting the Secretary of each chapter should present the report of the activities of the chapter for the past one year and the treasurer should present the audited financial report for the past financial year. In the event of their inabilities to attend the A.G.M., they may nominate a member of the chapter to attend the meeting and present the reports. Each chapter shall conduct periodic academic meetings and symposiums on its own or in

collaboration with other scientific organizations. It shall also endeavor to carry out other activities such as those outlined earlier.

Each chapter shall have its own Executive Committee consisting of President, Vice-President, Secretary, Joint Secretary, Treasurer and atleast three Executive Committee members.

Each chapter shall conduct elections once in 4 years within one month after the election of National Executive Committee, so that the term of its Executive Committee synchronises with that of the National Executive Committee.

The elections to the EC of the Chapters shall be held in the same manner as prescribed for the NEC.

In case of any conflict between the provisions applicable to Chapters and the provisions applicable to the IDA Association, the latter shall prevail.

The Chapters shall actively promote the participation by all their members in the activities of the Association at the National level.

SECTION – IV

4.1 Statutory Bodies of the Association

The Association shall consist of the following statutory bodies :

- General Body (GB)
- National Council (NC)
- National Executive Committee (NEC)
- Officers

SECTION – V

5.1 The General Body (GB)

All the Members of the Association, including the members without voting rights, constitute the General Body.

Annual Conference and Annual General Meetings

The Annual Conference is an Annual Scientific Session that is open to all categories of members of the Society, and others, including practitioners and students of medical and allied sciences on payment of appropriate delegate fees.

The Association shall try to organize the presentation of papers and/or special lectures and/or seminars/ workshops and receive and implement suggestions in this regard from the General Body.

There shall be atleast ONE Meeting of the General Body (Annual General Meeting) every year.

NOTES

The National Executive Committee shall fix the time and place of the Meetings of the General Body.

Notice of the AGM with the Agenda shall be posted on the Official Websites of the Association or in the alternative, sent to all the eligible members, atleast twenty three days prior to the date of the AGM.

The AGM shall, if not otherwise found inconvenient by the NEC, be held simultaneously with *i.e.*, immediately before or after, an Annual Conference.

The business of the Annual General Meeting shall ordinarily be as follows :

- Address by the President;
- Announcement of the names of the awardees for the various prizes instituted by the Association;
- Reports of the registration board;
- Resolutions and recommendations as approved and passed by the NEC and Council;
- Annual reports of the Hony. General Secretary and the Hony. Treasurer;
- The National Election Officer shall declare the results of the election (only during the election year);
- Reports of affiliated chapters/member organizations;
- Any other matters that form part of the Agenda circulated alongwith the Notice of AGM.

The Quorum for the AGM is 1/3rd of all members registered to attend the AGM. However, if there is deficient quorum at the Scheduled time of the meeting, the AGM shall be adjourned and reconvened after 30 minutes. At such reconvened meeting, business shall be proceeded with as per the Agenda, even in the absence of a quorum.

The Annual General Meetings and other General meetings shall be chaired by the President of the Association, and in her absence, by such person as appointed at the start of the meeting by the NEC members attending that meeting.

SECTION – VI

6.1 The National Council (NC)

All the members of the Association who are eligible to vote comprise the National Council.

Only those members of the Association who have been members for a continuous period of atleast One calendar year and who have no dues owing to the Association are eligible to vote.

There shall be atleast ONE Meeting of the Council (Annual Council Meeting) every year.

The Annual Council meeting shall, if not otherwise found inconvenient by the NEC, be held simultaneously with *i.e.*, immediately before or after, an Annual Conference.

The Council shall delegate to the NEC the powers to implement its policy decisions.

Carrying out of the programmes and policy of the Association.

The POWERS and DUTIES of the Council shall include the following :

- To receive reports and accounts from the Executive Committee and the Standing Committees.
- To decide the policy and work of the Association.
- To be responsible for the management of the Association.
- To decide the types of membership and their fees.
- To elect the Executive Committee.
- To approve the budgetary allocations and programmes of the Association.
- To choose the venue, time and theme of the Annual Conference.
- To make decisions relating to the Constitution and Byelaws of the Association.
- To ratify resolutions.
- To approve the Minutes of the previous Council Meetings.
- To Consider the reports of the chapters, different committees of the Association and IDA representatives at international dietetic associations.
- To approve the Report of the Registration Board.
- To approve the Report of the Editor-in-Chief and the Publication Secretary.
- To appoint the Auditors for the next year.
- To do any other business inherent to its nature as the democratic body of the Association.
- To do such acts as are necessary for the needs and running of the Association.

The National Executive Committee shall fix the time and place of the Meetings of the Council.

Notice of the ACM with the Agenda shall be posted on the Official Website of the Association or in the alternative, sent to all the eligible members, at least twenty three days prior to the date of the ACM.

NOTES

The ACM shall, if not otherwise found inconvenient by the NEC, be held simultaneously with i.e. immediately before or after, an Annual Conference.

The Quorum for the ACM is 1/3rd of members. However, if there is deficient quorum at the Scheduled time of the meeting, the ACM shall be adjourned and reconvened after 30 minutes. At such reconvened meeting, business shall be proceeded with as per the Agenda, even in the absence of a quorum.

The Annual Council Meetings and other Council meetings shall be chaired by the President of the Association, and in her absence, by such person as appointed by the NEC.

The Council shall not vote or decide on any new issue other than those specified in the Agenda circulated alongwith the Notice of ACM.

All proposals for amendments to the Constitution and Bye-laws shall be circulated to all the Members of the Association by the NEC atleast 2 months before the General Body meeting and Council Meeting.

Such proposals shall be accepted if 2/3rd of the members present and voting, vote in favour of the proposals.

The Amended Constitution and Bye-Laws shall be posted on the official website of the Association or, in the alternative, circulated to all the Members o0f the Association by the NEC within 2 months of the ACM at which the amendments are accepted.

SECTION – VII

7.1 The National Executive Committee (NEC)

There shall be a National Executive Committee for implementing the policies of the NEC and for the conduct of the affairs of the Association.

The Executive Committee shall consist of the following Elected, nominated and Ex-Officio Members:

(a) Elected Members:

The following members shall be elected:

- The Honorary President
- Three Honorary Vice-Presidents
- The Honorary General Secretary
- The Honorary Joint Secretary (from Headquarters)
- The Honorary Treasurer (from Headquarters)
- Three Executive Committee Members (from Headquarters)

NOTES

- Seven Executive Committee Members
- Publication Secretary

Every member who has been a member of the Association for a continuous period of atleast three years is eligible to contest the election for any post or be nominated to any post, save as follows:

Only a member residing in the District where the Headquarters of the Association is situated is eligible to contest the election for the following posts: ONE post of Honorary Joint Secretary, ONE post of Honorary Treasurer, TWO posts of Executive Committee Members. To clarify, FOUR posts as described above are open to contest only by eligible members residing in the District where the Headquarters of the Association is situated, and not by members residing anywhere else.

For the avoidance of doubt, it is clarified that this provision is inserted to enable the records of the Association to be maintained at the Headquarters conveniently and with the assistance of members getting elected to the above posts.

All the other elected posts are open to contest by eligible members residing anywhere in India.

(b). Only a Life Member who is a senior dietician, nutritionist and/or senior academician and who has been a Member of the Association for atleast 5 (five) Continuous years at any point of time and has been a member of the NEC at any point of time before is eligible to contest the election for the post of President.

7.2 Co-opted Non-elected Members :

- Honorary Joint Secretary
- Editor-in-Chief
- All members who have been a member of the Association for a continuous period of atleast three years are eligible to be nominated.
- The Editor-in-Chief shall be nominated by the Publication Secretary.
- The Honorary Joint Secretary shall be co-opted by the President. The co-opted members are eligible to attend the EC meetings, but do not have the right to vote at such meetings.

7.3 Ex-Officio Members :

- The Immediate Past President of the Association
- A member designated by the Executive Committee to represent IDA at International Conference of Dietitians Associations (ICDA).

- Any member of the association who is an officer or EC Member of the International Dietetic Association (ICDA), during the period she holds office in the international forum
- Presidents of affiliated chapters

Invitees :

As and when the Executive Committee feels the need it may invite any member to attend an EC meeting, but such member shall not have the right to vote at such meetings.

There shall be atleast two meetings of the NEC every year; the one immediately preceding the Annual Convention and two, after six months of the Annual Convention.

SECTION – VIII

8.1 Officers

The Officers of the Association shall be :

- The President
- The Immediate Past President
- The Vice-President
- The General Secretary
- The Honorary Treasurer
- The Joint Secretaries
- Chairperson, Registration Board
- Publication Secretary
- The Officers have the duty of implementing the decisions of the Executive Committee.

8.2 Duties of the Officers :

8.2.1 The President

- The President shall represent the Association.
- The President shall chair all the meetings of the General Body, the National Council and the National Executive Committee.
- The President shall be supervise the functioning of the Association.
- The President shall run the association in co-ordination with the NEC.
- The President shall coordinate the work of the Standing Committees. She shall be an ex-officio member of all such committees.

8.2.2 The Immediate Past President (IPP)

The Person holding the post of the President at the time immediately before the new National Executive Committee is announced for every term shall be the Immediate Past President.

The IPP shall be an Ex-officio member of the new NEC.

She shall be the National Election Officer (NEO) and in overall charge of conducting the elections of the Association.

The President with the Secretary and the Treasurer, acting jointly, are authorized to represent the Association in all legal or Court related matters.

8.2.3 The Vice-Presidents

The Vice-Presidents shall perform all duties assigned to them either by the President or the Executive Committee. They will have the responsibility to coordinate regional activities, ensuring two-way dissemination of information between IDA and the region they are assigned to and maintaining contact between members.

In the event of the absence, death or enforced withdrawal of the President, the Executive Committee shall have the power to decide which of the Vice-Presidents shall assume the office of the acting President until the next meeting of the Council when elections take place.

8.2.4 The General Secretary

The General Secretary shall conduct all her duties from the Headquarters of the Association. She shall maintain all the records of the Association in a suitable form for ready reference. She shall be responsible for sending the Notices together with the relevant documents and keeping a record of all the proceedings and minutes of the Association, General Body, Council and Executive Committee.

Together with the Honorary Treasurer she shall be responsible for maintaining an up-to-date Member's Register. She shall conduct any correspondence on behalf of the Association as directed by the President or EC. She shall keep a suspense sum not exceeding Rs. 500/- for expense, reimbursement of which is subsequent to approval of the Executive Committee. She shall report to the President and EC as and when required. She shall prepare the annual report of the Association and present it at the Annual General Meeting. She shall also perform any duties inherent to her position.

8.2.5 The Honorary Treasurer

The Treasurer shall operate funds of the Society. She will be responsible for / to: — Investments and disbursements as per decisions of EC, Sending reminder

of dues to defaulting members, maintaining account books, getting the audits done, ensuring that all bank accounts are operated as per authorizations only.

The Treasurer shall be responsible, in consultation with the EC, for all matters concerning the finances of the Association including the presentation of the Annual Financial audited Report and Budget.

She shall keep all funds and other properties of the Association and collect all dues and donations.

She shall disburse money as authorized by the NEC and submit a financial statement as and when required and discharge all other functions inherent to the position.

She shall maintain an up-to-date list of all members and submit to the General Secretary, every month, a list of new members and a statement of the payment and dues against each member.

She shall operate bank accounts jointly with the President or the general Secretary.

8.2.6 The Joint Secretaries

The Joint Secretary (Headquarters), will assist the General Secretary in the execution of her duties. She shall also be a member of the Publication Committee and work in close coordination with the Editor of the Journal.

The Joint Secretary (President's Secretariat) will perform all secretarial duties assigned to her by the President. She shall be in charge of the President's Secretariat and will work in close cooperation with Headquarters.

8.2.7 The Joint Treasurer

The Joint Treasurer (President's Secretariat) will perform any duties, inherent to her position, which may be assigned to her by the President.

8.2.8 Chairperson, Registration Board

She will be in overall charge of the working of the Registration Board and report to the Executive Committee.

8.2.9 Publication Secretary

She shall convene all meetings of the Editorial Board.

She shall work in co-ordination with the Chief Editor.

SECTION – IX

9.1 Term of Office

The Term of Office of the Executive Committee shall be four years starting from the first of January nearest to the day of election.

NOTES

The same person shall not contest for or hold the post of President of the Association for two consecutive terms.

No person shall hold the same post in the EC for more than two consecutive terms

If the President resigns/retires/dies/ is expelled during her term one of the two Vice Presidents (non headquarters) shall act as the President for the remainder of the term.

If the Secretary resigns/retires/dies/ is expelled during her term the elected Joint Secretary shall act as the Secretary for the remainder of the term.

If the Treasurer resigns/retires/dies/ is expelled during her term the Joint Treasurer available as the treasure nominated by the president shall act as the Treasurer for the remainder of the term.

No person shall hold two posts at one time at the NEC. Any person holding a post at the NEC shall not contest for a post in the Chapter EC.

SECTION – X

Committees of the Association

Standing Committees :

10.1 The Publication Committee :

A Publication Committee shall be constituted to help in bringing out the publications of the Association. The Committee shall consist of

- Chairman — President
- Convener — Editor-in-Chief
- Members — General Secretary and Honorary Treasurer
- Publication Secretary and two members appointed by the EC.

The Editor-in-Chief shall be the Chief Editor of the Official Journal and its printer and publisher. He/She shall be in overall charge of the publications of the Association.

There shall be an Editorial Board to help edit the official journal of the Association. Editors on this Board shall be nominated by the President in consultation with the Editor-in-Chief who will be the chairperson of this Board.

10.2 The IDA Registration Board :

The Registration Board will consist of the President, the Chairperson,and five members out of which three shall be senior practicing dieticians and two senior academicians. The outgoing Chairperson will be an ex-officio member.

The Chairpersons shall convene the meetings of the RD Board.

The members will be appointed by the EC in consultation with the Registration Board Chairperson.

The Registration Board Chairperson and its Five members shall be elected bysecret ballot. The elections shall be conduced by the President. The NEO shall send the biodata of the candidates standing for election to all NEC members.

In the event that the candidates standing for election of the post of RB chairperson and the Five constituent members are less than the requisite number of posts, the newly elected RB chairperson shall fill in the remaining seats by co – opting any member with the approval of the EC.

The Board shall be responsible for all the activities related to the selection, curriculum, examination etc., required for registration of dieticians. The Executive Committee shall pass the syllabus.

The tenure of the RD Board shall run concurrently with the tenure of the NEC.

The Disciplinary Committee:

The Disciplinary Committee shall consist of three members of the Association who have been members for atleast 10 years.

The Disciplinary Committee shall be appointed by the EC immediately after the new EC is elected.

If a complaint is made to the Disciplinary Committee about one of its members, such member shall not sit on the Disciplinary Committee while she is being enquired against.

The Disciplinary Committee shall hear all the complaints of indiscipline against any member of the Association, in a fair and just manner.

The Disciplinary Committee shall recommend the punishments.

The Disciplinary Committee shall endeavour to dispose of every complaint brought before it. Within a period of three months from the date of the complaint.

The Disciplinary Committee shall adopt its procedure to hear complaints.

The Grievance Redressal Committee:

The Grievance Redressal Committee shall consist of three members of the Association who have been members for atleast 10 years.

The Grievance Redressal Committee shall be appointed by the President immediately after the new President is elected.

The Grievance Redressal Committee shall endeavour to settle all disputes within the Association through Mediation and Reconciliation. The Grievance

NOTES

Redressal Committee will be looked upon to ensure the smooth functioning of the Association's activities in a spirit of co-operation and camaraderie between all members and chapters.

All disputes arising out or in relation to these byelaws, including the interpretation hereof, and all disputes between the members, Chapter or Statutory Bodies. In any combination whatsoever, pertaining to the affairs of the Association shall be decided by Arbitration to be conducted by the Grievance Redressal Committee. In the event that any or all members of the GRC is/are personally involved in the dispute. The president shall appoint other members to take the place of such members for the settlements of such dispute. The decision of the GRC shall be binding on the members.

The terms of these Standing Committees shall synchronize with that of the Executive Committee. They shall function as per the rules and guidelines laid down by the Council from time to time.

Other Committees :

The President in consultation with the EC may appoint the following committees whenever needed :

- Advisory Committee
- Finance Committee
- Membership Committee
- Programmes Committee
- Publicity Committee
- International Relations Committee
- Any other Committee as shall be required by the Association.

The members of the Finance and Programme Committees shall be selected out of the EC members. The members of the other Committees need not be EC members.

The members of the International Relations Committee shall be appointed by the NEC in consultation with the ICDA representative who will also be nominated by the NEC.

The President shall be the ex-officio Chairperson of all the Other Committees. In the absence of the President the Convener will chair that session.

The Convener of all the Committees, other than the Nominating Committee and the IDA Registration Board, shall be appointed by the EC.

The words EC and NEC are interchangeable within these byelaws.

NOTES

SECTION – XI

11.1 Elections :

The Elections to the Executive Committee shall be held after every four years.

The persons who holds the post of Immediate past president at the time the election process to elect the New EC is to begin shall be the National Election Officer and she shall be responsible for the conduct of the Elections.

The National Election Officer shall not be eligible to contest for any post in the elections conducted by her.

No member is eligible to contest for more than one post in the same election.

The members of the National Council alone shall vote at the elections for appointing the EC.

Elections shall be held by postal ballot, prior to the Annual Council Meeting of the Quadrennial Year.

The NEC shall have the right to change the logistics of the election process in the face of exigencies or introduce byelaws for other modes of voting like electronic voting as and when required, after the approval of the General Body.

The Election Process:

The election process should start not later than ten months before the annual convention in which election results are to be announced, referred to here as the "quadrennial election convention".

11.2 Electoral Roll :

The NEO shall prepare the Electoral Roll. As prelude to finalisation of electoral roll, all chapter Presidents shall prepare the list of eligible life members of her/his chapter after the due scrutiny of names and addresses, and dispatch the same, by registered post, to NEO within 31st March of the election year.

All chapters 'voters' lists shall next be scrutinized by NEO at her/his end and then all such lists together shall be treated as electoral roll under the authority of NEO.

Invitation for Nominations:

The NEO shall notify the date of the Proposed Election, Posts open for Election and invite nominations duly proposed and seconded by Life Members along with consent of candidate with brief biodata in prescribed format.

Such Notifications shall be, by Registered Post Acknowledgement Due or by courier where necessary sent directly by the NEO to the member's address for Communication;

NOTES

The Election Notification shall be accompanied by the Nomination and Consent Forms and shall specify the Last date and time for receipt of the Nominations by the NEO.

Candidates desiring for contesting the election must note that they can submit their nominations for one post only. Nomination paper in the name of one individual candidate for more than one post will not be treated as valid.

Nominations unaccompanied by consent letter are to be treated as invalid.

Compilation of Candidates' List:

After the last date specified for the receipt of Nominations, the NEO shall compile a List of candidates seeking election for each post and post it on the Official website. NEC is not eligible to vote.

11.3 Ballot Paper – Dispatch and Collection :

The NEO shall prepare a Final List of Candidates contesting the Elections and such List shall be printed on the Ballot Papers. The NEO shall prepare the Ballot Papers and dispatch the same to every eligible voter after affixing her/his signature by hand on every Ballot papers. The NEO shall issue clear instructions to all voters on the method of signing and return of the Ballot papers. The signed Ballot papers shall be returned to the NEO by each voter seperately in a Sealed Envelope, and another signature affixed over the seal of the envelope.

Ballot papers/envelopes of more than one voter if sent back in one cover (joint dispatch) would make all those votes invalid.

NEO shall prepare a sheet regarding total number of ballot papers dispatched and returned undelivered and keep the sheet ready for use during counting of Ballots at AGM.

The NEO is not eligible to vote in the election.

11.4 Voting:

Members shall vote by marking their candidates of choice for the various posts on the ballot paper and then follow the instructions of the NEO regarding dispatching the ballot envelope.

11.5 Counting of Votes :

The NEO shall keep the ballot envelopes unopened 'as received' after clearly indicating the date they were received. By signing across the pasted portion on receipt of the envelope along with the date.

The Votes shall be counted one day prior to the Annual General Meeting.

The NEO shall use the assistance of the Election Officer chosen by her/him in a representative manner from the different Chapters to scrutinize all the Envelopes from outside to rule out tampering. The Election Officers so Chosen shall only be from among Members who are not contesting for any post during that election.

Then the seals shall be removed and the ballot envelopes again checked for validity before removing the ballot papers. Finally the NEO shall supervise the counting of the votes.

The results of the elections shall be tabulated on a sheet of paper and attested by all those present during counting and shall be declared by the NEO at the AGM.

11.6 Documents, Records and Accounts :

The NEO shall keep custody of all papers, documents and records in connection with the election held under her supervision for a period of Six months after the election results are declared. Thereafter, the NEO shall hand over all these papers with proper authentication to the General Secretary of the New EC for safe and record keeping until the next election.

SECTION - XII

12.1 Special Meetings

Special meetings of the Association may be convened by the EC or requisitioned by at least 50 Council members for transacting specific business. The request for requisition shall be addressed to the General Secretary specifying the object for which the meeting is to be convened. On receipt of such a requisition, the EC shall cause a meeting to be convened within two months of its receipt at such time and place as may be decided by the EC. The General Secretary shall give at least three weeks notice to the members and no business, other than that notified, shall be transacted at this meeting.

SECTION - XIII

13.1 Awards and Recognitions

For the present the Association may confer the following awards and recognitions instituted by :

A. The Association

- The IDA President's Award
- A Suitable prize will be awarded at the Annual IDA Convention to the winner of the best poster, sponsored from the President's chapter.

B. Donors

- K.G. Naidu Memorial Trust Medal
- J.N. Bose Memorial Medal

- Sagarmal Goenka Award
- A.N. Radha Award
- Dr. Amiya Kumar Bose Memorial Lecture (Sponsored by Bengal Chapter)

The Executive Committee in consultation with the donors will frame rules and Guidelines for giving awards and prizes.

Any body who wishes to institute a new Award shall contribute a minimum of Rs.25,000 as a corpus fund. The Award shall be given from the interest accrued.

The Council shall be the final authority for approving the institution of any prize, award or recognition by the Association.

SECTION – XIV

Journal of the Association

The name of the official journal of the Association shall be "Journal of the Indian Dietetic Association".

For the present it shall be published twice a year.

The EC shall decide on the subscription and other guidelines for the publication of the Journal, which may be changed from time to time.

The Editor-in-Chief will be the chief editor, printer and publisher of the Association's official journal.

In consultation with the Editor-in-Chief, the EC shall form an Editorial Board, which will be responsible for the publication of the journal. The Board members shall be nominated from among the members of the Association. The term of the Editorial Board shall synchronize with that of the Executive Committee.

SECTION – XV

15.1 Fiscal Year

The fiscal year shall begin on the First of April every year.

15.2 Authorised Signatories

The Association shall be represented in any Legal proceeding or before any Court / Tribunal / Authority of Law only by the President, and General Secretary all acting jointly. In a situation where one of the above is unable to sign, the NC shall decide on an alternative signatory.

All bank accounts, deposits and other investments shall be opened, kept and made in the name of the Indian Dietetic Association.

The bank accounts shall be operated as follows:

- All cheques shall be signed only by the Treasure acting jointly with the President or the Secretary. In a situation where one of the above is unable to sign, the NEC shall decide on an alternative signatory.

- All Bills for amounts exceeding Rs. 5000/- (Rs. Five Thousands only) shall be paid only after obtaining the prior approval of the NEC.
- Members attending the NEC meetings are entitled to the epenses for travel from residence to the meeting destination at AC 2 tier train ticket rate. The travel expenses for the National President, Vice-President, Secretary ; Joint Secretary (Headquarters) and Publication Secretary shall be borne by the Headquarters. The travel expenses for the Executive Committee members (Headquarters) shall be borne by the Chapter which hosts the headquarters. The travel expenses of the RD Board members shall be borne by the RD Board and that of the ICDA representative by the ICDA Account. The travel expenses of all other EC members shall be borne by their respective Chapters.
- The accounts of the Association shall be consolidated with all the accounts of all the Chapters duly audited of each respective chapter.
- The accounts shall be circulated to all members of the EC prior to the EC meeting and such accounts shall be placed before the NEC for approval.
- No members of the NEC shall be responsible for any arising in any audited accounts of any chapter and each Chapter president and the members of the local chapter shall be responsible for the same.
- All Chapter treasures shall furnish the accounts of the Chapter at the end of every year after approval by the executive committee of the Treasure of the Association duly audited every year.
- Title to the real property of the Association shall be vested in the Executive Committee who shall have power to sell, lease. Mortgage or otherwise deal with the same only with the prior approval of the NEC.
- The Exective shall hold all funds of the Association using the capital and/ or income from them for the purpose for which they are held. All life membership amounts shall be invested as fixed securities and the interest utilized for Association purposes.
- At the end of every convention the auditor of the local chapter shall audit the accounts of the convention and send it to President for placing the same before the NEC. These audited accounts shall be independent of the regular accounts of the chapter.
- Budgets shall be prepared in consultation with the secretary and Treasurer and placed before the NEC for approval.
- On the recommendations of the EC, the Council shall appoint the auditors.
- Auditors appointed at one annual council meeting will audit all papers and books of accounts of the Association for one year and are eligible for reappointment .

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SECTION – XVI

Indemnity

Members of the Association, Executive Committee and the Officers and their heirs, executors and/or administrators shall be indemnified and saved harmless out all actions, costs, losses, damages etc., resulting from execution of their duties of the Association.

CHAPTERS:

The chapter shall translate the sprit of these byelaws at the Chapter level. The chapter shall hold atleast FOUR academic meeting in a Year to update its members in the field of dietetics and nutrition.

The chapter shall co-operate with the NEC to ensure the smooth administration of the affairs of the Association and shall endeavor to enroll new members and shall endeavour to create an atmosphere of Study , Research and Discussion. The Chapters shall celebrate all nutrition and dietetics related important events.

SECTION – XVII

Dissolution

In the event of need to wind up the Association, a special general Council meeting shall be called expressly for this purpose, that is after due notice to all the members of the Council.

Resolutions on the dissolution of the Association shall require a three-fourths majority of the votes of the Council members present.

In the event of dissolution of the Association, the Executive Committee shall decide the manner in which claims and liabilities are to be disposed off. The remaining assets, if any, shall be distributed to organizations or institutions with similar objectives as decided by the Executive Committee.

The President of the Association shall be the liquidator.

STUDENT ACTIVITY

1. Discuss the History of Dietetics.

2. Discuss about Indian Dietetics Association.

1.5 DIETICIAN

A dietician is an expert in food and nutrition. Dieticians help promote good health through proper eating. They supervise the preparation and service of food, develop modified diets, participate in research, and educate individuals and groups on good nutritional habits. In a medical setting, a dietitian may provide specific artificial nutritional needs to patients unable to consume food normally. Dietary modification to address medical issues involving dietary intake is also a major part of dietetics. The goals of the dietary department are to provide medical nutritional intervention, obtain, prepare, and serve flavorsome, attractive, and nutritious food to patients, family members, and health care providers.

The work of the clinical dietician is based upon the fundamental human need for energy and nutrition. To have one's energy and nutritional requirements fulfilled is an undisputed human right. This is also obvious from a general, ethical point of view. Illness, and medical treatment linked to illness, can however make it difficult or even impossible for an individual to meet these requirements without assistance. At the same time the illness itself may change the requirements. Social, psychological and cultural factors also influence the conditions for an optimal energy and nutritional intake. Food, mealtimes and eating also involve many aspects and can be organized in many formats. Taken together, all these factors constitute the foundations of the profession of the clinical dietitian. The work of a clinical dietitian is based on the science of nutrition. Professional practice requires an academic education which includes clinical training. This makes the dietitian uniquely qualified to carry out prevention and treatment of nutritionally related conditions.

In many countries only people who have specified educational credentials can call themselves "dietitians" – the title is legally protected. The term "nutritionist" is also widely used; however, the term nutritionist is not regulated as dietitian is. People may call themselves nutritionists without the educational and professional requirements of registered dietitians. Dietetic technicians are not the same as dietitians in terms of responsibilities and qualifications. Different professional terms are used in different countries. Dietitians are a valuable member of the medical multi-disciplinary team providing nutritional knowledge and acting as consultants to other health care professionals.

Definitions of the Term Dietician

A number of definitions of "dietician" from other organisations given below:

The European Federation of the Association of Dietitians (EFAD):

"A dietician with an education focused on clinical nutrition and dietetics with responsibility for dietary prevention and treatment of individuals, in an institution or a community".

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In this Code, the term 'clinical dietitian' refers to a 'registered dietitian with protected professional title'.

- **Clinical Dietician:** a dietician who has responsibility for planning, education, supervision and evaluation of a clinically devised eating plan to restore the client's/patient's functional health. Clinical dietitians can work in primary care as well as in institutions.
- **Public Health or Community Dietician:** a dietician directly involved in health promotion and policy formulation that leads to the promotion of food choice amongst individuals and groups to improve or maintain the nutritional health and minimize risk from nutritionally derived illness.

The International Confederation of Dietetic Associations (ICDA):

- A dietitian is a person with a qualification in Nutrition and Dietetic recognized by national authority(s). The dietitian applies the science of nutrition to feeding and education of groups of people and individuals in health and disease.
- The scope of dietetic practice is such that dietitians may work in a variety of settings and have a variety of work functions.

ASPEN Standards of Practice for Nutrition Support Dietitians:

- The NSD is a registered dietitian with clinical expertise or credentialing in nutrition support obtained through education, training, or experience in this field.
- The NSD assures optimal nutrition support through *(a)* individualized nutrition screening and assessment; *(b)* development of a medical nutrition therapy (MNT) care plan and its implementation; *(c)* monitoring and reassessment of an individual's response to the nutrition care delivered; and *(d)* development of a transitional feeding care plan or termination of a nutrition support care plan, as appropriate. Other activities may include management of nutrition support services, including developing policies and procedures and supervising personnel and budgets; recommending and maintaining enteral and parenteral formulas; evaluating equipment for enteral feeding delivery; participating in nutrition support committees; and assuring optimal reimbursement for nutrition support activities.
- A dietetics professional is a person who, by virtue of academic and clinical training and appropriate certification and/or licensure, is uniquely qualified to provide a comprehensive array of professional services relating to prevention and treatment of nutritional related conditions.

Qualifications and Professional Associations

There are two routes to qualification as a dietician. You can either take a four-year degree course at a polytechnic or university offering the subject or,

after a first degree in another, appropriate subject such as nutrition or biochemistry a two-year postgraduate course.

To start a degree course in dietetics, you will normally be expected to have obtained two A level passes, in chemistry and one other science or mathematics. Two AS level passes may be offered as an alternative to one A level.

Remember, however, that colleges and universities consider each applicant individually and other qualifications may be acceptable.

A dietician's education in health science involves significant scientific based knowledge in anatomy, chemistry, biochemistry, biology, physiology, nutrition, medical science. It is these strong foundations in advanced scientific knowledge and an internship that equipped with counseling skills and aspects of psychology enable a Registered Dietitian to assess, analyze, intervene, and educate a patient in relation to the diet and disease.

There are a few different academic routes to becoming a fully qualified registrable dietitian:

- A professional bachelor degree in Dietetics which requires four years of studies

or

- A bachelor of science degree and a postgraduate diploma in Dietetics

or

- A bachelor of science degree and a master's degree in Dietetics
- Internship is also essential to become a fully qualified Dietitian. The internship process differ in different countries.

Classification

The majority of dieticians are clinical, or therapeutic, dieticians. Clinical dieticians review medical charts and talk with patients' families. They work with other health care professionals and community groups to provide nourishment, nutritional programs and instructional presentations to benefit people of all ages, and with a variety of health conditions. This is accomplished by developing individual plans to meet nutritional needs. These plans include nourishment, tube feedings (called enteral nutrition), intravenous feedings (called parenteral nutrition) such as total parenteral nutrition (TPN) or peripheral parenteral nutrition (PPN), diets, and education. Clinical dietitians provide individual and group educational programs for patients and family members about their nutrition and health.

Clinical Dieticians

Clinical dieticians work in hospitals and other health care facilities to provide nutrition therapy to patients according to the disease processes, provide individual

dietary consultations to patients and their family members and also conduct group educations for other health workers, patients and the public. They coordinate both medical records and nutritional needs to assess the patients and make a plan based on their findings. Some clinical dietitians have dual responsibilities with medical nutrition therapy and in foodservice, described below. In addition, clinical dietitians in smaller facilities will also provide or create outpatient education programs. They work as a team with the physicians, physical therapists, occupational therapists, pharmacists, speech therapists, social workers and nurses to provide care to the patients.

Community Dieticians

Community dieticians work with wellness programs and international health organizations. These dietitians apply and distribute knowledge about food and nutrition to specific life-styles and geographic areas. They coordinate nutritional programs in public health agencies, daycare centers, health clubs, and recreational camps and resorts. Some community dietitians carry out clinical based patient care in the form of home visits for patients who are too physically ill to attend consultation in health facilities.

Foodservice Dieticians

Foodservice dieticians or managers are responsible for large-scale food planning and service. They coordinate, assess and plan foodservice processes in health care facilities, school food service programs, prisons, cafeterias and restaurants. These dietitians will also perform audits of their departments, train other food service workers and use marketing skills to launch new menus and various programs within their institution. They direct and manage the operational and nutrition services staffs such as kitchen staffs, delivery staffs and dietary assistants or diet aides.

Gerontological Dieticians

Gerontological dieticians are specialist in nutrition and aging. They are Board certified in Gerontological Nutrition with the American Dietetic Association. They work in government agencies in aging policy, and in a regulatory capacity in the oversight of nursing homes and community-based care facilities. They work as Consultants in Nursing Homes, and in higher education in the field of Gerontology (the study of Aging.)

Pediatric Dieticians

Pediatric dieticians provide health advice for persons under the age of 18.

Research Dieticians

Research dieticians are mostly involved with dietary related research in the clinical aspect of nutrition in disease states, public aspect on primary, secondary and sometimes tertiary health prevention and foodservice aspect in issues involving the food prepared for patients. Many registered dietitians also work with the biochemical aspects of nutrient interaction within the body. Research Dietitians normally work in a hospital or university research facilities. It should be noted that some Clinical dietitian's roles also involve research other than the normal clinical workload. Quality improvement in dietetics services is also one area of research.

Administrative Dieticians

Administrative, or manager or Director of Dietetics Department or Nutrition Services are sometimes also known as Manager instead of Director depending on the size, number of dietitians in the department and also the organizational structure adopted by the Health facilities or Hospital. Director or Manager acts as head of the dietitians. They also hire, train, direct and supervise employees and manage dietary departments. Administrative dietitians may also apply procedure and policy as part of their management job.

Business Dieticians

Business dieticians serve as resource people for the media. Dietitians' expertise in nutrition is often taped for TV, radio, and newspapers — either as an expert guest opinion, regular columnist or guest, or for resource, restaurant, or recipe development and critique. Dietitians have served as show hosts on major television stations and as drive-time radio news anchors. Dietitians write books, appear on television cooking channels, and author corporate newsletters on nutrition and wellness. They also work as sales representatives for food manufacturing companies that provide nutritional supplements and tube feeding supplies.

Consultant Dieticians

Consultant dieticians work under private practice. The title 'consultant' in this case should not be confused with the identical title given to certain medical doctors in countries such as the United Kingdom and Ireland. The term consultant in this instance is synonymous with the title attending as used in countries such as the United States. Consultant dietitians contract independently to provide nutrition services and educational programs to individuals, nursing homes, and in health care facilities. As recent studies have shown the importance of diet in both preventing and managing disease, many US states have moved towards covering medical nutrition therapy under the Medicaid/Medicare making consulting a much more lucrative option for dietitians due to insurance reimbursement.

