formation on nutrition books, visit our website www.ejaypee.com, for detailed information on nutrition books, visit our website www.ejaypee.com

Basics of Clinical Nutrition

YK Joshi
Jaya Benjamin

3rd Edition

JAYPEE

Contents

1. Food and Nutrient Requirements **1**

Puja Bhatia Kapoor, Kajal Jain

- Recommendation on Energy Requirement *1*
- Recommendation on Protein Requirement *1*
- Recommendation on Fats and Oil Requirement *1*
- Recommendation on Vitamins and Mineral Requirement *4*
- Food Groups *5*
- Planning of Balanced Diets *5*
- Meal Planning for Various Age Groups *9*
- Dietary Considerations and Requirements *9*
- Dietary Considerations *18*

2. Carbohydrates, Proteins and Fats **21**

Namrata Singh, Medha Gupta

- Carbohydrates *21*
- Proteins *29*
- Fats *35*
- Interrelated Metabolism of Carbohydrates, Fats and Proteins *46*

3. Vitamins **51**

Sabiha Khan, Nandini Saxena

Fat Soluble Vitamins

- Fat Soluble Vitamins
- Vitamin A *53*
- Vitamin D *58*
- Vitamin E *60*
- Vitamin K *61*

Water Soluble Vitamins

- Thiamin (B_1) *63*
- Riboflavin (B_2) *65*
- Niacin *66*
- Vitamin B_6 *68*
- Biotin *69*
- Vitamin B_{12} *70*
- Folic Acid *72*
- Vitamin C *73*
- Role of Vitamin Supplements *76*

Less-Known Vitamins

- Pantothenic Acid *76*
- Choline *76*
- Anti-inflammatory Potential of Vitamins *77*

4. Minerals **79**

Monita Gahlot

- Functions of Minerals *79*
- Mineral Metabolism *80*
- Calcium *80*
- Phosphorus *82*
- Sodium *84*
- Potassium *85*
- Magnesium *87*

Trace Minerals

- Iron *89*
- Iodine *92*
- Zinc *95*
- Selenium *97*
- Copper *99*
- Manganese *101*
- Fluorine *102*
- Chromium *103*

5. Energy in Health and Disease **106**

Varsha

- Concept of Energy and Human Life *106*
- Conservation of Energy *106*
- Bioenergetics *108*
- Energy and Life Concept in Relation to Health *111*
- Energy and Life Concept in Relation to Disease *111*
- Scalar Energy (SE)—A New Paradigm *113*
- Food Fuels *118*
- Body Fuels *121*
- Recapitulation of Concept of Bioenergetics *124*
- Energy Utilization in the Body *126*
- Energy Balance *129*
- Basal Metabolism (BM) *131*
- Basal Metabolic Rate (BMR) *131*
- Energy Expenditure *137*
- Thermic Effect of Food (TEF) *141*
- Energy Recommendations *141*

6. Starvation, Stress and Disease: Consequences 145

Varsha

Metabolism Overview

- Role of Nutrition in Health and Disease *145*
- Effects of Starvation Fasting *150*
- Effects of Stress and Disease *151*
- Effects of Infection *153*
- Consequences of Malnutrition *153*

7. Assessment of Nutritional Status in the Clinical Setting 156

Rekha Ramot

- Definition of Nutritional Status *156*
- Need to Assess Nutritional Status *156*
- Nutritional Risk Screening *156*
- Methods of Assessment of Nutritional Status *157*
- Body Composition *159*
- Measurements from DXA Scans *161*
- Magnetic Resonance Tomography (MRT) and Computed Tomography (CT) *163*
- Other Methods of Nutritional Assessment *164*
- Methods of Dietary Assessments: Dietary History and Current Dietary Intake *169*

8. Assessment of Nutritional Status in the Community Setting 178

Ajeet Singh Bhadoria, Aakriti Jasrotia, Parteek Walia

- Nutritional Assessment *178*
- Optimal Nutritional Status *178*
- Nutritional Assessment Systems *179*
- Purpose of Nutritional Assessment *180*
- Methods Used in Nutritional Assessment (ABCD) *181*
- Direct Methods *181*
- Indirect Methods *188*

9. Enteral Nutrition: A Comprehensive Overview 191

Vanessa Fuchs-Tarlovsky

- History *192*
- Nutrition Components *193*
- Macronutrients in Enteral Nutrition *193*
- Micronutrients in Enteral Nutrition *196*
- Enteral Nutrition Modalites of Delivery *197*
- Physical Characteristics of Enteral Formulas *198*
- Feeding Containers *199*
- Enteral Feeding Pumps *199*

- Factors to Consider for Enteral Feeding *199*
- Complications of Enteral Nutrition *200*
- Drug and Nutrient Delivery when Administering Medications Concomitantly *201*
- Enteral Formulas *202*

10. Parenteral Nutrition: An Overview **208**

Deven Juneja, Prashant Nasa

- Indications *208*
- Composition *209*
- Timing *211*
- Administration *211*
- Initiation and Monitoring *213*

11. Enteral and Parenteral Nutrition Formulas **220**

Shefali Sharma

- Enteral Formulas *220*
- Parenteral Formulas *220*
- Nutrient Composition of Enteral Formulas *221*
- Physical Characteristics of a Formula *224*
- Classification *224*
- Enteral Feeding in Patients with Allergies *239*
- Parenteral Formulas *239*
- Home Total Parenteral Nutrition (Home TPN) *244*

12. Trace Elements in Enteral and Parenteral Nutrition **256**

Krishnan Sriram, Chitra Mahesh

- Trace Elements *256*
- Zinc *259*
- Selenium *261*
- Copper *262*
- Chromium *263*
- Manganese *264*
- Iron *265*
- Megadoses of Trace Elements not Indicated *265*
- Trace Elements in Renal and Hepatic Insufficiency *265*
- Trace Elements in Obesity *265*
- Ultratrace Elements *265*

13. Probiotics **268**

Harshita Tripathi

- Composition of Probiotics *268*
- Availability *268*

- Probiotics Food Supplements *268*
- Carriers for Probiotics *269*
- Mechanism of Action *269*
- Dosage *272*
- Factors Impacting Probiotic Function *272*
- Benefits of Probiotics *272*
- Probiotics in Gastrointestinal Diseases *272*
- Probiotics in Extraintestinal Diseases *274*
- Conditions for which Probiotics are Ineffective *277*
- Safety *277*
- New Directions in Probiotic Research *277*
- Fecal Microbiota Transplantation *277*

14. Antioxidants **280**

✍ ***Payal Bhardwaj, Raj Kumar Yadav***

- Free Radicals *280*
- Antioxidants *284*
- Dietary Antioxidants and their Mechanism of Action *285*
- Foods/Food-Derived Antioxidants *289*
- Role of Antioxidants in Health and Disease: Current Perspective *289*

15. Immunonutrition **295**

✍ ***Nida Athar, Uma Kanal***

- Nutrition and the Immune System *295*
- Role of Immunonutrition in Critical Illness *299*

