

A winter landscape with snow, bare trees, and a bright sun in the sky. The sun is in the upper left, creating a lens flare. The text "Good Morning" is overlaid in the center.

Good Morning

D Y PATIL DENTAL SCHOOL

DEPARTMENT OF
PUBLIC HEALTH DENTISTRY

Nutrition and Oral Health

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Contents

- ▶ Introduction
- ▶ Definitions of the terms used
- ▶ Functions of food
- ▶ Nutrient values of food
- ▶ Food guide pyramid
- ▶ Recommended dietary allowance
- ▶ Effects of processing on nutritive value of foods

- ▶ Carbohydrates – classification, sources, function, metabolism
- ▶ Protein- classification, sources, function, metabolism
- ▶ Lipids - classification, sources, function, metabolism
- ▶ Vitamins
- ▶ Minerals
- ▶ Calcium metabolism.
- ▶ Water and electrolyte
- ▶ Nutrition and dental caries

- ▶ Nutrition in growth and development of oral tissue
- ▶ Proteins and oral health
- ▶ Fats and oral health
- ▶ Water and electrolyte balance and oral health
- ▶ Effects of trace elements on oral health
- ▶ Interaction of immunity, infection & nutritional status
- ▶ Lab and clinical assessment of nutrition
- ▶ Pediatric nutritional disorders
- ▶ Nutritional management of acute mucosal problems
- ▶ Conclusion
- ▶ References

Introduction



Definitions of the terms used

Nutrition:

Oxford dental dictionary: the sum process in the growth, maintenance and repair of living body as a whole or its constituent parts.

W.H.O: nutrition is the science of food and its relationship to health. It is concerned primarily with the part played by the nutrient in body growth, development & maintenance.

NIZEL 1989: the science which deals with the study of nutrient and foods and their effects on the nature & function of organism under different conditions of age, health & disease.

DCNA 2003: the science, how the body utilizes food to meet the requirements for development, growth, repair & maintenance.

Food:

Oxford dental dictionary: Any substance which when taken into the body or an organ may be used either to supply energy or build a tissue.

Nizel 1989 : any thing that is eaten , drunk or absorbed for maintenance of life, growth & repair of the tissue.

Diet:

Oxford dental dictionary : referred to as food & drink regularly consumed.

Nizel (1989): total oral intake of a substance that provides nourishment & supply.

P.M Randelph(1981) : It is the total intake of substance that furnish nourishment & or calories to the body.

BALANCED DIET:

is the one which contains varieties of foods in such quantities & proportion that are need for energy.

Stewart: one providing each nutrient in the (neither deficient nor excess) needed to maintain optimum health.

CHILD DIET: Defined as combination of food consumed and the nutrients contained there in , has the profound ability to influence cognition, behavior, & ultimate physical growth & development (DCNA 2003)

Functions of the food

*

- ▶ Physiological function
- ▶ Social function
- ▶ Psychological function

PHYSIOLOGICAL FUNCTION:

- supply energy.
- build & maintain the cells & tissues
- regulate body process - movement of fluids
 - control acid & base balance
 - coagulation of blood
 - activation of enzyme

SOCIAL FUNCTIONS OF FOOD:

- Acts as media to develop social rapport in the society.
- Is an integral part of social phase of university living.

PSYCHOLOGICAL FUNCTION OF FOOD:

- Satisfies of certain emotional needs.
- Used to express feelings:
 - a) Token of friendship
 - b) Serving of favorite foods - expression of special attention.
 - c) Withholding of wanted foods - punishment.

Nutrient values of food *

Energy from the food - 4 basic forms:

- chemical – synthesis of new compounds
- mechanical – for muscle contraction.
- electrical – for brain & nerve activity
- thermal – regulation of body temperature

25% of chemical energy from the food we eat -
mechanical energy.

Rest - heat

Energy- kilocalories

Kilocalories - the amount of heat required to raise the temperature of 1kg (2.2lb) of water in 1°C(14.5-15.5)

- SI -Joule

- 1 kcal= 4.18kJ

Atwater - calorimetry in US, corrected caloric values of avg heat of combustion of pure nutrients.

- Carbohydrates ---- 4.1 kcal/g

- Fats ----- 9.45 kcal/g

- Proteins ----- 5.65 kcal/g

- Alcohol ----- 7.1 kcal/g

ATWATER corrected caloric values:

- Carbohydrates -- 4 kcal/g or 17 kJ
- Fats -- 9 kcal/g or 38 kJ
- Proteins-- 4 kcal/g or 4 kJ
- Alcohol -- 7 kcal/g or 30 kJ

Energy needs of the body:

Calculated by sum of 3 factors:

- a) Basal metabolism.
- b) Energy for physical activity.
- c) Small amount of additional energy expended during digestion and absorption of carbohydrates, proteins fats in GIT – specific dynamic action (SDA) of food.