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Responsibilities

A combination of clinical and nutritional expertise enables the dietician to assess each patient's dietary needs. To this knowledge base, the successful dietician must add the skill of establishing an individual rapport with each patient to enable appropriate advice to be understood and accepted.

Dieticians may be based in clinical or community settings. Their patients and clients come from every age group and from all walks of life. While some dieticians choose to specialise in the treatment of, say children or people with renal disease, others continue to whose need for dietary advice may be vital, important or simply desirable.

Some very sick patients are unable to take food by mouth. Then the dietician's expertise is required to formulate an appropriate liquid feed, conditions, such as and the dietician continues to provide support and advice relevant to the patient's lifestyle. For other patients, pregnant women for example, the dietician may need to offer appropriate advice for a comparatively short time.

Personal Qualities

One should enjoy communicating with people from all walks of life. If he/she is able to explain complex things in a simple manner and can encourage people to take his/her advice, he/she could become an excellent dietician. The dietician will need an understanding, non-judgemental approach. Patience and a sense of humour help dietician rise to the demands of the job. Good writing skills are also an advantage, as dieticians produce leaflets, articles and teaching packs as well as writing reports and letters. The dietician should be committed to using scientific knowledge to improve the quality of people's lives. If he/she is interested in pursuing a career as a dietician, it might help him/her to speak to a local hospital dietetic department where he/she will be able to learn about the work of dieticians.

Dietitians provide advice and education about nutrition and its affect on health. They work in schools, educational administration, research, hospitals, clinics, industry, food service management, and private practice. Dietitians:

To prevent and alleviate illness/symptoms by means of nutritional treatment through oral, enteral1 and/or parenteral nutrition.

- To inform and educate on issues of nutrition.
- To act as a resource for the health and medical care services and society concerning nutritional issues.
- To participate in and follow research in the field of nutrition.
- Organize and supervise food service operations and meal preparation.

- Make dietary recommendations.
- Counsel patients and clients about diets.
- Supervise food purchasing.
- Study the nutritional value of food and how it is used by the body.

Code of Ethics and Obligations

The work of the clinical dietitian is based on nutrition, which comprises the science of energy metabolism and of nutrients and their function in the human body. Nutritional treatment is considered to be part of medical care. Within the realm of care, nutrition can be seen from different perspectives. For the clinical dietitian the main perspective is the nutritional treatment of the patient. The medical perspective of the doctor and the care perspective of the nurse also include concerns about nutrition. The different perspectives are sometimes overlapping and complementary.

The dietitian's professional competence is a synthesis of knowledge regarding food composition, nutritional requirements, foodstuffs, food choices, food preparation and meal planning, as well as knowledge about psychological and sociological factors which can control appetite and eating within the context of health and illness.

Dietetics is the integration and application of principles derived from several disciplines – including nutrition, biochemistry, physiology, food science and food composition, management of foodservices, as well as behavioural and social sciences – in order to achieve and maintain optimal human health.

Dietitians interpret the scientific evidence concerning human nutritional requirements and use this information to influence food intake and food choices within the population.

Medical Nutrition Therapy comprises nutritional diagnostics, therapy and advice for the purpose of treating illness. The dietitian's area of expertise involves a full command of current nutritional therapies as well as the prescription of a specific personalised treatment based on one or more of these therapies. The dietitian must also have a solid knowledge base regarding the different products available for the respective forms of therapy.

This professional competence is unique and therefore distinguishes the dietitian from other health care and medical professionals and is the reason for the dietitian's specific responsibility for nutritional treatment.

Professional Ethics

The clinical dietitian's work involves a responsibility towards all the different parties concerned. The most important responsibility for the clinical dietitian is

always towards the patient. The clinical dietitian fulfils this responsibility in various ways, for example by basing the treatment on scientific evidence and proven experience and by following scientific developments in the field. The clinical dietitian provides treatment and gives information in an environment that requires co-operation and respect between various professional groups and where all involved work towards a common goal.

The clinical dietitian must also fulfill professional demands for quality of care. All of these demands – responsibilities towards parties concerned as well as professional demands for quality – are justified and meeting them, as far as is possible, characterises an ethically correct care. They may, however, conflict making it necessary for the clinical dietitian to weigh them against each other when deciding further action.

Aims and Structure of the Code

The clinical dietitian encounters various ethical problems in his or her daily work. The Association of Clinical Dietitians has prepared this Code to serve not only as a guideline for the individual clinical Dietitian, but also to clarify which principles the Association considers should characterise the work of the clinical dietitian. In addition, the purpose of the code is to raise attention to ethical issues and problems in the clinical Dietitian's work, thereby contributing to lively discussion of such ethical issues amongst clinical dietitians. The Association of Clinical dietitians regards an ethical code as a step towards a common system of values within the profession.

The professional Code of Ethics of the Association of Clinical Dietitians has the following structure. Firstly, the clinical dietitian's profession is described in terms of its aims and tasks. Secondly, a number of obligations are specified; those that the clinical dietitian has to the profession itself as well as those to the parties involved in the clinical dietitian's work: the patient, the next-of-kin, colleagues, the various professional groups that the clinical dietitian co-operates with, the employer and society. It is important to emphasize that it is not only respect for the different parties concerned that puts ethical demands on the clinical dietitian. It is also unethical not to endeavor to meet the goals of the profession or the specific demands of the profession for quality of care. In this respect, the requirements for competence and quality are also ethical requirements.

Application and Limitations

A professional code of ethics can never be complete in the sense that all the ethical issues that the clinical dietitian must consider are specified in the text or formulated as rules. Reality is far too complex for this to be possible. In any particular situation, different ethical requirements and obligations may come into

conflict with one another, whereby it may be impossible to meet them all. In such cases the code cannot provide complete answers as to how to weigh up the different demands. A general rule, however, is that consideration towards the patient carries greater weight than that towards others concerned. Similarly, the clinical dietitian must not ignore strictly professional obligations. However, in specific cases the balancing of demands must be left to the clinical dietitian's own judgement. This does not imply that the formulation of the various considerations and requirements of a code becomes unimportant. On the contrary, the formulation of specific demands related to the parties concerned or and to the profession constitutes a means of support and a good starting point when it comes both to observing ethical problems in one's own work and to describing and analysing a problem in order to reach a solution.

A collection of examples giving common situations of different types which involve ethical problems or conflicts in professional practice has been produced to serve as help and guidance for training how to make good ethical analyses.

The Code of Ethics presumes that the clinical dietitian, in his or her work, respects current laws and regulations. The Code is not a legal document. Its rules should rather be seen as supplementing what is laid down in law.

The term patient is used throughout this Code to refer to the individual receiving treatment from the clinical dietitian. In certain situations other terms may be commonly used and more suitable. The ethical content of the Code can also be expressed using these terms.

The Dietician's Obligations

- To provide treatment or information based on scientific evidence and proven experience and actively represent this professional expertise in contacts within health and medical care and society in general.
- To endeavor at all times to adhere to the profession's established and accepted correct use of language, in an understandable form, when informing patients and staff, in written publications and in contact with the media.
- To be aware of one's role as a representative of the profession in official situations, and in contact with the media, and to strive for objectivity and a serious image.
- To understand the complexity of illness processes and the limits of the clinical dietitian's own competence and role in the treatment of patients.
- To seek advice and knowledge from colleagues as well as from other professions when required.

- To document the nutritional treatment.
- To transfer relevant information to others involved in the treatment of the patient.
- Not to allow the clinical dietitian's duties and work to be used to meet other needs of the patient.
- To maintain personal levels of competence by continually following the scientific and other literature within the nutritional and relevant medical fields, as well as participating in further education.
- To work using the nutritional care process as the overall structure.
- Not to allow thoughts of personal gain to influence the treatment and information given.
- To maintain a rational and critical position with regard to all forms of marketing and information and to be aware of the commercial interests behind parts of this information.

Obligations Towards Parties Concerned

Obligations towards parties concerned apply independent of their sex, age, ethnicity, colour of skin, sexual orientation, religion, political and social affiliations.

Obligations Towards the Patient

- To regard the patient as autonomous, *i.e.*, to see the patient as having the capability to make decisions concerning his/her own life.
- To keep informed at all times; of the patient's diagnosis, treatment and needs and of various factors concerning the patient's situation that may influence the formulation of the nutritional treatment.
- To give advice and propose treatment based on one's own professional knowledge and experience as well as on knowledge of the patient's wishes and situation and to establish goals together with the patient.
- To respect the patient's wishes as far as possible considering the demands of the profession.
- To balance the consequences of the nutritional treatment against those of other treatment of the patient so that an overall positive outcome has priority.
- To take into consideration that there may be situations where the correct course of action is to end or not to start a nutritional treatment.
- To actively support and encourage the patient in the nutritional treatment

and, as far as can be considered realistic, try to motivate the patient by means of information to carry out and complete the treatment.

- To make sure that the treatment is carried out with the patient's informed consent. This means that the patient is capable of making decisions, understands the information and is under no form of compulsion when accepting the treatment. If the patient is a child the informed consent must be obtained from its guardian. When the child is mature enough to give its own consent it must be obtained together with that of the guardian. Where the patient is an adult incapable of making decisions, an informed consent must be obtained from the patient's next-of-kin or guardian. In the absence of a guardian or next-of-kin, or if a certificate exists for compulsory admission to a psychiatric hospital in accordance with the law regulating compulsory psychiatric care, responsibility for deciding proper treatment rests with the clinical dietitian and physician.
- To treat patient information confidentially and to observe legally binding professional secrecy. Transfer of information over and above professional obligations may occur only after having obtained the patient's consent.
- To ensure that documentation is factual, relevant and observes the patient's integrity and dignity. The dietitian has a legal obligation to keep documented records.

Obligations Towards the Next-of-Kin or Equivalent

- To be explicit to the next-of-kin that the clinical dietitian represents the patient.
- To inform the next-of-kin concerned of the treatment only after having obtained the patient's consent.
- To involve the next-of-kin in the treatment only after having obtained the patient's consent.

Obligations Towards Colleagues and Other Professional Groups

- To work for a trustful co-operation towards common goals.
- To work jointly with colleagues or other professional groups in order to promote the interests of the patient in the best way possible.
- To explain one's own field of competence and to fulfill the obligations associated with the position.
- To respect the competence and field of responsibility of other professionals.
- To assist other clinical dietitians or representatives of other professions by

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giving advice and sharing knowledge and experience as requested. It is particularly important to introduce and support new and less experienced colleagues.

Obligations Towards the Employer

To adhere to the employer's guidelines and to show loyalty towards the employer as far as this is consistent with other demands of professional ethics.

Obligations Towards Society

To take responsibility towards society, mainly through providing information on matters concerning nutrition and nutritional treatment, using one's own competence based on scientific evidence and proven experience.

Research Ethics

- When the clinical dietitian pursues research, he/she is bound by the law concerning vetting of the ethics of research involving humans and by the ethical rules and guidelines that govern medical and social science research in India.
- When research is carried out in connection with care, the patient's wellbeing and rights must at all times precede research demands.
- When research is carried out as part of training, the final responsibility for adherence to research ethics rests with the supervisor.

EFAD and ICDA have adopted the International Code of Ethics:

Dietitians practice in a just and equitable manner to improve the nutrition of the world by:

1. Being competent, objective and honest in our actions
2. Respecting all people and their needs
3. Collaborating with others
4. Striving for positive nutrition outcomes for people
5. Doing no harm
6. Adhering to the standards of good practice in nutrition and dietetics

International Code of Good Practice:

Provision of Service and application of knowledge

1. Provide high quality, cost efficient services in nutrition and dietetics
2. Provide services based on the expectation and needs of the community or client

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3. Competently apply the knowledge of nutrition and dietetics and integrate this knowledge with other disciplines in health and social sciences
4. Work cooperatively with others to integrate nutrition and dietetics into overall care/service regardless of context
5. Work in partnership with clients and users of the service

Developing Practice and Application of Research

1. Interpret, apply, participate in or generate research to enhance practice
2. Develop a unique body of knowledge
3. Have an in depth scientific knowledge of food and human nutrition
4. Develop practice based on evidence

Communication

1. Communicate effectively through nutrition education, education and training, development of policy and programs
2. Advocate for nutrition and dietetics, the alleviation of hunger and the value of services
3. Advance and promote the dietetics profession

Quality in Practice

1. Systematically evaluate the quality of practice and revise practice on the basis of this feedback
2. Strive to improve services and practice at all times
3. Maintain continued competence to practice

Professional Accountability

1. Ensure accountability to the public
2. Accept responsibility for ensuring practice meets legislative requirements
3. Maintain continued competence by being responsible for lifelong learning and engaging in self-development.

1.6 DIETETICS

Dietetics is the study of the relationship between nutrition and health. Dieticians specialize in applying their knowledge to issues ranging from rescription diets for people suffering from specific medical conditions to ecommendations which are designed to improve the health of entire communities hrough dietary changes. This branch of the medical field has a number of pplications, including clinical treatment, research, and community outreach.

Individuals who practice dietetics usually have a bachelor's degree at a minimum, and many complete licensure requirements so that they can become registered dieticians. In many nations, the term "registered dietician" is protected by law, and only people who have satisfied certain requirements may use it. A dietician may also use the term "nutritionist," although it is possible for someone to be a nutritionist without having a background in dietetics.

What people eat can have a profound impact on their level of general health. Dieticians study nutritional needs at all stages of life, and in a variety of settings, so that they can understand the unique nutritional needs of specific patients, as well as nutritional trends in particular communities. A 90 year old woman has very different dietary requirements than a 25 year old male athlete, and a dietitian can determine what those needs are, and what the best source of nutrition might be.

Some dieticians work in hospitals and other clinical environments, working with specific patients. They can prescribe meal plans to help manage and prevent disease, and they can also prescribe enteral nutrition to patients who cannot eat normally. In clinical settings, dieticians work with doctors and other members of the medical team to ensure that their patients receive the best and most appropriate treatment.

Dietetics is also used in residential facilities such as colleges and nursing homes to ensure that residents get the nutrition they need, and in facilities such as schools and cafeterias, to provide a balanced, healthy diet which will promote health among customers. Dieticians are also an important part of public outreach programs related to nutrition, utilizing their skills to explain how people can eat healthier diets and maintain health. Research dieticians work in labs and similar settings to research nutrition, health, and emerging issues which pertain to the field.

A career in dietetics can be very interesting. Candidates who are interested in working in this field should be interested in science, medicine, and food, and it helps to have good people skills and excellent communication abilities.

1.7 CLASSIFICATION OF FOODS

Foods are substances from animal and plant sources that yield heat and energy when ingested and absorbed by the body. Food nutrients build and renew tissues and regulate the body processes. The unit commonly used for describing energy intake and energy expenditure is the calorie. Good food sources contain substantial amounts of nutrients in relation to caloric content and provide upwards of 10 percent of Recommended Dietary Allowance for each specific nutrient. Most people can get enough of each required nutrient daily by eating a wide variety of foods.

PROTEINS

Proteins are the "building blocks" of the body and provide important required nutritive elements. Proteins are needed for growth, maintenance, and replacement of body cells, and they form hormones and enzymes used to regulate body processes. Extra protein is either used to supply energy or is changed into body fat. Found in both the animal and plant kingdoms, all proteins are composed of amino acids. Some amino acids are absolutely essential to maintain life and are necessary for repair, growth, and body development. Of the approximately 20 amino acids, our body can produce all but nine. These nine amino acids are termed "essential amino acids." We must get them from food, and we need all nine at one time so our body can use them effectively.

Proteins, which promote tissue growth and renewal, have long been recognized as the main structural unit of all living cells. Each gram *(g)* of protein yields 4 calories in the process of metabolism. Although proteins yield energy, they are an expensive source. If sufficient carbohydrates are not supplied, the body will use protein for energy requirements. This protein may be obtained from muscle tissue, producing the "wasting effect" of long-term starvation and some diseases.

A constant protein source is required in the daily diet. The normal daily protein intake for adults should be 0.8 gram per kilogram *(g/kg)* (2.2 lbs) of body weight, or 12 percent of the total caloric intake. Pregnant women require an additional 10 grams of protein a day over the normal daily intake.

Proteins play an important role in recovering from fractures, burns, and infections. They are also important in healing wounds and recovering from surgical procedures. In cases of recovery, protein intake should be increased in accordance with the severity of the condition, and carbohydrates and fats can be added liberally. While proteins can supply energy, they are not a main source of energy like carbohydrates and fat.

Ideally, the patient should receive protein by mouth; however, it is sometimes necessary to meet the minimum requirements parenterally. Glucose parenteral solution, given during an acute emergency period, will prevent some loss of protein, Protein deficiency may stunt growth, promote a secondary anemia, or induce nutritional edema. Dietary sources of protein and the nine essential amino acids are milk, yogurt, eggs, meats, fish, cheese, poultry, peanut butter, legumes, and nuts. Protein from plant sources is best when combined with animal protein, such as milk plus peanut butter, or when legumes are combined with grains, such as Navy beans plus rice.

FATS

The chief functions of fats are to supply energy and transport fat-soluble vitamins. Each gram of fat yields 9 calories. Fats provide the most concentrated

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source of calories (and, therefore, energy) of all the food nutrients. Fats are found in both the animal and vegetable kingdoms. Fatty acids and glycerol are the end products of the digestion of fats.

Many fats act as carriers for the fat-soluble vitamins A, D, E, and K. They also act both as a padding for vital organs, particularly the kidneys, and as subcutaneous tissue to help conserve body heat. Fat is stored as adipose (fatty) tissue to form a reserve supply in time of need. Dietary fats delay gastric emptying and promote a feeling of fullness. Excess calories from fats may produce obesity, the forerunner of arteriosclerosis, hypertension, gallbladder disease, and diabetes. A diet high in fat, especially saturated fat and cholesterol, contributes to elevated blood cholesterol levels in many people. Adults over the age of 30 should have a serum cholesterol level of less than 200 mg/dl. Health experts agree that less than 30 percent of our total calories per day should come from fat. Saturated fat intake should be no more than 10 percent of the total calories.

Reducing dietary fat is also a good way to limit calories. Decreased fat intake results in fewer calories without a reduction of most nutrients. Too little fat in the diet may lead to being underweight, having insufficient padding for the vital organs, and lowered energy. Butter, margarine, cream cheese, fatty meats, whole milk, olives, avocados, egg yolks, nuts, commercial bakery products, and vegetable oils are all sources of dietary fat.

Carbohydrates

Carbohydrates (sugar and starches) are the most efficient sources of energy and are known as the "fuel of life." They are abundantly found in most plant food sources. Complex carbohydrates (starches) are in breads, cereals, pasta, rice, dry beans and peas, and other vegetables, such as potatoes and corn. Simple carbohydrates are found in sugars, honey, syrup, jam, and many desserts. The new nutritional guidelines established by the Food and Drug Administration (FDA) recommend that complex carbohydrates and naturally occurring sugars (found primarily in fruit) make up approximately 50 percent of one's total caloric intake. It is also recommended that refined and processed sugars make up no more than 10 percent of the calories in one's diet. Each gram of carbohydrate yields 4 calories in the process of its metabolism. Carbohydrates must be reduced to glucose before the body can use them. Carbohydrates are stored in the muscles to fuel their movement, and in the liver as glycogen, which is then broken down and released as glucose at the exact rate needed by the body. This latter mechanism is controlled largely by insulin from the pancreas. During fasting, liver glycogen is rapidly depleted, leading the body to use its fat for energy. Carbohydrates that are not needed for energy are converted to and stored as adipose (fat) tissue.

The main functions of carbohydrates are to :

- furnish the main source of energy for muscular work and nutritive processes,
- help maintain body temperature,
- form reserve fuel,
- assist in oxidation of fats, and
- spare protein for growth and repair.

Minerals

Although mineral elements constitute only a small portion of the total body weight, they enter into the activities of the body to a much greater degree than their weight would indicate. Certain mineral elements are essential for specific body functions. While it is not yet known exactly how many of the mineral elements are indispensable to the body functions, seemingly small changes of mineral concentration can be fatal. These essential inorganic elements contribute overwhelmingly to the skeletal framework of the body and teeth, and they are an essential part of many organic compounds.

Minerals form an integral part of basic cell structure and circulate in body fluids. They also exercise specific physiologic influences on the function of body tissues. For mineral needs to be met satisfactorily, consumption of each element must be sufficient to cover body tissue requirements and to meet changing physiological needs. At one time, it was erroneously believed that any diet adequate in other respects would also provide an adequate intake of essential minerals. This is not so. Foods vary greatly in their mineral—as well as their overall nutritional—content, depending on growing conditions, storage, and preparation procedures. Among the major minerals are calcium, phosphorus, iron, potassium, zinc, and magnesium.

Vitamins

Vitamins are essential compounds that are present in food in minute quantities. Although vitamins do not furnish energy or act as tissue-building materials, they do act as catalysts in many body chemical reactions and are necessary for normal metabolic functions, growth, and the health of the human body. Their absence results in malnutrition and specific deficiency diseases. Vitamin chemistry is complex and nutritional experimentation is difficult, so our knowledge of them is being continually supplemented and revised. It is quite possible that additional vitamins will be discovered or that some of those already recognized may prove to contain more than one factor.

Vitamins are so widely distributed in food that a properly prepared normal diet usually provides an adequate amount. Vitamins can be destroyed during the preparation or preservation of certain foods; however, manufacturers frequently add vitamins to their products to replace those destroyed or removed in processing. Since fat-soluble vitamins can be stored in the body, it is possible to develop hypervitaminosis by consuming excessive amounts of these nutrients, and death may result in extreme cases. Fat-soluble vitamins include A, D, E, and K.

- Vitamin A is involved in the formation and maintenance of healthy skin, hair, and mucous membranes. Vitamin A helps us to see in dim light and is necessary for proper bone growth, tooth development, and reproduction. Good sources of vitamin A include yellow, orange, and dark green vegetables; fruits; and liver, eggs, cheese, butter, and milk.
- Vitamin D promotes calcium and phosphorus absorption and is required for the formation of healthy bones and teeth. Good sources include fortified milk, egg yolk, liver, tuna, and cod liver oil. Vitamin D is produced in the body on exposure to sunlight.
- Vitamin E protects vitamin A and essential fatty acids from oxidation in the body cells and prevents breakdown of body tissues. Good sources include vegetable oils, fortified cereals, whole-grain cereals and bread, nuts, wheat germ, and green leafy vegetables.
- Vitamin K includes a group of vitamins that promote normal clotting of the blood and helps maintain normal liver functions. Good sources are green leafy vegetables, liver, soybean, and other vegetable products.

Water-soluble vitamins, such as vitamin C and the B-complex vitamins, are not stored in the body to any great extent. Rather, they are used as necessary by the body, and any amounts that remain are excreted in the urine. As a result, these vitamins must be replenished daily to ensure optimum health.

- Vitamin C (ascorbic acid) is necessary for normal growth and cell activity and is important for maintaining blood vessel strength. It helps the body resist upper respiratory infections and is necessary for the proper development of teeth and gums. Wounds and burns require vitamin C for healing. A deficiency of ascorbic acid causes an individual to bruise easily. A severe deficiency leads to a condition known as scurvy. Good sources include citrus fruits, raw leafy vegetables, and tomatoes.
- Vitamin B (Complex) includes more than 12 separate B vitamins. Some of the more common B vitamins are :

- Thiamin (B_1) is necessary for normal growth, normal carbohydrate metabolism and normal functioning of the heart, nerves, and muscles. Thiamin deficiency results in retarded growth and nerve disorders, and a condition known as beriberi. Good sources include pork, fish, eggs, and whole-grain cereals.
- Riboflavin (B_2) is required for normal growth, vigor, healthy skin and mucosa, and normal eye function. Riboflavin is found in milk products, green leafy vegetables, and eggs. Other good sources of vitamin B_2 are the organ meats, heart, kidney, and liver.
- Niacin (B_3) is necessary for normal growth and skin health, normal functioning of the stomach and intestines, nervous and circulatory systems, and for carbohydrate, fat, and protein metabolism. The best sources are meat, liver, poultry, and peanuts.
- Pyridoxine (B_6) is necessary for fat, carbohydrate, and protein metabolism, and is sometimes used to treat nausea in pregnancy. Sources include liver, yeast, wheat germ, pork, potatoes, and milk. Vitamin B_6 is usually prescribed with Isonizid (INH) treatment since INH often causes a pyridoxine deficiency.
- Cyanocobalamin (B_{12}) is necessary for the health of nervous tissue and assists in iron metabolism and the maturation process of red blood cells. B_{12} is used to prevent pernicious anemia. The best sources are liver and kidneys, milk, eggs, fish, and cheese.

Vitamin and Mineral Supplements

Vitamin supplements are usually not necessary if a diet includes a wide variety of foods. Exceptions may occur in prenatal diets in which iron is low, as well as in patients who are deficient in a specific vitamin. Vitamin supplements should be taken only on a physician or dietitian's recommendation.

Vitamin and mineral supplements are being widely used by physically active people because of all the performance-enhancing claims made by supplement manufacturers.

It is estimated that 40–50 percent of athletes use some form of vitamin/ mineral supplements. Some doses range from amounts similar to the Recommended Dietary Allowances (RDA) up to levels many times the RDA. Supplements are useful under a variety of conditions, such as if an individual

- has an existing vitamin or mineral deficiency;
- has poor nutrient intake and/or dietary habits; or
- is exposed to extreme environmental conditions, such as altitude.

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Often, laxatives are prescribed in conjunction with some medical treatments and may cause decreased absorption of vitamins, loss of minerals and electrolytes, or inhibition of glucose uptake. Therefore, any patient on laxatives should be carefully monitored, and supplementary nutritives should be administered as necessary.

Taking a general multivitamin supplement appears to be without measurable performance enhancing effects in healthy, well-nourished, physically active personnel. Similarly, no improvements in muscle strength or endurance have been noted in strength athletes, such as body builders, who tend to use megadoses of vitamin and mineral supplements. The indiscriminate use of high-potency vitamins and minerals is of growing concern since excessive amounts of vitamins and/or minerals can be harmful and may result in nutrient imbalances. Excessive intake of some vitamin and mineral supplements can result in adverse—and possibly toxic–side effects.

Water

Water is often called the "forgotten nutrient." Water is needed to replace body fluids lost primarily in urine and sweat. A person can survive weeks without food but only days without water. Water makes up 70 percent of body weight and is found in every cell in the body. It is the medium through which nutrients are transported from the digestive tract to the cells where they are needed. Water is also the medium through which the by-products of cell metabolism are removed.

Water also serves as the medium in which the chemical processes of life take place. It is normally taken into the body in beverages, soups, and in the form of solid foods. Fluid needs are increased with sweating, vomiting, diarrhoea, high-protein diets, and in hot environments. An insufficient intake may cause dehydration, evidenced by loss of weight, increased body temperature, and dizziness.

Calculating a therapeutic diet can be complicated and is best left to dietitians. It is now common practice for dietitians or dietary kitchens to select foods for diets using various food groups. These foods are classified according to their nutritional value and the number of servings that should be eaten each day.

Dietary Guidelines

The food pyramid graphically communicates the message of the Dietary Guidelines. Diets should be built upon a base of complex carbohydrates and less fats. The placement of the food groups starting at the base of the pyramid conveys the current recommendations. These recommendations are as follows:

- Eat more grains, vegetables, and fruits
- Eat moderate amounts of lean meats and dairy foods
- Use sweets, fats, and oils sparingly

1.8 DIET THERAPY AND PREPARATION OF NORMAL DIET

It is often necessary to cater to a patient's appetite, since many individuals become especially hard to please when sick. In some disease states, such as cancer, patients experience marked taste changes. Because of the importance of the nutritional elements in feeding the sick, try to carry out the patient's wishes whenever possible. A tactful and observant Hospital Corpsman can be of great benefit to the physician and dietitian in carrying out the dietary regimen. You must be aware of what comprises a well-balanced diet and should be able to recognize when dietary adjustments need to be made in special situations. This is important to meet the changing needs of the diseased body's ability to make use of foods.

The patient should be made to feel that the utmost cleanliness and care have been observed in the preparation and service of their food. The patient's face and hands should be cleaned before food is served, and the lips and teeth cleaned before and after the meal. If the mouth is dry, it should be moistened periodically.

When special or modified diets are ordered, check the contents of the tray with the written orders. An error in serving a special diet may cause discomfort, serious illness, or even death.

Objectives of Diet Therapy

The objectives of diet therapy are as follows:

- To increase or decrease body weight
- To rest a particular organ
- To adjust the diet to the body's ability to use certain foods
- To produce a specific effect as a remedy (*e.g.*, regulation of blood sugar in diabetes)
- To overcome deficiencies by the addition of food rich in some necessary element (*e.g.*, supplementing the diet with iron in treating macrocytic anemia)
- To provide ease of digestion by omitting irritating substances, such as fiber, spices, or high-fat foods.

TYPES OF DIETS

Diets used in the treatment of disease are often spoken of by specific names that show a special composition and often indicate the purpose for which the diet is intended.

Regular Diet

The regular diet is composed of all types of foods and is well balanced and capable of maintaining a state of good nutrition. It is intended for convalescing patients who do not require a therapeutic diet.

Modified or Therapeutic Diets

Modified or therapeutic diets are modifications of the regular diet and are designed to meet specific patient needs. These include :

- method of preparation (*e.g.*, baking, boiling, or broiling),
- consistency (*e.g.*, ground or chopped),
- total calories (*e.g.*, high or low calorie),
- nutrients (*e.g.*, altering carbohydrate, protein, fat, vitamins, and minerals), and
- allowing only specific foods (*e.g.*, diabetic diet).

SOFT DIET—The soft diet is soft in texture and consists of liquids and semi-solid foods. It is indicated in certain postoperative cases, for convalescents who cannot tolerate a regular diet, in acute illnesses, and in some gastrointestinal disorders. A soft diet is an intermediate step between a liquid and regular diet and is low in connective tissue and indigestible dietary fiber. Little or no spices are used in its preparation.

The soft diet includes all liquids other than alcohol, and foods that may be incorporated into a soft diet include well-cooked cereals, pastas, white bread and crackers, eggs, cottage cheese, tender meat, fish, poultry, and vegetables (including baked, mashed, and scalloped potatoes). Vegetables can be puréed and meats ground for dental patients. Permitted desserts are custards, gelatin puddings, soft fruits, and simple cakes and cookies. Foods prohibited in a soft diet include fried foods, raw vegetables, and nuts.

LIQUID DIET—A liquid diet consists of foods that are in a liquid state at body temperature. This type of diet is indicated in some postoperative cases, in acute illnesses, and in inflammatory conditions of the gastrointestinal (GI) tract. It is important that feedings consisting of 6 to 8 ounces or more be given every 2 to 3 hours while the patient is awake.

Liquid diets are usually ordered as clear, full, or dental liquid. A clear liquid diet includes clear broth, black tea or coffee, plain gelatin, and clear fruit juices (apple, grape, and cranberry), popsicles, fruit drinks, and soft drinks. This diet is inadequate in all nutrients. A full liquid diet includes all the liquids served on a clear liquid diet, with the addition of strained cream soups, milk and milk drinks, ice cream, puddings, and custard. The full liquid diet is inadequate in iron, niacin, and possibly Vitamin A and thiamin. A dental liquid diet includes regular foods blended and strained in liquid form and all foods allowed on clear and full liquid diets. Vitamin and mineral supplements may be necessary with the dental liquid diet if the recommended amounts of food are not tolerated.

HIGH-CALORIE DIET—The high-calorie diet is of a higher caloric value than the average patient normally requires. A high-calorie diet is indicated when an increase of total calories is required by malnourished, underweight, postsurgical, or convalescing patients, especially those recovering from acute illnesses such as infections, burns, and fevers. The increase in calories is obtained by supplementing or modifying the regular diet with high-calorie foods or commercial supplements, by giving larger portions, or by adding snacks. It is given to meet a need for energy caused by the more rapid metabolism that accompanies certain diseases (especially fever, hyperthyroidism, poliomyelitis, and tuberculosis). In the liquid or soft diet, adding fats and carbohydrates increases the caloric value. The high-calorie diet is often ordered along with high protein. Proteins are added to prevent depletion of proteins in the plasma (a condition known as hypoproteinemia). As the patient progresses, a more solid diet is given.

Good sources of high-calorie foods are whole milk, cream, sweets, butter, margarine, fried foods, gravy, sauces, and ice cream. Between-meal feedings consisting of milk, milkshakes, cheese, cookies, or sandwiches are recommended, but these feedings should not interfere with the patient's appetite at mealtime.

HIGH-PROTEIN DIET—As previously stated, protein is essential for tissue growth and regeneration. A high-protein diet is indicated in almost all illnesses (*e.g.*, nephrosis, cirrhosis of the liver, infectious hepatitis, burns, radiation injury, fractures, some GI disorders, conditions in which the protein blood level is low, and in preoperative and postoperative cases).

In some acute illnesses and disorders, such as infectious hepatitis, GI disorders, and postoperative conditions, patients may be unable to consume solid foods or the daily requirement of protein and calories because of pain or nausea. In these cases, intravenous fluids with nutrient additives are required for the patient to receive the required amount of protein.

Protein-calorie deficiency is a definite factor in postoperative wound disruption. This disruption can best be prevented by preemptive nutritional

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measures before surgery. Antibody production will be decreased if the patient receives inadequate protein. Remember, the daily recommended intake of proteins for adults is at least 0.8 g/kg of body weight (approximately 56 g). A high-protein diet should provide a minimum of 1.5 g of protein per kg of body weight (approximately 105 g). The seriously burned and radiation injury patients should receive at least 3.0 g/kg daily.

Supplement the regular diet with high-quality protein foods, such as meat, fish, cheese, milk, and eggs.

LOW-CALORIE DIET--The low-calorie diet is useful in the treatment of obesity, but it may also be used to control weight in medical conditions such as arthritis, hypertension, diabetes, cardiac disease, or hypothyroidism. A loss of 1 to 2 pounds per week is the medically acceptable limit for weight reduction. A low-calorie diet consists of 1,000 to 1,800 calories per day. Calorie levels are determined by physicians and dietitians to help meet specific individual patient weight-loss goals. The daily intake of proteins should be at least 0.8 g/kg of standard body weight. Supplemental vitamins may be ordered if the prescribed diet is less than 1,200 calories.

Patients on low-calorie diets should be instructed by the dietitian (if available) or other medical personnel knowledgeable in proper eating habits. The dietitian conducts patient interviews to learn the patient's eating behaviours, usual portions, preparation of foods, meal patterns, nutritional adequacy, exercise, and so forth. Individual programs should then be recommended to assist patients to attain and maintain their ideal weight.

The Handbook of Clinical Dietetics, published by the American Dietetic Association, lists the following formula for determining ideal body weight. For females, the basic weight for 5 feet is 100 pounds. Add 5 pounds for every inch over 5 feet. For males, the basic weight for 5 feet is 106 pounds, with 6 pounds added for every inch over 5 feet. Adjustments must be made for body build. Reduce desired weight by 10 percent for a small frame; increase it by 10 percent for a large frame. Total caloric requirements are based on ideal body weight plus activity.

Many patients on low-calorie diets experience hunger. To satisfy this hunger or appetite, low-calorie foods such as raw vegetables, broth, black coffee or tea, and other unsweetened or diet beverages should be provided. Water and sodium need not be restricted unless there are cardiac complications or edema, and the restrictions are ordered by the physician.