16. Nutrition in Diabetes **303**

✍ ***Rekha Sharma, Swapna Chaturvedi***

- Classification *303*
- Etiology and Pathophysiology *303*
- Diagnostic Criteria *305*

Management of Diabetes Mellitus

- Medical Nutrition Therapy (MNT) in Diabetes *306*
- Exercise and Physical Activity *318*
- Medication/Insulin Therapy *319*
- Behavior Modification in Diabetes *320*

17. Nutrition and Obesity **323**

✍ ***Naval K Vikram, Divya Tripathi, Payal, Mansi Mittal***

- Obesity *323*
- Mechanisms of Weight Gain—Pathophysiology of Obesity *324*
- Control of Food Intake *327*

- Assessment of Nutritional Status in Obese *327*
- Sarcopenic Obesity *330*
- Impact of Obesity on Health *331*
- Management of Obesity *333*
- Weight Loss and Weight Maintenance *340*
- Nutritional Management of Critically Obese Patients *341*
- Directions for Future Research in Obesity *342*

18. Nutrition in Bariatric Surgery 346

Sarfaraz Jalil Baig, Deeba Siddiqui

- Bariatric Procedures and Physiological Alterations *346*
- Preoperative Management *350*
- Immediate Postoperative Nutritional Management *351*
- Follow Up *353*
- Clinical Evaluation in Follow-up: Our Protocol *357*
- History *360*
- Examination *362*
- Laboratory Tests *365*

19. Nutrition in Cardiovascular Diseases 368

Kanika Jain Bansal

- What are Cardiovascular Diseases? *368*
- Pathophysiology of Atherosclerosis and Coronary Heart Disease *369*
- Blood Lipids and Lipoproteins *369*
- Difference between Triglycerides and Cholesterol *371*
- Lipoprotein and Metabolism *372*
- Serum Biomarkers for Cardiovascular Disease *372*
- Prevention of Modifiable Risk Factors of CVD *373*
- Medical Nutritional Therapy for CVD *376*

20. Nutrition in Kidney Diseases 382

Varsha Shasthry

- Functions of Kidney *382*
- Diseases of Kidney *382*
- Epidemiology of CKD in India *384*
- Renal Replacement Therapy (Dialysis) in Kidney Diseases *384*
- Nutritional Issues in AKD and CKD *385*
- Malnutrition in Kidney Diseases *385*
- Nutritional Management in Kidney Diseases *386*
- Nutritional Recommendations in Patients with CKD *391*
- Monitoring Dietary Adequacy and Compliance *394*
- Nutritional Requirements in Patients on Dialysis *394*

- Nutritional Recommendations in Post-renal Transplant Recipient *398*
- Nutritional Requirements in Nephrotic Syndrome *400*
- Recurrent Renal Calculi (Nephrolithiasis) *401*

21. Nutrition in Liver Diseases **406**

✍ ***Puja Bhatia Kapoor***

- Chronic Liver Disease *406*
- Acute Hepatitis *409*
- Alcoholic Liver Disease *409*
- Cholestatic Liver Disease *411*
- Nonalcoholic Fatty Liver Disease *413*
- Wilson's Disease *417*

22. Nutrition in Pancreatic Diseases **421**

✍ ***Shallu Midha, Jaya Benjamin, Namrata Singh***

- Acute Pancreatitis *422*
- Classification of AP *423*
- Pathophysiology of Malnutrition in Acute Pancreatitis *425*
- Tools Used for Nutritional Screening in Acute Pancreatitis *427*
- Debatable Issues with Nutrition Support in Acute Pancreatitis *432*
- Nutritional Requirement of Patients with Acute Pancreatitis *433*
- Methods of Nutritional Support in Acute Pancreatitis *433*
- Enteral Formulas Available for Feeding *434*
- Types of Parenteral Nutrition for Patients with Acute Pancreatitis *435*
- Immunonutrition and its Requirement in Acute Pancreatitis *435*
- Nutrition Therapy in Acute Pancreatitis under Special Conditions *435*
- Chronic Pancreatitis *436*
- Methods of Nutritional Assessment in Patients with Chronic Pancreatitis *439*
- Dietary Recommendations for Patients with Chronic Pancreatitis *442*
- Nutritional Treatment for Pancreatic Exocrine Insufficiency and/or Undernourished Patients *442*
- Treatment for Osteopathy in Patients with Chronic Pancreatitis *443*
- Micronutrient Supplementation Required for Patients with Chronic Pancreatitis *444*
- Treatment of Diabetes in Patients with CP (Type 3c Diabetes Mellitus) *444*
- Lifestyle Modification in Patients with Chronic Pancreatitis *444*

23. Nutrition in Inflammatory Bowel Disease **455**

✍ ***Neelanjana Singh***

- Role of Diet in the Etiology of IBD *455*
- Other Diet Protocols for IBD Patients *461*

- Diet and Disease Activity, EDIP Score *462*
- Diet as Therapy: A Recap *462*
- Asian Working Group Guidelines 2019 *464*
- Nutritional Management Plan *464*
- Special Situations *469*
- Case-based Solutions *472*
- Bio-active Compounds in IBD *473*

24. Nutrition in Celiac Disease 476

Vidyut Bhatia, Anuja Agarwala

- Pathophysiology and Etiology of Celiac Disease *476*
- Common Symptoms and Complications *477*
- Diagnosis and Screening *478*
- Nutritional Management of Celiac Disease *480*
- Nutritional Deficiencies in Celiac Disease *484*
- Compliance with Gluten-free Diet *485*
- Follow-up Care and Multidisciplinary Approach *486*
- Can Celiac Disease be Prevented? *488*
- Sample of an Ideal Diet *489*
- Gluten-free Recipes *490*

25. Nutrition in Surgery 495

Dharmendra Dangi, Shridhar Satsukar

- Pathophysiology of Surgical Stress *495*
- Importance of Preoperative Nutritional Status *496*
- Acute and Chronic Pancreatitis *499*
- Special Situation *500*
- Chronic Pancreatitis and Surgery *501*
- Pancreatic Exocrine Insufficiency and Pancreatic Enzyme Replacement Therapy *503*
- Hepatobiliary Surgery and Obstructive Jaundice *504*
- Biliary Tract Surgery *504*
- Metabolic Surgery *505*
- Postsurgery Diet Progression *505*
- Diarrhea and Flatulence *506*
- Postoperative Nutritional Follow-up *507*
- Liver Transplantation *508*
- Chyle Leak *509*
- Nutritional Assessment *510*
- Anthropometry *511*
- Current Guidelines: ESPEN *512*
- Nutritional Recommendations in Surgery *512*

26. Nutrition in Cancer 520

Puja Sahai, KM Mohandas, Hanuman Prasad Yadav

- Cancer Cachexia *520*
- Approach to Nutritional Management in Patients with Cancer *522*
- Nutrition and Cancer Prevention *531*