Energy requirement= BM + Physical Activity + SDA

•B.M.R:

Metabolic rate at basal conditions.

Basal condition is a condition when the subject is at complete mental, physical rest (but not sleeping) 12-14 hrs after the last meal, at ambient temp of about 25°C & free from all illness.

•Males – 40 kcal (168kJ/sq m/hr)

•Females- 37 kcal(155 kJ/sq m /hr)

NORMAL BMR VALUES FOR DIFFERENT AGE GROUP AND SEX.

Age in yrs	Male kcal	female kcal
5	53	51
10	49	46
15	46	40
20	41	36
30	39	36
40	38	36
50	37	34
60	35	33

Physical variations of BMR:

1. Surface area:

BMR is directly proportional to body surface.

Du Bois – formula to measure body surface area.

$$A = H^{0.725} \times 71.84 \times W^{0.425}$$

where, A-surface area in sq m

H – height in cm

W – weight in kg

2. Age:

max at the age of 5 yrs.

slight ↑ puberty.

3. **Sex:** - women < men

4. **Climate:** - slight higher in cold climate.

5. **Race:** - ↑ western races, But independent of effect of climate.

- Additional increment during pregnancy & lactation.
- Pregnant women – 10-20% cal > non pregnant.
- Lactating mother - 850 ml of milk requires about 100 additional Cal

PATHOLOGICAL variations of BMR:

- **Fever:** for every 1°C rise in body temp there is an \uparrow by 7%
- **Increased metabolic activity** –
leukemia, polycythmia, cardiac failure, hypertension, difficult breathing (Asthma) - \uparrow BMR
- **Endocrinal disturbances:**
 - Hyperthyroidism - \uparrow BMR by 75% or more.
 - Hypothyroidism- \downarrow BMR by 40 %
 - \uparrow - Cushing's syndrome, Acromegaly
 - \downarrow -Addison's disease.
- **major surgery , infections, & burns-** \uparrow BMR

Energy for Physical activity:

- Muscular activity - both energy expenditure & heat production.
- Energy expenditure \uparrow - muscular activity.
- Moderate activity- der 300 kcal
- Strenuous activity (laborer, athlete) – \uparrow 600-900 kcal

Specific Dynamic Action (SDA): the expenditure of calories during the digestion & absorption of food.

- SDA of diet – app10% of the consumed calories.
- E.g.; person energy needs is 2000kcal + 200 kcal (heat expended in SDA)

Food guide pyramid

*

Fats, Oils & Sweets
USE SPARINGLY

KEY

- Fat (naturally occurring and added)
- ▼ Sugars (added)

These symbols show fats and added sugars in foods.

Milk, Yogurt & Cheese Group
2-3 SERVINGS



Meat, Poultry, Fish, Dry Beans, Eggs & Nuts Group
2-3 SERVINGS



Vegetable Group
3-5 SERVINGS



Fruit Group
2-4 SERVINGS



Bread, Cereal, Rice & Pasta Group
6-11 SERVINGS



Recommended Dietary Allowance

RDA – nutrient amounts in excess of what 98% of the population requires to maintain health ; they are not minimum requirements or optimal intakes for all people.

- ▶ Average daily intake energy and nutrient level intake estimates for healthy populn , through consumption of variety of foods.
- ▶ Food & nutrition board --- Dietary reference intakes.

DRI- 4 set of different values:

- **RDA**: the average daily dietary intake of a nutrient that is sufficient to meet the requirement of nearly all healthy people.
- **Adequate Intake (AI)**: a level of nutrient based on observed intakes of groups of healthy people when RDA cannot be established.
- **Tolerable upper intake level**: the highest daily intake of a nutrient that is likely to pose no risk of toxicity for most healthy people
- **Estimated average requirement (EAR)**: the amount of nutrient estimated to meet the requirement for half of all healthy people in a population.