LOW-PROTEIN DIET—As the name implies, the low-protein diet is made up of foods that furnish only small amounts of protein and consist largely of

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carbohydrates and fats (*e.g.*, foods such as marshmallows, hard candy, and butter). This diet is used in renal diseases associated with nitrogen retention or liver disorders. Limited amounts of protein are sometimes advocated in certain kidney diseases (such as chronic nephrotic edema). Low-protein diets for renal failure are usually restricted in sodium and potassium, because these two elements are not excreted properly during this condition. In some cases of chronic renal insufficiency, the protein content of the diet is varied, usually between 40-60 g per day, so that there will be sufficient complete protein to maintain nitrogen equilibrium.

In some metabolic disturbances, such as amino acids in the urine, protein restriction may be of therapeutic value.

HIGH-RESIDUE DIET—The high-residue (high-bulk, high-fiber, high-roughage) diet is indicated in atonic constipation, spastic colon, irritable bowel syndrome, and diverticulosis. This diet encourages regular elimination by stimulating muscle tone, creating softer and larger stools that are more easily propelled through the colon, thereby reducing the pain and cramping that accompany spastic colon or irritable bowel syndrome.

The patient is given a regular diet, with the inclusion of high-residue foods. The main sources of fiber are whole-grain breads and cereals, bran cereals, fresh fruits, and vegetables that are raw or cooked until tender. Whole grain breads and cereals that contain wheat bran have a greater laxative effect than fruits and vegetables, because the bran acts to absorb water within the colon, creating a bulk effect. Fiber intake should be increased gradually to minimize potential side effects of bloating, cramps, and diarrhoea. At least one serving of 100 percent wheat bran cereal is recommended daily. Cereals such as raisin bran, Bran Flakes, Shredded Wheat, and oatmeal may be used occasionally, but they contain less than half the amount of fiber found in All-Bran or Bran Buds. Fresh fruits and vegetables with edible skins, such as apples and grapes, are higher in fiber content than canned fruits or vegetables and their juices.

Dietary intake of refined sugars and starches should be decreased because they are poor sources of fiber. Also, limit white flour products, refined cereals, pies, cakes, and cookies.

Too little fluid in the high-residue diet may cause dehydration and lead to constipation. The patient must drink at least eight 8-ounce glasses of water or other fluids daily, particularly when consuming the recommended amount of bran. Drinking too much alcohol, beverages containing caffeine (such as coffee, cola, tea, and soft drinks), however, can irritate a sensitive colon and can cause dehydration. When possible, use decaffeinated coffee. One or two glasses of water

in the morning help to stimulate peristalsis. Excessive intake of foods like dried beans, fruits with seeds and skins, nuts, popcorn, and strong spices may cause irritability, especially during the inflammation period of colon disease states. These foods should be individualized to the patient.

When one is progressing from a low-residue diet after an acute infection or diverticulitis, increase fiber in the diet gradually. Start by adding one serving of 100 percent bran cereal and three servings of whole-grain bread to the low-residue menu pattern. Gradually increase the amount of raw vegetables and fresh fruits to at least four servings per day.

LOW-RESIDUE DIET—The low-residue diet is indicated in ulceration, inflammation, and other gastric disorders (such as partial intestinal obstruction or diverticulitis). It is also used in certain posto- perative states that affect any part of the GI tract, *e.g.*, a hemorrhoidectomy. Low-residue diets are also used in treating dysenteries of long duration.

The purpose of this diet is to provide nonstimulating, non-irritating, and easily digested material that leaves little residue, thus avoiding mechanical irritation of the GI tract. Various commercially prepared low-residue elemental diet supplements may be given to provide complete nutrition.

LOW-SODIUM DIET—A low-sodium diet consists of foods containing a very small percentage of sodium, with no salt added in preparation or by the patient. It is impossible to prepare an absolutely sodium-free diet.

The low-sodium diet is indicated when edema is present, in renal diseases, hypertension, and certain cardiac conditions.

The nephrotic patient is often unable to excrete sodium in a normal manner because the kidneys' retention of sodium leads to edema. A low-sodium diet is thus indicated, with no restriction on salt-free liquids. Such patients should be encouraged to drink 2,000 to 3,000 milliliters (ml) of low-sodium fluids daily.

The allowance of sodium in a strict low-sodium diet is 250 to 1,000 mg daily. The allowance of sodium in a moderate low-sodium diet is 2,000 mg or 2 g. Regular diets with no salt added contain 2.4 to 4.5 g of sodium.

Any diet in which the amount of sodium is drastically reduced has possible side effects. The patient who is on this diet regimen must be constantly observed—particularly in warm climates—for lassitude, complaints of weakness, anorexia, nausea and vomiting, mental confusion, abdominal cramps, and aching skeletal muscles. Electrolyte imbalances can have serious consequences. If you observe symptoms such as those described above, notify a medical officer.

BLAND DIET—A bland diet may be helpful for gastritis, hyperacidity, hemorrhoids, peptic ulcers, and other GI disorders. Dietary management of patients with chronic ulcer disease has been the subject of much controversy.

Bland diets have traditionally been used for these patients. However, experiments show that there is no significant difference in the response of patients with an active duodenal ulcer to a bland diet. Known irritants to the gastric mucosa include alcohol, black pepper, caffeine, chili powder, cocoa, coffee, certain drugs, and tea.

Emphasizing how to eat is as important as indicating what foods to eat, since there are individual responses to bland diets. Offer the following suggestions to the patient :

- Avoid worry and emotional upsets at mealtime
- Chew food well and eat slowly
- Rest before and after meals
- Avoid foods of extreme temperatures

If fruits and juices between meals cause distress, try including them with meals. Meals must be kept small to reduce gastric acidity and distention. Among foods to avoid in the bland diet are :

- fatty meats,
- fried foods,
- whole-grain breads and cereals,
- dried beans and peas,
- cabbage-family vegetables,
- chocolate,
- nuts and seeds, and
- carbonated beverages, caffeine, coffee, and tea.

Patients on a bland diet may use spices and condiments such as allspice, cinnamon, mace, paprika, sage, thyme, catsup, cranberry or mint jelly, and extract and flavorings without chocolate or vinegar.

The bland diet allows a more liberal food selection than other restrictive diets. This diet reduces the number of meals to three, and increases the quantity of foods given. Individualize the diet to the patient.

The "Regular-No Stimulants Diet" (also called "liberal bland"), a type of bland diet, eliminates only those items that have been shown scientifically to irritate the gastric mucosa (*i.e.*, alcohol, black pepper, caffeine, chili powder, cocoa, coffee, certain drugs, and tea).

Decaffeinated coffee may be restricted in most types of bland diets. Recent studies show that it causes increased gastric acid secretion and esophageal pressure causing gastric acid reflux in the esophagus. Decaffeinated coffee is only offered on the bland diet and the regular-no stimulants diet if it is tolerated by the patient.

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Chronic and excessive use of antacids to treat hyperacidity and related conditions may result in thiamin deficiency, presumably because of alkaline destruction of thiamin within the bowel lumen. Excessive intake of milk with antacids may cause systemic alkalosis and hypercalcemia. Milk may be contraindicated in patients with allergic reactions or lactose intolerance.

LOW-CARBOHYDRATE, HIGH-PROTEIN DIET—A low-carbohydrate, high-protein diet is used in the treatment of hypoglycemia. This diet limits simple carbohydrates that are quickly absorbed into the blood. A marked rise in blood sugar stimulates the pancreas to overproduce insulin, which leads to a hypoglycemic state as too much sugar is transported out of the blood.

Individualize the diet to the patient, since hypoglycemic reactions may occur at any time for various reasons. For example, meal skipping, inadequate calorie intake with excessive energy expenditure, and drinking alcohol may precipitate a low-blood-sugar reaction.

The foods may be divided into three to six or more small meals. Liberal amounts of protein and fat are used, as they are more slowly digested and absorbed. The diet includes meats, fish, poultry, cheese, eggs, fats, low-starch vegetables, and limited amounts of unsweetened fruit and juices, breads, cereals, and high-starch-content vegetables (like corn, peas, and potatoes). Because milk contains the sugar lactose, limit it to 2 cups a day for an adult.

Sweets such as candy, sugar, jams, jellies, soft drinks, and pastries should be avoided to help prevent hypoglycemic reactions. They should be consumed only when necessary to quickly increase blood-sugar levels during a hypoglycemic reaction. If reactions are frequent, it is helpful to carry hard candy for quick and easy use. Handy high-protein snacks to help prevent hypoglycemic reactions may include cheese, peanut butter, milk, and hard-boiled eggs.

STUDENT ACTIVITY

1. Discuss the responsibilities of a dietician.

2. Outline the classification of Foods.

1.9 PRINCIPLES IN FORMULATION OF THERAPEUTIC DIETS AND FACTORS TO BE CONSIDERED FOR THERAPEUTIC DIETS

Indian Dietetics Association, Dietician and Dietetics

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Diet therapy is a broad term for the practical application of nutrition as a preventative or corrective treatment of disease. This usually involves the modification of an existing dietary lifestyle to promote optimum health. However, in some cases, an alternative dietary lifestyle plan may be developed for the purpose of eliminating certain foods in order to reclaim health. For example, the latter kind of diet therapy is often recommended for those who suffer from allergies, including those that are not food-related. Elimination diet therapy is often found to be helpful in improving symptoms associated with attention deficit disorder and hyperactivity in children.

In an ideal world, all the nutrients we require in the correct amounts would be taken in with our food as part of a balanced diet.

Unfortunately, in our fast and furious modern society this is not always the case. Farming and environmental issues mean that many foods have suffered a natural decline in nutrient levels. Plus, the "age of the ready meal" has brought not only speed and convenience but also a whole host of chemical additives, preservatives, colourings and flavourings all convincingly packaged to disguise often poor nutritional content. With foods like these being eaten everyday it is no surprise that chronic health problems such as obesity, diabetes and heart disease are becoming 21st century epidemics.

Malnutrition is definitely not a thing of the past; it comes in many different guises and surrounds us all everyday. To add to the impacts of our depleted diets, environmental pollution and the use of antibiotics and other drugs can reduce the body's ability to absorb and use nutrients correctly. This further diminishes our chances of optimum health and contributes to illness.

Therapeutics diets takes all of these things into account and each patient is treated as an individual:

- Everyone's nutritional needs are different and each food and supplement programme is carefully tailored to suit the particular needs and wishes of that person.
- Therapeutics diet identifies and addresses the underlying cause of a health problem, not merely treating the symptoms as many medications do.
- Each person is treated as a whole. All aspects are considered to account for the fact that the body functions through a complex web of interactions that need to be working in harmony to achieve optimal health.
- A diet therapist may recommend levels of nutrients way above RDA's (Recommended Daily Amounts) as set by the government. RDA's are

simply a guide to the minimum amounts of nutrients we require each day to prevent diseases such as scurvy. Therapeutic levels of nutrients help address disease and promote optimum health.

- As the old saying goes, prevention is better than cure. Diet therapy works to prevent disease and maintain health as well as tackling illness.

Principles of therapeutic diets planning also include:

A. **Adequacy** – provides sufficient energy and enough of the nutrients to meet the needs of most healthy people to maintain their health.

B. **Balance** – eat a variety of foods to obtain all the nutrients.

C. **Nutrient Density** – a measure of the nutrients a food provides relative to the energy it provides. The more nutrients and the fewer kcalories, the higher the nutrient density.

D. **Energy control** – managing energy intake.

E. **Moderation** – select foods low in fat and sugar, they promote weight gain.

F. **Variety** – select foods from different food groups.

1.10 SUMMARY

- Dietetics (from the Greek diaita, meaning "mode of life") has been implicated in the cause, cure and prevention of disease from earliest recorded history.
- In 1963, a band of nutritionists, dietitians and workers in the allied health fields resolved to form a scientific body to highlight the importance of dietetics and nutrition in the maintenance of health, and in the prevention and treatment of diseases. Thus, the Indian Dietetic Association was founded.
- A dietician is an expert in food and nutrition. Dieticians help promote good health through proper eating. They supervise the preparation and service of food, develop modified diets, participate in research, and educate individuals and groups on good nutritional habits.
- The dietitian's professional competence is a synthesis of knowledge regarding food composition, nutritional requirements, foodstuffs, food choices, food preparation and meal planning, as well as knowledge about psychological and sociological factors which can control appetite and eating within the context of health and illness.
- Diet therapy is a broad term for the practical application of nutrition as a preventative or corrective treatment of disease. This usually involves the modification of an existing dietary lifestyle to promote optimum health.

1.11 GLOSSARY

- **Dietetics:** Derived from the Greek diaita, meaning "mode of life".
- **Dietician:** An expert in food and nutrition.
- **Clinical Dietician:** A dietician who has responsibility for planning, education, supervision and evaluation of a clinically devised eating plan to restore the client's/patient's functional health.
- **Community Dietician:** A dietician directly involved in health promotion and policy formulation.
- **Gerontological Dieticians:** A specialist in nutrition and aging.
- **Foodservice Dieticians:** Responsible for large-scale food planning and service.
- **Research Dieticians:** They are mostly involved with dietary related research in the clinical aspect of nutrition in disease states.
- **Dietetics:** It is the study of the relationship between nutrition and health.

1.12 REVIEW QUESTIONS

1. Discuss the historical developments took place in the field of dietetics in 19th century.
2. What are the important objectives of Indian Dietetics Association? Discuss.
3. Give an appropriate definition of dietecian.
4. What are the main obligations of a dietician? Discuss.
5. Describe the classification of foods.
6. Why is diet planning important?
7. How is diet therapy helpful?

1.13 FURTHER READINGS

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UNIT– II

OBESITY AND UNDERWEIGHT

OBJECTIVES

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

- state the etiology of obesity and underweight;
- discuss the physiology and metabolic changes in obesity;
- understand the complications of obesity and underweight;
- explain the dietary modifications during obesity and underweight.

STRUCTURE

2.1 INTRODUCTION

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.

Underweight refers to a human who is considered to be under a healthy weight. The definition is usually made with reference to the body mass index (BMI). A BMI of under 18.5 is usually referred to as underweight. This medical definition of underweight may differ from other uses of the term, such as those based on attractiveness.

Obesity is excess body fat; consequences depend not only on the absolute amount but also on the distribution of the fat. Complications include cardiovascular disorders, diabetes mellitus, many cancers, cholelithiasis, fatty liver and cirrhosis, osteoarthritis, reproductive disorders in men and women, psychologic disorders, and premature death. Diagnosis is based on body mass index (BMI—calculated from height and weight) and waist circumference. BP, fasting plasma glucose, and lipid levels should be measured. Treatment includes physical activity, dietary and behavioural modification, and sometimes drugs or surgery.

Prevalence is more than twice as high at age 55 as at age 20. Obesity is twice as common among women in a lower socioeconomic group as among those in a higher group. Prevalence among black and white men does not differ significantly, but it is higher among black women than white women. More than 50% of black women > 40 yr are obese; > 80% are overweight.

In the US, obesity and its complications cause as many as 300,000 premature deaths each year, making it second only to cigarette smoking as a preventable cause of death.

2.2 ETIOLOGY OF OBESITY

Almost all cases of obesity result from a combination of genetic predisposition and a chronic imbalance between energy intake, energy utilization for basic metabolic processes, and energy expenditure from physical activity.

Genetic factors: Heritability of BMI is about 66%. Genetic factors may affect the many signaling molecules and receptors used by parts of the hypothalamus and GI tract to regulate food intake. Rarely, obesity results from abnormal levels of peptides that regulate food intake (*e.g.*, leptin) or abnormalities in their receptors (*e.g.*, melanocortin-4 receptor).

Genetic factors also regulate energy expenditure, including BMR, diet-induced thermogenesis, and nonvoluntary activity–associated thermogenesis. Genetic factors may have a greater effect on the distribution of body fat, particularly abdominal fat, than on the amount of body fat.

Environmental factors: Weight is gained when caloric intake exceeds energy needs. Important determinants of energy intake include portion sizes and the energy density of the food. High-fat foods, processed foods, and diets high in refined carbohydrates, soft drinks, fruit juices, and alcohol promote weight gain. Diets high in fresh fruit and vegetables, fiber, and complex carbohydrates, with water as the main fluid consumed, minimize weight gain. A sedentary lifestyle promotes weight gain.

Regulatory factors: Prenatal maternal obesity, prenatal maternal smoking, intrauterine growth restriction, and insufficient sleep can disturb weight regulation. About 15% of women permanently gain > 20 lb with each pregnancy. Obesity that persists beyond early childhood makes weight loss in later life more difficult.

Drugs, including corticosteroids, lithium, traditional antidepressants (tricyclics, tetracyclics, and monoamine oxidase inhibitors [MAOIs]), benzodiazepines, and antipsychotic drugs, often cause weight gain.

Uncommonly, weight gain is caused by one of the following disorders:

- Brain damage caused by a tumor (especially a craniopharyngioma) or an infection (particularly those affecting the hypothalamus), which can stimulate consumption of excess calories
- Hyperinsulinism due to pancreatic tumors
- Hypercortisolism due to Cushing's syndrome, which produces predominantly abdominal obesity
- Hypothyroidism (rarely a cause of substantial weight gain)

Eating disorders: At least 2 pathologic eating patterns may be associated with obesity:

- Binge eating disorder is consumption of large amounts of food quickly with a subjective sense of loss of control during the binge and distress after it. This disorder does not include compensatory behaviours, such as

vomiting. Prevalence is 1 to 3% among both sexes and 10 to 20% among people entering weight reduction programs. Obesity is usually severe, large amounts of weight are frequently gained or lost, and pronounced psychologic disturbances are present.

- Night-eating syndrome consists of morning anorexia, evening hyperphagia, and insomnia. At least 25 to 50% of daily intake occurs after the evening meal. About 10% of people seeking treatment for severe obesity may have this disorder. Rarely, a similar disorder is induced by use of a hypnotic such as zolpidem.

Similar but less extreme patterns, classified as eating disorders not otherwise specified (EDNOS), probably contribute to excess weight gain in more people. For example, nocturnal eating contributes to excess weight gain in many people who do not have night-eating syndrome.

2.3 COMPLICATIONS

Complications of obesity include the following:

- Metabolic syndrome
- Diabetes mellitus
- Cardiovascular disease
- Non alcoholic steatohepatitis (fatty liver)
- Gallbladder disease
- Gastroesophageal reflux
- Obstructive sleep apnea
- Reproductive system disorders
- Many cancers
- Osteoarthritis
- Social and psychologic problems

Insulin resistance, dyslipidemias, and hypertension (the metabolic syndrome) develop, often leading to diabetes mellitus and coronary artery disease. These complications are more likely in patients with fat that is concentrated abdominally, a high plasma triglyceride level, a family history of type 2 diabetes mellitus or premature cardiovascular disease, or a combination of these risk factors.

Obesity is also a risk factor for nonalcoholic steatohepatitis (which may lead to cirrhosis) and for reproductive system disorders, such as a low plasma testosterone level in men and polycystic ovary syndrome in women.

Obstructive sleep apnea can result if excess fat in the neck compresses the airway during sleep. Breathing stops for moments, as often as hundreds of times a night. This disorder, often undiagnosed, can cause loud snoring and excessive daytime sleepiness and increases the risk of hypertension, cardiac arrhythmias, and metabolic syndrome.

Obesity may cause the obesity-hypoventilation syndrome (Pickwickian syndrome). Impaired breathing leads to hypercapnia, reduced sensitivity to CO_2 in stimulating respiration, hypoxia, cor pulmonale, and risk of premature death. This syndrome may occur alone or secondary to obstructive sleep apnea.

Osteoarthritis and tendon and fascial disorders may result from obesity. Skin disorders are common; increased sweat and skin secretions, trapped in thick folds of skin, are conducive to fungal and bacterial growth, making intertriginous infections especially common. Being overweight probably predisposes to cholelithiasis, gout, deep venous thrombosis and pulmonary embolism, and many cancers (especially colon and breast cancers).

Obesity leads to social, economic, and psychologic problems as a result of prejudice, discrimination, poor body image, and low self-esteem. For example, people may be underemployed or unemployed.

2.4 DIAGNOSIS OF OBESITY

- BMI
- Waist circumference
- Sometimes body composition analysis

BMI

Body mass index or BMI is a simple and widely used method for estimating body fat mass. BMI was developed in the 19th century by the Belgian statistician and anthropometrist Adolphe Quetelet. BMI is an accurate reflection of body fat percentage in the majority of the adult population. It however is less accurate in people such as body builders and pregnant women. A formula combining BMI, age and gender can be used to estimate a person's body fat percentage to an accuracy of 4%.

In adults, BMI, defined as weight (kg) divided by the square of the height (m^2), is used to screen for overweight or obesity. BMI of 25 to 29.9 kg/m^2 indicates overweight; BMI – 30 kg/m^2 indicates obesity (see Table 1: Obesity and the Metabolic Syndrome: Body Mass Index (BMI). However, BMI is a crude screening tool and has limitations in many subpopulations. BMI is age- and race-specific; its use is limited in children and the elderly. In children and adolescents, overweight is BMI at the e" 95th percentile based on age- and sex-specific CDC growth charts.

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Asians, Japanese, and many aboriginal populations have a lower cut-off (23 kg/m^2) for overweight. In addition, BMI may be high in muscular athletes who lack excess body fat, and normal or low in formerly overweight people who have lost muscle mass.

The risk of metabolic and cardiovascular complications due to obesity is determined more accurately by the following:

- Other risk factors, particularly a family history of type 2 diabetes or premature cardiovascular disease
- Waist circumference
- Plasma triglycerides

TABLE 1. BODY MASS INDEX (BMI)

	Normal*	Overweight	Obese	
BMI'	*19–24*	*25–29*	*30–34*	*35–39*
Height(inches)			*Body Weight (pounds)*	
60–61	97–127	128–153	153–180	179–206
62–63	104–135	136–163	164–191	191–220
64–65	110–144	145–174	174–204	204–234
66–67	118–153	155–185	186–217	216–249
68–69	125–162	164–196	197–230	230–263
70–71	132–172	174–208	209–243	243–279
72–73	140–182	184–219	221–257	258–295
74–75	148–192	194–232	233–272	272–311
76	156–197	205–238	246–279	287–320

*BMIs less than those listed as normal are considered underweight.

The waist circumference that increases risk of complications due to obesity varies by ethnic group and sex:

- **White men:** > 93 cm (> 36.6 in), particularly > 101 cm (> 39.8 in)
- **White women:** > 79 cm (> 31.1 in), particularly > 87 cm (> 34.2 in)
- **Indian men:** > 78 cm (> 30.7 in), particularly > 90 cm (> 35.4 in)
- **Indian women:** > 72 cm (> 28.3 in), particularly > 80 cm (> 31.5 in)

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity. The Japanese have defined obesity as any BMI greater than 25 while China uses a BMI of greater than 28.

Body composition analysis: Body composition—the percentage of body fat and muscle—is also considered when obesity is diagnosed. Although probably

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unnecessary in routine clinical practice, body composition analysis can be helpful if clinicians question whether elevated BMI is due to muscle or excessive fat.

The percentage of body fat can be estimated by measuring skinfold thickness (usually over the triceps) or determining mid upper arm area.

Bioelectrical impedance analysis (BIA) can estimate percentage of body fat simply and noninvasively. BIA estimates percentage of total body water directly; percentage of body fat is derived indirectly. BIA is most reliable in healthy people and in people with only a few chronic disorders that do not change the percentage of total body water (*e.g.*, moderate obesity, diabetes mellitus). Whether measuring BIA poses risks in people with implanted defibrillators is unclear.

Underwater (hydrostatic) weighing is the most accurate method for measuring percentage of body fat. Costly and time-consuming, it is used more often in research than in clinical care. To be weighed accurately while submerged, people must fully exhale beforehand.

Imaging procedures, including CT, MRI, and dual-energy x-ray absorptiometry (DEXA), can also estimate the percentage and distribution of body fat but are usually used only for research.

Waist Circumference and Waist–Hip Ratio

The waist circumference (>102 cm in men and >88 cm in women) and the waist–hip ratio (the circumference of the waist divided by that of the hips of >0.9 for men and >0.85 for women) are both used as measures of central obesity.

In those with a BMI under 35, intra-abdominal body fat is related to negative health outcomes independent of total body fat. Intra-abdominal or visceral fat has a particularly strong correlation with cardiovascular disease. In a study of 15,000 people, waist circumference also correlated better with metabolic syndrome than BMI. Women with abdominal obesity have a cardiovascular risk similar to that of men. In people with a BMI over 35, measurement of waist circumference however adds little to the predictive power of BMI as most individuals with this BMI have an abnormal waist circumferences.

Body Fat Percentage

Body fat percentage is total body fat expressed as a percentage of total body weight. It is generally agreed that men with more than 25% body fat and women with more than 33% body fat are obese. Body fat percentage can be estimated from a person's BMI by the following formula:

$$\text{Bodyfat\%} = (1.2 * \text{BMI}) + (0.23 * \text{age}) - 5.4 - (10.8 * \text{gender})$$

where gender is 0 if female and 1 if male.

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This formula takes into account the fact that body fat percentage is 10 percentage points greater in women than in men for a given BMI. It recognizes that a person's percentage body fat increases as they age even if their weight remains constant. The results of this formula have an accuracy of 4%.

There are many other methods used to determine body fat percentage. Hydrostatic weighing, one of the most accurate methods of body fat calculation, involves weighting a person underwater. Two other simpler and less accurate methods have been used historically but are now not recommended. The first is the skinfold test, in which a pinch of skin is precisely measured to determine the thickness of the subcutaneous fat layer. The other is bioelectrical impedance analysis which uses electrical resistance. Bioelectrical impedance has not been shown to provide an advantage over BMI.

Body fat percentage measurement techniques used mainly for research include computed tomography (CT scan), magnetic resonance imaging (MRI), and dual energy X-ray absorptiometry (DEXA). These techniques provide very accurate measurements, but it can be difficult to obtain in the severely obese due to weight limits of most equipment and insufficient diameter of many CT or MRI scanners.

Other Testing: Obese patients should be screened for obstructive sleep apnea with an instrument such as the Epworth Sleepiness Scale and often the apnea-hypopnea index (total number of apnea or hypopnea episodes occurring per hour of sleep. This disorder is often underdiagnosed, and obesity increases the risk.

Fasting plasma glucose and lipids should be measured routinely in patients with a large waist circumference or a family history of type 2 diabetes mellitus or premature cardiovascular disease.

Prognosis Untreated, obesity tends to progress. The probability and severity of complications are proportional to the absolute amount of fat, the distribution of the fat, and absolute muscle mass. After weight loss, most people return to their pretreatment weight within 5 yr, and accordingly, obesity requires a lifelong management program similar to that for any other chronic disorder.

2.5 TREATMENT

- Nutrition management
- Physical activity
- Behavioural therapy
- Drugs (*e.g.*, sibutramine)
- Bariatric surgery

Weight loss of even 5 to 10% improves overall health and well-being and in particular helps reduce risk of cardiovascular disorders and type 2 diabetes. Weight loss can lead to improvement in patients with obstructive sleep apnea, but sometimes a lot of weight must be lost for the disorder to resolve.

Support from health care practitioners, peers, and family members and various structured programs can help with weight loss and weight maintenance.

Nutrition

A normal eating pattern is important. People who miss breakfast tend to passively consume too many calories later in the day. Patients should eat small meals and avoid or carefully choose snacks. Low-fat (particularly very low saturated fat), high-fiber diets with modest calorie restriction (by 600 kcal/day) and substitution of some protein for carbohydrate appear to have the best long-term outcome. Fresh fruits and vegetables and salads should be substituted for refined carbohydrates and processed food, and water for soft drinks or juices. Alcohol consumption should be limited to moderate levels. Foods with a low glycemic index and marine fish oils or monounsaturated fats derived from plants (*e.g.*, olive oil) reduce the risk of cardiovascular disorders and diabetes. Low-fat dairy products are also part of a healthy diet. Patients need an adequate amount of vitamin D, preferably obtained by exercising outdoors in the sunshine.

Use of meal replacements has proven efficacy; use can be ongoing or intermittent. Diets that require unusual eating habits should be avoided. They are unlikely to be maintained, and weight increases when patients resume previous poor eating habits. Diets of < 1200 kcal/day cannot be sustained, but such diets are sometimes needed to achieve rapid short-term weight loss (*e.g.*, to prepare for surgery, to lessen obstructive sleep apnea). Diets of < 800 kcal/day do not produce greater weight loss and are less well tolerated.

Physical Activity

Exercise increases energy expenditure, BMR, and diet-induced thermogenesis. Exercise also seems to regulate appetite to more closely match caloric needs. Other benefits include

- Increased insulin sensitivity
- Improved plasma lipid profile
- Lower BP
- Better aerobic fitness
- Improved psychologic well-being

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Strengthening (resistance) exercises increase muscle mass. Because muscle tissue burns more calories at rest than does fat tissue, increasing muscle mass produces lasting increases in BMR. Exercise that is interesting and enjoyable is more likely to be sustained. A combination of aerobic and resistance exercise is better than either alone.

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.

The city of Bogota, Colombia blocks off 113 kilometers (70 miles) of roads every Sunday and on holidays to make it easier for it citizens to get exercise. These pedestrian zones are part of an effort to combat chronic diseases, including obesity.

Behavioural Therapy

Behavioural therapy aims to improve eating habits and physical activity level. Rigid dieting is discouraged in favour of healthy eating. Common-sense measures include the following :

- Avoiding high-calorie snacks
- Choosing healthful foods when dining out
- Eating slowly
- Substituting a physically active hobby for a passive one

Social support, cognitive therapy, and stress management may help, particularly during the lapses usually experienced during any long-term weight loss program. Self-monitoring is useful, and maintenance of a diet diary is particularly effective.

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DRUGS

Drugs may be used if BMI is > 30 or if BMI is > 27 and patients have complications (*e.g.*, hypertension, insulin resistance). Most weight loss due to drug treatment is modest (5 to 10%) at best and occurs during the first 6 months; not all patients benefit. Drugs are more useful for maintaining weight loss but must be continued indefinitely for weight loss to be maintained. Premenopausal women taking systemically acting drugs for weight control should use contraception.

Sibutramine is a centrally acting appetite suppressant that produces dose-related weight loss. The usual starting dose is 10 mg po once/day; the dose can be decreased to 5 mg or increased to 15 mg. Common adverse effects are headache, dry mouth, insomnia, and constipation; the most common serious one is hypertension. Cardiovascular disorders, particularly poorly controlled hypertension, are contraindications.

Orlistat inhibits intestinal lipase, decreasing fat absorption and improving blood glucose and lipids. Because orlistat is not absorbed, systemic effects are rare. Flatus, oily stools, and diarrhea are common but tend to resolve during the 2nd yr of treatment. A dose of 120 *mg po tid* should be taken with meals that include fat. A vitamin supplement should be taken at least 2*h* before or after taking orlistat Malabsorption and cholestasis are contraindications; irritable bowel syndrome and other GI disorders may make orlistat difficult to tolerate. Orlistat is available OTC.

Other OTC weight-loss drugs are not recommended. Some (*e.g.*, caffeine, ephedrine, guarana, phenylpropanolamine) may be marginally effective, but their adverse effects outweigh their advantages. Others (*e.g.*, brindleberry, L-carnitine, chitosan, pectin, grapeseed extract, horse chestnut, chromium picolinate, fucus vesiculosus, ginkgo biloba) have not been shown to be effective and may have adverse effects.

SURGERY

Surgery is the most effective treatment for extremely obese patients.

2.6 OBESITY IN SPECIAL POPULATIONS

Obesity is a particular concern in children and the elderly.

CHILDREN

Childhood obesity is even more worrisome than adult obesity. For obese children, complications are more likely because they are obese longer. About 20 to 25% of children and adolescents are overweight or obese. Risk factors for obesity in infants are low birth weight and maternal obesity, diabetes, and smoking. After

puberty, food intake increases; in boys, the extra calories are used to increase protein deposition, but in girls, fat storage is increased.

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For obese children, psychologic complications (*e.g.*, poor self-esteem, social difficulties, depression) and musculoskeletal complications can develop early. Some musculoskeletal complications, such as slipped capital femoral epiphyses, occur only in children. Other early complications may include obstructive sleep apnea, insulin resistance, hyperlipidemia, and nonalcoholic steatohepatitis. Risk of cardiovascular, respiratory, metabolic, hepatic, and other obesity-related complications increases when these children become adults.

The healthy BMI range varies with the age and sex of the child. Obesity in children and adolescents is defined as a BMI greater than the 95th percentile. The reference data that these percentiles are based on is from 1963 to 1994 and thus has not been affected by the recent increases in rates of obesity.

Childhood obesity has reached epidemic proportions in 21st century with rising rates in both the developed and developing world. Rates of obesity in Canadian boys have increased from 11% in 1980s to over 30% in 1990s, while during this same time period rates increased from 4 to 14% in Brazilian children.

As with obesity in adults many different factors contribute to the rising rates of childhood obesity. Changing diet and decreasing physical activity are believed to be the two most important in causing the recent increase in the rate of obesity. Activities from selfpropelled transport, to school physical education, and organized sports has been declining in many countries.

Because childhood obesity often persists into adulthood, and is associated with numerous chronic illnesses, it is important that children who are obese be tested for hypertension, diabetes, hyperlipidemia, and fatty liver.

Risk of obesity persisting into adulthood depends partly on when obesity first develops:

- During infancy: Low risk
- Between 6 months and 5 yr: 25%
- After 6 yr: > 50%
- During adolescence if a parent is obese: > 80%

In children, preventing further weight gain, rather than losing weight, is a reasonable goal. Diet should be modified, and physical activity increased. Increasing general activities and play is more likely to be effective than a structured exercise program. Participating in physical activities during childhood may promote a lifelong physically active lifestyle. Drugs and surgery are avoided but, if complications of obesity are life threatening, may be warranted.

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Measures that control weight and prevent obesity in children may benefit public health the most. Such measures should be implemented in the family, schools, and primary care programs.

THE ELDERLY

The percentage of obese elderly people has been increasing.

With aging, body fat increases and is redistributed to the abdomen, and muscle mass is lost, largely because of physical inactivity, but decreased androgens and growth hormone (which are anabolic) and inflammatory cytokines produced in obesity may also play a role.

Risk of complications depends on

- Body fat distribution (increasing with a predominantly abdominal distribution)
- Duration and severity of obesity
- Associated sarcopenia

Increased waist circumference, suggesting abdominal fat distribution, predicts morbidity (*e.g.*, hypertension, diabetes mellitus, coronary artery disease) and mortality risk better in the elderly than BMI.

For the elderly, increased physical activity is usually preferable to dietary restriction unless restricted mobility prohibits activity; in such cases, caloric restriction may be needed to reduce weight enough to restore mobility. Physical activity also improves muscle strength, endurance, and overall well-being. Activity should include strengthening and endurance exercises.

Regardless of whether caloric restriction is considered necessary, nutrition should be optimized.

Weight-loss drugs such as sibutramine or fluoxetine are not recommended for the elderly because the possible benefits do not outweigh the adverse effects. However, orlistat may be useful for obese elderly patients, particularly those with diabetes mellitus or hypertension. Surgery is usually best avoided, although it has proven efficacy and benefits outweigh risks in carefully selected patients.

OBESITY IN INDIA

In 1997, an article in the Indian Express dated July 19th stated that the incidence of obesity in India is 7-9 % and comprising mainly of urbanites. Although this number is small as compared to America and other countries, it is significant due to the sheer size of the population in India. With such large numbers, India has been requested to join the International Congress on Obesity (ICO) for further

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study on the risk and management of the overweight in a developing economy. With this, India would be one of the first nations from the developing countries in Asia to be put on the obesity map.