27. Nutrition in Critical Illness 536

Harshita Tripathi

- Defining Critically Ill *536*
- Pathophysiology of Change of Metabolic Response to Critical Illness *536*
- Determining Nutrition Risk in Critically Ill Patients *537*
- Assessment of Biochemical/Immune Competence of Critically Ill Patients *539*
- Requirements in Critically Ill *539*
- Route of Administration *541*
- Selection of Formula *542*

28. Imbalances in Mineral and Trace Elements: Management in Acute Care Setting 546

Vandana Saluja, Gagan Pal Singh

Trace Elements

- Iron *546*
- Iodine *549*
- Copper *550*
- Zinc *551*
- Selenium *551*
- Chromium *552*

Minerals

- Sodium *552*
- Potassium *555*
- Calcium *557*
- Magnesium *558*
- Phosphorus *559*

29. Nutrition in Geriatrics 564

Chitra Mahesh, Seema Puri, Krishnan Sriram

- The Aging Process *565*
- Etiology of Malnutrition in Elderly *568*
- Malnutrition Screening and Assessment *568*
- Protein and Calorie Requirements *568*
- Micronutrients *569*
- Anemia Due to Nutritional Deficiencies *571*

- Nutritional Support *571*
- Outcomes from Nutritional Support in the Elderly *572*

30. Nutrition in Anxiety-Related Disorders 576

Mohit Varshney, Sabin Syed

- Evidence that Food Deficiency Impacts Depression and Anxiety *576*
- Evidence that Food Supplementation Improves Depression and Anxiety *577*
- Diet for Depression and Anxiety Treatment *580*
- Food and Cognitive Impacts in Young and Older Adults *581*
- Role of the Gut Microbiome *581*
- Challenges *582*

31. Nutrition Support Teams 585

Krishnan Sriram, Chitra Mahesh

- Need for Nutritional Support Teams *586*
- Functions of Nutrition Support Team *586*
- General Responsibilities of NST *591*
- Justification for NST *592*
- Tele-Nutrition, E-Nutrition *592*

32. Drug Nutrient Interactions 595

Himika Wasan, KH Reeta, YK Gupta

- Classification of Drug Nutrient Interactions *596*
- Effect of Nutritional Status on Drug Disposition *597*
- Effect of Food on Drug Disposition *598*
- Effects of Dietary Supplement Ingredients on Drug Disposition *603*
- Influence of Drugs on Global Nutritional Status *604*
- Influence of Medication on the Status of Specific Nutrients *605*
- Clinical Approaches towards Drug Nutrient Interactions *607*
- Research Directions *608*

33. Types of Diets 615

Vinita Jaiswal

- Atkins Diet *615*
- Mediterranean Diet *617*
- Ketogenic Diet *620*
- Intermittent Fasting Diet *624*
- Dukan Diet *626*
- Zone Diet *627*
- Therapeutic Diets *627*
- Gluten-free Diet *630*

34. Methods of Food Preparation **634**

Poonam Pareek

- Methods of Heat Transfer *635*
- Methods of Cooking *635*
- Cooking in Oil *639*
- Cooking by using Microwave *640*
- Cooking using Air Fryer *641*
- Effect of Cooking on Nutrients *641*

35. Role of Nurse in Nutritional Management **646**

Sarita Nadiya

- Nutrition Care Process *646*
- Nutritional Assessment *647*
- Nutrition Diagnosis *651*
- Nutrition Intervention *651*
- Nutrition Monitoring and Evaluation *654*
- Levels of Prevention and Role of Nurse in Context of Nutrition *654*
- Therapeutic Diet *655*
- Feeding the Helpless Patient *656*
- Nutrition Counseling by Nurse *657*
- Drug-food Interaction and Role of Nurse *657*
- Nursing Approach for Nutritional Management of Patients with Obesity *659*
- Respect and Uphold Human Rights during Nutrition *659*

36. Nutrigenomics **662**

Nishu Choudhary, Ashi Mittal, Shvetank Sharma

- Nutrigenomics and Health *664*
- Gut Microbiome and Nutrigenomics *669*
- Approaches to Study the Gene-Nutrition Interaction *671*
- Database and Tools for Nutrigenomics *674*
- Challenges *675*
- Future Aspects *676*

Annexures **685**

1. Know Your Food Exchanges and the Nutritional Content *685*
2. Example of Nutritional Process *687*
3. An Example of Format for Nutritional Assessment used by Nurses *690*
4. Diet Plan *694*

Index 695

Chapter 6

Starvation, Stress and Disease: Consequences

✍ *Varsha*

Metabolism Overview

ROLE OF NUTRITION IN HEALTH AND DISEASE

Nutrition is a prime example of a science in which an integrated approach is necessary. Nutritional needs of a single cell can be studied, although not in any real sense of a molecule, but most cells require a constant supply of nutrients to keep them alive in cell-culture systems. Cells will die within a day or two if nutrients are not provided. A human being, in contrast can survive for perhaps 2 months without food, because of the coordination that occurs between different cells. Description of metabolism at the level of the cell, the tissues and organs and the whole body, is the approach that would help one to see the grander picture of life: there is more to it than molecules.[1]

Nutrition is defined as only source of exogenous compounds needed for metabolism. Metabolic pathways are an elegant example of nature's ability to synthesize, interconvert, and degrade a myriad of compounds in a very narrow range of temperature, pH, and ionic conditions. However, if minimal quantities of certain compounds are not added to the body and made available to metabolic pathways, illness and death occur.

Provision of metabolic substrates from the food/diet are a complex matrix of macronutrients carbohydrate, fat, and protein, which are ingested, digested, absorbed, and delivered to the blood or lymph and then the tissues.

The substrates used to fuel metabolism are dietary carbohydrates and fat. Protein oxidation supplies approximately 20% of daily energy expenditure. Nutrients used to fuel metabolism are used in adenosine triphosphate (ATP)-generating pathways for immediate needs or stored in the body's fuel depots for later use. The intermittent ingestion of nutrients is accompanied by a metabolism cycle of absorptive state and fasting state that is modulated by the frequency of nutrient ingestion. Humans can survive extended periods of time without ingesting food by alternating fuel utilization patterns in several tissues.[2]

Resting metabolism—resting metabolic rate—the minimum amount of work that must be performed to maintain tissue integrity and a body temperature of 37°C. Resting metabolism is not available for work, but is a necessary condition for life. Transport of ions, energy substrates, and other essential metabolites to their proper location to maintain normal cellular function requires a major fraction of the resting metabolic rate.[3–5]

Flow of metabolic substrates through metabolic pathways is controlled by:

- The availability of substrates
- Allosteric activation and inhibition of enzymes

JAYPEE BROTHERS

- Covalent modification of enzymes
- Induction-repression of enzyme synthesis

These mechanisms operate on different time scales and allow the body to adapt to a wide variety of physiological situations.