DCNA 2003

Values –mg

Dietary reference intakes by age group^a

Age	Female	Male
14–18 years		
Calcium	1300	1300
Phosphorus	1250	1250
Magnesium	360	410
Vitamin D ^b	5	5
Fluoride	3	3
Thiamin	1	1.2
Riboflavin	1	1.3
Niacin	14	16
Vitamin B ₆	1.2	1.3
Folate ^c	400	400
Vitamin B ₁₂	2.4	2.4
Pantothenic acid	5	5
Biotin	25	25
Choline	400	550
Vitamin C	65	75
Vitamin E	15	15
Selenium	55	55
19–30 years		
Calcium	1000	1000
Phosphorus	700	700
Magnesium	310	400
Vitamin D ^b	5	5
Fluoride	3	4
Thiamin	1.1	1.2
Riboflavin	1.1	1.3
Niacin	14	16
Vitamin B ₆	1.3	1.3
Folate ^c	400	400
Vitamin B ₁₂	2.4	2.4
Pantothenic acid	5	5
Biotin	30	30
Choline	425	550
Vitamin C	75	90
Vitamin E	15	15
Selenium	55	55

31–50 years

Calcium	1000	1000
Phosphorus	700	700
Magnesium	320	420
Vitamin D ^b	5	5
Fluoride	3	4
Thiamin	1.1	1.2
Riboflavin	1.1	1.3
Niacin	14	16
Vitamin B ₆	1.3	1.3
Folate ^c	400	400
Vitamin B ₁₂	2.4	2.4

Pantothenic acid	5	5
Biotin	30	30
Choline	425	550
Vitamin C	75	90
Vitamin E	15	15
Selenium	55	55

^a Values are given in milligrams.

^b As cholecalciferol. 1 mg cholecalciferol = 40 IU vitamin D.

^c As dietary folate equivalents (DFE). 1 DFE = 1 mg food folate = 0.6 mg of folic acid from fortified foods or as a supplement consumed with food. In view of evidence linking folate intake with neural tube defects in the fetus, it is recommended that all women capable of becoming pregnant consume 400 mg from supplements or fortified foods in addition to intake of food folate from a varied diet.

Recommended dietary allowance for Indians

RECOMMENDED DIETARY ALLOWANCES FOR INDIANS

Group	Particulars	Body wt kg	Net energy Kcal/d	Protein g/d	Fat g/d	Calcium mg/d	Iron mg/d	Vit. A. $\mu\text{g}/\text{d}$		Thi- min mg/d	Ribo- flavin mg/d	Nico- tinic acid mg/d	Pyri- doxin mg/d	Ascor- bic acid mg/d	Folic acid $\mu\text{g}/\text{d}$	Vit. B-12 $\mu\text{g}/\text{d}$
								Reti- nol	β -caro- tene							
Man	Sedentary work		2425							1.2	1.4	16				
	Moderate work	60	2875	60	20	400	28	600	2400	1.4	1.6	18	2.0	40	100	1
	Heavy work		3800							1.6	1.9	21				
Woman	Sedentary work		1875							0.9	1.1	12				
	Moderate work	50	2225	50	20	400	30	600	2400	1.1	1.3	14	2.0	40	100	1
	Heavy work		2925							1.2	1.5	16				
	Pregnant woman	50	+300	+15	30	1000	38	600	2400	+0.2	+0.2	+2	2.5	40	400	1
	Lactation															
	0-6 months	50	+550	+25	45	1000	30	950	3800	+0.3	+0.3	+4	2.5	80	150	1.5
	6-12 months		+400	+18						+0.2	+0.2	+3				
Infants	0-6 months	5.4	108/kg	2.05/kg		500				55 $\mu\text{g}/\text{kg}$	65 $\mu\text{g}/\text{kg}$	710 $\mu\text{g}/\text{kg}$	0.1	25	25	0.2
	6-12 months	8.6	98/kg	1.65/kg				350	1200	50 $\mu\text{g}/\text{kg}$	60 $\mu\text{g}/\text{kg}$	650 $\mu\text{g}/\text{kg}$	0.4			
Children	1-3 years	12.2	1240	22			12	400	1600	0.6	0.7	8	0.9	40	30	
	4-6 years	19.0	1690	30	25	400	18	400	2400	0.9	1.0	11		40	40	0.2-1.0
	7-9 years	26.9	1950	41			26	600	2400	1.0	1.2	13	1.6	60		
Boys	10-12 years	35.4	2190	54			34			1.1	1.3	15		70		
Girls	10-12 years	31.5	1970	57	22	600	19	600	2400	1.0	1.2	13	1.6	40	40	0.2-1.0
Boys	13-15 years	47.8	2450	70			41			1.2	1.5	16		100		
Girls	13-15 years	46.7	2060	65	22	600	28	600	2400	1.0	1.2	14	2.0	40	40	0.2-1.0
Boys	16-18 years	57.1	2640	78			50			1.3	1.6	17		100		
Girls	16-18 years	49.9	2060	63	22	500	30	600	2400	1.0	1.2	14	2.0	40	40	0.2-1.0

Effects of processing on nutritive value of foods *

Beneficial effects of cooking:

- ▶ Improves the cooking
- ▶ Improves palatability.
- ▶ Cooking eggs-destroys avidin (binds to biotin)
- ▶ Autoclaving legumes- destroy the trypsin inhibitor, improve protein availability.
- ▶ Destroys harmful food- borne microorganisms.