In the urban population of India, refined wheat and rice have virtually displaced coarse grains and millets as the staple cereal, resulting in a substantial reduction in fibre content in the diet and possibly, also the content of micronutrients such as vitamin B-complex, zinc and chromium, etc. As the population ascends the socio-economic scale, cereal intake declines and the intake of sugar and fats generally increase. Convenience and fast foods find increasing acceptance, especially in the context of globalisation.

While a third of India's population still falls below the poverty line, there has been a steady growth of the relatively affluent urban middle class, now estimated to number over 200 million. Those who have achieved affluence within a lifetime constitute a good proportion of this middle class.

The Nutrition Foundation of India has just completed a study of the prevalence of obesity in urban Delhi.

It would appear from the results of this study that nearly a third of the males and more than half of females belonging to what may be termed the 'upper middle class' in India are currently overweight (BMI>25). The prevalence of abdominal obesity in this group is even higher. Assuming that the 'upper middle class' in India number around 100 million (half the number of middle class), it may be computed that there are roughly 40 to 50 million overweight subjects belonging to the upper middle class in the country today. If present trends continue, the situation can get worse within a decade, and overweight could emerge as the single most important public health problem in adults. Overweight/obesity may not be considered as a specific disease but it is certainly the mother of important degenerative diseases in adult life. Prevention and control of this problem must, therefore, claim priority attention.

Measuring Body Weight

The definition of ideal or desirable body weight changes from time to time as seen from periodic revisions in the various height-weight tables. These tables are usually derived from mortality data; they under represent the lower socio - economic class, minorities and elderly with arbitrary definition of body frame size.

The desirable body weight is different for different people. Conforming strictly to a printed chart should therefore not be an obsession.

On a more scientific basis, obesity is expressed in terms of body mass index (BMI): –

This is calculated in the following way:

$$BMI = Weight\ in\ kilograms / (Height\ in\ meters)^2$$

The BMI can be compared with the following ranges:

Below 20	*Underweight*
20-25	Acceptable
25-30	Overweight
30-40	Obese
Over 40	Very obese

2.7 PATHOPHYSIOLOGY

Flier summarizes the many possible pathophysiological mechanisms involved in the development and maintenance of obesity. This field of research had been almost unapproached until leptin was discovered in 1994. Since this discovery, many other hormonal mechanisms have been elucidated that participate in the regulation of appetite and food intake, storage patterns of adipose tissue, and development of insulin resistance. Since leptin's discovery, ghrelin, insulin, orexin, PYY 3-36, cholecystokinin, adiponectin, as well as many other mediators have been studied. The adipokines are mediators produced by adipose tissue; their action is thought to modify many obesity-related diseases.

Leptin and ghrelin are considered to be complementary in their influence on appetite, with ghrelin produced by the stomach modulating short-term appetitive control (*i.e.*, to eat when the stomach is empty and to stop when the stomach is stretched). Leptin is produced by adipose tissue to signal fat storage reserves in the body, and mediates long-term appetitive controls (*i.e.*, to eat more when fat storages are low and less when fat storages are high). Although administration of leptin may be effective in a small subset of obese individuals who are leptin deficient, most obese individuals are thought to be leptin resistant and have been found to have high levels of leptin. This resistance is thought to explain in part why administration of leptin has not been shown to be effective in suppressing appetite in most obese people.

While leptin and ghrelin are produced peripherally, they control appetite through their actions on the central nervous system. In particular, they and other appetite-related hormones act on the hypothalamus, a region of the brain central to the regulation of food intake and energy expenditure. There are several circuits within the hypothalamus that contribute to its role in integrating appetite, the melanocortin pathway being the most well understood. The circuit begins with an area of the hypothalamus, the arcuate nucleus, that has outputs to the lateral

hypothalamus (LH) and ventromedial hypothalamus (VMH), the brain's feeding and satiety centers, respectively.

The arcuate nucleus contains two distinct groups of neurons. The first group coexpresses neuropeptide Y (NPY) and agouti-related peptide (AgRP) and has stimulatory inputs to the LH and inhibitory inputs to the VMH. The second group coexpresses pro-opiomelanocortin (POMC) and cocaine- and amphetamine-regulated transcript (CART) and has stimulatory inputs to the VMH and inhibitory inputs to the LH. Consequently, NPY/AgRP neurons stimulate feeding and inhibit satiety, while POMC/CART neurons stimulate satiety and inhibit feeding. Both groups of arcuate nucleus neurons are regulated in part by leptin. Leptin inhibits the NPY/AgRP group while stimulating the POMC/CART group. Thus a deficiency in leptin signaling, either via leptin deficiency or leptin resistance, leads to overfeeding and may account for some genetic and acquired forms of obesity.

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2.8 MANAGEMENT

The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the shortterm, 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.

2.9 DIETING

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.

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.

2.10 PREVENTION

Regular physical activity and healthy eating improve general fitness, can control weight, and help prevent obesity and diabetes mellitus. Even without weight loss, exercise decreases the risk of cardiovascular disorders. Dietary fiber decreases the risk of colon cancer and cardiovascular disorders. Sufficient and

good-quality sleep, management of stress, and moderation of alcohol intake are also important.

NOTES

Weight Loss Programs

Weight loss programs often promote lifestyle changes and diet modification. This may involves eating smaller meals, by cutting down on certain types of food, and making a conscious effort to exercise more. These program enables people to connect with a group of others that are attempting to loss weight in the hopes that they will encourage and help each other out.

A number of different programs exist including Weight Watchers, Overeaters Anonymous, and Jenny Craig. They appear to provide modest weight loss (2.9 kg, 6.4 lb) over dieting on ones own (0.2 kg, 0.4 lb) over a two year period. Internet based programs appear to be ineffective. In China, the government has introduced a number of fat farms where obese children go for reinforced exercise, and has passed a law which requires students to exercise or play sports for an hour a day at school.

Medication

Only two anti-obesity medications are currently approved by the FDA for longterm use. One is orlistat (Xenical), which reduces intestinal fat absorption by inhibiting pancreatic lipase; the other is sibutramine (Meridia), which acts in the brain to inhibit deactivation of the neurotransmitters norepinephrine, serotonin, and dopamine (very similar to some anti-depressants), therefore decreasing appetite. Rimonabant (Acomplia), a third drug, works via a specific blockade of the endocannabinoid system. It has been developed from the knowledge that cannabis smokers often experience hunger, which is often referred to as "the munchies". It has been approved in Europe for the treatment of obesity but has not yet received approval in the United States or Canada due to safety concerns.

Weight loss with these drugs is modest; over the longer term, average weight loss on orlistat is 2.9 kg (6.4 lb), sibutramine is 4.2 kg (9.3 lb) and rimonabant is 4.7 kg (10.4 lb). Orlistat and rimonabant lead to a reduced incidence of diabetes, and all three drugs have some effect on cholesterol. There is however little data on how these drugs affect the longer-term complications or outcomes of obesity.

There are a number of less commonly used medication. Some are approved for only shortterm use, others are used off label, and still others are used illegally. Most are appetite suppressants that act on either one or more neurotransmitters. Phendimetrazine (Bontril), diethylpropion (Tenuate), and phentermine (Adipex-*P*) are approved by the FDA for shortterm use while bupropion (Wellbutrin), topiramate (Topamax), and zonisamide (Zonegran) are sometimes used off label.

Certain drugs are useful depending on the comorbities present. Metformin (Glucophage) is preferred in overweight diabetics as it may lead to mild weight loss in comparison to sulfonylureas or insulin. The thiazolidinediones, on the other hand, may cause weight gain, but decrease central obesity. Diabetics also achieve modest weight loss with fluoxetine (Prozac), orlistat and sibutramine over 12–57 weeks. The long-term health benefits of theses treatments however remains unclear.

Fenfluramine and dexfenfluramine were withdrawn from the market in 1997, while ephedrine (Ma Huang) was removed from the market in 2004. Dexamphetamines are not approve by the FDA for the treatment of obesity due to concerns regarding addiction. These drug are all not recommended due to potential side effects. People however do occasionally use these drug illegally.

Surgery

Bariatric surgery ("weight loss surgery") is the use of surgical interventions in the treatment of obesity. As every operation may have complications, surgery is only recommended for severely obese people (BMI > 40) who have failed to lose weight with dietary modification and pharmacological treatment. Weight loss surgery relies on various principles; the most common approaches are reducing the volume of the stomach, producing an earlier sense of satiation (*e.g.*, by adjustable gastric banding and vertical banded gastroplasty) and reduce the length of bowel that food will be in contact with, directly reducing absorption (gastric bypass surgery). Band surgery is reversible, while bowel shortening operations are not. Some procedures can be performed laparoscopically. Complications from weight loss surgery are frequent.

Surgery for severe obesity is associated with long-term weight loss and decreased overall mortality. One study found a weight loss of between 14% and 25% at 10 years depending on the type of procedure performed and a 29% reduction in all cause mortality when compared to standard weight loss measures. A marked decrease in the risk of diabetes mellitus, cardiovascular disease and cancer has also been found after bariatric surgery. Weight loss is marked in the first few months after surgery and is sustained in the longterm. In one study there was an unexplained increase in deaths from accidents and suicide but this did not outweigh the benefit in terms of disease prevention. When the two main techniques are compared gastric bypass procedures are found to lead to 30% more weight loss than banding procedures one year after surgery.

The effects of liposuction on obesity are less well determined. Some small studies show benefits while others show none. A treatment involving the placement of an intragastric balloon via gastroscopy has shown promise. One

type of ballon lead to a weight loss of 5.7 BMI units over 6 months or 14.7 kg (32.4 lb). Regaining of lost weight is however common after removal and 4.2% of people were intolerant of the device.

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2.11 CLINICAL PROTOCOLS

In a clinical practice guideline by the American College of Physicians, the following five recommendations are made :

1. People with a BMI of over 30 should be counseled on diet, exercise and other relevant behavioural interventions, and set a realistic goal for weight loss.
2. If these goals are not achieved, pharmacotherapy can be offered. The person needs to be informed of the possibility of side-effects and the unavailability of long-term safety and efficacy data.
3. Drug therapy may consist of sibutramine, orlistat, phentermine, diethylpropion, fluoxetine, and bupropion. For more severe cases of obesity, stronger drugs such as amphetamine and methamphetamine may be used on a selective basis. Evidence is not sufficient to recommend sertraline, topiramate, or zonisamide.
4. In people with a BMI over 40 who fail to achieve their weight loss goals (with or without medication) and who develop obesity-related complications, referral for bariatric surgery may be indicated. The person needs to be aware of the potential complications.
5. Those requiring bariatric surgery should be referred to high-volume referral centers, as the evidence suggests that surgeons who frequently perform these procedures have fewer complications.

STUDENT ACTIVITY

1. Outline the causes of obesity.

2. Discuss the essential dieting plan for obesity.

PUBLIC HEALTH

The World Health Organization (WHO) predicts that overweight and obesity may soon replace more traditional public health concerns such as undernutrition and infectious diseases as the most significant cause of poor health. Obesity is a public health and policy problem because of its prevalence, costs, and health effects. Public health efforts seek to understand and correct the environmental factors responsible for the increasing prevalence of obesity in the population. Solutions look at changing the factors that cause excess calorie consumption and inhibit physical activity. Efforts include federally reimbursed meal programs in schools, limiting direct junk food marketing to children, and decreasing access to sweetened beverages in schools. When constructing urban environments, efforts have been made to increase access to parks and to develop pedestrian routes.

2.12 UNDERWEIGHT PROBLEMS – BODY MASS INDEX (BMI)

Being underweight would indicate that your Body Mass Index, or BMI, is below the standard weight. The BMI is calculated to estimate how heavy you are, given your height – or your mass spread over the square of your height. The importance of this measurement has ramifications for your health as a lack of proper nutrition or deficient intake means that you cannot perform certain body functions as normal. The way to come back to a normal weight is simple. All you have to do is eat, but that is where the simplicity ends. Eating just to put on weight could have serious consequences for your health if you do not do it correctly. For example, you could get really heavy by just eating fat, but that would be the wrong way to gain weight and you would just end up with flab and no muscle. Therefore, eating correctly and exercising correctly is the secret to good health.

WEIGHT GAIN DIETS – FOODS, NUTRITION TO GAIN WEIGHT

The basic food groups that you have to concentrate on gain weight again is protein, carbohydrates, vitamins, minerals and very little fat. Fat is not just in the form of cheese and butter, but also in the form of cream and oil. To begin with, start to eat food with a limited amount of oil. The oil itself should be light plant oil like safflower or olive oil. Protein is one thing that you can actually gorge on and this can be in the form of animal protein, like dairy and meat, and plant protein like soy and legumes. Carbohydrates intake is important from the perspective of every day fuel required to get through the day. Therefore, the carbohydrate sources should only come from food, and not carbonated drinks and food with high sugar content. Vitamins and minerals will come from a good vegetarian diet of

leafy green vegetables, soy, dairy, and if necessary supplements. Supplements are one addition that you cannot do away with because humans do not have the capability to acquire vitamins from plant sources— like vitamin B12. This supplement can only come from an animal source like meat and dairy.

NOTES

Body Weight Exercise

Exercise is the final part of the puzzle. The day you start your diet is the day that you start exercising as well. This should start with simple toning exercises like walking and then progressing everyday to activities like jogging and running that eventually progresses to some basic weightlifting as well.

2.13 ETIOLOGY OF UNDERWEIGHT

The most common cause of a person being underweight is primarily malnutrition caused by the unavailability of adequate food, which can run as high as 50% in parts of sub-Saharan Africa and south Asia. The effects of primary malnutrition may be amplified by disease; even easily treatable diseases such as diarrhoea may lead to death.

In the presence of adequate food resources, being underweight can sometimes be the result of mental or physical disease. There are hundreds of possible causes for excessive weight loss or a person being underweight. Some of the more prevalent include:

- Poverty
- Anorexia nervosa
- Bulimia nervosa
- Cancer or Cancer Treatment
- Tuberculosis
- Hyperthyroidism
- Type 1 Diabetes
- Anxiety and depressive disorders
- Drug use, especially stimulants
- Inflammatory bowel disease
- Superior Mesenteric Artery Syndrome
- Malfunctioning digestive organs
- Dental pain
- Over-training (endurance sports)

NOTES

- HIV/AIDS
- Genetics / Naturally light weight
- Puberty (height increases, body cannot catch up with muscle/fat growth)

2.14 PROBLEMS

The most immediate problem with underweight is that it might be secondary to, and/or symptomatic of, an underlying disease. Unexplained weight loss requires professional medical diagnosis.

Underweight can also be a primary causative condition. Severely underweight individuals may have poor physical stamina and a weak immune system, leaving them open to infection. According to Robert E. Black of the Johns Hopkins School of Public Health, "Underweight status ... and micronutrient deficiencies also cause decreases in immune and non-immune host defenses, and should be classified as underlying causes of death if followed by infectious diseases that are the terminal associated causes." People who are malnutrative underweight raise special concerns, as not only gross caloric intake may be inadequate, but also intake and absorption of other vital nutrients, especially essential amino acids and micronutrients such as vitamins and minerals.

In women, being grossly underweight can result in amenorrhea (absence of menstruation) and possible complications during pregnancy. It can also cause anemia and hair loss.

Underweight is an established risk factor for osteoporosis, even for young people. This is a particular insidious consequence, because the affected persons do not notice the danger, they can feel fit and may be brilliant for example; in endurance sports. After the occurrence of first spontaneous fractures the damage is often already irreversible.

Weight gain

If an individual is severely underweight to the point where problems with his or her health develop, it may be necessary for the person to make a concentrated effort to gain weight. The treatment for an underweight individual is to increase the food energy intake so that more food energy is consumed than is being used as work. It is usually suggested that weight training is also to be undertaken to increase muscle mass.

If weight loss results from a disease, resolving the illness and consuming adequate calories can bring many underweight individuals to a healthy body weight.

2.15 UNDERWEIGHT SOLUTIONS

NOTES

Underweight may be due to eating disorders high basal metabolism malnutrition digestive tract disorder or born with few fat cells.

Diseases and Medicines

One may be underweight due to an eating disorder such as anorexia and bulimia, over active thyroids, hormonal imbalance, depression, or taking certain medications. You should consult a physician to determine the cause and subsequent remedies.

Metabolism

You may have high basal metabolism. This can be easily resolved by taking high calorie diet and life style changes.

Malnutrition

Malnutrition can cause underweight. A balanced high calorie diet is needed. You may also consider taking a daily multi vitamin and mineral supplement and a daily omega 3 supplement.

Nutrition Absorption Digestive Tracts

You need a diet high in fiber and fluids for proper stool density and to expel. Omega 3 fatty acids help lubricate colon. For Omega 3, eat nuts and high fat fish. The intestines, specially the colon area is filled with bacteria. Eat yogurt with live culture to ensure presence of good bacteria. Enzymes are needed to breakdown carbohydrates, proteins and fats. Most of these enzymes are only present in raw foods. Eat fresh fruits (Papayas are specially good), and vegetables.

Fat cells

All of us are all born with a given number of fat cells. The fat gathers around these cells making a fat tissue (Adipose). You may be born with low number of fat cells. Unfortunately, you cannot increase the number of fat cells. The diet can help increase the size of adipose to certain extent.

2.16 GRADES OF OBESITY

Grade I obesity

People having a Body Mass Index (BMI), more than 25 but less than 29.9. Overweight does not affect their health, they lead a normal health and life expectancy is above normal. They may reduce weight on their own.

NOTES

GRADE II OBESITY

The BMI is between 30 and 39.9. Doctors and dieticians should treat these patients.

Although they appear to be in good health, they have reduced tolerance to exercise and with shortness of breath on exertion, they are unduly fatigued. This is due to the burden of increased weight they carry always and reduced capacity of the circulatory and respiratory systems that are handicapped by masses of internal fat and fatty infiltration of muscle. For mechanical and metabolic reasons these patients are at increased risk of diabetes, artheroscelerosis, hypertension, fatty liver, gall bladder diseases, osteoarthritis, hernias and varicose veins. Mortality rate also increases.

GRADE III OBESITY

The body mass index is above 40; these patients are in pathetic condition. Their day to day activities are restricted due to their enormous mass and susceptible to diseases mentioned in grade II. They are susceptible to artherosclerosis, prone to accidents and have serious psychological disturbances.

STUDENT ACTIVITY

1. Discuss the characteristics of underweight.

2. Point out the basic causes of underweight.

2.17 SUMMARY

NOTES

- 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.
- Almost all cases of obesity result from a combination of genetic predisposition and a chronic imbalance between energy intake, energy utilization for basic metabolic processes, and energy expenditure from physical activity.
- Body mass index or BMI is a simple and widely used method for estimating body fat mass. BMI was developed in the 19th century by the Belgian statistician and anthropometrist Adolphe Quetelet.
- Diets to promote weight loss are generally divided into four categories: low-fat, low-carbohydrate, low-calorie, and very low calorie.
- The most common cause of a person being underweight is primarily malnutrition caused by the unavailability of adequate food, which can run as high as 50% in parts of sub-Saharan Africa and south Asia.

2.18 GLOSSARY

- **Obesity:** a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health.
- **Body mass index or BMI:** a simple and widely used method for estimating body fat mass.
- **Body fat percentage:** it is total body fat expressed as a percentage of total body weight.
- **Low-fat diet:** it involves the reduction of the percentage of fat in one's diet.
- **Low carbohydrate diet:** it is diet low in carbohydrate and are relatively high in fat and protein.
- **Low-calorie diet:** a diet that usually produces an energy deficit of 500–1,000 calories per day.
- **Very low calorie diet:** a diet that provides 200–800 kcal/day, maintaining protein intake.

2.19 REVIEW QUESTIONS

1. What is obesity? Define.
2. How is obesity measured?
3. What is the condition of extreme obesity?

4. What are the main causes of obesity? Explain.
5. How is obesity treated?
6. What is the status of obesity in India?
7. Why is underweight dangerous?

2.20 FURTHER READINGS

- *Dictionary of foods, nutrition* and *dietetics.*
- Kawshik, V.K. Haroco, *problems of food nutrition and dietetics,* Book enclave, Jain Bhavan 1999.
- Mahatab S., Bamji, Prahlad Rao, Vinodini Reddy, *Text of human nutrition,* Oxford and IBM publishing Co., Pvt. Ltd.
- *Special Diet Solutions* by Carol Fenster.

UNIT – III

FOOD ALLERGY AND INTOLERANCE

OBJECTIVES

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

- state the causes and symptoms of food allergy;
- discuss the types of allergens;
- explain the metabolic changes during food allergy;
- understand the diagnostic tests and dietary management during food allergy.

STRUCTURE

3.1 INTRODUCTION

A food allergy is an adverse immune response to a food protein. Food allergy is distinct from other adverse responses to food, such as food intolerance, pharmacologic reactions, and toxin-mediated reactions.

Food Allergy	*Pharmacologic*	*Toxins*	*Intolerance*
adverse immune response to a food protein	caffeine tremors, cheese/wine (tyramine) migraine, scombroid (histamine) fish poisoning	bacterial food poisoning, staphylotoxin	lactose intolerance (lactase deficiency)

The food protein triggering the allergic response is termed a food allergen. It is estimated that up to 12 million Americans have food allergies, and the prevalence is rising. Six to eight percent of children under the age of three have food allergies and nearly four percent of adults have them. Food allergies cause roughly 30,000 emergency room visits and 100 to 200 deaths per year in the United States. The most common food allergies in adults are shellfish, peanuts, tree nuts, fish, and eggs, and the most common food allergies in children are milk, eggs, peanuts, and tree nuts.

Treatment consists of avoidance diets, in which the allergic person avoids all forms of the food to which they are allergic. For people who are extremely sensitive, this may involve the total avoidance of any exposure with the allergen, including touching or inhaling the problematic food as well as touching any surfaces that may have come into contact with it. Areas of research include anti-IgE antibody (omalizumab, or Xolair) and specific oral tolerance induction (SOTI), which have shown some promise for treatment of certain food allergies. People diagnosed with a food allergy may carry an autoinjector of epinephrine such as an EpiPen or Twinject, wear some form of medical alert jewellery, or develop an emergency action plan, in accordance with their doctor.

3.2 SIGNS AND SYMPTOMS

Classic immunoglobulin-E (IgE)-mediated food allergies are classified as type-I immediate hypersensitivity reactions. These allergic reactions have an acute onset (from seconds to one hour) and may include:

- **Angioedema:** soft tissue swelling, usually involving the eyelids, face, lips, and tongue. Angioedema may result in severe swelling of the tongue as well as the larynx (voice box) and trachea, resulting in upper airway obstruction and difficulty breathing.
- **Hives**
- Itching of the mouth, throat, eyes, skin
- Nausea, vomiting, diarrhoea, stomach cramps, and/or abdominal pain. This group of symptoms is termed gastrointestinal hypersensitivity.

- Rhinorrhoea, nasal congestion
- Wheezing, scratchy throat, shortness of breath, or difficulty swallowing
- **Anaphylaxis:** a severe, whole-body allergic reaction that can result in death.

The reaction may progress to anaphylactic shock: A systemic reaction involving several different bodily systems including hypotension (low blood pressure),loss of consciousness, and possibly death. Allergens most frequently associated with this type of reaction are peanuts, nuts, milk, egg, and seafood, though many food allergens have been reported as triggers for anaphylaxis.

Food allergy is thought to develop more easily in patients with the atopic syndrome, a very common combination of diseases: allergic rhinitis and conjunctivitis, eczema and asthma. The syndrome has a strong inherited component; a family history of allergic diseases can be indicative of the atopic syndrome

Conditions caused by food allergies are classified into 3 groups according to the mechanism of the allergic response:

1. *IgE-mediated (classic):*
   - Type-I immediate hypersensitivity reaction (symptoms described above)
   - Oral allergy syndrome
2. *IgE and/or non-IgE-mediated:*
   - Allergic eosinophilic esophagitis
   - Allergic eosinophilic gastritis
   - Allergic eosinophilic gastroenteritis
3. *Non-IgE mediated:*
   - Food protein-induced Enterocolitis syndrome (FPIES)
   - Food protein proctocolitis/proctitis
   - Food protein-induced enteropathy. An important example is Coeliac disease, which is an adverse immune response to the protein gluten.
   - Milk-soy protein intolerance (MSPI) is a non-medical term used to describe a non-IgE mediated allergic response to milk and/or soy protein during infancy and early childhood. Symptoms of MSPI are usually attributable to food protein proctocolitis or FPIES.
   - Heiner syndrome - lung disease due to formation of milk protein/IgG antibody immune complexes (milk precipitins) in the blood stream after

it is absorbed from the GI tract. The lung disease commonly causes bleeding into the lungs and results in pulmonary hemosiderosis.

3.3 TYPE OF ALLERGENS

The most common food allergies are:

- Dairy allergy
- Egg allergy
- Peanut allergy
- Tree nut allergy
- Seafood allergy
- Shellfish allergy
- Soy allergy
- Wheat allergy

These are often referred to as "the big eight." They account for over 90% of the food allergies in the India.

The top allergens vary somewhat from country to country but milk, eggs, peanuts, treenuts, fish, shellfish, soy, wheat and sesame tend to be in the top 10 in many countries. Allergies to seeds — especially sesame — seem to be increasing in many countries.

More Rare Food Allergies

Likelihood of allergy can increase with exposure. For example, rice allergy is more common in East Asia where rice forms a large part of the diet.

In Central Europe, celery allergy is more common. In Japan, allergy to buckwheat flour, used for Soba noodles, is more common.

Red meat allergy is extremely rare in the general population, but a geographic cluster of people allergic to red meat has been observed in Sydney, Australia. There appears to be a possible association between localised reaction to tick bite and the development of red meat allergy.

Fruit allergies exist, such as to apples, pears, jackfruit, strawberries, etc.

Corn allergy may also be prevalent in many populations, although it may be difficult to recognize in areas such as the United States and Canada where corn derivatives are common in the food supply.

3.4 DIAGNOSIS

The best method for diagnosing food allergy is to be assessed by an allergist. The allergist will review the patient's history and the symptoms or reactions that

have been noted after food ingestion. If the allergist feels the symptoms or reactions are consistent with food allergy, he/she will perform allergy tests.

Examples of allergy testing include:

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- Skin prick testing is easy to do and results are available in minutes. Different allergists may use different devices for skin prick testing. Some use a "bifurcated needle", which looks like a fork with 2 prongs. Others use a "multi-test", which may look like a small board with several pins sticking out of it. In these tests, a tiny amount of the suspected allergen is put onto the skin or into a testing device, and the device is placed on the skin to prick, or break through, the top layer of skin. This puts a small amount of the allergen under the skin. A hive will form at any spot where the person is allergic. This test generally yields a positive or negative result. It is good for quickly learning if a person is allergic to a particular food or not, because it detects allergic antibodies known as IgE. Skin tests cannot predict if a reaction would occur or what kind of reaction might occur if a person ingests that particular allergen. They can however confirm an allergy in light of a patient's history of reactions to a particular food. Non-IgE mediated allergies cannot be detected by this method.
- Blood tests are another useful diagnostic tool for evaluating IgE-mediated food allergies. For example, the RAST (RadioAllergoSorbent Test)detects the presence of IgE antibodies to a particular allergen. A CAP-RAST test is a specific type of RAST test with greater specificity: it can show the amount of IgE present to each allergen. Researchers have been able to determine "predictive values" for certain foods. These predictive values can be compared to the RAST blood test results. If a persons RAST score is higher than the predictive value for that food, then there is over a 95% chance the person will have an allergic reaction (limited to rash and anaphylaxis reactions) if they ingest that food. Currently, predictive values are available for the following foods: milk, egg, peanut, fish, soy, and wheat. Blood tests allow for hundreds of allergens to be screened from a single sample, and cover food allergies as well as inhalants. However, non-IgE mediated allergies cannot be detected by this method.
- Food challenges, especially double-blind placebo-controlled food challenges (DBPCFC), are the gold standard for diagnosis of food allergies, including most non-IgE mediated reactions. Blind food challenges involve packaging the suspected allergen into a capsule, giving it to the patient, and observing the patient for signs or symptoms of an allergic reaction. Due to the risk of anaphylaxis, food challenges are usually conducted in a hospital environment in the presence of a doctor.

- Additional diagnostic tools for evaluation of eosinophilic or non-IgE mediated reactions include endoscopy, colonoscopy, and biopsy.

Important differential diagnoses are:

- Lactose intolerance; this generally develops later in life but can present in young patients in severe cases. This is due to an enzyme deficiency (lactase) and not allergy. It occurs in many non-Western people.
- Celiac disease; this is an autoimmune disorder triggered by gluten proteins such as gliadin (present in wheat, rye and barley). It is a non-IgE mediated food allergy by definition.
- Irritable bowel syndrome (IBS)
- C1 esterase inhibitor deficiency (hereditary angioedema); this rare disease generally causes attacks of angioedema, but can present solely with abdominal pain and occasional diarrhoea.

3.5 PATHOPHYSIOLOGY

Generally, introduction of allergens through the digestive tract is thought to induce immune tolerance. In individuals who are predisposed to developing allergies (atopic syndrome), the immune system produces IgE antibodies against protein epitopes on non-pathogenic substances, including dietary components. The IgE molecules are coated onto mast cells, which inhabit the mucosal lining of the digestive tract.

Upon ingesting an allergen, the IgE reacts with its protein epitopes and release (degranulate) a number of chemicals (including histamine), which lead to oedema of the intestinal wall, loss of fluid and altered motility. The product is diarrhoea.

Any food allergy has the potential to cause anaphylaxis, which in rare cases may be fatal.

3.6 CAUSES

The immune system's eosinophils, once activated in a histamine reaction, will register any foreign proteins they see. One theory regarding the causes of food allergies focuses on proteins presented in the blood along with vaccines, which are designed to provoke an immune response. Influenza vaccines and the Yellow Fever vaccine are still egg-based, but the Measles-Mumps-Rubella vaccine stopped using eggs in 1994. However large scientific studies do not support this theory, especially as it applies to autoimmune disease.

Another theory focuses on whether an infant's immune system is ready for complex proteins in a new food when it is first introduced.

One hypothesis at this time is the Hygiene hypothesis. While there is no proof for the hygiene hypothesis, people speculate that in modern, industrialized nations, such as the United States, food allergies are more common due to the lack of early exposure to dirt and germs, in part due to the over-use of antibiotics and antibiotic cleansers. This hypothesis is based partly on studies showing less allergy in third world countries. Some research suggests that the body, with less dirt and germs to fight off, turns on itself and attacks food proteins as if they were foreign invaders.

Antibiotics have also been implicated in Leaky Gut Syndrome which is another possible cause of food allergies.

A lower incidence of food allergies in the developing world could also be due to differences in diet from the West and less exposure to food allergens.

Others have found that food allergies are due to widespread usage of baby skin-care products that contain allergens, such as lotions based upon peanut oil. These skin-care products are cheaper to manufacture than non-allergenic ones and using them sensitizes the baby, which later develops into a food allergy. This theory has yet to come with sufficient explanation as to why the occurrence of allergies has been on a steady rise in the last two decades.

3.7 PREVENTION

According to a report issued by the American Academy of Pediatrics, "There is evidence that breastfeeding for at least 4 months, compared with feeding infants formula made with intact cow milk protein, prevents or delays the occurrence of atopic dermatitis, cow milk allergy, and wheezing in early childhood."

Student Activity

1. Discuss the causes of food allergy.

2. Outline the symptoms of food allergy.

3.8 TREATMENT

The mainstay of treatment for food allergy is avoidance of the foods that have been identified as allergens.

If the food is accidentally ingested and a systemic reaction (anaphylaxis) occurs, then epinephrine (best delivered with an autoinjector of epinephrine such as an Epipen or Twinject) should be used. It is possible that a second dose of epinephrine may be required for severe reactions. The patient should also seek medical care immediately.

At this time, there is no cure for food allergies. There are no allergy desensitization or allergy "shots" available for food allergies. Some doctors feel they do not work in food allergies because even minute amounts of the food in question or even food extracts (as in the case of allergy shots) can cause an allergic response in many sufferers.

Ronald van Ree of Amsterdam University expects that vaccines can in theory be created using genetic engineering to cure allergies. If this can be done, food allergies could be eradicated in about ten years.

3.9 FOOD ALLERGY IN CHILDREN

Milk and soy allergies in children can often go undiagnosed for many months, causing much worry for parents and health risks for infants and children. Many infants with milk and soy allergies can show signs of colic, blood in the stool, mucous in the stool, reflux, rashes and other harmful medical conditions. These conditions are often misdiagnosed as viruses or colic.

Some children who are allergic to cow's milk protein also show a cross sensitivity to soy-based products. There are infant formulas in which the milk and soy proteins are degraded so when taken by an infant, their immune system does not recognize the allergen and they can safely consume the product. Hypoallergenic infant formulas can be based on hydrolyzed proteins, which are proteins partially predigested in a less antigenic form. Other formulas, based on free amino acids, are the least antigenic and provide complete nutrition support in severe forms of milk allergy.

Seventy-five percent of children who have allergies to milk protein are able to tolerate baked-in milk products, *i.e.*, muffins, cookies, cake. About 50% of children with allergies to milk, egg, soy, and wheat will outgrow their allergy by the age of 6. Those that don't, and those that are still allergic by the age of 12 or so, have less than an 8% chance of outgrowing the allergy.

Peanut and tree nut allergies are less likely to be outgrown, although evidence now shows that about 20% of those with peanut allergies and 9% of those with

tree nut allergies will outgrow their allergies. In such a case, they need to consume nuts in some regular fashion to maintain the non-allergic status. This should be discussed with a doctor.

NOTES

Those with other food allergies may or may not outgrow their allergies.

3.10 DIETARY MANAGEMENT

Dietary management is very important to avoid food allery. To avoid allergic foods, one should learn the terms used to describe these foods on foods labels, for example:

- **Milk protein** - milk, non-fat milk solids, cheese, yoghurt, caseinates, whey, lactose.
- **Egg** - eggs, egg albumen, egg yolk, egg lecithin
- **Lactose** - milk, lactose.
- **Gluten** - wheat, barley, rye, triticale, wheat bran, malt, oats, cornflour, oatbran.
- **Soy** -soybeans, hydrolysed vegetable protein, soy protein isolate, soy lecithin.
- **Salicylates** - strawberries and tomatoes.

3.11 FOOD INTOLERANCE

Food intolerance or non-allergic food hypersensitivity is a delayed, negative reaction to a food, beverage or food additive. It can involve symptoms in one or more body organs and systems, but is not considered directly related to the immune system. Intolerance can result from the absence of specific chemicals or enzymes needed to digest a food substance, or from reactions to naturally occurring chemicals in foods. The precise distinction between food intolerance and a food allergy is often missed.

Non-allergic food hypersensitivity is the medical name for food intolerance, loosely referred to as food hypersensitivity, or previously as pseudo-allergic reactions. Non-allergic food hypersensitivity should not be confused with allergic responses to foods as occurs with a food allergy.

A food allergy is an immunological hypersensitivity which occurs most commonly in response to food proteins such as those found in egg, milk, seafood, shellfish, tree nuts, soya, wheat and peanuts. True allergies are associated with a fast-acting immunoglobulin E (IgE) antibody response.

A non-allergic food hypersensitivity is an abnormal physiological response to food that does not involve an actual allergy. It can be difficult to determine the

offending food causing an intolerance reaction because if the immune system is involved, the response is likely to be IgG-mediated and takes place slowly. Thus the causative agent and the response are separated in time, and may not be obviously related. Food intolerance reactions can include pharmacologic, metabolic, and toxic responses to foods or food components. Food intolerance does not include psychological responses.