Metabolic factors controlling availability of fuel substrates from either diet or from storage depots require **integration of metabolism** in different organs and can be considered as a team effort in which one tissue may provide substrates to another tissue or process compounds, produced by other tissues. There are four main members of the fuel metabolism team—brain, liver, adipose tissue, and muscle. As the "executive director" of the body, the brain receives a top priority rating when its preferred fuel, glucose, is limited. The liver has a critical role in distribution of nutrients to the rest of the body. Adipose tissue is distributed throughout the body and provides a depot for fuel storage. Muscle plays the lead role in fight or flight as directed by the decisions of the brain.

To execute their roles, the metabolism of each tissue is specially designed. Liver metabolism must be able to interconvert and process fuel substrates for use by other tissues. Adipose tissue metabolism must be able to efficiently store as many calories as possible in a small space and to rapidly mobilize it in response to signals from other tissues that fuel is running low. Brain metabolism must be focused on its complex duties as executive director and thus, have a streamlined fuel metabolism using a minimal number of different substrates. Muscle metabolism carries its own small fuel depot in the form of glycogen for use when a large amount of fuel is needed on very short notice. Muscle fuel metabolism must be equipped to use a variety of fuel substrates to sustain a long-term fight or flight. However, much of muscle metabolism is focused on the proteins needed to implement the mechanics of fight or flight. Under physiological conditions such as the post-trauma state, the muscle proteins will not be used for fight or flight and become a source of fuel for other tissues during the immediate recovery phase.

Critical communication among the tissues depends upon the nervous system, availability of circulating substrates, and variation in plasma hormone concentrations. Integration of energy metabolism, which should be focus herein, depends upon changes in the circulating concentration of insulin, glucagon, catecholamines, and growth hormone. Varying ratios of these plasma hormones are required to store energy when food is available in abundance and to use stored energy when food is unavailable during severe injury and during fight or flight situations.

During starvation the decreased insulin-to-glucagon ratio and the decreased availability of circulating substrates puts the fasting person in a catabolic state characterized by degradation of triacylglycerol, glycogen, and protein. An exchange of substrates among liver, adipose tissue, muscle, and brain is designed to maintain adequate plasma concentrations of glucose to sustain energy metabolism of the brain and other glucose requiring tissues. The substrate exchange also mobilizes fatty acids from adipose tissue and ketone bodies from liver to supply energy to other tissues.[6–8]

Though it is beyond the scope of this presentation to detail individual macronutrient (carbohydrate, fat, protein) metabolism, but it would be appropriate at this juncture to however place on record that its study is vital to understand:

- Metabolism of the fed state
- Metabolism during starvation

Summarized below through **Figures 6.1 to 6.3** is the fuel metabolism team and metabolism of the fed state and during starvation as reflected in the interaction of the main four-team members.

JAYPEE BROTHERS

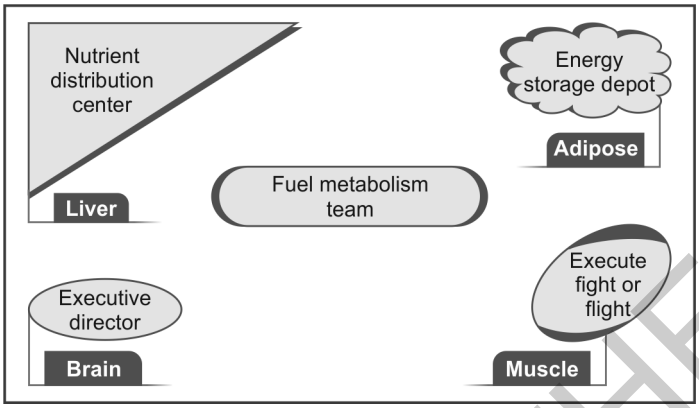

Fig. 6.1: Fuel metabolism team.

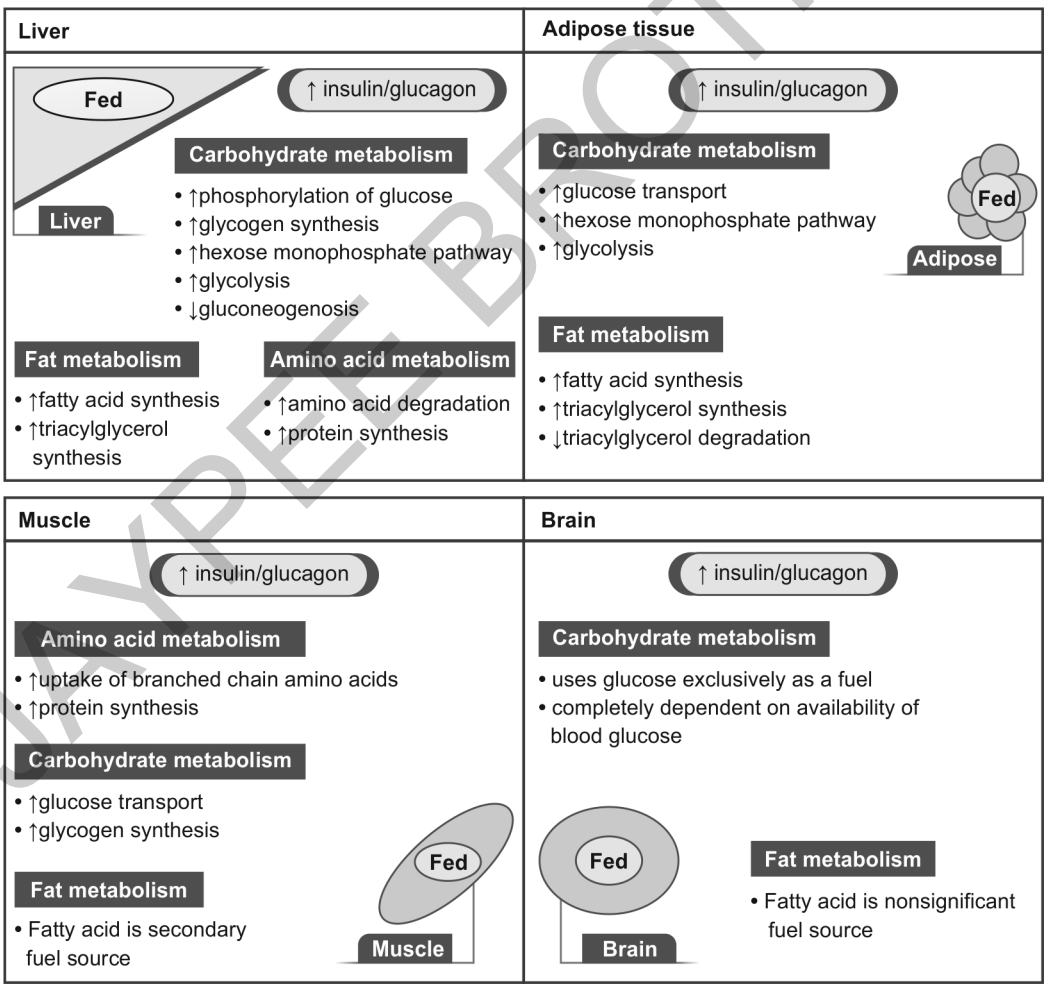

Fig. 6.2: Metabolism during the fed state.