Adverse effects of processing:

Loss of nutrients – depends on

- Temperature
- Duration of cooking
- Nutrient involved.

Loss of nutrients occurs :

- By discarding the water used for cooking.
- Loss of K Ca – leaching.
- Loss of vitamin C – cutting the vegetables into very small pieces, exposed to environment before serving
- Preliminary washing of Rice - loss of thiamine, nicotinic acid 40%.
- Fat- repeated heating during frying.

CARBOHYDRATES

Contain carbon & hydrogen & oxygen in the same ratio as in water

- ▶ Major source of energy:
- ▶ Avg adult stores - 300g in liver & muscle tissue as glycogen.

▶ Functions:

- ▶ To provide fuel to the body.
- ▶ To facilitate oxidation of fats
- ▶ Contribute to the body structure.
- ▶ Provide energy to the oral and intestinal bacteria
- ▶ Provide protein, vitamins, minerals.
- ▶ Furnish fiber for normal peristalsis.

Sources of energy:

- Cereals
- Bread
- Vegetable- root tuber, seed,
- leafy types-glucose and fructose.
- Fruits.- fresh and dried-fructose, cellulose, hemi cellulose, dextrin, pectin.
- Sugar & corn syrup- combn of mono &disaccharide, - highest % of carbohydrate on a dry weight basis.

CLASSIFICATION:

➤ 3 groups:

- Monosaccharide
- Oligosaccharides
- Polysaccharides

OVERVIEW OF METABOLISM *

Metabolic pathways – 3 categories

1. Anabolic pathway
2. Catabolic pathway
3. Amphibolic pathway

Anabolic pathway: those involved in the synthesis of compounds. protein synthesis, such a pathway as in the synthesis of fuel reserve of triacylglycerol & glycogen.

Catabolic pathway : involved in breakdown of larger molecules, commonly involving oxidative reactions.

Amphibolic pathway: occurs at the cross roads of metabolism. Act as links between anabolic & catabolic pathway. E.g.; citric acid cycle.

CARBOHYDRATE METABOLISM :

(centered on the provision & fate of glucose)