- Metabolic food reactions are due to inborn or acquired errors of metabolism of nutrients, such as in diabetes melitus, lactase deficiency, phenylketonuria and favism. Toxic food reactions are caused by the direct action of a food or additive without immune involvement.
- Pharmacological reactions are generally due to low-molecular-weight chemicals which occur either as natural compounds, such as salicylates and amines, or to artificially added substances, such as preservatives, colouring, emulsifiers and taste enhancers (including glutamate [MSG]). These chemicals are capable of causing drug-like (biochemical) side effects in susceptible individuals.
- Toxins may either be present naturally in food, be released by bacteria, or be due to contamination of food products.
- Psychological reactions involve manifestation of clinical symptoms caused not by food but by emotions associated with food. These symptoms do not occur when the food is given in an unrecognisable form.

Elimination diets are useful to assist in the diagnosis of food allergies and pharmacological food intolerance. Metabolic, toxic and psychological reactions can be diagnosed by other means.

Signs and Symptoms

Non-IgE-mediated food hypersensitivity (food intolerance) is more chronic, less acute, less obvious in its presentation, and often more difficult to diagnose than a food allergy. Symptoms of food intolerance vary greatly, and can be mistaken for the symptoms of a food allergy. While true allergies are associated with fast-acting immunoglobulin IgE responses, it can be difficult to determine the offending food causing a food intolerance because the response generally takes place over a prolonged period of time. Thus the causative agent and the response are separated in time, and may not be obviously related. Food intolerance symptoms usually begin about half an hour after eating or drinking the food in question, but sometimes symptoms may delayed up to 48 h.

Food intolerance can present with symptoms affecting the skin, respiratory tract, gastrointestinal tract (GIT) either individually or in combination. On the

skin may include skin rashes, urticaria (hives), angioedema, dermatitis, eczema. Respiratory tract symptoms can include nasal congestion, sinusitis, pharyngeal irritations, asthma and an unproductive cough. GIT symptoms include mouth ulcers, abdominal cramp, nausea, gas, intermittent diarrhoea, constipation, and irritable bowel syndrome., and may include anaphylaxis.

Food intolerance has been found associated with; irritable bowel syndrome and inflammatory bowel disease, chronic constipation, chronic hepatitis C infection, eczema, NSAID intolerance and respiratory complaints including asthma., rhinitis and headache, functional dyspepsia, eosinophilic esophagitis and ENT illnesses.

Causes

Reactions to chemical components of the diet are more common than true food allergies. They are caused by various organic chemicals occurring naturally in a wide variety of foods, both of animal and vegetable origin more often than to food additives, preservatives, colourings and flavourings, such as sulfites or dyes. Both natural and artificial ingredients may cause adverse reactions in sensitive people if consumed in sufficient amount, the degree of sensitivity varying between individuals.

Chemical intolerance can occur in individuals from both allergic and non-allergic family backgrounds. Symptoms may begin at any age, and may develop quickly or slowly. Triggers may range from a viral infection or illness to environmental chemical exposure. It occurs more commonly in women and may be because of hormone differences, as many food chemicals mimic hormones.

A deficiency in digestive enzymes can also cause some types of food intolerances. Dietary carbohydrate intolerances include, Lactose intolerance is a result of the body not producing sufficient lactase to digest the lactose in milk; dairy foods which are lower in lactose, such as cheese, are less likely to trigger a reaction in this case. Coeliac disease (gluten intolerance) results in damage to villi in the small intestine, which makes it difficult for the body to absorb water and nutrients from foods,and fructose intolerance.

The most widely distributed naturally occurring food chemical capable of provoking reactions is salicylate, although tartrazine and benzoic acid are well recognised in susceptable individuals. Benzoates and salicylates occur naturally in many different foods, including fruits, juices, vegetables, spices, herbs, nuts, tea, wines, and coffee. Salicylate sensitivity causes reactions to not only aspirin and NSAID's but also foods in which salicylates naturally occur, such as cherries.

Other natural chemicals which commonly cause reactions and cross reactivity include amines, nitrates, sulphites and some anti-oxidants. Chemicals involved in aroma and flavour are often suspect.

NOTES

The classification or avoidance of foods based on botanical families bears no relationship to their chemical content and is not relevant in the management of food intolerance.

Salicylate-containing foods include apples, citrus fruits, strawberries, tomatoes, and wine, while reactions to chocolate, cheese, bananas, avocado, tomato or wine point to amines as the likely food chemical. Thus exclusion of single foods does not necessarily identify the chemical responsible as several chemicals can be present in a food, the patient may be sensitive to multiple food chemicals and reaction more likely to occur when foods containing the triggering substance are eaten in a combined quantity that exceeds the patient's sensitivity thresholds. People with food sensitivities have different sensitivity thresholds, and so more sensitive people will react to much smaller amounts of the substance.

Pathogenesis

The term food allergy is widely misused for all sorts of symptoms and diseases caused by food. Food allergy (FA) is an adverse reaction to food (food hypersensitivity) occurring in susceptible individuals, which is mediated by a classical immune mechanism specific for the food itself. The best established mechanism in FA is due to the presence of IgE antibodies against the offending food. Food intolerance (FI) are all non-immune-mediated adverse reactions to food. The subgroups of FI are enzymatic (*e.g.*, lactose intolerance due to lactase deficiency), pharmacological (*e.g.*, reactions against biogenic amines, histamine intolerance), and undefined food intolerance (*e.g.*, against some food additives).

Food intolerances are mainly caused by enzymatic defects in the digestive system, (*e.g.*, lactose (milk sugar)intolerance), but may also result from pharmacological effects of vasoactive amines present in foods (*e.g.*, Histamine).

A frequent misconception among the general public is confusion between cow's milk allergy (CMA) and cow's milk intolerance, which is mainly intolerance to lactose. There are at least two, and possibly more, distinct pathologies. Hypersensitivity to milk is often broadly classified into immunoglobulin E (IgE)-mediated allergy and non-IgE-mediated allergy/intolerance. The immunopathological mechanisms of non-IgE-mediated allergy/intolerance in particular remain poorly understood, and this has hindered the development of simple and reliable diagnostics. Adults with non-IgE-mediated allergy/intolerance to milk tend to suffer ongoing allergy without the development of milk tolerance. The precise immunopathological mechanisms of non-IgE-mediated intolerance remain unclear. A number of mechanisms have been implicated, including type-1 T helper cell (Th1) mediated reactions, the formation of immune complexes leading to the activation of Complement, or T-cell/mast cell/neuron interactions

inducing functional changes in smooth muscle action and intestinal motility. Food antigens contact the immune system throughout the intestinal tract via the gut associated lymphoid system (GALT), where interactions between antigen presenting cells and T cells direct the type of immune response mounted. Unresponsiveness of the immune system to dietary antigens is termed "oral tolerance" and is believed to involve the deletion or switching off of reactive antigen-specific T cells and the production of regulatory T cells (T reg) that quell inflammatory responses to benign antigens. In the case of IgE-mediated allergies, a deficiency in regulation and a polarisation of specific effector T cells towards type-2 T helper cells (Th2) lead to signalling of B-cells to produce milk protein-specific IgE. Where as non-IgE-mediated reactions (intolerances) may be due to Th1 mediated inflammation. Dysfunctional T reg cell activity has been identified as a factor in both allergy/intolerance mechanisms.

Diagnosis

Diagnosis is made using medical history and cutaneous and serological tests to exclude other causes, but to obtain final confirmation a Double Blind Controlled Food Challenge must be performed. It is important to be able to distinguish between food allergy, food intolerance, and autoimmune disease in the management of these disorders. Non-IgE-mediated food hypersensitivity (food intolerance) is more chronic, less acute, less obvious in its clinical presentation, and often more difficult to diagnose than allergy, skin tests and immunological studies are not helpful.

Diagnosis can include an elimination diet and challenge testing. The antigen leukocyte cellular antibody test (ALCAT) has been commercially promoted as an alternative, but has not been reliably shown to be of clinical value. Clinical investigation is generally undertaken only for more serious cases, as for minor complaints which do not significantly limit the person's lifestyle the cure may be more inconvenient than the problem. Treatment can involve avoidance, and re-establishing a level of tolerance.

Testing of IgG4 to foods is considered irrelevant in the laboratory work-up of food intolerance and should not be performed in case of food-related complaints. In contrast to the disputed beliefs, IgG4 against foods indicates that the person has been repeatedly exposed to food components, recognized as foreign proteins by the immune system. Its presence should not be considered as a factor which induces hypersensitivity, but rather as an indicator for immunological tolerance, linked to the activity of T cells. In conclusion, food-specific IgG4 does not indicate (imminent) food allergy or intolerance, but rather a physiological response of the immune system after exposure to food components.

NOTES

PREVENTION

There is emerging evidence from studies of cord bloods that both sensitization and the acquisition of tolerance can begin in pregnancy, however the window of main danger for sensitization to foods extends prenatally, remaining most critical during early infancy when the immune system and intestinal tract are still maturing. There is no conclusive evidence to support the restriction of dairy intake in the maternal diet during pregnancy in order to prevent. This is generally not recommended since the drawbacks in terms of loss of nutrition can out-weigh the benefits. However, further randomised, controlled trials are required to examine if dietary exclusion by lactating mothers can truly minimize risk to a significant degree and if any reduction in risk is out-weighed by deleterious impacts on maternal nutrition.

A Cochrane review has concluded feeding with a soy formula cannot be recommended for prevention of allergy or food intolerance in infants. Further research may be warranted to determine the role of soy formulas for prevention of allergy or food intolerance in infants unable to be breast fed with a strong family history of allergy or cow's milk protein intolerance. In the case of allergy and celiac disease others recommend a dietary regimen is effective in the prevention of allergic diseases in high-risk infants, particularly in early infancy regarding food allergy and eczema. The most effective dietary regimen is exclusively breastfeeding for at least 4–6 months or, in absence of breast milk, formulas with documented reduced allergenicity for at least the first 4 months, combined with avoidance of solid food and cow's milk for the first 4 months

STUDENT ACTIVITY

1. Discuss the food allergy in children.

2. Write a short note on food intolerance.

NOTES

TREATMENT OR MANAGEMENT

Individuals can try minor changes of diet to exclude foods causing obvious reactions, and for many this may be adequate without the need for professional assistance. For reasons mentioned above foods causing problems may not be so obvious since food sensitivities may not be noticed for hours or even days after one has digested food. Persons unable to isolate foods and those more sensitive or with disabling symptoms should seek expert medical and dietitian help. The dietetic department of a teaching hospital is a good start. (see links below)

Guidance can also be given to your general practitioner to assist in diagnosis and management. Food elimination diets have been designed to exclude food chemicals likely to cause reactions and foods commonly causing true allergies and those foods where enzyme deficiency cause symptoms. These elimination diets are not every-day diets but intended to isolate problem foods and chemicals. Avoidance of foods with additives is also essential in this process.

Individuals and practitioners need to be aware that during the elimination process patients can display aspects of food addiction, masking, withdrawals, and further sensitization and intolerance. Those foods that an individual considers as 'must have every day' are suspect addictions, this includes tea, coffee, chocolate and health foods and drinks, as they all contain food chemicals. Individuals are also unlikely to associate foods causing problems because of masking or where separation of time between eating and symptoms occur. The elimination process can overcome addiction and unmask problem foods so that the patients can associate cause and effect.

It takes around five days of total abstinence to unmask a food or chemical, during the first week on an elimination diet withdrawal symptoms can occur but it takes at least two weeks to remove residual traces. If symptoms have not subsided after six weeks, food intolerance is unlikely involved and a normal diet should be restarted. Withdrawals are often associated with a lowering of the threshold for sensitivity which assists in challenge testing, but in this period individuals can be ultra-sensitive even to food smells so care must be taken to avoid all exposures.

After two or more weeks if the symptoms have reduced considerably or gone for at least five days then challenge testing can begin. This can be carried out with selected foods containing only one food chemical, so as to isolate it if reactions occur. In Australia, purified food chemicals in capsule form are available to doctors for patient testing. These are often combined with placebo capsules for control purposes. This type of challenge is more definitive. New challenges should only be given after 48 hours if no reactions occur or after five days of no symptoms if reactions occur.

Once all food chemical sensitivities are identified a dietitian can prescribe an appropriate diet for the individual to avoid foods with those chemicals. Lists of suitable foods are available from various hospitals and patient support groups can give local food brand advice. A dietitian will ensure adequate nutrition is achieved with safe foods and supplements if need be.

Over a period of time it is possible for individuals avoiding food chemicals to build up a level of resistance by regular exposure to small amounts in a controlled way, but care must be taken, the aim being to build up a varied diet with adequate composition.

3.12 SUMMARY

- A food allergy is an adverse immune response to a food protein. Food allergy is distinct from other adverse responses to food, such as food intolerance, pharmacologic reactions, and toxin-mediated reactions.
- Classic immunoglobulin-E (IgE)-mediated food allergies are classified as type-I immediate hypersensitivity reactions.
- Upon ingesting an allergen, the IgE reacts with its protein epitopes and release (degranulate) a number of chemicals (including histamine), which lead to oedema of the intestinal wall, loss of fluid and altered motility. The product is diarrhoea.
- The mainstay of treatment for food allergy is avoidance of the foods that have been identified as allergens.
- Food intolerance or non-allergic food hypersensitivity is a delayed, negative reaction to a food, beverage or food additive.
- Food intolerance (FI) are all non-immune-mediated adverse reactions to food.

3.13 GLOSSARY

- **Food allergy:** an adverse immune response to a food protein.
- **Angioedema:** soft tissue swelling, usually involving the eyelids, face, lips, and tongue.
- **Anaphylaxis:** a severe, whole-body allergic reaction.
- **Food intolerance:** it is a delayed, negative reaction to a food, beverage or food additive.

3.14 REVIEW QUESTIONS

1. What is food allergy?
2. What are the principal signs and symptoms of food allergy?
3. How is food intolerance different from food allergy?

4. Discuss the dietary management for food allergy.
5. How is food allergy treated?
6. How to prevent food intolerance?

3.15 FURTHER READINGS

NOTES

- *Dietetics* By B Srilakshmi, Published at New Age International.
- *Food Allergies and Food Intolerance*, By Jonathan Brostoff, M.D. , Linda Gamlin, Published at Healing Arts Press.
- *Food Allergy Field Guide* by Theresa Willingham.
- Rose and Wilson, *Anatomy and Physiology in health and illness*, ELBS in print, 7th edition, 1990.

UNIT— IV

GASTRO INTESTINAL DISORDERS

OBJECTIVES

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

- state the fundamental concepts of Dyspepsia;
- discuss the causes, symptoms and treatment of Gastritis;
- understand the signs and syptoms, complications etc. of Ulcer;
- eaplain the causes and clinical features of Malabsorbption syndrome;
- state the diagnosis, treatment and prevention of Sprue and Diverticular disorders.

STRUCTURE

4.1 INTRODUCTION

All diseases that pertain to the gastrointestinal tract are labelled as digestive diseases. This includes diseases of the esophagus, stomach, first, second and third part of the duodenum, jejunum, ileum, the ileo-cecal complex, large intestine (ascending, transverse and descending colon) sigmoid colon and rectum.

4.2 DYSPEPSIA

Dyspepsia popularly known as indigestion, meaning hard or difficult digestion, is a medical condition characterized by chronic or recurrent pain in the upper abdomen, upper abdominal fullness and feeling full earlier than expected when eating. It can be accompanied by bloating, belching, nausea or heartburn. Dyspepsia is a common problem, and is frequently due to gastroesophageal reflux disease (GERD) or gastritis, but in a small minority may be the first symptom of peptic ulcer disease (an ulcer of the stomach or duodenum) and occasionally cancer. Hence, unexplained newly-onset dyspepsia in people over 55 or the presence of other alarm symptoms may require further investigations.

Signs and Symptoms

The characteristic symptoms of dyspepsia are upper abdominal pain, bloating, fullness and tenderness on palpation. Pain worsened by exertion and associated with nausea and perspiration may also indicate angina.

Occasionally dyspeptic symptoms are caused by medication, such as calcium antagonists (used for angina or high blood pressure), nitrates (used for angina), theophylline (used for chronic lung disease), bisphosphonates, corticosteroids and non-steroidal anti-inflammatory drugs (NSAIDs, used as painkillers).

The presence of gastrointestinal bleeding (vomit containing blood), difficulty swallowing, anorexia (loss of appetite), unintentional weight loss, abdominal swelling and persistent vomiting are suggestive of peptic ulcer disease or malignancy, and would necessitate urgent investigations.

Diagnosis

People under 55 years, without alarm symptoms, can be treated without investigation. People over 55 years with recent onset dyspepsia or those with alarm symptoms should be urgently investigated by upper gastrointestinal endoscopy. This will rule out peptic ulcer disease, medication-related ulceration, malignancy and other rarer causes.

People under the age of 55 years with no alarm features do not need endoscopy but are considered for investigation for peptic ulcer disease caused by

Helicobacter pylori infection. Investigation for H.pylori infection is usually performed when there is a moderate to high prevalence of this infection in the local community or the person with dyspepsia has other risk factors for H. pylori infection, related for example to ethnicity or immigration from a high-prevalence area. If infection is confirmed it can usually be eradicated by medication.

Medication-related dyspepsia is usually related to non-steroidal anti-inflammatory drugs (NSAIDs) and can be complicated by bleeding or ulceration with perforation of stomach wall.

Treatment

Functional and undifferentiated dyspepsia have similar treatments. Decisions around the use of drug therapy are difficult because trials included heartburn in the definition of dyspepsia. This led to the results favoring proton pump inhibitors (PPIs), which are questionably effective for the treatment of heartburn.

Traditional therapies used for this diagnosis include lifestyle modification, antacids, H2-receptor antagonists (H2-RAs), prokinetic agents, and antiflatulents. It has been noted that one of the most frustrating aspects of treating functional dyspepsia is that these traditional agents have been shown to have little or no efficacy.

Antacids and sucralfate were found to be no better than placebo in a literature review. H2-RAs have been shown to have marked benefit in poor quality trials (30% relative risk reduction), but only a marginal benefit in good quality trials. Prokinetic agents would empirically seem to work well since delayed gastric emptying is considered a major pathophysiological mechanism in functional dyspepsia. They have been shown in a meta-analysis to produce a relative risk reduction of up to 50%, but the studies evaluated to come to this conclusion used the drug cisapride which has since been removed from the market (now only available as an investigational agent due to serious adverse events such as torsades, and publication bias has been cited as a potential partial explanation for such a high benefit. Modern prokinetic agents such as metoclopramide, erythromycin and tegaserod have little or no established efficacy and often result in substantial side effects. Simethicone has been found to be of some value, as one trial suggests potential benefit over placebo and another shows equivalence with cisapride. So, with the somewhat recent advent of the proton pump inhibitor (PPI) class of medications, the question of whether these new agents are superior to traditional therapy has arisen.

A 2004 meta-analysis, pooling data from three double-blind placebo-controlled studies, found the multiple herbal extract Iberogast to be significantly

more effective than placebo (p value = .001) at treating patients with functional dyspepsia through the targeting of multiple dyspeptic pathologies. This German-made phytopharmaceutical was found to be equivalent to cisapride and significantly superior to metoclopramide at reducing the symptoms of functional dyspepsia over a four week period. Retrospective surveillance of 40,961 children (12 years and under) found no serious side-effects.

Currently, PPIs are, depending on the specific drug, FDA indicated for erosive esophagitis, gastroesophageal reflux disease (GERD), Zollinger-Ellison syndrome, eradication of H. pylori, duodenal and gastric ulcers, and NSAID-induced ulcer healing and prevention, but not functional dyspepsia. There are, however, evidence-based guidelines and literature that evaluate the use of PPIs for this indication. A helpful chart summarizing the major trials is available from the functional dyspepsia guidelines published in the World Journal of Gastroenterology in 2006.

The CADET study was the first to compare a PPI (omeprazole 20mg daily) to both an H2-RA (ranitidine 150mg BID) as well as a prokinetic agent (cisapride 20mg BID) alongside placebo. The study evaluated these agents in patients at 4 weeks and 6 months and noted that omeprazole had a significantly better response at 6 months (31%) than cisapride (13%) or placebo (14%) (p = .001) while it was just above the cutoff for being statistically significantly better than ranitidine (21%) (p = .053). Omeprazole also showed a significant increase in quality of life scores over the other agents and placebo in all but one category measured (p = .01 to .05).

The ENCORE study, which was a follow-up of patients from the OPERA study, showed responders to omeprazole therapy had fewer clinic visits than non-responders (1.5 vs 2.0) over a three month period (p < .001).

4.3 GASTRITIS

Gastritis is not a single disease, but several different conditions that all have inflammation of the stomach lining. Gastritis can be caused by drinking too much alcohol, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin or ibuprofen, or infection with bacteria such as Helicobacter pylori (H. pylori). Sometimes gastritis develops after major surgery, traumatic injury, burns, or severe infections. Certain diseases, such as pernicious anemia, autoimmune disorders, and chronic bile reflux, can cause gastritis as well.

The most common symptoms are abdominal upset or pain. Other symptoms are belching, abdominal bloating, nausea, and vomiting or a feeling of fullness or of burning in the upper abdomen. Blood in your vomit or black stools may be a sign of bleeding in the stomach, which may indicate a serious problem requiring immediate medical attention.

NOTES

CAUSES AND TREATMENT

Acute

Erosive gastritis is gastric mucosal erosion caused by damage to mucosal defenses. Alcohol consumption does not cause chronic gastritis. It does, however, erode the mucosal lining of the stomach; low doses of alcohol stimulate hydrochloric acid secretion. High doses of alcohol do not stimulate secretion of acid. NSAIDs inhibit cyclooxygenase-1, or COX-1, an enzyme responsible for the biosynthesis of eicosanoids in the stomach, which increases the possibility of peptic ulcers to form. Also, NSAIDs, such as aspirin, reduce a substance that protects the stomach called prostaglandin. These drugs used in a short period of time are not typically dangerous. However, regular use can lead to gastritis.

Regardless of common misconceptions, "Gastritis" is not related to the skin and nervous condition "Gastroitus" which can affect the spinal cord as well as nerve endings in the lower half of the cerebral cortex. Gastroitus can be identified as a series of inflamed marks on the hand, and is most certainly, not in any way related.

Chronic

If the cardiac sphincter fails to do its job properly, some stomach acid can escape up the esophagus. This causes very painful "heartburn" or "gastritis" in the chest as the esophageal walls are eroded by the hydrochloric acid. Chronic gastritis refers to a wide range of problems of the gastric tissues that are mainly the result of H. pylori infection. The immune system makes proteins and antibodies that fight infections in the body to maintain a homeostatic condition. In some disorders, the body accidentally targets the stomach, believing it is a foreign protein or pathogen. It makes antibodies against, severely damages, and may even destroy the stomach and/or its lining. In some cases, bile, normally used to aid digestion in the small intestine, will enter through the pyloric valve of the stomach, because it had been removed during surgery or may not work properly. This also leads to gastritis. Gastritis may also be caused by other medical conditions, including HIV/AIDS, Crohn's disease, certain connective tissue disorders, or liver/kidney failure.

Metaplasia

Mucous gland metaplasia, the reversible replacement of differentiated cells, occurs in the setting of severe damage of the gastric glands, which then waste away (atrophic gastritis), which are progressively replaced by mucous glands. Gastric ulcers may develop; it is unclear if they are the causes or the consequences. Intestinal metaplasia typically begins in response to chronic mucosal injury in the antrum, and may extend to the body. Gastric mucosa cells change to resemble intestinal mucosa and may even assume absorptive characteristics. Intestinal

metaplasia is classified histologically as complete or incomplete. With complete metaplasia, gastric mucosa is completely transformed into small-bowel mucosa, both histologically and functionally, with the ability to absorb nutrients and secrete peptides. In incomplete metaplasia, the epithelium assumes a histologic appearance closer to that of the large intestine and frequently exhibits dysplasia.

Helicobacter pylori

Helicobacter pylori colonizes the stomach of more than half of the world's population, and the infection continues to play a key role in the pathogenesis of a number of gastroduodenal diseases. Colonization of the gastric mucosa with Helicobacter pylori results in the development of chronic gastritis in all infected individuals and in a subset of patients chronic gastritis progresses to complications (*i.e.*, ulcer disease, gastric neoplasias, some distinct extra gastric disorders). However, gastritis has no adverse consequences for most hosts and emerging evidence suggests that *Helicobacter pylori* prevalence is inversely related to gastroesophageal reflux disease and allergic disorders. These observations indicate that eradication may not be appropriate for certain populations due to the potentially beneficial effects conferred by persistent gastric inflammation.

TREATMENT

Over-the-counter antacids in liquid or tablet form are a common treatment for mild gastritis. Antacids neutralize stomach acid and can provide fast pain relief. When antacids don't provide enough relief, medications such as cimetidine, ranitidine, nizatidine or famotidine that helps reduce the amount of acid the stomach produces are often prescribed. An even more effective way to limit stomach acid production is to shutdown the acid "pumps" within acid-secreting stomach cells. Proton pump inhibitors reduce acid by blocking the action of these small pumps. This class of medications includes omeprazole, lansoprazole, rabeprazole, and esomeprazole. Proton pump inhibitors also appear to inhibit *Helicobacter pylori* activity. Cytoprotective agents are designed to help protect the tissues that line your stomach and small intestine. They include the medications sucralfate and misoprostol. If NSAIDs are being taken regularly, one of these medications to protect the stomach may also be taken. Another cytoprotective agent is bismuth subsalicylate. In addition to protecting the lining of stomach and intestines, bismuth preparations appear to inhibit *Helicobacter pylori* activity as well. Several regimens are used to treat *Helicobacter pylori* infection. Most use a combination of two antibiotics and a proton pump inhibitor. Sometimes bismuth is also added to the regimen. The antibiotic aids in destroying the bacteria, and the acid blocker or proton pump inhibitor relieves pain and nausea, heals inflammation, and may increase the antibiotic's effectiveness.

SYMPTOMS

Severe gastritis is possible when the stomach is viewed without symptoms being present and may be present despite only minor changes in the stomach lining. Seniors have a higher likelihood of developing painless stomach damage. They may have no symptoms at all, such as an absence of vomiting or pain, until they are suddenly taken ill with internal bleeding. Pain in the upper abdomen is the most common symptom. The pain is usually in the upper central portion of the abdomen, the "pit" of the stomach. Gastritis pain can occur in the left upper portion of the abdomen and in the back. The pain seems to travel from the belly to the back. The pain is typically vague, but can be a sharp pain. Belching either doesn't relieve pain or only relieves it for a moment. The vomit is either clear, green or yellow, has a bloody streak in it, or is completely bloody, depending on the severity of inflammation. Bloating and a feeling of fullness or burning in the upper abdomen are also signs of moderate gastritis. Severe gastritis presents pallor, sweating, rapid heart beat, feeling faint or short of breath, severe chest or stomach pain, vomiting large amounts of blood, or bloody or dark, sticky, foul-smelling bowel movements. Masking the pain can be achieved by sitting in a hot shower.

DIAGNOSIS

Typically, a diagnosis is made based on the patients description of his or her symptoms. If a diagnosis is not possible based on these symptoms, however, other methods are used. Tests for blood cell count, H. pylori, and pregnancy; and liver, kidney, gallbladder, and pancreas functions, may be ordered. Urinalysis may be used, or a stool sample taken, to look for blood in the stool. X-rays may be ordered, as well as ECGs. If none of these tests are able to be used for diagnosis, the patient may be recommended to a gastroenterologist. An endoscopy may be performed, where a flexible probe with a camera on the end is sent into the stomach to check for stomach lining inflammation and mucous erosion. At the same time, a stomach biopsy may be taken to test for gastritis and a variety of other conditions.

4.4 ULCER

A stomach ulcer (also called a peptic ulcer) is a small erosion (hole) in the gastrointestinal tract. The most common type, duodenal, occurs in the first 12 inches of small intestine beyond the stomach. Ulcers that form in the stomach are called gastric ulcers. An ulcer is not contagious or cancerous. Duodenal ulcers are almost always benign, while stomach ulcers may become malignant.

A peptic ulcer, also known as ulcus pepticum, PUD or peptic ulcer disease, is an ulcer of an area of the gastrointestinal tract that is usually acidic and thus

extremely painful. As many as 80% of ulcers are associated with Helicobacter pylori, a spiral-shaped bacterium that lives in the acidic environment of the stomach, however only 20% of those cases go to a doctor. Ulcers can also be caused or worsened by drugs such as aspirin and other NSAIDs.

Contrary to general belief, more peptic ulcers arise in the duodenum (first part of the small intestine, just after the stomach) than in the stomach. About 4% of stomach ulcers are caused by a malignant tumor, so multiple biopsies are needed to exclude cancer. Duodenal ulcers are generally benign.

CLASSIFICATION

- Stomach (called gastric ulcer)
- Duodenum (called duodenal ulcer)
- Oesophagus (called Oesophageal ulcer)
- Meckel's Diverticulum (called Meckel's Diverticulum ulcer)

Types of peptic ulcers:

- Type I: Ulcer along the lesser curve of stomach
- Type II: Two ulcers present — one gastric, one duodenal
- Type III: Prepyloric ulcer
- Type IV: Proximal gastroesophageal ulcer
- Type V: Anywhere along gastric body, NSAID induced

SIGNS AND SYMPTOMS

Symptoms of a peptic ulcer can be

- abdominal pain, classically epigastric with severity relating to mealtimes, after around 3 hours of taking a meal (duodenal ulcers are classically relieved by food, while gastric ulcers are exacerbated by it);
- bloating and abdominal fullness;
- waterbrash (rush of saliva after an episode of regurgitation to dilute the acid in esophagus);
- nausea, and copious vomiting;
- loss of appetite and weight loss;
- hematemesis (vomiting of blood); this can occur due to bleeding directly from a gastric ulcer, or from damage to the esophagus from severe/ continuing vomiting.
- melena (tarry, foul-smelling feces due to oxidized iron from hemoglobin);

- rarely, an ulcer can lead to a gastric or duodenal perforation. This is extremely painful and requires immediate surgery.

A history of heartburn, gastroesophageal reflux disease (GERD) and use of certain forms of medication can raise the suspicion for peptic ulcer. Medicines associated with peptic ulcer include NSAID (non-steroid anti-inflammatory drugs) that inhibit cyclooxygenase, and most glucocorticoids (*e.g.*, dexamethasone and prednisolone).

In patients over 45 with more than two weeks of the above symptoms, the odds for peptic ulceration are high enough to warrant rapid investigation by EGD.

The timing of the symptoms in relation to the meal may differentiate between gastric and duodenal ulcers: A gastric ulcer would give epigastric pain during the meal, as gastric acid is secreted, or after the meal, as the alkaline duodenal contents reflux into the stomach. Symptoms of duodenal ulcers would manifest mostly before the meal—when acid (production stimulated by hunger) is passed into the duodenum. However, this is not a reliable sign in clinical practice.

Complications

- Gastrointestinal bleeding is the most common complication. Sudden large bleeding can be life-threatening. It occurs when the ulcer erodes one of the blood vessels.
- Perforation (a hole in the wall) often leads to catastrophic consequences. Erosion of the gastro-intestinal wall by the ulcer leads to spillage of stomach or intestinal content into the abdominal cavity. Perforation at the anterior surface of the stomach leads to acute peritonitis, initially chemical and later bacterial peritonitis. The first sign is often sudden intense abdominal pain. Posterior wall perforation leads to pancreatitis; pain in this situation often radiates to the back.
- Penetration is when the ulcer continues into adjacent organs such as the liver and pancreas.
- Scarring and swelling due to ulcers causes narrowing in the duodenum and gastric outlet obstruction. Patient often presents with severe vomiting.
- Pyloric stenosis

Pathophysiology

Tobacco smoking, not eating properly, blood group, spices and other factors that were suspected to cause ulcers until late in the 20th century, are actually of relatively minor importance in the development of peptic ulcers.

A major causative factor (60% of gastric and up to 90% of duodenal ulcers) is chronic inflammation due to Helicobacter pylori that colonizes the antral mucosa. The immune system is unable to clear the infection, despite the appearance of antibodies. Thus, the bacterium can cause a chronic active gastritis (type B gastritis), resulting in a defect in the regulation of gastrin production by that part of the stomach, and gastrin secretion can either be decreased (most cases) resulting in hypo- or achlorhydria or increased. Gastrin stimulates the production of gastric acid by parietal cells and, in H. pylori colonization responses that increase gastrin, the increase in acid can contribute to the erosion of the mucosa and therefore ulcer formation. Studies have shown eating cabbage or cabbage juice can increase the mucosa lining in the stomach.

Another major cause is the use of NSAIDs (see above). The gastric mucosa protects itself from gastric acid with a layer of mucus, the secretion of which is stimulated by certain prostaglandins. NSAIDs block the function of cyclooxygenase 1 (cox-1), which is essential for the production of these prostaglandins. Newer NSAIDs (celecoxib, rofecoxib) only inhibit cox-2, which is less essential in the gastric mucosa, and roughly halve the risk of NSAID-related gastric ulceration. As the prevalence of H. pylori-caused ulceration declines in the Western world due to increased medical treatment, a greater proportion of ulcers will be due to increasing NSAID use among individuals with pain syndromes as well as the growth of aging populations that develop arthritis.

The incidence of duodenal ulcers has dropped significantly during the last 30 years, while the incidence of gastric ulcers has shown a small increase, mainly caused by the widespread use of NSAIDs. The drop in incidence is considered to be a cohort-phenomena independent of the progress in treatment of the disease. The cohort-phenomena is probably explained by improved standards of living which has lowered the incidence of H. pylori infections.

Glucocorticoids lead to atrophy of all epithelial tissues. Their role in ulcerogenesis is relatively small.

Smoking leads to atherosclerosis and vascular spasms, causing vascular insufficiency and promoting the development of ulcers through ischemia. Nicotine contained in cigarettes can increase parasympathetic nerve activity to the gastrointestinal tract by acting on the nicotinic receptors at synapses — increased stimulation to the enterochromaffin-like cells and G cells increases the amount of histamine and gastrin secreted and therefore increases the acidity of the gastric juice.

A family history is often present in duodenal ulcers, especially when blood group O is also present. Inheritance appears to be unimportant in gastric ulcers.

Gastrinomas (Zollinger Ellison syndrome), rare gastrin-secreting tumors, cause multiple and difficult to heal ulcers.

Stress

Despite the finding that a bacterial infection is the cause of ulcers in 80% of cases, bacterial infection does not appear to explain all ulcers and researchers continue to look at stress as a possible cause, or at least a complication in the development of ulcers.

There is debate as to whether psychological stress can influence the development of peptic ulcers. Burns and head trauma, however, can lead to physiologic stress ulcers, which are reported in many patients who are on mechanical ventilation.

An expert panel convened by the Academy of Behavioural Medicine Research concluded that ulcers are not purely an infectious disease and that psychological factors do play a significant role. Researchers are examining how stress might promote H. pylori infection. For example, Helicobacter pylori thrives in an acidic environment, and stress has been demonstrated to cause the production of excess stomach acid.

A study of peptic ulcer patients in a Thai hospital showed that chronic stress was strongly associated with an increased risk of peptic ulcer, and a combination of chronic stress and irregular mealtimes was a significant risk factor.

A study on mice showed that both long-term water-immersion-restraint stress and H. pylori infection were independently associated with the development of peptic ulcers.

Differential Diagnosis of Epigastric Pain

- Peptic ulcer
- Gastritis
- Stomach cancer
- Gastroesophageal reflux disease
- Pancreatitis
- Hepatic congestion
- Cholecystitis
- Biliary colic
- Inferior myocardial infarction
- Referred pain (pleurisy, pericarditis)
- Superior mesenteric artery syndrome

Diagnosis

An esophagogastroduodenoscopy (EGD), a form of endoscopy, also known as a gastroscopy, is carried out on patients in whom a peptic ulcer is suspected. By direct visual identification, the location and severity of an ulcer can be described. Moreover, if no ulcer is present, EGD can often provide an alternative diagnosis.