JAYPEE BROTHERS

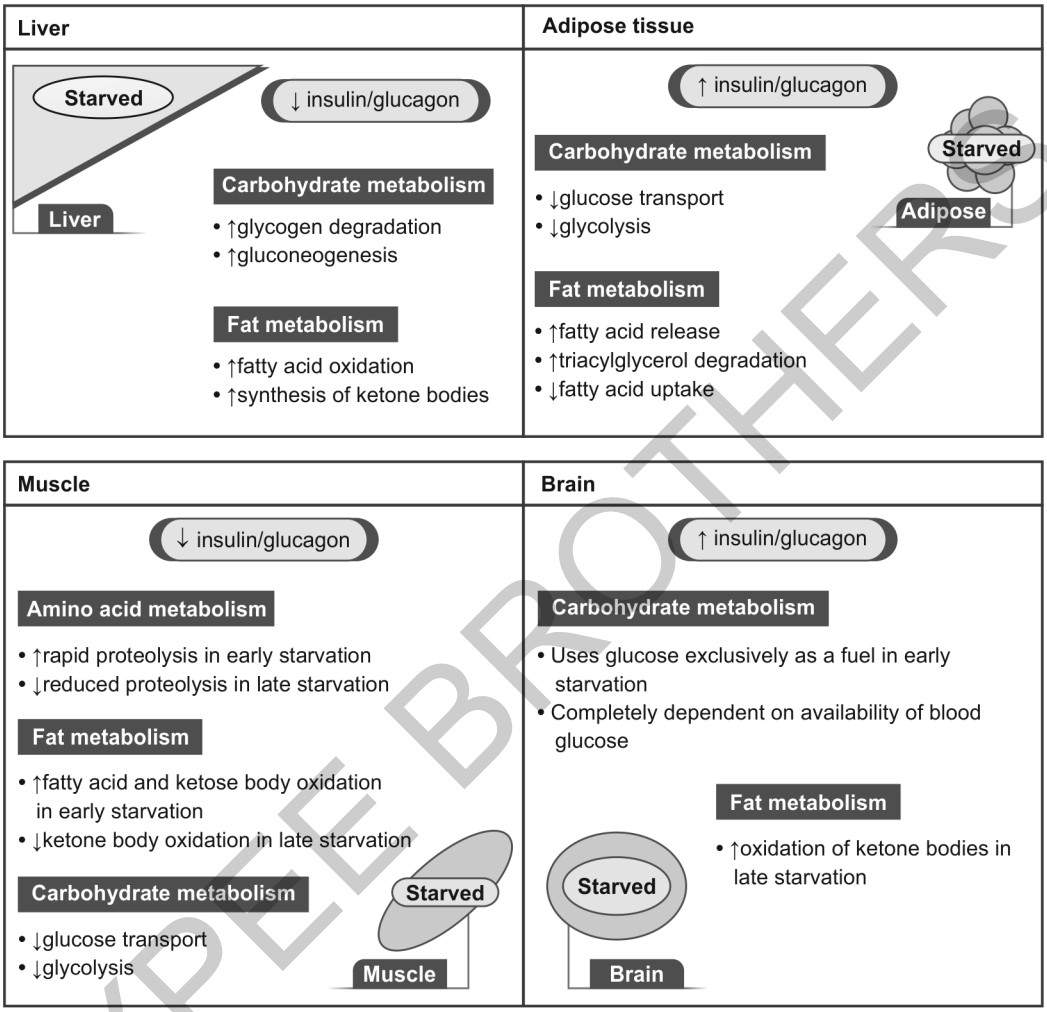

Fig. 6.3: Metabolism during starvation.

Autonomic Nervous System[9-11]

The role of the autonomic nervous system is to exercise the control of the central nervous system on internal homeostasis. Under normal conditions, numerous functions such as cardiac rate, vascular tones, gastrointestinal motility and the majority of the endocrine functions are under the tonic influence of one or another of the two divisions of the autonomic nervous system. The sympathetic system is primarily responsible for mediating the stress response. The clinical characteristics of the stress response, such as sweating, acceleration of the heart rate, peripheral vasoconstriction and increase in intestinal motility are governed by the sympathetic nervous system. Catecholamine levels appear to be correlated with the severity of stress while elevated levels reflect its duration. On one hand, catecholamines modulate the cardiovascular response. On the other hand, these mediators raise blood glucose concentration by promoting

JAYPEE BROTHERS

glycogenolysis in the liver and muscle as well as increasing the release of free fatty acids from the adipose tissue. The combined effect of adrenaline-induced insulin suppression and glucagon stimulation increases gluconeogenesis in the liver and accounts for the hyperglycemic conditions encountered during stress.

Hormonal Response[9–11]

In the responses to stress, all the hormones secreted by the hypophysis adrenocorticotropic hormone (ACTH), growth hormone (GH), luteinic hormone (LH) and prolactin (PRL) may play a role but only the effect of ACTH is well-documented. Increased secretion of ACTH and glucocorticoids is observed in trauma, extensive burns, infections and after surgery. To some extent, the physiological effects of the glucocorticoids on metabolism are basically opposed to the effect of insulin. They affect the regulation of carbohydrate, protein, lipid and nucleic acid metabolism. Glucocorticoids increase gluconeogenesis. As regards protein metabolism, corticosteroids favor the breakdown of muscle proteins, thereby furnishing the precursors for gluconeogenesis. Also, the permissive effect of glucocorticoids on the action of catecholamines is necessary for the mobilization of triglycerides. As regards body composition, the overall effect of corticosteroids is a loss in lean body mass, while the adipose tissue may be preserved. The retention of extracellular water may also be promoted. Corticosteroids in conjunction with catecholamines have a catabolic effect, transferring energy substrates from the muscular carcass and adipose tissue to the viscera as a matter of biological priority.

Cytokine Response[9-11]

Cytokines are immune-derived endogenous polypeptide mediators, which initiate immune, hematological and metabolic alterations that are integral to the response of the organism to stress conditions. Cytokines are products of immunocompetent cells and act at very low concentrations to regulate the biological functions of other cells and organs. These mediators participate in the development of both the beneficial and injurious sequel of immune system activation. They are essential to the recovery process, but lethal in high doses. Their beneficial properties are, for example, the stimulation of antimicrobial function, wound healing, myelostimulation and mobilization of fuel stores. On the other hand, cytokines are capable of triggering a state of chronic cachexia or acute septic shock, followed by tissue injury, multiple organ failure and eventually even death of the patient. Through a cascade system, different cytokines can stimulate each other's production.

One cytokine in particular seems to occupy a pivotal role in the development of the metabolic and pathological consequences of the stress response: tumor necrosis factor (TNF), also known as cachectin. TNF is probably the most potent mediator of septic shock. It has been detected in the serum of patients experiencing various diseases, such as parasitic or bacterial infections, tumor-bearing disease, burns and acute hepatic failure. TNF also exerts influences on the metabolism of the organism. It increases degradation of protein in skeletal muscle but also stimulates the production of acute phase proteins in the liver. TNF also induces changes in lipid metabolism. There is further a loss of triglycerides from the adipocytes and an increase of cellular lipolysis.

These phenomena together account for hypertriglyceridemia and loss of body fat. Overall, the beneficial effects of TNF predominate at low tissue levels, while higher concentrations contribute to the development of cachexia and septic shock syndrome.

JAYPEE BROTHERS

Other important cytokines are interleukin-1 (IL-1) and interleukin-6 (IL-6). Both mediators are induced by the presence of TNF. IL-1 has been shown to stimulate the proliferation of T-cells and to promote myelopoiesis. Furthermore, it is able to induce fever and anorexia. Like TNF, IL-1 exerts beneficial influences during injury, but chronic IL-1 release has also been implicated in the pathogenesis of different degenerative diseases. In patients developing sepsis in the intensive care unit, high TNF serum levels were found in patients with septic shock. There was a correlation between TNF level and sepsis severity score and mortality; no correlation, however, was found with IL-1 values.

IL-6 is also rapidly released into the circulation in response to injury. It is the main promoter of hepatic acute phase protein synthesis. IL-6 is also an endogenous pyrogen. Like TNF and IL-1, IL-6 is an early response in the cascade of host mediators after injury. Its influence is rather beneficial to the host by enhancing immune function.

Nutritional factors may have an influence on cytokine synthesis and release. Furthermore, nutrition can affect the direct and indirect actions on target tissues. Especially lipids exert an influence on cytokine production and inflammation. Lipids are known to be able to modify cell membrane structure and to alter eicosanoid metabolism. Protein-energy malnutrition probably also influences cytokines in such a way that their levels are lowered. Preliminary data seem to indicate that overnutrition might increase cytokine production.

Starving/fasting and stress have opposite influences on the energy expenditure of the human organism. The healthy human body is capable of passing from a state involving three regular food intakes to a state of short-term fasting and even prolonged fasting, as a result of precise metabolic regulation. In these cases, the organism will save as much energy as possible, thus reducing energy expenditure.

However, in stress conditions, energy expenditure is markedly increased. As a result, the body's metabolism will be converted into a catabolic state, the gravity of which is determined by the nature and degree of the injury and type and severity of underlying disease.

EFFECTS OF STARVATION FASTING[12-13]

In theory, if a person having 15 kg of adipose triglycerides, i.e., 140,000 kcal of reserves in the form of fats, and energy requirements of 1,800 kcal/day, begins to fast, he should be capable to withstanding 75 days of total fasting. However, in practice, an abstinence from feeding leads to death after about 50 days of total fasting. In other words, the theoretical value of the energy reserve cannot be used in its entirety, because death intervenes beforehand due to partial depletion of functional tissue proteins.

In the case of abstinence or fasting, endogenous energy stores are used for metabolic process. Fat, stored in different fat tissues, is the major source of energy. Energy can also be derived from protein; however, there is no indifferent protein tissue and as a consequence the loss of protein always leads to a loss of organ function.

Hence, in the case of fasting in healthy persons, the metabolism is aimed at keeping the loss of protein, as low as possible by lowering the metabolism and the gluconeogenesis. The loss of nitrogen is reduced in the case of complete fasting from 10 g per day to 4–5 g a day after weeks. Fat stores are depleted faster with the purpose of providing energy.

Many organs including the heart, kidneys and muscles can use either fatty acids or ketone bodies, derived from the partial oxidation of fatty acids, directly as energy substrates. The central

JAYPEE BROTHERS

nervous system, on the other hand, and the red blood cells can only use glucose as an energy substrate. For example, during a 24 hour fast, the brain will consume 150 g of glucose and the other organs about 36 g, i.e., a total of 186 g of glucose per day. Since the body is incapable to synthesizing glucose from fat, it uses other substrates for gluconeogenesis. In fact, the glycogen reserves are sufficient to cover the requirements just for a day or two. The most important substrate for gluconeogenesis is provided by amino acids, to a minor extent, by glycerol derived from the triglycerides.

In short-term fasting, some of the glucose required by the brain is provided by liver glycogen, the reserve being exhausted within 48 hours. If the human body is to withstand fasting, it must mobilize 1,800 kcal/day and produce 186 g of glucose mainly for the central nervous system. About 80% of the energy requirements are provided by lipolysis of adipose tissue where 160 g of triglycerides are split into fatty acids and glycerol. Approximately 75 g of muscle proteins, i.e. nearly 300 g of muscle, per day are mobilized to provide the substrate for gluconeogenesis. If protein breakdown were to continue at the initial rate, roughly one-third of the total body proteins would be exhausted in 3 weeks, which is incompatible with survival.

So, if fasting is prolonged, a major metabolic adaptation occurs. The central nervous system begins to use ketone bodies as an energy substrate, thereby reducing glucose requirements. Therefore, in prolonged fasting, there is a shift from the use of protein as an energy source towards the use of fats (in the form of ketone bodies). This adaptation permits protein sparing and preserves the protein's functional role. Nevertheless, obligatory proteolysis always persists, amounting in the foregoing example to at least 20 g of protein daily.

Metabolic processes respond to internal signals. During fasting, blood glucose levels fall with a consequent reduction in the secretion of insulin and an increase in glucagon, two hormones with antagonistic actions on energy metabolism. As a result of the decrease in the circulating insulin level, triglyceride catabolism increases, causing the release of free fatty acids and glycerol. The raised glucagon levels lead initially to a distinct increase in liver glycogenolysis. Further, gluconeogenesis is stimulated by glucagon, which inhibits protein synthesis and stimulates muscular proteolysis, thereby furnishing the amino acid substrate.

There is therefore a metabolic adaptation to prolonged fasting, resulting in a reduction of energy expenditure of up to 40%. These mechanisms, which tend to limit proteolysis in the healthy person, are defective or nonoperative in cases of disease or stress **(Table 6.1)**.

EFFECTS OF STRESS AND DISEASE[12-14, 16-19]

Metabolic responses to disease and stress depend on a sequence of events under the control of the central nervous system, the purpose of which is to mobilize endogenous energy substrates. The nature and magnitude of these neurohumoral signals emitted by the brain depend on the degree of stress, which in turn is a function of the extent of the lesions of the tissue. The patient's age, physiological reserves, nutritional status and latent diseases may modulate these responses. The complexity of the metabolic regulation, which culminates in the hypermetabolism induced by certain disorders, is illustrated by the example of severe acute trauma.

Cuthbertson has intensively studied the metabolic response to injury. In the course of post-traumatic metabolism, two phases may be distinguished in the stress response: the ebb and the flow phase. The ebb or shock phase is transitory and generally completed in 24 hours, but may continue for a few days. This phase is immediate and characterized by a general depression of

JAYPEE BROTHERS

Table 6.1: Metabolic physiology.