The diagnosis of Helicobacter pylori can be made by:

- Urea breath test (noninvasive and does not require EGD);
- Direct culture from an EGD biopsy specimen; this is difficult to do, and can be expensive. Most labs are not set up to perform H. pylori cultures;
- Direct detection of urease activity in a biopsy specimen by rapid urease test;
- Measurement of antibody levels in blood (does not require EGD). It is still somewhat controversial whether a positive antibody without EGD is enough to warrant eradication therapy;
- Stool antigen test;
- Histological examination and staining of an EGD biopsy.

The possibility of other causes of ulcers, notably malignancy (gastric cancer) needs to be kept in mind. This is especially true in ulcers of the greater (large) curvature of the stomach; most are also a consequence of chronic H. pylori infection.

If a peptic ulcer perforates, air will leak from the inside of the gastrointestinal tract (which always contains some air) to the peritoneal cavity (which normally never contains air). This leads to "free gas" within the peritoneal cavity. If the patient stands erect, as when having a chest X-ray, the gas will float to a position underneath the diaphragm. Therefore, gas in the peritoneal cavity, shown on an erect chest X-ray or supine lateral abdominal X-ray, is an omen of perforated peptic ulcer disease.

Macroscopic Appearance

Gastric ulcers are most often localized on the lesser curvature of the stomach. The ulcer is a round to oval parietal defect ("hole"), 2 to 4 cm diameter, with a smooth base and perpendicular borders. These borders are not elevated or irregular in the acute form of peptic ulcer, regular but with elevated borders and inflammatory surrounding in the chronic form. In the ulcerative form of gastric cancer the borders are irregular. Surrounding mucosa may present radial folds, as a consequence of the parietal scarring.

Microscopic Appearance

A gastric peptic ulcer is a mucosal defect which penetrates the muscularis mucosae and muscularis propria, produced by acid-pepsin aggression. Ulcer margins are perpendicular and present chronic gastritis. During the active phase, the base of the ulcer shows 4 zones: inflammatory exudate, fibrinoid necrosis, granulation tissue and fibrous tissue. The fibrous base of the ulcer may contain vessels with thickened wall or with thrombosis.

Treatment

Younger patients with ulcer – like symptoms are often treated with antacids or H_2 antagonists before EGD is undertaken. Bismuth compounds may actually reduce or even clear organisms, though it should be noted that the warning labels of some bismuth subsalicylate products indicate that the product should not be used by someone with an ulcer.

Patients who are taking nonsteroidal anti-inflammatories (NSAIDs) may also be prescribed a prostaglandin analogue (Misoprostol) in order to help prevent peptic ulcers, which may be a side-effect of the NSAIDs.

When H. pylori infection is present, the most effective treatments are combinations of 2 antibiotics (*e.g.*, Clarithromycin, Amoxicillin, Tetracycline, Metronidazole) and 1 proton pump inhibitor (PPI), sometimes together with a bismuth compound. In complicated, treatment-resistant cases, 3 antibiotics (*e.g.*, amoxicillin + clarithromycin + metronidazole) may be used together with a PPI and sometimes with bismuth compound. An effective first-line therapy for uncomplicated cases would be Amoxicillin + Metronidazole + Pantoprazole (a PPI). In the absence of *Helicobacter pylori*, long-term higher dose PPIs are often used.

Treatment of *Helicobacter pylori* usually leads to clearing of infection, relief of symptoms and eventual healing of ulcers. Recurrence of infection can occur and retreatment may be required, if necessary with other antibiotics. Since the widespread use of PPI's in the 1990s, surgical procedures (like "highly selective vagotomy") for uncomplicated peptic ulcers became obsolete.

Perforated peptic ulcer is a surgical emergency and requires surgical repair of the perforation. Most bleeding ulcers require endoscopy urgently to stop bleeding with cautery, injection, or clipping.

Epidemiology

The lifetime risk for developing a peptic ulcer is approximately 10%.

In Western countries the prevalence of Helicobacter pylori infections roughly matches age *(i.e., 20% at age 20, 30% at age 30, 80% at age 80 etc).* Prevalence is

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higher in third world countries. Transmission is by food, contaminated groundwater, and through human saliva (such as from kissing or sharing food utensils.)

According to Mayo Clinic, however, there is evidence that the infection can be transmitted by kissing.

A minority of cases of Helicobacter infection will eventually lead to an ulcer and a larger proportion of people will get non-specific discomfort, abdominal pain or gastritis.

STUDENT ACTIVITY

1. Discuss the diagnosis of Dyspepsia.

2. Outline the causes of Gastritis.

3. Discuss the complications of Ulcer.

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4.5 MALABSORPTION

Malabsorption is a state arising from abnormality in absorption of food nutrients across the gastrointestinal (GI) tract.

Impairment can be of single or multiple nutrients depending on the abnormality. This may lead to malnutrition and variety of anaemias.

Malabsorption syndrome is an alteration in the ability of the intestine to absorb nutrients adequately into the bloodstream. Malabsorption may be due to an abnormality of the gut wall, failure to produce enzymes or bile to aid digestion, or there may be abnormalities of the flora of the gut. Malabsorption may occur for many nutrients or for specific carbohydrates, fats or micronutrients. Protein, fats, and carbohydrates normally are absorbed in the small intestine — the small bowel also absorbs about 80 percent of the eight to ten liters of fluid ingested daily.

Many different conditions affect fluid and nutrient absorption by the intestine. A fault in the digestive process may result from failure of the body to produce the enzymes needed to digest certain foods. Congenital structural defects or diseases of the pancreas, gall bladder, or liver may alter the digestive process. Inflammation, infection, injury or surgical removal of portions of the intestine may inhibit absorption abilities. Reduced length or surface area of intestine available for fluid and nutrient absorption can also result in malabsorption.

Radiation therapy may injure the mucosal lining of the intestine, resulting in diarrhoea that may not become evident until several years later. Furthermore, the use of some antibiotics can also affect the bacteria that normally live in the intestine, thus affecting intestinal function.

Common disorders that can lead to malabsorption syndrome include cystic fibrosis, chronic pancreatitis, celiac disease or gluten enteropathy, short bowel syndrome, intestinal lymphangiectasia, Whipple's disease, inflammatory bowel disease and irritable bowel syndrome.

Malabsorption causes weight loss, glossitis, carpopedal spasms, absent tendon reflexes, cutaneous bruising, flatulence and abdominal distention, bloating or discomfort resulting from increased intestinal bulk and gas production.

The most common symptoms of malabsorption include:

- anemia, with weakness and fatigue due to inadequate absorption of vitamin B-12, iron, and folic acid
- diarrhoea, steatorrhoea (excessive amount of fat in the stool), and abdominal distention with cramps, bloating, and gas due to impaired water and carbohydrate absorption and irritation from unabsorbed fatty acids.

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The individual may also report explosive diarrhoea with greasy, foul-smelling stools

- edema from decreased protein absorption
- malnutrition and weight loss due to decreased fat, carbohydrate, and protein absorption
- muscle cramping from decreased vitamin D, calcium, and potassium levels
- muscle wasting and atrophy due to decreased protein absorption and metabolism
- perianal skin burning, itching or soreness due to frequent loose stools

Irregular heart rhythms may also result from inadequate levels of potassium and other electrolytes. Blood clotting disorders may occur due to a vitamin K deficiency.

Secondary nutritional deficiencies develop in proportion to the severity of the primary disease and the area of the GI tract involved. Many patients with malabsorption are anemic, usually because of deficiencies of iron (microcytic anemia) and folic acid (megaloblastic anemia) but also from the B vitamins, calcium, vitamin D, vitamin K (mainly fat-soluble), and niacin.

Protein malabsorption may lead to hypoproteinemic edema, usually of the lower limbs, and secondary endocrine deficiencies may result from malnutrition due to the high risk of polyunsaturated fatty acid (PUFA) deficiency. Moreover, patients with malabsorption will become immunosuppressed due to the nutritional deficiencies.

Fluid and nutrient monitoring and replacement is essential for any individual with malabsorption syndrome. Hospitalization may be required to treat severe fluid and electrolyte imbalances. Consultation with a dietitian to assist with nutritional support and meal planning is helpful. If the patient is able to eat, the diet and supplements should provide bulk and be rich in carbohydrates, proteins, fats, minerals, and vitamins. It is often recommended that those suffering from malabsorption syndrome eat foods as close to already digested form as possible, and eat several small, frequent meals throughout the day, avoiding fluids and foods that promote diarrhoea. Intake and output should be monitored, along with the number, colour, and consistency of stools.

Vitamin and mineral supplements are advised. Other helpful supplements may include probiotics and prebiotics.

"There is no doubt that a healthy digestive system is critical for proper nutrient absorption," says Tim Gamble, vice president of sales and marketing for Nutraceutix, Inc. "A healthy gut to some extent means that

there is a healthy, naturally occurring population of probiotic bacteria present in the intestines. Research indicates that viable, properly delivered probiotics can play a key role in strengthening the function of the intestines from digestion of food to prohibiting pathogenic bacterial growth to immune system stimulation. If one is hoping to gain weight by manipulating or supplementing one's diet, perhaps beyond that which is considered normal or routine, a healthy gut is critical to allowing one to do so without digestive discomfort or dysfunction. Probiotic supplementation is probably a wise choice before and during a weight gain regimen."

Medical management for malabsorption syndrome is dependent upon the cause. (Treatment for tropical sprue consists of folic acid supplements and long-term antibiotics. Whipple's disease also may require long-term use of antibiotics, such as tetracycline). Management of some individuals may require injections of vitamin B-12 and oral iron supplements. The doctor may also prescribe enzymes to replace missing intestinal enzymes, or antispasmodics to reduce abdominal cramping and associated diarrhoea. People with cystic fibrosis and chronic pancreatitis require pancreatic supplements. Those with lactose intolerance or gluten enteropathy will have to modify their diets to avoid foods that they cannot properly digest.

Classification

Some prefer to classify malabsorption clinically into three basic categories:

(1) selective, as seen in lactose malabsorption;

(2) partial, as observed in a-Beta-lipoproteinemia, and

(3) total as in coeliac disease.

Pathophysiology

The main purpose of the gastrointestinal tract is to digest and absorb nutrients (fat, carbohydrate, and protein), micronutrients (vitamins and trace minerals), water, and electrolytes. Digestion involves both mechanical and enzymatic breakdown of food. Mechanical processes include chewing, gastric churning, and the to-and-fro mixing in the small intestine. Enzymatic hydrolysis is initiated by intraluminal processes requiring gastric, pancreatic, and biliary secretions. The final products of digestion are absorbed through the intestinal epithelial cells.

Malabsorption constitutes the pathological interference with the normal physiological sequence of digestion (intraluminal process), absorption (mucosal process) and transport (postmucosal events) of nutrients.

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Intestinal malabsorption can be due to:

- Mucosal damage (enteropathy)
- Congenital or acquired reduction in absorptive surface
- Defects of specific hydrolysis
- Defects of ion transport
- Pancreatic insufficiency
- Impaired enterohepatic circulation

Causes

Due to infective agents

- Whipple's disease
- Intestinal tuberculosis
- HIV related malabsorption
- Tropical sprue
- Traveller's diarrhoea
- Parasites *e.g.*, Giardia lamblia, fish tape worm (B12 malabsorption); roundworm, hookworm (Ancylostoma duodenale and Necator americanus)

Due to structural defects

- Blind loops
- Inflammatory bowel diseases commonly in Crohn's Disease
- Intestinal hurry from Post-gastrectomy; post-vagotomy, gastro-jejunostomy
- Fistulae, diverticulae and strictures,
- Infiltrative conditions such as amyloidosis, lymphoma, Eosinophilic gastroenteropathy
- Radiation enteritis
- Systemic sclerosis and collagen vascular diseases
- Short gut syndrome

Due to mucosal abnormality

- Coeliac/Celiac disease
- Cows' milk intolerance

- Soya milk intolerance
- Fructose malabsorption

Due to enzyme deficiencies

- Lactase deficiency inducing lactose intolerance (constitutional, secondary or rarely congenital)
- Sucrose intolerance
- Intestinal disaccharidase deficiency
- Intestinal enteropeptidase deficiency

Due to digestive failure

- Pancreatic insufficiencies:
  - cystic fibrosis
  - chronic pancreatitis
  - carcinoma of pancreas
  - Zollinger-Ellison syndrome
- Bile salt malabsorption
  - terminal ileal disease
  - obstructive jaundice
  - bacterial overgrowth

Due to other systemic diseases affecting GI tract

- Hypothyroidism and hyperthyroidism
- Addison's disease
- Diabetes mellitus
- Hyperparathyroidism and Hypoparathyroidism
- Carcinoid syndrome
- Malnutrition
- Abeta-lipoproteinemia

Clinical Features

It can present in variety of ways and features might give clue to underlying condition. Symptoms can be intestinal or extra-intestinal—the former predominates in severe malabsorption.

- Diarrhoea, often steatorrhoea is the most common feature. Watery, diurnal and nocturnal, bulky, frequent stools are the clinical hallmark of overt

malabsorption. It is due to impaired water, carbohydrate and electrolyte absorption or irritation from unabsorbed fatty acid. Latter also results in bloating, flatulence and abdominal discomfort. Cramping pain usually suggests obstructive intestinal segment *e.g.*, in Crohn's disease, especially if it persists after defecation.

- Weight loss can be significant despite increased oral intake of nutrients.
- Growth retardation, failure to thrive, delayed puberty in children
- Swelling or oedema from loss of protein
- Anaemias, commonly from vitamin B12, folic acid and iron deficiency presenting as fatigue and weakness.
- Muscle cramp from decreased vitamin D, calcium absorption. Also lead to osteomalacia and osteoporosis
- Bleeding tendencies from vitamin K and other coagulation factor deficiencies.
- Low serum tryptophan and clinical depression, as can happen with fructose malabsorption

Diagnosis

There is no specific test for Malabsorption. As for most medical conditions, investigation is guided by symptoms and signs. Moreover, tests for pancreatic function are complex and varies widely between centres.

Blood Tests

- Routine blood tests may reveal anaemia, high ESR or low albumin; which has high sensitivity for presence of organic disease. In this setting, microcytic anaemia usually implies iron deficiency and macrocytosis can be from impaired folic acid or B12 absorption or both. Low cholesterol or triglyceride may give clue toward fat malabsorption as low calcium and phosphate toward osteomalacia from low vitamin D.
- Specific vitamins like vitamin D or micro nutrient like zinc levels can be checked. Fat soluble vitamins (A, D, E & K) are affected in fat malabsorption. Prolonged prothrombin time can be from vitamin K deficiency.
- Serological studies

Specific tests are carried out to determine underlying cause. IgA tissue trans glutamate or IgA antiendomysium assay for gluten sensitive enteropathy.

Stool studies

- Microscopy is particularly useful in diarrhoea, may show protozoa like giardia, ova, cyst and other infective agents.

- Fecal fat study to diagnose steatorrhoea is less frequently performed nowadays.
- Low elastase is indicative of pancreatic insufficiency. Chymotrypsin and pancreolauryl can be assessed as well

Radiological studies

- Barium follow through is useful in delineating small intestinal anatomy. Barium enema may be undertaken to see colonic or ileal lesions.
- CT abdomen is useful in ruling out structural abnormality, done in pancreatic protocol when visualising pancreas.
- Magnetic resonance cholangiopancreatography (MRCP) to complement or as an alternative to ERCP

Interventional studies

Biopsy of small bowel showing coeliac disease manifested by blunting of villi, crypt hyperplasia, and lymphocyte infiltration of crypts.

- Endoscopy is frequently undertaken, but to visualise small intestine, which can be up to 7m long, is indeed a daunting task.
  - OGD to reveal duodenal lesion also for D2 biopsy (for celiac disease, tropical sprue, Whipple's disease, A-b-lipoproteinemia etc.)
  - Enteroscopy for enteropathy and jejunal aspirate and culture for bacterial overgrowth
  - Colonoscopy is helpful in colonic or ileal lesion.
- ERCP

Other investigations

- Radio isotope tests *e.g.*, 75SeHCAT, 95mTc to exclude terminal ileal disease.
- Sugar probes or sub 51Cr-EDTA to determine intestinal permeability.
- Glucose hydrogen breath test for bacterial overgrowth
- D-xylose absorption test. lower level in urine after ingestion indicates bacterial overgrowth or reduced absorptive surface. normal in pancreatic insufficiency.
- Bile salt breath test to determine bile salt malabsorption.
- Schilling test to establish cause of B12 deficiency.
- Lactose H_2 breath test for lactose intolerance.

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MANAGEMENT

Treatment is directed largely towards management of underlying cause.

- Replacement of nutrients, electrolytes and fluid may be necessary. In severe deficiency, hospital admission may be required for parenteral administration, often advice from dietitian is sought. People whose absorptive surface are severely limited from disease or surgery may need longterm total parenteral nutrition. Pancreatic enzymes are supplemented orally in insufficiencies.
- Dietary modification is important in some conditions. Life-long avoidance of particular food or food constituent may be needed in Celiac disease or lactose intolerance.
- Bacterial overgrowth usually respond well to course of antibiotic. Use of cholestyramine to bind bile acid will help reducing diarrhoea in bile acid malabsorption.

4.6 CELIAC SPRUE

Celiac sprue, also known as celiac disease, gluten-sensitive enteropathy, and gluten-induced enteropathy, is a chronic disease of the digestive tract that interferes with the digestion and absorption of nutrients from food. People with celiac sprue cannot tolerate gluten, a protein commonly found in wheat, rye, barley, and to some degree, oats. When affected individuals ingest foods containing gluten, the lining (mucosa) of the intestine becomes damaged due to the body's immune reaction. Because the lining of the intestine contains essential enzymes for digestion and absorption, its destruction leads to malabsorption, a difficulty in absorption of food and essential nutrients. As result, celiac sprue is often considered a malabsorption disorder.

Persons with celiac sprue experience improvement in the condition when on a strict, gluten-free diet and relapse when dietary gluten is reintroduced. With treatment, celiac sprue is rarely fatal. However, untreated and unrecognized celiac disease may slightly increase the risk of developing intestinal lymphoma, a form of cancer.

Celiac sprue is a genetic disease; the genes for this condition may be transmitted to some family members and not to others. Sometimes the disease is triggered, or becomes apparent for the first time, after surgery, pregnancy, childbirth, viral infection, or severe emotional stress. Celiac sprue is rare in persons with an African American, Caribbean, or Asian background. Females are slightly more affected than males. Although celiac sprue can manifest at any age, the detection of this disease usually peaks at 8-12 months and in the third to fourth decade of life.

The true prevalence of celiac sprue is not known. The increased awareness and the availability of better diagnostic tests have led to the realization that the disease is relatively common. The highest prevalence is in Western Europe and in places where Europeans emigrated, notably North America and Australia. In these regions, celiac sprue affects approximately 1 of every 250-300 individuals. In the United States, the estimated prevalence is 1 case per 3,000 in the population. However, this rate is likely an underestimation; a recent study involving 2,000 healthy blood donors showed an estimated prevalence of 1 case in 300.

CAUSES

Celiac sprue results from a combination of immunological responses to an environmental factor (gluten) and genetic factors. People need both a genetic predisposition and the exposure to gluten in order to develop celiac sprue.

Immune mechanisms

The interaction of gliadin (a specific gluten present in certain grain products) with the lining of the small intestine is critical in the development of celiac sprue. When people with celiac sprue eat foods containing gluten, gliadin is identified by the immune system as a threat. As a result, the body produces antibodies called antigliadin antibodies. Antigliadin antibodies are directed against gliadin.

Two additional antibodies have been identified in the bloodstream of people with celiac disease. In contrast to antigliadin antibodies, these antibodies target the person's own body and are referred to as autoantibodies (antibodies against our own cells and organs). The first antibody targets endomysium, a small intestinal smooth muscle component. The second antibody targets an enzyme called tissue transglutaminase. The presence of these autoantibodies suggests that autoimmunity plays a role in the disease process of celiac sprue.

Genetic factors

Genes play an important role in celiac sprue. Celiac disease occurs much more frequently in relatives of persons with celiac sprue than in the general population. Celiac disease may occur in up to 10% of close family members of persons with celiac sprue.

CELIAC SPRUE SIGNS AND SYMPTOMS

Gastrointestinal symptoms in children

Because celiac sprue affects the absorption of nutrients essential for growth, children who are affected may have impaired growth and consequently short stature. Other common signs and symptoms include the following:

- Abdominal pain

- Vomiting
- Diarrhoea
- Behavioural disturbances, including depression, irritability, and poor school performance

The onset of the symptoms is usually gradual and coincides with the introduction of cereal into the diet. The symptoms usually diminish in adolescence.

Gastrointestinal symptoms in adults

Celiac sprue usually affects adults in the third to fourth decade of life but sometimes later. The signs and symptoms of celiac sprue are variable and may include the following:

- Diarrhoea
- Abdominal discomfort
- Bloating
- Steatorrhoea, or fatty stools (caused by malabsorption of ingested fat)

Malabsorption of ingested fat results in the delivery of excessive dietary fat to the large bowel. The bacteria in the colon feast on the fats and other undigested and unabsorbed nutrients, generating intestinal gas resulting in bloating and flatulence. In addition, other substances are released, causing secretion of fluid into the intestine and hence diarrhoea. Fatigue (tiredness) and weakness can result from the loss of electrolytes, such as potassium and magnesium, due to the diarrhoea.

Nutrient and vitamin deficiencies

Iron and folic acid are essential for the production of normal red blood cells (erythrocytes). Abnormalities in the absorption of iron or folic acid may result in anemia (low red blood cell count). Vitamin B-12 deficiencies can also contribute to the anemia noticed in affected persons with a mechanism similar to that of iron and folic acid deficiencies.

Vitamin deficiencies may develop when malabsorption is present. Vitamins soluble in fat are commonly malabsorbed. These include vitamins K and D.

- Vitamin K is essential for the production of clotting proteins. As a result, vitamin K deficiency causes a bleeding tendency among persons with celiac sprue.
- Vitamin D is essential for the absorption of calcium, which is required for appropriate bone growth. As a result, vitamin D deficiency may cause low blood calcium levels (hypocalcemia). This predisposes children with

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celiac sprue to bone disorders such as rickets. Adults with celiac sprue have decreased calcium in the bones, a condition referred to as osteomalacia, and may develop fractures. Loss of protein and calcium may lead to osteoporosis.

Non-gastrointestinal (extraintestinal) features

Skin disorders can complicate the course of the celiac sprue. These conditions include dermatitis herpetiformis, an itchy skin condition characterized by a rash or blisters involving the extremities, the trunk, the buttocks, the scalp, and the neck.

Neurologic (nervous system) symptoms include weakness, problems with balance, and sensory changes (for example, sensation of touch and pain).

Hormonal disorders, such as loss of menstruation (amenorrhoea) and infertility in women, and impotence and infertility in men, are very uncommon.

When to Seek Medical Care

Celiac sprue can be a debilitating condition, especially if the diagnosis is not considered early in the course of the disease. As a result, persons with any of the symptoms mentioned above (see Signs and Symptoms) or those with a family history of the disease are encouraged to seek medical advice. Because celiac sprue is hereditary, close family members of persons with celiac sprue should be tested for the disease. About 10% of an affected person's first-degree relatives (parents, siblings, or children) will also have the disease.

Females who are pregnant and have worsening anemia should seek medical care. This diagnosis should be considered in females with significant worsening of anemia during pregnancy.

Diagnosis

There are several tests that can be used to assist in diagnosis. The level of symptoms may determine the order of the tests, but all tests lose their usefulness if the patient is already taking a gluten-free diet. Intestinal damage begins to heal within weeks of gluten being removed from the diet, and antibody levels decline over months. For those who have already started on a gluten-free diet, it may be necessary to perform a re-challenge with 10 g of gluten (four slices of bread) per day over 2–6 weeks before repeating the investigations. Those who experience severe symptoms (*e.g.*, diarrhoea) earlier can be regarded as sufficiently challenged and can be tested earlier.

Combining findings into a prediction rule to guide use of endoscopy reported a sensitivity of 100% (it would identify all the cases) and specificity of

61% (it would be incorrectly positive in 39%). The prediction rule recommends that patients with high-risk symptoms or positive serology should undergo endoscopy. The study defined high-risk symptoms as weight loss, anaemia (haemoglobin less than 120 g/l in females or less than 130 g/l in males), or diarrhoea (more than three loose stools per day).

The likelihood of celiac sprue determines the approach to diagnosis. If a low or moderate suspicion exists that celiac disease is present, a blood test for tissue transglutaminase (tTG) or antiendomysial antibody is performed. If the likelihood that someone has celiac disease is very high or the blood test result is positive, then biopsies of the small intestinal should be performed.

Genetic testing is only performed in certain circumstances.

Blood tests

Blood chemistry, red blood cell, and clotting test results suggest but do not confirm the diagnosis of celiac sprue. The same abnormalities may be seen in many other diseases.

- Electrolyte imbalances, such as low potassium level (hypokalemia), low calcium level (hypocalcemia), and low magnesium level (hypomagnesemia), may be present.
- Sometimes, malnutrition includes a low albumin level (hypoalbuminemia), a low total protein level (hypoproteinemia), and a low cholesterol level (hypocholesterolemia).
- Anemia due to deficiency in iron, folate, or vitamin B-12 may be present.
- A low serum iron level is common.
- Malabsorption of vitamin K may cause abnormal clotting test results such as a prolonged prothrombin time.

Serologic tests

The best diagnostic tests for celiac sprue include measurements of antibody levels to endomysium and to an enzyme called tissue transglutaminase (tTG). The 2 tests are very specific for celiac disease in persons who are untreated.

Measurements of antibodies to gliadin and reticulin (a part of the cell structure) are other diagnostic tests that are less specific for celiac disease.

Small intestinal imaging tests

Radiology tests, such as small-bowel barium studies and abdominal/pelvic CT scanning, are usually not helpful in establishing the diagnosis of celiac disease. In video capsule endoscopy, a tiny camera in a capsule films the small intestine as the camera moves through it; however, this study cannot examine the tissue

microscopically. These tests should be considered in the evaluation of persons with suspected celiac sprue and those who have dramatic weight loss, severe abdominal pain, intestinal bleeding, significant decrease in albumin levels, and intestinal obstruction. These symptoms may suggest the presence of tumors or ulcers in the small intestine.

Small intestinal biopsy

The lining of the small intestine usually consists of finger like projections called villi. The villi contain digestive enzymes and provide the large absorptive surface of the small intestine. In celiac sprue, the villi are destroyed because of the inflammatory and autoimmune process. Once the villi are destroyed, nutrients cannot be absorbed. Biopsy samples of the small intestine show mild, moderate, or severe destruction of the villi depending on the severity of the inflammation. Biopsy samples of the small intestine are obtained by introducing a small, flexible endoscope through the mouth, the stomach, and into the small intestine while the patient is sedated.

Treatment

The treatment for celiac sprue is strict avoidance of gluten in the diet.

- Removal of gluten from the diet is essential. Because a gluten-free diet is a lifetime commitment, is more expensive than a normal diet, and has social implications, it should not be recommended unless the diagnosis is firmly established. Complete avoidance of gluten-containing grain products takes significant effort. Maintaining a strict, gluten-free diet may be relatively difficult because certain products, such as wheat flour, are so common in the Western diet.
- Improvement in symptoms begins within days of starting the gluten-free diet. Complete healing of the small intestine, meaning the villi are intact and working, usually occurs in 3-6 months, although it may take up to 2 years in older persons.

Because lactose intolerance is common in people with celiac sprue, vital improvements occur when a lactose-free diet is introduced.

Self-Care

For the most part, successful control of celiac sprue consists of what happens at home to modify diet and to select foods that can be eaten. Many resources are available to assist a person with choosing appropriate foods and modifying recipes to work within his or her diet.

Food labels should be read carefully. Wheat and rye flours, barley, and oats are common ingredients in many products. Many products a person would not

suspect contain flour, such as salad dressings. In addition, barley is used in the brewing process of beer. The following substitutions may be tried:

- Rice flour and bread made with rice flour may be found at local specialty grocery stores.
- Cornstarch may be substituted for thickening sauces or gravies.
- Sorghum may also be substituted.

Endoscopy

An upper endoscopy with biopsy of the duodenum (beyond the duodenal bulb) or jejunum is performed. It is important for the physician to obtain multiple samples (four to eight) from the duodenum. Not all areas may be equally affected; if biopsies are taken from healthy bowel tissue, the result would be a false negative.

Most patients with coeliac disease have a small bowel that appears normal on endoscopy; however, five concurrent endoscopic findings have been associated with a high specificity for coeliac disease: scalloping of the small bowel folds (pictured), paucity in the folds, a mosaic pattern to the mucosa (described as a "cracked-mud" appearance), prominence of the submucosa blood vessels, and a nodular pattern to the mucosa.

Until the 1970s, biopsies were obtained using metal capsules attached to a suction device. The capsule was swallowed and allowed to pass into the small intestine. After x-ray verification of its position, suction was applied to collect part of the intestinal wall inside the capsule. One often-utilised capsule system is the Watson capsule. This method has now been largely replaced by fibre-optic endoscopy, which carries a higher sensitivity and a lower frequency of errors.

Dietary Treatment

At present, the only effective treatment is a life-long gluten-free diet. No medication exists that will prevent damage or prevent the body from attacking the gut when gluten is present. Strict adherence to the diet allows the intestines to heal, leading to resolution of all symptoms in most cases and, depending on how soon the diet is begun, can also eliminate the heightened risk of osteoporosis and intestinal cancer. Dietician input is generally requested to ensure the patient is aware which foods contain gluten, which foods are safe, and how to have a balanced diet despite the limitations. In many countries, gluten-free products are available on prescription and may be reimbursed by health insurance plans.

The diet can be cumbersome; failure to comply with the diet may cause relapse. The term gluten-free is generally used to indicate a supposed harmless level of gluten rather than a complete absence. The exact level at which gluten is

harmless is uncertain and controversial. A recent systematic review tentatively concluded that consumption of less than 10 mg of gluten per day is unlikely to cause histological abnormalities, although it noted that few reliable studies had been done. Regulation of the label gluten-free varies widely by country. For example, in the United States, the term gluten-free is not yet regulated. The current international Codex Alimentarius standard, established in 1981, allows for 50 mg N/100 g on dry matter, although a proposal for a revised standard of 20 ppm in naturally gluten-free products and 200 ppm in products rendered gluten-free has been accepted. Gluten-free products are usually more expensive and harder to find than common gluten-containing foods. Since ready-made products often contain traces of gluten, some coeliacs may find it necessary to cook from scratch.

Even while on a diet, health-related quality of life (HRQOL) may be lower in people with coeliac disease. Studies in the United States have found that quality of life becomes comparable to the general population after staying on the diet, while studies in Europe have found that quality of life remains lower, although the surveys are not quite the same. Men tend to report more improvement than women. Some have persisting digestive symptoms or dermatitis herpetiformis, mouth ulcers, osteoporosis and resultant fractures. Symptoms suggestive of irritable bowel syndrome may be present, and there is an increased rate of anxiety, fatigue, dyspepsia and musculoskeletal pain.

Everyone is different, but many people with coeliac disease also have one or more additional food allergies or food intolerances, which may include milk protein (casein), corn (maize), soy, amines, or salicylates.

Student Activity

1. Outline the characteristics of Malabsorption syndrome.

2. Discuss the signs and symptoms of Celiac Sprue.

4.7 DIVERTICULAR DISORDERS

Diverticular disorders are divided into two main types of disorders: the more serious diverticulitis and the milder form called diverticulosis. The disorder of diverticulosis starts off when the inner lining of the large bowel protrudes and pushes through the muscular layer that normally envelopes it, this leads to the formation of pouches called-diverticula's, these pouches range in sizes from minute, pea-sized pouches to more those that are more than an inch in across. Food particles can become entangled and trapped in these pouches, even through there are no symptoms of diverticulosis as such, the pouches become inflamed from having the food particles stuck in them which then become inflamed, this inflammation leads to infection, and soon diverticulitis results, the pain and the symptoms now become inescapable.

A low-fiber diet is the typical cause of many types of diverticulosis, the deficiency of fiber in the diet can mean passing stool becomes harder as the colon must now work harder, and there is a lot of straining involved during bowel movements this can complicate and aggravate the symptoms of the condition. The chances of diverticulitis increases in people who have very little fiber in the diet, because waste moves slowly within the body without fiber, this gives more time to food particles to become trapped and causes an inflammation or infection in the pouches. The colon may also perform sluggishly due to the lack of physical exercises in certain people. This condition can strike across all members of a family and tends to run in families, as members are more likely to have similar food habits.

Causes

Diverticular disease essentially results from eating a diet with too little fiber.

Fiber itself is not digested. It passes through the intestines pretty much unchanged, softening the stools and their passage. Lack of fiber begins a sequence of events:

- Without fiber, the stools are dry and small, and the intestinal muscles must contract with greater force to pass the stools along, generating a higher pressure in the large intestines.
- The excess pressure leads to weak spots in the colon walls that eventually bulge out and form pouches called diverticula.
- Existing weakness in the colon walls-either from age or, in younger people, or from collagen disorders like Marfan's syndrome-also contribute to the development of diverticula.
- Most often, the pouches form in the sigmoid colon, which is the lower left

part of the colon that connects to the rectum. This area of the colon is subject to the highest amount of pressure because it is the narrowest portion of the large intestine.

SYMPTOMS

Most patients with diverticulosis have few or no symptoms. The diverticulosis condition in these individuals is found incidentally during tests for other intestinal problems. Twenty percent of patients with diverticulosis will develop symptoms related to diverticulosis. The most common symptoms of diverticular disease include :

- abdominal cramping,
- constipation, and
- diarrhoea.

These symptoms are related to difficulty of passing stool along the left colon narrowed by diverticular disease.

More serious complications include :

- diverticulitis,
- collection of pus (abscess) in the pelvis,
- colon obstruction,
- generalized infection of the abdominal cavity (bacterial peritonitis), and
- bleeding into the colon.

A diverticulum can rupture, and the bacteria within the colon can spread into the tissues surrounding the colon causing diverticulitis. Constipation or diarrhoea may also occur. A collection of pus can develop around the inflamed diverticulum, leading to formation of an abscess, usually in the pelvis. On rare occasions, the inflamed diverticula can erode into the urinary bladder, causing bladder infection and passing of gas during urination. Inflammation in the colon can also lead to bowel obstruction. Infrequently, a diverticulum ruptures freely into the abdominal cavity causing a life threatening infection called peritonitis.

Diverticular bleeding occurs when the expanding diverticulum erodes into a blood vessel at the base of a diverticulum. Rectal passage of red, dark or maroon-coloured blood and clots occur without any associated abdominal pain. Rarely, blood may be black from a diverticulum of the right colon. Bleeding may be continuous or intermittent, lasting several days.

Patients with active bleeding usually are hospitalized for monitoring. Intravenous fluids are given to support the blood pressure. Blood transfusions

are necessary for those with moderate to severe blood loss. In a rare individual with brisk and severe bleeding, the blood pressure can drop, causing dizziness, shock, and loss of consciousness. In most patients, bleeding stops spontaneously and they are sent home after several days in the hospital. Patients with persistent, severe bleeding require surgical removal of the bleeding diverticula.

Diagnosis

Once suspected, the diagnosis of diverticular disease can be confirmed by a variety of tests. Barium x-rays (barium enemas) can be performed to visualize the colon. Diverticula are seen as barium filled pouches protruding from the colon wall.

Direct visualization of the intestine can be done with flexible tubes inserted through the rectum and advanced into the colon. Either short tubes (sigmoidoscopes) or longer tubes (colonoscopes) may be used to assist in the diagnosis and to exclude other diseases that can mimic diverticular disease.

In patients suspected of having diverticular abscess causing persistent pain and fever, ultrasound and CT scan examinations of the abdomen and pelvis can be done to detect collections of pus fluid.

Treatment

Many patients with diverticulosis have minimal or no symptoms, and do not require any specific treatment. A high fiber diet and fiber supplements are advisable to prevent constipation and the formation of more diverticula.

Patients with mild symptoms abdominal pain due to muscular spasm in the area of the diverticula may benefit from anti-spasmodic drugs such as:

- chlordiazepoxide (Librax),
- dicyclomine (Bentyl),
- hyoscyamine, atropine, scopolamine, phenobarb (Donnatal), and
- hyoscyamine (Levsin).

Some doctors also recommend avoidance of nuts, corn, and seeds to prevent complications of diverticulosis. Whether these dietary restrictions are beneficial is uncertain.

When diverticulitis occurs, antibiotics are usually needed. Oral antibiotics are sufficient when symptoms are mild. Some examples of commonly prescribed antibiotics include:

- ciprofloxacin (Cipro),
- metronidazole (Flagyl),

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- cephalexin (Keflex), and
- doxycycline (Vibramycin).

Liquid or low fiber foods are advised during acute attacks of diverticulitis. This is done to reduce the amount of material that passes through the colon, which at least theoretically, might aggravate the diverticulitis. In severe diverticulitis with high fever and pain, patients are hospitalized and given intravenous antibiotics. Surgery is needed for those with persistent bowel obstruction or abscess not responding to antibiotics.

Surgery

Diverticulitis that does not respond to medical treatment requires surgical intervention. Surgery usually involves drainage of any collections of pus and resection (surgical removal) of that segment of the colon containing the diverticuli, usually the sigmoid colon. Therefore, surgical removal of the bleeding diverticula is necessary for those with persistent bleeding. In patients needing surgery to stop persistent bleeding, exact localization becomes crucial to guide the surgeon.

Sometimes, diverticula can erode into the adjacent bladder, causing severe recurrent urine infection and passage of gas during urination. This situation also requires surgery.

Sometimes, surgery may be suggested for patients with frequent, recurrent attacks of diverticulitis leading to multiple courses of antibiotics, hospitalizations, and days lost from work. During surgery, the goal is to remove all, or almost all, of the colon containing diverticula in order to prevent future episodes of diverticulitis. There are few long-term consequences of resection of the sigmoid colon for diverticulitis, and the surgery often can be done laparoscopically, which limits post operative pain and time for recovery.

Prevention

Once formed, diverticula are permanent. No treatment has been found to prevent complications of diverticular disease.

Diets high in fiber increases stool bulk and prevents constipation, and theoretically may help prevent further diverticular formation or worsening of the diverticular condition. Some doctors recommend avoiding nuts, corn, and seeds which can plug diverticular openings and cause diverticulitis. Whether avoidance of such foods is beneficial is unclear.

Patients with known diverticular disease who develop unexplained fever, chills or abdominal pain should notify their doctor because of the possibility of the complication of diverticulitis. A better understanding of the way diverticula form and become infected will hopefully lead to discovery of more effective ways to manage these common conditions.

4.8 SUMMARY

- Dyspepsia popularly known as indigestion, meaning hard or difficult digestion, is a medical condition characterized by chronic or recurrent pain in the upper abdomen, upper abdominal fullness.
- Gastritis can be caused by drinking too much alcohol, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin or ibuprofen, or infection with bacteria such as Helicobacter pylori (H. pylori). Sometimes gastritis develops after major surgery, traumatic injury, burns, or severe infections.
- A stomach ulcer (also called a peptic ulcer) is a small erosion (hole) in the gastrointestinal tract. The most common type, duodenal, occurs in the first 12 inches of small intestine beyond the stomach.
- Malabsorption is a state arising from abnormality in absorption of food nutrients across the gastrointestinal(GI) tract.
- Celiac sprue, also known as celiac disease, gluten-sensitive enteropathy, and gluten-induced enteropathy, is a chronic disease of the digestive tract that interferes with the digestion and absorption of nutrients from food.
- Diverticular disorders are divided into two main types of disorders: the more serious diverticulitis and the milder form called diverticulosis. The disorder of diverticulosis starts off when the inner lining of the large bowel protrudes and pushes through the muscular layer that normally envelopes it.

4.9 GLOSSARY

- **Dyspepsia:** popularly known as indigestion, meaning hard or difficult digestion, is a medical condition characterized by chronic or recurrent pain in the upper abdomen.
- **Gastritis:** it is a disease caused by drinking too much alcohol, prolonged use of nonsteroidal anti-inflammatory drugs (NSAIDs) such as aspirin or ibuprofen, or infection with bacteria such as Helicobacter pylori (H. pylori).
- **Ulcer:** a small erosion (hole) in the gastrointestinal tract. The most common type, duodenal, occurs in the first 12 inches of small intestine beyond the stomach.
- **Malabsorption:** it is a state arising from abnormality in absorption of food nutrients across the gastrointestinal(GI) tract.
- **Celiac Sprue:** it is a chronic disease of the digestive tract that interferes with the digestion and absorption of nutrients from food.

- **Diverticular Disorders:** it is a disorder that starts off when the inner lining of the large bowel protrudes and pushes through the muscular layer.

4.10 REVIEW QUESTIONS

1. How is Dyspepsia treated? Discuss.
2. How does Gastritis develop? Point out it's causes.
3. What are the signs and symptoms of Ulcer?
4. Discuss the diagnosis of Malabsorption.
5. What are the features of Celiac Sprue?
6. How is Celiac Sprue prevented?
7. What are the principal signs and symptoms of Diverticular disorders?

4.11 FURTHER READINGS

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UNIT– V

LIVER, GALL BLADDER AND BILLIARY TRACK DISORDERS

OBJECTIVES

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

- understand the causes, symptoms and dietary treatment of Jaundice;
- state the signs, symptoms and treatment of Hepatitis;
- explain the complications, causes and diagnosis of Cirrhosis;
- discuss the characteristics, signs and treatments of Cholelithiasis;
- understand the reasons behind Pancreatitis and its dietary treatment.

STRUCTURE

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5.1 INTRODUCTION

Located in the upper right portion of the abdomen, the liver and gallbladder are interconnected by ducts known as the biliary tract, which drains into the first segment of the small intestine (the duodenum). Although the liver and gallbladder participate in some of the same functions, they are very different.

A number of structural and microscopic changes occur as the liver ages. For example, the colour of the liver changes from lighter to darker brown. Its size and blood flow decrease. However, liver function test results generally remain normal.

The ability of the liver to metabolize many substances decreases with aging. Thus, some drugs are not inactivated as quickly in older people as they are in younger people. As a result, a drug dose that would not have side effects in younger people may cause dose-related side effects in older people. Thus, drug dosages often need to be decreased in older people. Also, the liver's ability to withstand stress decreases. Thus, substances that are toxic to the liver can cause more damage in older people than in younger people. Repair of damaged liver cells is also slower in older people.

The production and flow of bile decrease with aging. As a result, gallstones are more likely to form.

The gallbladder is a small, pear-shaped, muscular storage sac that holds bile. Bile is a greenish yellow, thick, sticky fluid. It consists of bile salts, electrolytes (dissolved charged particles, such as sodium and bicarbonate), bile pigments, cholesterol, and other fats (lipids). Bile has two main functions: aiding in digestion and eliminating certain waste products (mainly hemoglobin and excess cholesterol) from the body. Bile salts aid in digestion by making cholesterol, fats, and fat-soluble vitamins easier to absorb from the intestine. The main pigment in bile, bilirubin, is a waste product that is formed from hemoglobin (the protein that carries oxygen in the blood) and is excreted in bile. Hemoglobin is released when old or damaged red blood cells are destroyed.

Bile flows out of the liver through the left and right hepatic ducts, which come together to form the common hepatic duct. This duct then joins with a duct connected to the gallbladder, called the cystic duct, to form the common bile duct. The common bile duct enters the small intestine at the sphincter of Oddi (a ring-shaped muscle), located a few inches below the stomach.

About half the bile secreted between meals flows directly through the common bile duct into the small intestine. The rest of the bile is diverted through the cystic duct into the gallbladder to be stored. In the gallbladder, up to 90% of the water in bile is absorbed into the bloodstream, making the remaining bile

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very concentrated. When food enters the small intestine, a series of hormonal and nerve signals triggers the gallbladder to contract and the sphincter of Oddi to relax and open. Bile then flows from the gallbladder into the small intestine to mix with food contents and perform its digestive functions.

After bile enters and passes down the small intestine, about 90% of bile salts are reabsorbed into the bloodstream through the wall of the lower small intestine. The liver extracts these bile salts from the blood and resecretes them back into the bile. Bile salts go through this cycle about 10 to 12 times a day. Each time, small amounts of bile salts escape absorption and reach the large intestine, where they are broken down by bacteria. Some bile salts are reabsorbed in the large intestine. The rest are excreted in the stool.

The gallbladder, although useful, is not necessary. If the gallbladder is removed (for example, in a person with cholecystitis), bile can move directly from the liver to the small intestine.

Hard masses consisting mainly of cholesterol (gallstones) may form in the gallbladder or bile ducts. Gallstones usually cause no symptoms. However, gallstones may block the flow of bile from the gallbladder, causing pain (biliary colic) or inflammation. They may also migrate from the gallbladder to the bile duct, where they can block the normal flow of bile to the intestine, causing jaundice (a yellowish discolouration of the skin and whites of the eyes) in addition to pain and inflammation. The flow of bile can also be blocked by tumors. Other causes of blocked flow are less common.

5.2 JAUNDICE

Jaundice, also known as icterus (attributive adjective: icteric), is a yellowish discolouration of the skin, the conjunctival membranes over the sclerae (whites of the eyes), and other mucous membranes caused by hyperbilirubinemia (increased levels of bilirubin in the blood). This hyperbilirubinemia subsequently causes increased levels of bilirubin in the extracellular fluids. Typically, the concentration of bilirubin in the plasma must exceed 1.5 mg/dL, three times the usual value of approximately 0.5 mg/dL, for the colouration to be easily visible. Jaundice comes from the French word jaune, meaning yellow.

One of the first tissues to change color as bilirubin levels rise in jaundice is the conjunctiva of the eye, a condition sometimes referred to as scleral icterus. However, the sclera themselves are not "icteric" (stained with bile pigment) but rather the conjunctival membranes that overlie them. The yellowing of the "white of the eye" is thus more properly conjunctival icterus.

Normal Physiology

In order to understand how jaundice results, the pathological processes that cause jaundice to take their effect must be understood. Jaundice itself is not

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a disease, but rather a sign of one of many possible underlying pathological processes that occur at some point along the normal physiological pathway of the metabolism of bilirubin.

When red blood cells have completed their life span of approximately 120 days, or when they are damaged, their membranes become fragile and prone to rupture. As each red blood cell traverses through the reticuloendothelial system, its cell membrane ruptures when its membrane is fragile enough to allow this. Cellular contents, including hemoglobin, are subsequently released into the blood. The hemoglobin is phagocytosed by macrophages, and split into its heme and globin portions. The globin portion, a protein, is degraded into amino acids and plays no role in jaundice.

Two reactions then take place with the heme molecule. The first oxidation reaction is catalyzed by the microsomal enzyme heme oxygenase and results in biliverdin (green colour pigment), iron and carbon monoxide. The next step is the reduction of biltverdin to a yellow colour tetrapyrol pigment called bilirubin by cytosolic enzyme biliverdin reductase. This bilirubin is "unconjugated," "free" or "indirect" bilirubin. Approximately 4 mg per kg of bilirubin is produced each day. The majority of this bilirubin comes from the breakdown of heme from expired red blood cells in the process just described. However approximately 20 percent comes from other heme sources, including ineffective erythropoiesis, and the breakdown of other heme-containing proteins, such as muscle myoglobin and cytochromes.

Hepatic Events

The unconjugated bilirubin then travels to the liver through the bloodstream. Because this bilirubin is not soluble, however, it is transported through the blood bound to serum albumin. Once it arrives at the liver, it is conjugated with glucuronic acid (to form bilirubin diglucuronide, or just "conjugated bilirubin") to become more water soluble. The reaction is catalyzed by the enzyme UDP-glucuronide transferase.

This conjugated bilirubin is excreted from the liver into the biliary and cystic ducts as part of bile. Intestinal bacteria convert the bilirubin into urobilinogen. From here the urobilinogen can take two pathways. It can either be further converted into stercobilinogen, which is then oxidized to stercobilin and passed out in the faeces, or it can be reabsorbed by the intestinal cells, transported in the blood to the kidneys, and passed out in the urine as the oxidised product urobilin. Stercobilin and urobilin are the products responsible for the colouration of faeces and urine, respectively.

Causes

When a pathological process interferes with the normal functioning of the metabolism and excretion of bilirubin just described, jaundice may be the result.

Jaundice is classified into three categories, depending on which part of the physiological mechanism the pathology affects. The three categories are:

- **Pre-hepatic:** The pathology is occurring prior the liver.
- **Hepatic:** The pathology is located within the liver.
- **Post-Hepatic:** The pathology is located after the conjugation of bilirubin in the liver.

Pre-hepatic

Pre-hepatic jaundice is caused by anything which causes an increased rate of hemolysis (breakdown of red blood cells). In tropical countries, malaria can cause jaundice in this manner. Certain genetic diseases, such as sickle cell anemia, spherocytosis and glucose 6-phosphate dehydrogenase deficiency can lead to increased red cell lysis and therefore hemolytic jaundice. Commonly, diseases of the kidney, such as hemolytic uremic syndrome, can also lead to colouration. Defects in bilirubin metabolism also present as jaundice. Jaundice usually comes with high fevers. Rat fever (leptospirosis) can also cause jaundice.

Laboratory findings include:

- **Urine:** no bilirubin present, urobilirubin > 2 units (except in infants where gut flora has not developed).
- **Serum:** increased unconjugated bilirubin.

Hepatic

Hepatic jaundice causes include acute hepatitis, hepatotoxicity and alcoholic liver disease, whereby cell necrosis reduces the liver's ability to metabolise and excrete bilirubin leading to a buildup in the blood. Less common causes include primary biliary cirrhosis, Gilbert's syndrome (a genetic disorder of bilirubin metabolism which can result in mild jaundice, which is found in about 5% of the population), Crigler-Najjar syndrome, metastatic carcinoma and Niemann-Pick disease, type C. Jaundice seen in the newborn, known as neonatal jaundice, is common, occurring in almost every newborn as hepatic machinery for the conjugation and excretion of bilirubin does not fully mature until approximately two weeks of age.

Laboratory findings include:

- **Urine:** Conjugated bilirubin present, urobilirubin > 2 units but variable (except in children).

Post-hepatic

Post-hepatic jaundice, also called obstructive jaundice, is caused by an interruption to the drainage of bile in the biliary system. The most common

causes are gallstones in the common bile duct, and pancreatic cancer in the head of the pancreas. Also, a group of parasites known as "liver flukes" can live in the common bile duct, causing obstructive jaundice. Other causes include strictures of the common bile duct, biliary atresia, ductal carcinoma, pancreatitis and pancreatic pseudocysts. A rare cause of obstructive jaundice is Mirizzi's syndrome.

The presence of pale stools and dark urine suggests an obstructive or post-hepatic cause as normal feces get their colour from bile pigments. Patients also can present with elevated serum cholesterol, and often complain of severe itching or "pruritus".

No one test can differentiate between various classifications of jaundice. A combinations of liver function tests is essential to arrive at a diagnosis.

Neonatal Jaundice

Neonatal jaundice is usually harmless, this condition is often seen in infants around the second day after birth, lasting until day 8 in normal births, or to around day 14 in premature births. Serum bilirubin normally drops to a low level without any intervention required: the jaundice is presumably a consequence of metabolic and physiological adjustments after birth. In extreme cases, a brain-damaging condition known as kernicterus can occur, leading to significant lifelong disability; there are concerns that this condition has been rising in recent years due to inadequate detection and treatment of neonatal hyperbilirubinemia.

5.3 HEPATITIS

Hepatitis (plural hepatitides) implies injury to the liver characterized by the presence of inflammatory cells in the tissue of the organ. The name is from ancient Greek hepar, the root being hepat- meaning liver, and suffix -itis, meaning "inflammation". The condition can be self-limiting, healing on its own, or can progress to scarring of the liver. Hepatitis is acute when it lasts less than six months and chronic when it persists longer. A group of viruses known as the hepatitis viruses cause most cases of liver damage worldwide. Hepatitis can also be due to toxins (notably alcohol), other infections or from autoimmune process. It may run a subclinical course when the affected person may not feel ill. The patient becomes unwell and symptomatic when the disease impairs liver functions that include, among other things, removal of harmful substances, regulation of blood composition, and production of bile to help digestion.

Causes

Acute

- Viral hepatitis: Hepatitis A through E (more than 95% of viral cause), Herpes simplex, Cytomegalovirus, Epstein-Barr, yellow fever virus, adenoviruses.

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- Nonviral infection: toxoplasma, Leptospira, Q fever, rocky mountain spotted fever
- Alcohol
- Toxins: Amanita toxin in mushrooms, carbon tetrachloride, asafetida
- Drugs: Paracetamol, amoxycillin, antituberculosis medicines, minocycline and many others (see longer list below).
- Ischemic hepatitis (circulatory insufficiency)
- Pregnancy
- Auto immune conditions, *e.g.*, Systemic Lupus Erythematosus (SLE)
- Metabolic diseases, *e.g.*, Wilson's disease

Chronic

- Viral hepatitis: Hepatitis B with or without hepatitis D, hepatitis C (neither hepatitis A nor hepatitis E causes chronic hepatitis)
- Autoimmune: Autoimmune hepatitis
- Alcohol
- Drugs: methyldopa, nitrofurantoin, isoniazid, ketoconazole
- Non-alcoholic steatohepatitis
- Heredity: Wilson's disease, alpha 1-antitrypsin deficiency
- Primary biliary cirrhosis and primary sclerosing cholangitis occasionally mimic chronic hepatitis

Symptoms

Acute

Clinically, the course of acute hepatitis varies widely from mild symptoms requiring no treatment to fulminant hepatic failure needing liver transplantation. Acute viral hepatitis is more likely to be asymptomatic in younger people. Symptomatic individuals may present after convalescent stage of 7 to 10 days, with the total illness lasting 2 to 6 weeks.

Initial features are of nonspecific flu – like symptoms, common to almost all acute viral infections and may include malaise, muscle and joint aches, fever, nausea or vomiting, diarrhoea, and headache. More specific symptoms, which can be present in acute hepatitis from any cause, are: profound loss of appetite, aversion to smoking among smokers, dark urine, yellowing of the eyes and skin (*i.e.*, jaundice) and abdominal discomfort. Physical findings are usually minimal, apart from jaundice (33%) and tender hepatomegaly (10%). There can be occasional lymphadenopathy (5%) or splenomegaly (5%).

Chronic

Majority of patients will remain asymptomatic or mildly symptomatic, abnormal blood tests being the only manifestation. Features may be related to the extent of liver damage or the cause of hepatitis. Many experience return of symptoms related to acute hepatitis. Jaundice can be a late feature and may indicate extensive damage. Other features include abdominal fullness from enlarged liver or spleen, low grade fever and fluid retention (ascites). Extensive damage and scarring of liver (*i.e.*, cirrhosis) leads to weight loss, easy bruising and bleeding tendencies. Acne, abnormal menstruation, lung scarring, inflammation of the thyroid gland and kidneys may be present in women with autoimmune hepatitis.

Findings on clinical examination are usually those of cirrhosis or are related to etiology.

TYPES

Viral

Most cases of acute hepatitis are due to viral infections:

- Hepatitis A
- Hepatitis B
- Hepatitis C
- Hepatitis B with D
- Hepatitis E
- Hepatitis F virus (existence unknown)
- Hepatitis G, or GBV-C
- In addition to the hepatitis viruses (please note that the hepatitis viruses are not all related), other viruses can also cause hepatitis, including cytomegalovirus, Epstein-Barr virus, yellow fever, etc.

Other viral causes

Other viral infections can cause hepatitis (inflammation of the liver):

- Mumps virus
- Rubella virus
- Cytomegalovirus
- Epstein-Barr virus
- Other herpes viruses

Alcoholic Hepatitis

Ethanol, mostly in alcoholic beverages, is a significant cause of hepatitis. Usually alcoholic hepatitis comes after a period of increased alcohol consumption.

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Alcoholic hepatitis is characterized by a variable constellation of symptoms, which may include feeling unwell, enlargement of the liver, development of fluid in the abdomen ascites, and modest elevation of liver blood tests. Alcoholic hepatitis can vary from mild with only liver test elevation to severe liver inflammation with development of jaundice, prolonged prothrombin time, and liver failure. Severe cases are characterized by either obtundation (dulled consciousness) or the combination of elevated bilirubin levels and prolonged prothrombin time; the mortality rate in both categories is 50% within 30 days of onset.

Alcoholic hepatitis is distinct from cirrhosis caused by long term alcohol consumption. Alcoholic hepatitis can occur in patients with chronic alcoholic liver disease and alcoholic cirrhosis. Alcoholic hepatitis by itself does not lead to cirrhosis, but cirrhosis is more common in patients with long term alcohol consumption. Patients who drink alcohol to excess are also more often than others found to have hepatitis C. The combination of hepatitis C and alcohol consumption accelerates the development of cirrhosis.

Drug Induced

A large number of drugs can cause hepatitis:

- Agomelatine (antidepressant)
- Allopurinol
- Amitriptyline (antidepressant)
- Amiodarone (antiarrhythmic)
- Atomoxetine
- Azathioprine
- Halothane (a specific type of anesthetic gas)
- Hormonal contraceptives
- Ibuprofen and indomethacin (NSAIDs)
- Isoniazid (INH), rifampicin, and pyrazinamide (tuberculosis-specific antibiotics)
- Ketoconazole (antifungal)
- Loratadine (antihistamine)
- Methotrexate (immune suppressant)
- Methyldopa (antihypertensive)
- Minocycline (tetracycline antibiotic)
- Nifedipine (antihypertensive)

- Nitrofurantoin (antibiotic)
- Paracetamol (acetaminophen in the United States) can cause hepatitis when taken in an overdose. The severity of liver damage may be limited by prompt administration of acetylcysteine.
- Phenytoin and valproic acid (antiepileptics)
- Troglitazone (antidiabetic, withdrawn in 2000 for causing hepatitis)
- Zidovudine (antiretroviral *i.e.*, against HIV)
- Some herbs and nutritional supplements

The clinical course of drug-induced hepatitis is quite variable, depending on the drug and the patient's tendency to react to the drug. For example, halothane hepatitis can range from mild to fatal as can INH-induced hepatitis.

Hormonal contraception can cause structural changes in the liver. Amiodarone hepatitis can be untreatable since the long half life of the drug (up to 60 days) means that there is no effective way to stop exposure to the drug. Statins can cause elevations of liver function blood tests normally without indicating an underlying hepatitis. Lastly, human variability is such that any drug can be a cause of hepatitis.

Other Toxins

Other Toxins can cause hepatitis:

- Amatoxin-containing mushrooms, including the Death Cap (Amanita phalloides), the Destroying Angel (Amanita ocreata), and some species of Galerina. A portion of a single mushroom can be enough to be lethal (10 mg or less of *alpha-amanitin*).
- White phosphorus, an industrial toxin and war chemical.
- Carbon tetrachloride ("tetra", a dry cleaning agent), chloroform, and trichloroethylene, all chlorinated hydrocarbons, cause steatohepatitis (hepatitis with fatty liver).
- Cylindrospermopsin, a toxin from the cyanobacterium Cylindrospermopsis raciborskii and other cyanobacteria.

Metabolic Disorders

Some metabolic disorders cause different forms of hepatitis. Hemochromatosis (due to iron accumulation) and Wilson's disease (copper accumulation) can cause liver inflammation and necrosis.

Non-alcoholic steatohepatitis (NASH) is effectively a consequence of metabolic syndrome.

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Obstructive

"Obstructive jaundice" is the term used to describe jaundice due to obstruction of the bile duct (by gallstones or external obstruction by cancer). If longstanding, it leads to destruction and inflammation of liver tissue.

Autoimmune

Anomalous presentation of human leukocyte antigen (HLA) class II on the surface of hepatocytes, possibly due to genetic predisposition or acute liver infection; causes a cell-mediated immune response against the body's own liver, resulting in autoimmune hepatitis.

Alpha 1-antitrypsin deficiency

In severe cases of alpha 1-antitrypsin deficiency (A1AD), the accumulated protein in the endoplasmic reticulum causes liver cell damage and inflammation.

Non-alcoholic fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is the occurrence of fatty liver in people who have no history of alcohol use. It is most commonly associated with obesity (80% of all obese people have fatty liver). It is more common in women. Severe NAFLD leads to inflammation, a state referred to as non-alcoholic steatohepatitis (NASH), which on biopsy of the liver resembles alcoholic hepatitis (with fat droplets and inflammatory cells, but usually no Mallory bodies).

The diagnosis depends on medical history, physical exam, blood tests, radiological imaging and sometimes a liver biopsy. The initial evaluation to identify the presence of fatty infiltration of the liver is medical imaging, including such ultrasound, computed tomography (CT), or magnetic resonance (MRI). However, imaging cannot readily identify inflammation in the liver. Therefore, the differentiation between steatosis and NASH often requires a liver biopsy. It can also be difficult to distinguish NASH from alcoholic hepatitis when the patient has a history of alcohol consumption. Sometimes in such cases a trial of abstinence from alcohol along with follow-up blood tests and a repeated liver biopsy are required.

NASH is becoming recognized as the most important cause of liver disease second only to hepatitis C in numbers of patients going on to cirrhosis.

Ischemic Hepatitis

Ischemic hepatitis is caused by decreased circulation to the liver cells. Usually this is due to decreased blood pressure (or shock), leading to the equivalent term "shock liver". Patients with ischemic hepatitis are usually very ill due to the underlying cause of shock. Rarely, ischemic hepatitis can be caused by local problems with the blood vessels that supply oxygen to the liver (such as

thrombosis, or clotting of the hepatic artery which partially supplies blood to liver cells). Blood testing of a person with ischemic hepatitis will show very high levels of transaminase enzymes (AST and ALT), which may exceed 1000 U/L. The elevation in these blood tests is usually transient (lasting 7 to 10 days). It is rare that liver function will be affected by ischemic hepatitis.

5.4 CIRRHOSIS

Cirrhosis is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrous scar tissue as well as regenerative nodules (lumps that occur as a result of a process in which damaged tissue is regenerated), leading to progressive loss of liver function. Cirrhosis is most commonly caused by alcoholism, hepatitis B and C, and fatty liver disease but has many other possible causes. Some cases are idiopathic, *i.e.*, of unknown cause.

Ascites (fluid retention in the abdominal cavity) is the most common complication of cirrhosis and is associated with a poor quality of life, increased risk of infection, and a poor long-term outcome. Other potentially life-threatening complications are hepatic encephalopathy (confusion and coma) and bleeding from esophageal varices. Cirrhosis is generally irreversible once it occurs, and treatment generally focuses on preventing progression and complications. In advanced stages of cirrhosis the only option is a liver transplant.

Signs and Symptoms

Some of the following signs and symptoms may occur in the presence of cirrhosis or as a result of the complications of cirrhosis. Many are nonspecific and may occur in other diseases and do not necessarily point to cirrhosis. Likewise, the absence of any does not rule out the possibility of cirrhosis.

- **Spider angiomata or spider nevi.** Vascular lesions consisting of a central arteriole surrounded by many smaller vessels due to an increase in estradiol. These occur in about 1/3 of cases.
- **Palmar erythema.** Exaggerations of normal speckled mottling of the palm, due to altered sex hormone metabolism.
- Nail changes.

—Muehrcke's nails - paired horizontal bands separated by normal colour due to hypoalbuminemia (inadequate production of albumin).

—Terry's nails - proximal two thirds of the nail plate appears white with distal one-third red, also due to hypoalbuminemia

—Clubbing - angle between the nail plate and proximal nail fold > 180 degrees

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- **Hypertrophic osteoarthropathy.** Chronic proliferative periostitis of the long bones that can cause considerable pain.
- **Dupuytren's contracture.** Thickening and shortening of palmar fascia that leads to flexion deformities of the fingers. Thought to be due to fibroblastic proliferation and disorderly collagen deposition. It is relatively common (33% of patients).
- **Gynecomastia.** Benign proliferation of glandular tissue of male breasts presenting with a rubbery or firm mass extending concentrically from the nipples. This is due to increased estradiol and can occur in up to 66% of patients.
- **Hypogonadism.** Manifested as impotence, infertility, loss of sexual drive, and testicular atrophy due to primary gonadal injury or suppression of hypothalamic or pituitary function.
- **Liver size.** Can be enlarged, normal, or shrunken.
- **Splenomegaly (increase in size of the spleen).** Due to congestion of the red pulp as a result of portal hypertension.
- **Ascites.** Accumulation of fluid in the peritoneal cavity giving rise to flank dullness (needs about 1500 mL to detect flank dullness). It may be associated with hydrocele and penile flomation (swelling of the penile shaft) in men.
- **Caput medusa.** In portal hypertension, the umbilical vein may open. Blood from the portal venous system may be shunted through the periumbilical veins into the umbilical vein and ultimately to the abdominal wall veins, manifesting as caput medusa.
- **Cruveilhier-Baumgarten murmur.** Venous hum heard in epigastric region (on examination by stethoscope) due to collateral connections between portal system and the remnant of the umbilical vein in portal hypertension.
- **Fetor hepaticus.** Musty odor in breath due to increased dimethyl sulfide.
- **Jaundice.** Yellow discolouring of the skin, eye, and mucus membranes due to increased bilirubin (at least 2–3 mg/dL or 30 mmol/L). Urine may also appear dark.
- **Asterixis.** Bilateral asynchronous flapping of outstretched, dorsiflexed hands seen in patients with hepatic encephalopathy.
- **Other.** Weakness, fatigue, anorexia, weight loss.

COMPLICATIONS

As the disease progresses, complications may develop. In some people, these may be the first signs of the disease.

- Bruising and bleeding due to decreased production of coagulation factors.
- Jaundice due to decreased processing of bilirubin.
- Itching (pruritus) due to bile salts products deposited in the skin.
- Hepatic encephalopathy - the liver does not clear ammonia and related nitrogenous substances from the blood, which are carried to the brain, affecting cerebral functioning: neglect of personal appearance, unresponsiveness, forgetfulness, trouble concentrating, or changes in sleep habits.
- Sensitivity to medication due to decreased metabolism of the active compounds.
- Hepatocellular carcinoma is primary liver cancer, a frequent complication of cirrhosis. It has a high mortality rate.
- Portal hypertension - blood normally carried from the intestines and spleen through the hepatic portal vein flows more slowly and the pressure increases; this leads to the following complications:
  - o Ascites - fluid leaks through the vasculature into the abdominal cavity.
  - o Esophageal varices - collateral portal blood flow through vessels in the stomach and esophagus. These blood vessels may become enlarged and are more likely to burst.
- Problems in other organs.
  - o Cirrhosis can cause immune system dysfunction, leading to infection. Signs and symptoms of infection may be aspecific are more difficult to recognize (*e.g.*, worsening encephalopathy but no fever).
  - o Fluid in the abdomen (ascites) may become infected with bacteria normally present in the intestines (spontaneous bacterial peritonitis).
  - o Hepatorenal syndrome - insufficient blood supply to the kidneys, causing acute renal failure. This complication has a very high mortality (over 50%).
  - o Hepatopulmonary syndrome - blood bypassing the normal lung circulation (shunting), leading to cyanosis and dyspnea (shortness of breath), characteristically worse on sitting up.
  - o Portopulmonary hypertension - increased blood pressure over the lungs as a consequence of portal hypertension.

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Causes

Cirrhosis has many possible causes; sometimes more than one cause is present in the same patient. In the Western World, chronic alcoholism and hepatitis C are the most common causes.

- **Alcoholic liver disease (ALD).** Alcoholic cirrhosis develops for between 10% and 20% of individuals who drink heavily for a decade or more. There is great variability in the amount of alcohol needed to cause cirrhosis (as little as 3-4 drinks a day in some men and 2-3 in some women). Alcohol seems to injure the liver by blocking the normal metabolism of protein, fats, and carbohydrates. Patients may also have concurrent alcoholic hepatitis with fever, hepatomegaly, jaundice, and anorexia. AST and ALT are both elevated but less than 300 IU/L with a AST:ALT ratio > 2.0, a value rarely seen in other liver diseases. Liver biopsy may show hepatocyte necrosis, Mallory bodies, neutrophilic infiltration with perivenular inflammation.
- **Chronic hepatitis C.** Infection with this virus causes inflammation of and low grade damage to the liver that over several decades can lead to cirrhosis. Can be diagnosed with serologic assays that detect hepatitis C antibody or viral RNA. The enzyme immunoassay, EIA-2, is the most commonly used screening test in the US.
- **Chronic hepatitis B.** The hepatitis B virus is probably the most common cause of cirrhosis worldwide, especially South-East Asia, but it is less common in the United States and the Western world. Hepatitis B causes liver inflammation and injury that over several decades can lead to cirrhosis. Hepatitis D is dependent on the presence of hepatitis B, but accelerates cirrhosis in co-infection. Chronic hepatitis B can be diagnosed with detection of HBsAG > 6 months after initial infection. HBeAG and HBV DNA are determined to assess whether patient will need antiviral therapy.
- **Non-alcoholic steatohepatitis (NASH).** In NASH, fat builds up in the liver and eventually causes scar tissue. This type of hepatitis appears to be associated with diabetes, protein malnutrition, obesity, coronary artery disease, and treatment with corticosteroid medications. This disorder is similar to that of alcoholic liver disease but patient does not have an alcohol history. Biopsy is needed for diagnosis.
- **Primary biliary cirrhosis.** May be asymptomatic or complain of fatigue, pruritus, and non-jaundice skin hyperpigmentation with hepatomegaly. There is prominent alkaline phosphatase elevation as well as elevations in

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cholesterol and bilirubin. Gold standard diagnosis is antimitochondrial antibodies with liver biopsy as confirmation if showing florid bile duct lesions. It is more common in women.

- **Primary sclerosing cholangitis.** PSC is a progressive cholestatic disorder presenting with pruritus, steatorrhoea, fat soluble vitamin deficiencies, and metabolic bone disease. There is a strong association with inflammatory bowel disease (IBD), especially ulcerative colitis. Diagnosis is best with contrast cholangiography showing diffuse, multifocal strictures and focal dilation of bile ducts, leading to a beaded appearance. Non-specific serum immunoglobulins may also be elevated.
- **Autoimmune hepatitis.** This disease is caused by the immunologic damage to the liver causing inflammation and eventually scarring and cirrhosis. Findings include elevations in serum globulins, especially gamma globulins. Therapy with prednisone +/- azathioprine is beneficial. Cirrhosis due to autoimmune hepatitis still has 10-year survival of 90%+. There is no specific tool to diagnose autoimmune but it can be beneficial to initiate a trial of corticosteroids.
- **Hereditary hemochromatosis.** Usually presents with family history of cirrhosis, skin hyperpigmentation, diabetes mellitus, pseudogout, and/or cardiomyopathy, all due to signs of iron overload. Labs will show fasting transferrin saturation of > 60% and ferritin > 300 ng/mL. Genetic testing may be used to identify HFE mutations. If these are present, biopsy may not need to be performed. Treatment is with phlebotomy to lower total body iron levels.
- **Wilson's disease.** Autosomal recessive disorder characterized by low serum ceruloplasmin and increased hepatic copper content on liver biopsy. May also have Kayser-Fleischer rings in the cornea and altered mental status.
- **Alpha 1-antitrypsin deficiency (AAT).** Autosomal recessive disorder. Patients may also have COPD, especially if they have a history of tobacco smoking. Serum AAT levels are low. Recombinant AAT is used to prevent lung disease due to AAT deficiency.
- **Cardiac cirrhosis.** Due to chronic right sided heart failure which leads to liver congestion.
- Galactosemia
- Glycogen storage disease type IV
- Cystic fibrosis

- Hepatotoxic drugs or toxins
- Certain parasitic infections (such as schistosomiasis)

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Diagnosis

The gold standard for diagnosis of cirrhosis is a liver biopsy, through a percutaneous, transjugular, laparoscopic, or fine-needle approach. Histologically cirrhosis can be classified as micronodular, macronodular, or mixed, but this classification has been abandoned since it is non-specific to the aetiology, it may change as the disease progresses, and serological markers are much more specific. However, a biopsy is not necessary if the clinical, laboratory, and radiologic data suggests cirrhosis. Furthermore, there is a small but significant risk to liver biopsy, and cirrhosis itself predisposes for complications due to liver biopsy.