Characteristic finding	*Starvation*	*Metabolic response to injury*
Energy needs	Decreased	Increased
Primary fuel (RQ*)	Lipids (0.75)	Mixed (0.85)
Insulin	Decreased	Increased (resistance)
Ketones	Present	Absent
Counterregulatory hormones	Basal	Increased
Total body water	Decreased	Increased
Proteolysis	Decreased	Accelerated
Glycogenolysis	Increased	Accelerated
Lipolysis	Increased	Increased
Body stores—skeletal muscle	Reduced	Reduced
Body stores—fat	Reduced	Reduced
Body stores—visceral protein	Preserved	Increased liver/immune
Refeeding response	Net anabolism	None (unless reversed)
Weight (lean tissue) loss	Gradual	Accelerated
Typical setting	Chronic diseases	Hospitalized ICU patient

the body's physiological functions. There is a progressive fall in blood flow, in body temperature and oxygen utilization.

A series of cardiovascular readjustments takes place, the cardiac output falls, the peripheral resistance increases and the blood is redistributed to the vital organs. The circulating levels of the so-called stress hormones, catecholamines, ACTH, growth hormone and glucocorticoids increase significantly. At the site of the injury, there is an accumulation of water, plasma protein and sodium, thus helping in the organism to retain sodium and conserve body water.

If these initial adjustments enable the traumatized patient to survive, a second phase of stress response follows. This is the flow phase, which is mainly characterized by a hypermetabolic state associated with increased losses of nitrogen and other body constituents. It is possible to divide the flow phase into a catabolic and an anabolic stage. The latter then heralds the final recovery of the patient. However, the flow phase is dominated by the catabolic stage, which is in fact an inflammatory response, a king of defense reaction in order to initiate repair of the damaged tissue.

There is also a rise in urinary excretion of nitrogen, sulfur, phosphorus, potassium, magnesium and creatinine. This goes together with an increased basal oxygen consumption. Usually, the catabolic processes reach a maximum 4–8 days after stress induction. In general, the more severe the injury longer the catabolic response and the more rigorous it is. With burns, for example, the time course can be extended by a few weeks. There is a new protein catabolism with urinary nitrogen loss, increased substrate cycling of lipids, carbohydrates and proteins, accounting for much of the increase in energy expenditure. The increase in amino acid turnover results in a negative balance. However, there is a qualitative predominance of proteolysis

JAYPEE BROTHERS

over protein synthesis. Both processes are stimulated: proteolysis can increase by as much as 45%, while the maximum losses can even be higher than 20 g of nitrogen per day, which corresponds to a loss of 600–800 g of muscle per day. The main goal of clinical nutrition is to support protein synthesis rather than to suppress proteolysis. This will help to initiate the anabolic stage of the flow phase, leading to the patient's convalescence.

The central nervous system processes the information received during stress and dictates the responses, which are transmitted on the one hand by the autonomic nervous system and on the other hand by the hypothalmopituitary axis. Hormones and cytokines are the mediators of these processes.

EFFECTS OF INFECTION[15]

Basically, the stress response due to an infection is the same as the response after injury, trauma or severe disease. As a matter of fact, the same mechanisms and mediators intervene. Hence, a catabolic response occurs with all infections, even when they are subclinical and not accompanied by symptoms.

The biological response is amplified by the production of chemical mediators (histamine, prostaglandin, serotonin, cytokines, etc.) and inflammatory processes. Cell-mediated immunity is depressed in malnutrition; delayed hypersensitivity skin responses to antigen are reduced. Malnutrition has differential effects on cytokine production. Production of interferon, prostaglandin E2 and interleukin-2 and interactions with T-lymphocytes appear to be compromised in severe malnutrition. In contrast, production of other cytokines are apparently unaffected by protein-energy malnutrition. For each degree centigrade above the normal value, and increase in energy expenditure of 12–13% is observed. Microbial infections frequently complicate the clinical profile of an ill, traumatized or surgical patient. Apart from local tissue destruction, the systemic effects of infections may have a pronounced effect on metabolic responses. Measurements of energy expenditure have shown that the development of an infection intensifies the metabolic response to stress. If the energy expenditure is measured before and after elective, uncomplicated surgery, the postoperative value is found to be some 10% higher. If, on the other hand, peritonitis supervenes, the postoperative increase will be 20–25% higher than the expected value. This observation may be confirmed by measuring the excretion of urinary nitrogen, the level of that is much higher in the presence of postoperative infection. The increase in nutrient requirements depends on the nature, duration and severity of the infection. Moreover, the type of infection, chronic or acute, and the nutritional state of the patient both influence the recovery, improvement of the nutritional status reduces morbidity and mortality from infection.

CONSEQUENCES OF MALNUTRITION[12-14]

Malnutrition is characterized by an impairment of a number of physiological functions. Strength is reduced and fatigue occurs much earlier. Respiratory muscle function is deteriorated, as are other bodily functions, such as cardiac, sexual and immune function as well as thermoregulation. Malnutrition leads to depression, irritability, anxiety, reduced ability to maintain concentration and reduced sexual drive. In children, malnutrition impairs growth and delays sexual maturation. The importance of feeding on neurodevelopment in preterm infants have been shown by the influence of the type of feeding (preterm formula vs. term formula) on the IQ eight years later.

JAYPEE BROTHERS

In the hospitalized patient, malnutrition combined with weight loss occurs as a consequence of an insufficient metabolism. Both insufficient food intake and chronic catabolic illness can cause this. The lack of minerals, vitamins or trace elements resulting from insufficient feeding is beyond the scope of this definition, because these shortages can be supplemented relatively easily. Especially in the feeding of the acutely ill patient, manipulation of the protein mass, however, forms the major problem.

Two main types of malnutrition can be discerned **(Table 6.2)**. Malnutrition may in form of protein-energy deficiency (marasmus-type) or only protein deficiency [kwashiorkor-type]. Besides these two types, marasmic kwashiorkor is defined as a combination of these two clinical states. Inadequate energy is termed marasmus, which is characterized by a weight less than 40% of the standard; absence of subcutaneous fat, muscle wasting, diminution in height/length; edema not noticeable. Marasmus (or simple starvation) is diagnosed relatively easily and is characterized by a gradual loss of lean body mass and adipose tissue, due to a chronic imbalance between protein and energy intake and requirements. The patient utilizes his endogenous energy reserves, including adipose and somatic muscle tissue with subsequent weight loss, muscle wasting and loss of fat depots. Marasmic malnutrition develops over a relatively long time period and patients are underweight and more or less cachectic. The diagnosis of kwashiorkor is often more difficult, since body protein is reduced but fat stores are normal. This type of malnutrition is caused by an unbalanced feeding pattern and is characterized by hypoalbuminemia. Other symptoms are hepatomegaly and hair and skin changes. Extracellular water spaces are expanded, with salt retention, edema and occasionally ascites. Marasmic kwashiorkor, an intermediate form characterized by wasting and hypoalbuminemia, with or without edema, is also relatively common in hospital patients. These patients are often overweight and at first do not necessarily appear malnourished.

Malnourished hospitalized patients have a diminished immune response and are at a greater risk of developing postoperative complications or infection.

Table 6.2: Description of types of malnutrition.

	Marasmus	*Kwashiorkor*
Clinical setting	↓ calories	↓ protein + stress
Time-course to develop	Months to years	Weeks to months
Clinical features	Starved appearance ↓ weight ↓ triceps skinfold (fat energy) ↓ mid arm circumference (muscle)	• Well nourished appearance • Edema • Loose Hair
Laboratory findings	• Unremarkable laboratory pattern • Serum albumin may be lowered	↓ serum albumin ↓ serum transferrin ↓ lymphocyte count Non-reactive skin test
Clinical course	Reasonable response to short-term stress	↓ wound healing ↓ immunocompetence ↑ infection/complications
Mortality rate	Low (unless related to underlying disease)	High

Source: Butterworth and Weinsier, 1980.

JAYPEE BROTHERS

REFERENCES

1. Borum PR. The Science and Practice of Nutrition Support—A Case-Based Core Curriculum. ASPEN. 2002;17-26.
2. Hirsch J, Leibel RL, Edens NK. Overview: cellular corollaries of energy expenditure. In: Kinney JM, Tucker HN (Eds). Energy Metabolism-Tissue Determinants and Cellular Corollaries. New York, NY: Raven Press, Ltd; 1992; pp:525-32.
3. Champe PC, Harvey RA. Metabolism in the well-fed state. In: Lippincott's Illustrated Reviews: Biochemistry. 2nd edition. Philadelphia, PA: J.B. Lippincott Company; 1994; pp:281-9.
4. Marks DB, Marks AD, Smith CM. The fed or absorptive state. In: Basic Medical Biochemistry-A Clinical Approach. Baltimore, MD: Williams and Wilkins;1996; pp:21-6. 4.
5. Poehlman ET, Horton ES. Energy needs: Assessment and requirements in humans. In: Shils ME, Olson JA, Shike M, Ross AC (Eds). Modern Nutrition in Health and Disease. Baltimore, MD: Williams and Wilkins; 1999; pp:95-104.
6. Voet D, Voet JG. Energy metabolism: Integration and organ specialization. In: Biochemistry. 2nd edition. New York, NY: John Wiley and Sons, Inc; 1995; pp:785-94.
7. Stryer L. Integration of metabolism. In: Biochemistry. 4th edition. New York, NY: W.H. Freeman and Company;1995; pp: 763-84.
8. Harris RA, Crabb DW. Metabolic interrelationships. In: Devlin TM (Ed). Textbook of Biochemistry with Clinical Cor relations. 4th edition. New York, NY: Wiley-Liss;1997; pp:525-62.
9. Gibney MJ, Macdonald IA, Roche HM. Nutrition and Metabolism. The Nutrition Society Textbook Series. Blackwell Publishing; 2003.
10. Fong Y, Moldawer LL, Shires GT, et al. The biologic characteristics of cytokines and their implication in surgical injury. Surg Gynecol Obster. 1990; 170:363-78.
11. Damas P, Reuter A, Gysen P, et al. Tumor necrosis factor and interleukin-1 serum levels during severe sepsis in human. Crit Care Med. 17:975-8.
12. The Merck Manual of Diagnosis and Therapy, 17th Centennial Edition; 1999.
13. ME, Shike M, Ross AC, Caballero B, Cousins RJ [Eds]. Modern Nutrition in Health and Disease, 10th edition. Lippincott Williams and Wilkins; 2006.
14. Dorlands Illustrated Medical Dictionary, 30th edition. Saunders; 2003.
15. Scrimshaw NS. Effect of infection on nutrient requirements. JPEN. 1991; 15:589-600.
16. Goldstein SA, Elwyn DH. The effects of injury and sepsis on fuel utilization. Ann Rev Nutr.1989; 9:445-73.
17. Cuthbertson DP, Tilstone WJ. Metabolism during the postinjury period. Adv Clin Chem;1969; 12:1-55.
18. Cuthbertson DP. Post-traumatic metabolism: A multidisciplinary challenge. Surg Clin North Am.1978; 58:1045-54.
19. Cuthbertson DP. The metabolic response to injury and its nutritional implications: retrospect and prospect. JPEN. 1979; 3:108-29.

JAYPEE BROTHERS

or detailed information on nutrition books, visit our website www.ejaypee.com, for detailed information on nutrition books, visit our website www

Basics of Clinical Nutrition

Salient Features

- This book covers all the important topics including the fundamental aspects as well as the emerging areas, all from a practical standpoint.
- All the information is backed by the latest scientific research, updated with recent references.
- All chapters are curated by practicing scientists, nutritionists, teaching faculty, physicians, nurses, and surgeons, with their knowledge and personal experience in the subject area.
- The book contains numerous tables, figures, and charts, allowing the readers to apply the information directly to clinical practice.
- It is an ideal book for students, be it nutrition, medical, or nursing.
- It is the only book on the subject of "clinical nutrition" written by Indian authors.

YK Joshi MD PhD is Emeritus Professor, Department of Hepatology and Clinical Nutrition, Institute of Liver and Biliary Sciences, New Delhi, India. He is an eminent gastroenterologist, distinguished medical educator, researcher, reviewer, author, and mentor to generations of gastroenterologists and hepatologists. He has served as Professor of Gastroenterology and Human Nutrition at the All India Institute of Medical Sciences (AIIMS), New Delhi, and as Senior Professor in the Department of Hepatology and Clinical Nutrition at the Institute of Liver and Biliary Sciences (ILBS), New Delhi.

He is a former Secretary-General and President of the Indian Society of Gastroenterology and the founding President of the Indian Society of Clinical Nutrition. His pioneering contributions to the field of nutrition include seminal research on waterborne non-A, non-B hepatitis virus (now known as Hepatitis E virus), pyrrolizidine alkaloid-induced toxic liver injury, and the role of casein proteins in liver disease. He has authored over 175 scientific publications, 26 book chapters, and 2 books and has led 25 research projects. He continues to contribute to the field as Emeritus Professor at ILBS, New Delhi.

Jaya Benjamin MSc PhD is Associate Professor, Department of Clinical Nutrition, Institute of Liver and Biliary Sciences, New Delhi, India. She is a young and dynamic academician and nutrition scientist with over 15 years of experience in the field of nutrition in liver and gastrointestinal diseases. A university gold medallist, she received her PhD from the All India Institute of Medical Sciences (AIIMS), New Delhi. Her areas of interest include nutrition in liver and gastrointestinal disorders, liver transplantation, critical illness, sarcopenia, frailty, branched chain amino acids, and energy metabolism. She has authored over 30 publications and 12 book chapters and has led 6 government funded extramural research projects. Additionally, she has mentored 4 PhD scholars, 5 PDCC fellows, and over 25 postgraduate students (DM, MCh, and DNB). She is an accomplished nutritionist, widely recognized for her research acumen, constructive reviewership, and engaging oratory.

Shelving Recommendation
NUTRITION

ISBN 978-93-5696-605-5

9 789356 966055

Buy from ejaypee

JAYPEE BROTHERS