Lab Findings

The following findings are typical in cirrhosis:

- **Aminotransferases** - AST and ALT are moderately elevated, with AST > ALT. However, normal aminotransferases do not preclude cirrhosis.
- **Alkaline phosphatase** - usually slightly elevated.
- **GGT – correlates with AP levels.** Typically much higher in chronic liver disease from alcohol.
- **Bilirubin** - may elevate as cirrhosis progresses.
- **Albumin** - levels fall as the synthetic function of the liver declines with worsening cirrhosis since albumin is exclusively synthesized in the liver
- **Prothrombin time** - increases since the liver synthesizes clotting factors.
- **Globulins** - increased due to shunting of bacterial antigens away from the liver to lymphoid tissue.
- **Serum sodium** - hyponatremia due to inability to excrete free water resulting from high levels of ADH and aldosterone.
- **Thrombocytopenia** - due to both congestive splenomegaly as well as decreased thrombopoietin from the liver. However, this rarely results in platelet count < 50,000/mL.
- **Leukopenia and neutropenia** - due to splenomegaly with splenic margination.
- **Coagulation defects** - the liver produces most of the coagulation factors and thus coagulopathy correlates with worsening liver disease.

There is now a validated and patented combination of 6 of these markers as non-invasive biomarker of fibrosis (and so of cirrhosis) : FibroTest.

Other laboratory studies performed in newly diagnosed cirrhosis may include:

- Serology for hepatitis viruses, autoantibodies (ANA, anti-smooth muscle, anti-mitochondria, anti-LKM)
- Ferritin and transferrin saturation (markers of iron overload), copper and ceruloplasmin (markers of copper overload)
- Immunoglobulin levels (IgG, IgM, IgA) - these are non-specific but may assist in distinguishing various causes
- Cholesterol and glucose
- Alpha 1-antitrypsin

Imaging

Ultrasound is routinely used in the evaluation of cirrhosis, where it may show a small and nodular liver in advanced cirrhosis along with increased echogenicity with irregular appearing areas. Ultrasound may also screen for hepatocellular carcinoma, portal hypertension and Budd-Chiari syndrome (by assessing flow in the hepatic vein).

A new type of device, the FibroScan (transient elastography), uses elastic waves to determine liver stiffness which theoretically can be converted into a liver score based on the METAVIR scale. The FibroScan produces an ultrasound image of the liver (from 20–80 mm) along with a pressure reading (in kPa.) The test is much faster than a biopsy (usually last 2.5–5 minutes) and is completely painless. It shows reasonable correlation with the severity of cirrhosis.

Other tests performed in particular circumstances include abdominal CT and liver/bile duct MRI (MRCP).

Endoscopy

Gastroscopy (endoscopic examination of the esophagus, stomach and duodenum) is performed in patients with established cirrhosis to exclude the possibility of esophageal varices. If these are found, prophylactic local therapy may be applied (sclerotherapy or banding) and beta blocker treatment may be commenced.

Rarely diseases of the bile ducts, such as primary sclerosing cholangitis, can be causes of cirrhosis. Imaging of the bile ducts, such as ERCP or MRCP (MRI of biliary tract and pancreas) can show abnormalities in these patients, and may aid in the diagnosis.

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Pathology

Macroscopically, the liver may be initially enlarged, but with progression of the disease, it becomes smaller. Its surface is irregular, the consistency is firm and the colour is often yellow (if associates steatosis). Depending on the size of the nodules there are three macroscopic types: micronodular, macronodular and mixed cirrhosis. In micronodular form (Laennec's cirrhosis or portal cirrhosis) regenerating nodules are under 3 mm. In macronodular cirrhosis (post-necrotic cirrhosis), the nodules are larger than 3 mm. The mixed cirrhosis consists in a variety of nodules with different sizes.

However, cirrhosis is defined by its pathological features on microscopy: (1) the presence of regenerating nodules of hepatocytes and (2) the presence of fibrosis, or the deposition of connective tissue between these nodules. The pattern of fibrosis seen can depend upon the underlying insult that led to cirrhosis; fibrosis can also proliferate even if the underlying process that caused it has resolved or ceased. The fibrosis in cirrhosis can lead to destruction of other normal tissues in the liver: including the sinusoids, the space of Disse, and other vascular structures, which leads to altered resistance to blood flow in the liver and portal hypertension.

As cirrhosis can be caused by many different entities which injure the liver in different ways, different cause-specific patterns of cirrhosis, and other cause-specific abnormalities can be seen in cirrhosis. For example; in chronic hepatitis B, there is infiltration of the liver parenchyma with lymphocytes; in cardiac cirrhosis there are erythrocytes and a greater amount of fibrosis in the tissue surrounding the hepatic veins; in primary biliary cirrhosis, there is fibrosis around the bile duct, the presence of granulomas and pooling of bile; and in alcoholic cirrhosis, there is infiltration of the liver with neutrophils.

Grading

The severity of cirrhosis is commonly classified with the Child-Pugh score. This score uses bilirubin, albumin, INR, presence and severity of ascites and encephalopathy to classify patients in class A, B or C; class A has a favourable prognosis, while class C is at high risk of death. It was devised in 1964 by Child and Turcotte and modified in 1973 by Pugh.

More modern scores, used in the allocation of liver transplants but also in other contexts, are the Model for End-Stage Liver Disease (MELD) score and its pediatric counterpart, the Pediatric End-Stage Liver Disease (PELD) score.

The hepatic venous pressure gradient, *i.e.*, the difference in venous pressure between afferent and efferent blood to the liver, also determines severity of cirrhosis, although hard to measure. A value of 16 mm or more means a greatly increased risk of dying.

PATHOPHYSIOLOGY

The liver plays a vital role in synthesis of proteins (*e.g.*, albumin, clotting factors and complement), detoxification and storage (*e.g.*, vitamin A). In addition, it participates in the metabolism of lipids and carbohydrates.

Cirrhosis is often preceded by hepatitis and fatty liver (steatosis), independent of the cause. If the cause is removed at this stage, the changes are still fully reversible.

The pathological hallmark of cirrhosis is the development of scar tissue that replaces normal parenchyma, blocking the portal flow of blood through the organ and disturbing normal function. Recent research shows the pivotal role of stellate cell, a cell type that normally stores vitamin A, in the development of cirrhosis. Damage to the hepatic parenchyma leads to activation of the stellate cell, which becomes contractile (called myofibroblast) and obstructs blood flow in the circulation. In addition, it secretes TGF-â$_1$, which leads to a fibrotic response and proliferation of connective tissue. Furthermore, it disturbs the balance between matrix metalloproteinases and the naturally occurring inhibitors (TIMP 1 and 2), leading to matrix breakdown and replacement by connective tissue-secreted matrix.

The fibrous tissue bands (septa) separate hepatocyte nodules, which eventually replace the entire liver architecture, leading to decreased blood flow throughout. The spleen becomes congested, which leads to hypersplenism and increased sequestration of platelets. Portal hypertension is responsible for most severe complications of cirrhosis.

TREATMENT

Generally, liver damage from cirrhosis cannot be reversed, but treatment could stop or delay further progression and reduce complications. A healthy diet is encouraged, as cirrhosis may be an energy-consuming process. Close follow-up is often necessary. Antibiotics will be prescribed for infections, and various medications can help with itching. Laxatives, such as lactulose, decrease risk of constipation; their role in preventing encephalopathy is limited.

Treating underlying causes

Alcoholic cirrhosis caused by alcohol abuse is treated by abstaining from alcohol. Treatment for hepatitis-related cirrhosis involves medications used to treat the different types of hepatitis, such as interferon for viral hepatitis and corticosteroids for autoimmune hepatitis. Cirrhosis caused by Wilson's disease, in which copper builds up in organs, is treated with chelation therapy (*e.g.*, penicillamine) to remove the copper.

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Preventing further liver damage

Regardless of underlying cause of cirrhosis, alcohol and paracetamol, as well as other potentially damaging substances, are discouraged. Vaccination of susceptible patients should be considered for Hepatitis A and Hepatitis B.

Preventing complications

Ascites

Salt restriction is often necessary, as cirrhosis leads to accumulation of salt (sodium retention). Diuretics may be necessary to suppress ascites.

Esophageal variceal bleeding

For portal hypertension, propranolol is a commonly used agent to lower blood pressure over the portal system. In severe complications from portal hypertension, transjugular intrahepatic portosystemic shunting is occasionally indicated to relieve pressure on the portal vein. As this can worsen encephalopathy, it is reserved for those at low risk of encephalopathy, and is generally regarded only as a bridge to liver transplantation or as a palliative measure.

Hepatic encephalopathy

High-protein food increases the nitrogen balance, and would theoretically increase encephalopathy; in the past, this was therefore eliminated as much as possible from the diet. Recent studies show that this assumption was incorrect, and high-protein foods are even encouraged to maintain adequate nutrition.

Hepatorenal syndrome

The hepatorenal syndrome is defined as a urine sodium less than 10 mmol/L and a serum creatinine > 1.5 mg/dl (or 24 hour creatinine clearance less than 40 ml/min) after a trial of volume expansion without diuretics.

Spontaneous bacterial peritonitis

Cirrhotic patients with ascites are at risk of spontaneous bacterial peritonitis.

TRANSPLANTATION

If complications cannot be controlled or when the liver ceases functioning, liver transplantation is necessary. Survival from liver transplantation has been improving over the 1990s, and the five-year survival rate is now around 80%, depending largely on the severity of disease and other medical problems in the recipient. In the United States, the MELD score (online calculator) is used to prioritize patients for transplantation. Transplantation necessitates the use of immune suppressants (cyclosporine or tacrolimus).

Decompensated Cirrhosis

In patients with previously stable cirrhosis, decompensation may occur due to various causes, such as constipation, infection (of any source), increased

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alcohol intake, medication, bleeding from esophageal varices or dehydration. It may take the form of any of the complications of cirrhosis listed above.

Patients with decompensated cirrhosis generally require admission to hospital, with close monitoring of the fluid balance, mental status, and emphasis on adequate nutrition and medical treatment — often with diuretics, antibiotics, laxatives and/or enemas, thiamine and occasionally steroids, acetylcysteine and pentoxifylline. Administration of saline is generally avoided as it would add to the already high total body sodium content that typically occurs in cirrhosis.

Epidemiology

Cirrhosis and chronic liver disease were the 10th leading cause of death for men and the 12th for women in the United States in 2001, killing about 27,000 people each year. Also, the cost of cirrhosis in terms of human suffering, hospital costs, and lost productivity is high.

Established cirrhosis has a 10-year mortality of 34-66%, largely dependent on the cause of the cirrhosis; alcoholic cirrhosis has a worse prognosis than primary biliary cirrhosis and cirrhosis due to hepatitis. The risk of death due to all causes is increased twelvefold; if one excludes the direct consequences of the liver disease, there is still a fivefold increased risk of death in all disease categories.

Little is known on modulators of cirrhosis risk, apart from other diseases that cause liver injury (such as the combination of alcoholic liver disease and chronic viral hepatitis, which may act synergistically in leading to cirrhosis). Studies have recently suggested that coffee consumption may protect against cirrhosis, especially alcoholic cirrhosis.

Cirrhosis Diet

Before following a Cirrhosis Diet, one needs to have a sound knowledge about what is Cirrhosis? Cirrhosis is the result of chronic liver disease when the liver tissue is damaged and fibrous scar tissue and lumps occur. This disease leads to the improper functioning of liver. The main and most common cause of cirrhosis is alcoholism and hepatitis C. So while following Cirrhosis diet, the first and foremost thing that one need to take care of is stopping any intake of alcohol.

Diet for Cirrhosis can be as simple and healthy as one can follow even if the person is not prone to the disease. A heart and liver-healthy diet is what one should follow when prone to Cirrhosis. A basic diet and nutrition regime for a cirrhosis patient will comprise intake of foods which are low in fats and should also have ample intake of vegetables, fruits, and whole grains as they provide the adequate nutrition. A patient following a healthy diet regime when prone to Cirrhosis should also try to maintain a healthy weight.

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Foods to be eaten while prone to Cirrhosis are:

1. Carbohydrates: Cereals, dried beans, grains, breads, pasta, legumes, peas and rice. These foods provide blood glucose or sugar.
2. Protein: soy, dairy products and fruits, vegetables. Protein helps to repair the body tissues and maintain the level of blood.
3. Foods with minerals like zinc, calcium, and magnesium, anti-oxidants should also be taken.
4. Herbs: Celery Seed, Dandelion, Green Tea, Licorice root, Milk thistle, Turmeric

Some special care and basic dietary habits that a Cirrhosis patient should follow is as follows:

1. Limiting the intake of salt, foods rich in iron and fats
2. Eating foods which are high in calories, carbohydrates and rich in protein
3. Last but not the least, eliminating intake of alcohol and shell fish
4. Intake of soft foods which are well cooked and appetizing

STUDENT ACTIVITY

1. Discuss the characteristics of Jaundice.

2. Point out signs and symptoms of Cirrhosis.

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5.5 CHOLILITHIASIS

In medicine, gallstones (choleliths) are crystalline bodies formed within the body by accretion or concretion of normal or abnormal bile components.

Gallstones can occur anywhere within the biliary tree, including the gallbladder and the common bile duct. Obstruction of the common bile duct is choledocholithiasis; obstruction of the biliary tree can cause jaundice; obstruction of the outlet of the pancreatic exocrine system can cause pancreatitis. Cholelithiasis is the presence of stones in the gallbladder or bile ducts: chole- means "bile", lithia means "stone", and "sis" means "process".

Characteristics

Size

A gallstone's size varies and may be as small as a sand grain or as large as a golf ball. The gallbladder may develop a single, often large stone or many smaller ones. They may occur in any part of the biliary system.

Content

Gallstones have different appearance, depending on their contents. On the basis of their contents, gallstones can be subdivided into the two following types:

- Cholesterol stones are usually green, but are sometimes white or yellow in colour. They are made primarily of cholesterol, the proportion required for classification as a cholesterol stone being either 70% (Japanese classification system) or 80% (US system)
- Pigment stones are small, dark stones made of bilirubin and calcium salts that are found in bile. They contain less than 20% of cholesterol. Risk factors for pigment stones include hemolytic anemia, cirrhosis, biliary tract infections, and hereditary blood cell disorders, such as sickle cell anemia and spherocytosis.

The proportions of these different types of stone found varies between samples, and is thought to be affected by the age and ethnic or regional origin of the patients.

Mixed stones

All stones are of mixed content to some extent. Those classified as mixed, however, contain between 30% and 70% of cholesterol. In most cases the other majority constituent is calcium salts such as Calcium carbonate, palmitate fosfate and/or bilirubinate. Because of their calcium content, they can often be visualized radiographically.

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CAUSES

Researchers believe that gallstones may be caused by a combination of factors, including inherited body chemistry, body weight, gallbladder motility (movement), and perhaps diet.

Pigment gallstones

People with erythropoietic protoporphyria (EPP) are at increased risk to develop gallstones.

Conditions causing hemolytic anemia can cause pigment gallstones.

Cholesterol gallstones

Cholesterol gallstones develop when bile contains too much cholesterol and not enough bile salts. Besides a high concentration of cholesterol, two other factors seem to be important in causing gallstones. The first is how often and how well the gallbladder contracts; incomplete and infrequent emptying of the gallbladder may cause the bile to become overconcentrated and contribute to gallstone formation. The second factor is the presence of proteins in the liver and bile that either promote or inhibit cholesterol crystallization into gallstones.

In addition, increased levels of the hormone estrogen as a result of pregnancy, hormone therapy, or the use of combined (estrogen-containing) forms of hormonal contraception, may increase cholesterol levels in bile and also decrease gallbladder movement, resulting in gallstone formation.

No clear relationship has been proven between diet and gallstone formation. However, low-fibre, high-cholesterol diets, and diets high in starchy foods have been suggested as contributing to gallstone formation. Other nutritional factors that may increase risk of gallstones include rapid weight loss, constipation, eating fewer meals per day, eating less fish, and low intakes of the nutrients folate, magnesium, calcium, and vitamin C. On the other hand, wine and whole grain bread may decrease the risk of gallstones.

The common mnemonic for gallstone risk factors refer to the "four F's": fat (*i.e.*, overweight), forty (an age near or above 40), female, and fertile (pre-menopausal); a fifth F, fair is sometimes added to indicate that the condition is more prevalent in Caucasians. The absence of these risk factors does not, however, preclude the formation of gallstones.

Interestingly, a lack of melatonin could significantly contribute to gallbladder stones, as melatonin both inhibits cholesterol secretion from the gallbladder, enhances the conversion of cholesterol to bile, and is an antioxidant, capable of reducing oxidative stress to the gallbladder.

SYMPTOMS

Gallstones usually remain asymptomatic initially. They start developing symptoms once the stones reach a certain size (>8 mm). A main symptom of gallstones is commonly referred to as a gallstone "attack", also known as biliary colic, in which a person will experience intense pain in the upper abdominal region that steadily increases for approximately thirty minutes to several hours. A patient may also experience pain in the back, ordinarily between the shoulder blades, or pain under the right shoulder. In some cases, the pain develops in the lower region of the abdomen, nearer to the pelvis, but this is less common. Nausea and vomiting may occur. Patients characteristically exhibit a positive Murphy's sign: the patient is instructed to breathe in while the gall bladder is deeply palpated. If the gallbladder is inflamed, the patient will abruptly stop inhaling due to the pain, a positive Murphy's sign.

These attacks are sharp and intensely painful, similar to that of a kidney stone attack. Often, attacks occur after a particularly fatty meal and almost always happen at night. Other symptoms include abdominal bloating, intolerance of fatty foods, belching, gas, and indigestion. If the above symptoms coincide with chills, lowgrade fever, yellowing of the skin or eyes, and/or clay-coloured stool, a doctor should be consulted immediately.

Some people who have gallstones are asymptomatic and do not feel any pain or discomfort. These gallstones are called "silent stones" and do not affect the gallbladder or other internal organs. They do not need treatment.

TREATMENT

Medical Options

Cholesterol gallstones can sometimes be dissolved by oral ursodeoxycholic acid, but it may be required that the patient takes this medication for up to two years. Gallstones may recur however, once the drug is stopped. Obstruction of the common bile duct with gallstones can sometimes be relieved by endoscopic retrograde sphincterotomy (ERS) following endoscopic retrograde cholangiopancreatography (ERCP). Gallstones can be broken up using a procedure called lithotripsy (Extracorporeal Shock Wave Lithotripsy), which is a method of concentrating ultrasonic shock waves onto the stones to break them into tiny pieces. They are then passed safely in the feces. However, this form of treatment is only suitable when there are a small number of gallstones.

Surgical Options

Cholecystectomy (gallbladder removal) has a 99% chance of eliminating the recurrence of cholelithiasis. Only symptomatic patients must be indicated to

surgery. The lack of a gall bladder does not seem to have any negative consequences in many people. However, there is a significant proportion of the population-between 5–40%—who develop a condition called postcholecystectomy syndrome which may cause gastrointestinal distress and persistent pain in the upper right abdomen. In addition, as many as 20% of patients develop chronic diarrhoea.

There are two surgical options for cholecystectomy :

- Open cholecystectomy: This procedure is performed via an incision into the abdomen (laparotomy) below the right lower ribs. Recovery typically consists of 3–5 days of hospitalization, with a return to normal diet a week after release and normal activity several weeks after release.
- Laparoscopic cholecystectomy: This procedure, introduced in the 1980s, is performed via three to four small puncture holes for a camera and instruments. Post-operative care typically includes a same-day release or a one night hospital stay, followed by a few days of home rest and pain medication. Laparoscopic cholecystectomy patients can generally resume normal diet and light activity a week after release, with some decreased energy level and minor residual pain continuing for a month or two. Studies have shown that this procedure is as effective as the more invasive open cholecystectomy, provided the stones are accurately located by cholangiogram prior to the procedure so that they can all be removed. The procedure also has the benefit of reducing operative complications such as bowel perforation and vascular injury.

Value

Gallstones are a valuable by-product of meat processing, fetching up to US$32–per–gram in their use as a purported antipyretic and antidote in the folk remedies of some cultures, particularly in China. The finest gallstones tend to be sourced from old dairy cows, which are called Niu-Huang (yellow thing of oxen) in Chinese. Those obtained from dogs, called Gou-Bao (treasure of dogs) in Chinese, are also used today. Much as in the manner of diamond mines, slaughterhouses carefully scrutinize offal department workers for gallstone theft.

5.6 PANCREATITIS

Pancreatitis is inflammation of the pancreas that can occur in two very different forms. Acute pancreatitis is sudden while chronic pancreatitis "is characterized by recurring or persistent abdominal pain with or without steatorrhoea or diabetes mellitus."

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Causes

Excessive alcohol use is often cited as the most common cause of acute pancreatitis, yet gallstones are actually the most common cause. Less common causes include hypertriglyceridemia (but not hypercholesterolemia) and only when triglyceride values exceed 1500 mg/dl (16 mmol/L), hypercalcemia, viral infection (*e.g.*, mumps), trauma (to the abdomen or elsewhere in the body) including post-ERCP (*i.e.*, Endoscopic Retrograde Cholangiopancreatography), vasculitis (*i.e.*, inflammation of the small blood vessels within the pancreas), and autoimmune pancreatitis. Pregnancy can also cause pancreatitis, but in some cases the development of pancreatitis is probably just a reflection of the hypertriglyceridemia which often occurs in pregnant women. Pancreas divisum, a common congenital malformation of the pancreas may underlie some cases of recurrent pancreatitis. Pancreatitis is less common in pediatric population.

The more mundane, but far more common causes of pancreatitis, as mentioned above, must always be considered first. However, the known porphyrinogenicity of many drugs, hormones, alcohol, chemicals and the association of porphyrias with autoimmune disorders and gallstones do not exclude the diagnosis of heme disorders when these explanations are used. A primary medical disorder, including an underlying undetected inborn error in metabolism, supersedes a secondary medical complication or explanation. As mentioned above, pancreatitis is less common in children but if seen, abuse or abdominal trauma should be suspected.

Autoimmune disorders, lipid disorders, gallstones, drug reactions and pancreatitis itself are not primary medical disorders.

It is worth noting that pancreatic cancer is seldom the cause of pancreatitis.

People with diabetes should promptly seek medical care if they experience unexplained severe abdominal pain with or without nausea and vomiting.

Porphyrias

Acute hepatic porphyrias, including acute intermittent porphyria, hereditary coproporphyria and variegate porphyria, are genetic disorders that can be linked to both acute and chronic pancreatitis. Acute pancreatitis has also occurred with erythropoietic protoporphyria.

Conditions that can lead to gut dysmotility predispose patients to pancreatitis. This includes the inherited neurovisceral porphyrias and related metabolic disorders. Alcohol, hormones and many drugs including statins are known porphyrinogenic agents. Physicians should be on alert concerning underlying porphyrias in patients presenting with pancreatitis and should investigate and eliminate any drugs that may be activating the disorders.

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Still, notwithstanding their potential role in pancreatitis, the porphyrias (as a group or individually) are considered to be rare disorders. However, since there are no systematic studies to determine the actual incidence of latent dominantly-inherited porphyrias in the world population, there is DNA or enzyme evidence of high rates of latency of classic textbook symptoms in families where porphyrias have been detected and the technology is not developed to detect all latent porphyrias, the diagnosis of underlying inborn errors of metabolism impacting heme should not be routinely eliminated in pancreatitis.

Medications

Many medications have been reported to cause pancreatitis. Some of the more common ones include the AIDS drugs DDI and pentamidine, diuretics such as furosemide and hydrochlorothiazide, the anticonvulsants divalproex sodium and valproic acid, the chemotherapeutic agents L-asparaginase and azathioprine, and estrogen. Just as is the case with pregnancy-associated pancreatitis, estrogen may lead to the disorder because of its effect of raising blood triglyceride levels. Pancreatitis due to statins first started appearing in the medical literature as early as 1990. All statins currently in use reportedly can cause pancreatitis, a not surprising observation when one considers that all statins are reductase inhibitors and can be expected to have similar side effect profiles.

Occasionally one statin will have a somewhat greater tendency for a side effect than another, like Baycol, removed from the market because of excess rhabdomyolysis deaths, but all statins cause this condition. The total rhabdomyolysis deaths seen today far exceed the 100 or so attributed to Baycol.

Genetics

Hereditary pancreatitis may be due to a genetic abnormality that renders trypsinogen active within the pancreas, which in turn leads to digestion of the pancreas from the inside.

Pancreatic diseases are notoriously complex disorders resulting from the interaction of multiple genetic, environmental and metabolic factors.

Three candidates for genetic testing are currently under investigation:

- Trypsinogen mutations (Trypsin 1)
- Cystic Fibrosis Transmembrane Conductance Regulator Gene (CFTR) mutations
- SPINK1 which codes for PSTI - a specific trypsin inhibitor.

Virus infection

Viruses can cause profound inflammation in, and destruction of, the pancreas. This is true of several viruses in the coxsackievirus group.

SYMPTOMS AND SIGNS

Severe upper abdominal pain, with radiation through to the back, is the hallmark of pancreatitis. Nausea and vomiting (emesis) are prominent symptoms. Findings on the physical exam will vary according to the severity of the pancreatitis, and whether or not it is associated with significant internal bleeding. The blood pressure may be high (when pain is prominent) or low (if internal bleeding or dehydration has occurred). Typically, both the heart and respiratory rates are elevated. Abdominal tenderness is usually found but may be less severe than expected given the patient's degree of abdominal pain. Bowel sounds may be reduced as a reflection of the reflex bowel paralysis (*i.e.*, ileus) that may accompany any abdominal catastrophe.

DIAGNOSIS

The diagnostic criteria for pancreatitis are "two of the following three features: (1) abdominal pain characteristic of acute pancreatitis, (2) serum amylase and/or lipase >3 times the upper limit of normal, and (3) characteristic findings of acute pancreatitis on CT scan."

Laboratory Tests

Most frequently, measurement is made of amylase and/or lipase, and often one, or both, are elevated in cases of pancreatitis. Two practice guidelines state:

It is usually not necessary to measure both serum amylase and lipase. Serum lipase may be preferable because it remains normal in some nonpancreatic conditions that increase serum amylase including macroamylasemia, parotitis, and some carcinomas. In general, serum lipase is thought to be more sensitive and specific than serum amylase in the diagnosis of acute pancreatitis".

Although amylase is widely available and provides acceptable accuracy of diagnosis, where lipase is available it is preferred for the diagnosis of acute pancreatitis (recommendation grade A)".

Most, but not all individual studies support the superiority of the lipase. In one large study, there were no patients with pancreatitis who had an elevated amylase with a normal lipase. Another study found that the amylase could add diagnostic value to the lipase, but only if the results of the two tests were combined with a discriminant function equation.

Conditions other than pancreatitis may lead to rises in these enzymes and, further, that those conditions may also cause pain that resembles that of pancreatitis (*e.g.*, cholecystitis, perforated ulcer, bowel infarction (*i.e.*, dead bowel as a result of poor blood supply), and even diabetic ketoacidosis.

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Imaging

Although ultrasound imaging and CT scanning of the abdomen can be used to confirm the diagnosis of pancreatitis, neither is usually necessary as a primary diagnostic modality. In addition, CT contrast may exacerbate pancreatitis, although this is disputed.

Prognosis

There are several scoring systems used to help predict the severity of an attack of pancreatitis. The Apache II has the advantage of being available at the time of admission as opposed to 48 hours later for the Glasgow criteria and Ranson criteria. However, the Glasgow criteria and Ranson criteria are easier to use.

APACHE II

Ranson criteria

At admission:

1. age in years > 55 years
2. white blood cell count > 16000 /mcL
3. blood glucose > 11 mmol/L (>200 mg/dL)
4. serum AST > 250 IU/L
5. serum LDH > 350 IU/L

After 48 hours:

1. Haematocrit fall > 11.3444%
2. increase in BUN by 1.8 or more mmol/L (5 or more mg/dL) after IV fluid hydration
3. hypocalcemia (serum calcium < 2.0 mmol/L (≤8.0 mg/dL))
4. hypoxemia (PO2 < 60 mmHg)
5. Base deficit > 4 Meq/L
6. Estimated fluid sequestration > 6 L

The criteria for point assignment is that a certain breakpoint be met at anytime during that 48 hour period, so that in some situations it can be calculated shortly after admission. It is applicable to both biliary and alcoholic pancreatitis.

Interpretation

- If the score e>3, severe pancreatitis likely.
- If the score < 3, severe pancreatitis is unlikely

Or

- Score 0 to 2: 2% mortality
- Score 3 to 4: 15% mortality
- Score 5 to 6: 40% mortality
- Score 7 to 8: 100% mortality

Glasgow Criteria

Glasgow's criteria: The original system used 9 data elements. This was subsequently modified to 8 data elements, with removal of assessment for transaminase levels (either AST (SGOT) or ALT (SGPT) greater than 100 U/L).

On Admission

1. Age >55 yrs
2. WBC Count >15 x10^9/L
3. Blood Glucose >200 mg/dL (No Diabetic History)
4. Serum Urea >16 mmol/L (No response to IV fluids)
5. Arterial Oxygen Saturation <76 mmHg

Within 48 hours

1. Serum Calcium <2 mmol/L
2. Serum Albumin <34 g/L
3. LDH >219 units/L
4. AST/ALT >96 units/L

COMPLICATIONS

Acute (early) complications of pancreatitis include

- shock,
- hypocalcemia (low blood calcium),
- high blood glucose,
- dehydration, and kidney failure (resulting from inadequate blood volume which, in turn, may result from a combination of fluid loss from vomiting, internal bleeding, or oozing of fluid from the circulation into the abdominal cavity in response to the pancreas inflammation, a phenomenon known as Third Spacing).
- Respiratory complications are frequent and are major contributors to the mortality of pancreatitis. Some degree of pleural effusion is almost ubiquitous in pancreatitis. Some or all of the lungs may collapse (atelectasis) as a result of the shallow breathing which occurs because of

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the abdominal pain. Pneumonitis may occur as a result of pancreatic enzymes directly damaging the lung, or simply as a final common pathway response to any major insult to the body (*i.e.*, ARDS or Acute Respiratory Distress Syndrome).

- Likewise, SIRS (Systemic inflammatory response syndrome) may ensue.
- Infection of the inflamed pancreatic bed can occur at any time during the course of the disease. In fact, in cases of severe hemorrhagic pancreatitis, antibiotics should be given prophylactically.

Late Complications

Late complications include recurrent pancreatitis and the development of pancreatic pseudocysts. A pancreatic pseudocyst is essentially a collection of pancreatic secretions which has been walled off by scar and inflammatory tissue. Pseudocysts may cause pain, may become infected, may rupture and hemorrhage, may press on and block structures such as the bile duct, thereby leading to jaundice, and may even migrate around the abdomen.

TREATMENT

The treatment of pancreatitis will, of course, depend on the severity of the pancreatitis itself. Still, general principles apply and include:

1. Provision of pain relief. In the past this was done preferentially with meperidine (Demerol), but it is now not thought to be superior to any narcotic analgesic. Indeed, given meperidine's generally poor analgesic charactersitics and its high potential for toxicity, it should not be used for the treatment of the pain of pancreatitis. The preferred analgesic is morphine for acute pancreatitis.
2. Provision of adequate replacement fluids and salts (intravenously).
3. Limitation of oral intake (with dietary fat restriction the most important point). NG tube feeding is the preferred method to avoid pancreatic stimulation and possible infection complications caused by bowel flora.
4. Monitoring and assessment for, and treatment of, the various complications listed above.
5. ERCP if gallstone pancreatitis

When necrotizing pancreatitis ensues and the patient shows signs of infection, it is imperative to start antibiotics such as Imipenem due to the high penetration of the drug in the pancreas. Floroquinolone + metronidazole is another treatment option.

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STUDENT ACTIVITY

1. Discuss the characteristics of Cholelithiasis.

2. Outline the basic causes of Pancreatitis.

5.7 SUMMARY

- Jaundice, also known as icterus (attributive adjective: icteric), is a yellowish discolouration of the skin, the conjunctival membranes over the sclerae (whites of the eyes), and other mucous membranes caused by hyperbilirubinemia (increased levels of bilirubin in the blood).
- Hepatitis (plural hepatitides) implies injury to the liver characterized by the presence of inflammatory cells in the tissue of the organ. The name is from ancient Greek hepar, the root being hepat- meaning liver, and suffix -itis, meaning "inflammation".
- Cirrhosis is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrous scar tissue as well as regenerative nodules (lumps that occur as a result of a process in which damaged tissue is regenerated), leading to progressive loss of liver function.
- Cirrhosis has many possible causes; sometimes more than one cause is present in the same patient. In the Western World, chronic alcoholism and hepatitis C are the most common causes.

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- In medicine, gallstones (cholelithsl are crystalline bodies formed within the body by accretion or concretion of normal or abnormal bile components.
- Pancreatitis is inflammation of the pancreas that can occur in two very different forms. Acute pancreatitis is sudden while chronic pancreatitis "is characterized by recurring or persistent abdominal pain with or without steatorrhea or diabetes mellitus."

5.8 GLOSSARY

- **Jaundice:** also known as icterus is a yellowish discolouration of the skin, the conjunctival membranes over the sclerae (whites of the eyes), and other mucous membranes caused by increased levels of bilirubin in the blood.
- **Hepatitis:** implies injury to the liver characterized by the presence of inflammatory cells in the tissue of the organ.
- **Cirrhosis:** it is a consequence of chronic liver disease characterized by replacement of liver tissue by fibrous scar tissue as well as regenerative nodules.
- **Gallstones (Cholelithsl:** they are crystalline bodies formed within the body by accretion or concretion of normal or abnormal bile components.
- **Pancreatitis:** it is inflammation of the pancreas that can occur in two very different forms. Acute pancreatitis is sudden while chronic pancreatitis "is characterized by recurring or persistent abdominal pain with or without steatorrhoea or diabetes mellitus."

5.9 REVIEW QUESTIONS

1. What are the signs and symptoms of Jaundice?
2. How is Jaundice treated?
3. Discuss the diagnosis of Hapatitis.
4. What are the primary causes of Cirrhosis?
5. How is Cirrhosis treated?
6. Outline the diet plan for a patient of Cirrhosis.
7. What are the complications of Pancreatitis?

5.10 FURTHER READINGS

- Wilson Fisher, *Principles of Nutrition*, Wilen Easten Pvt. Ltd.
- *Economics of Health and Nutrition* by Rani, Gopal Publications.