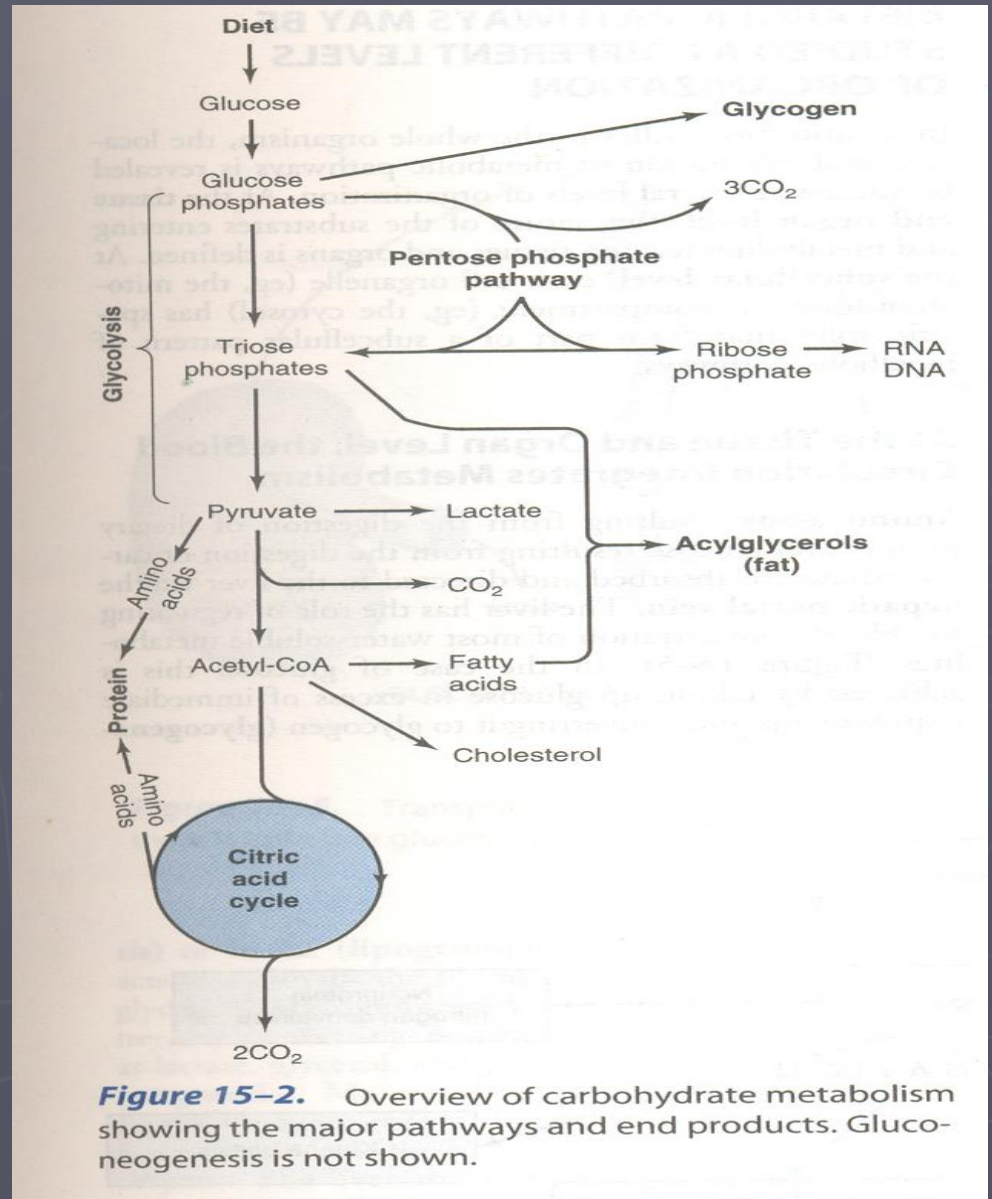


Figure 15-2. Overview of carbohydrate metabolism showing the major pathways and end products. Gluconeogenesis is not shown.

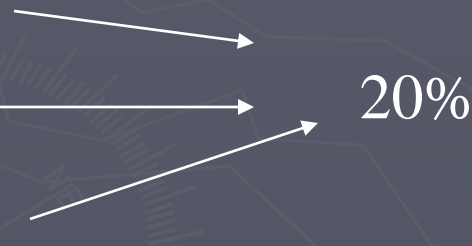
PROTEINS

- ▶ Derived – Greek word , ‘FIRST RANK’
- ▶ Next plentiful substance available in the body other than water.
- ▶ $\frac{1}{2}$ the dry weight of the body
- ▶ $\frac{1}{3}$ of body protein -- muscle
- ▶ $\frac{1}{5}$ -- bone and cartilage
- ▶ $\frac{1}{10}$ -- skin.
- ▶ Rest -- other tissue and body fluids.

FUNCTIONS:

1. Building, repair & replacement of body tissue.
2. Function as enzyme, hormone, regulators of fluid & acid base balance.
3. Transport molecules (hemoglobin).
4. Constituent of cytoplasm & nuclei of cells, serve as building block for cell membrane and tissue structure.
5. Serve as source of energy--carbohydrate and fats are inadequate.

SOURCES: In cytoplasm , cell membrane

- Mammalian muscle - 20%
 - Blood plasma- 7%
 - Cow's milk- 35%
 - Cereals- 12%
 - Beans
 - Nuts
 - Pulses
- 20%
- Enzyme, viruses, antibodies, hormones, wool, leather.
- 

Protein turnover:

- Mucous membrane of SI- 1-2days
 - Oral mucosa- within a week
 - RBC -120 days
 - 300g of the 10kg of body protein – replaced daily.
-
- Amino acids – metabolic pool.
 - Metabolic pool- aggregate of exogenous and endogenous protein metabolism,
 - a) amount and quality of dietary protein
 - b) rate of absorption of AA
 - c) endocrine balance.
 - AA – excess not stored.
degraded to a source of energy , excreted as urea.

NITROGEN BALANCE :

- Balance b /n intake and output of nitrogen in the body.
- Negative nitrogen balance – breakdown of body protein.
 - a) infection
 - b) fever
 - c) surgical trauma
 - d) blood loss
 - e) loss of plasma because of burns.
- Depends- amount and proportion of essential AA.
- Nitrogen equilibrium- adequate diet.

PROTEIN METABOLISM:

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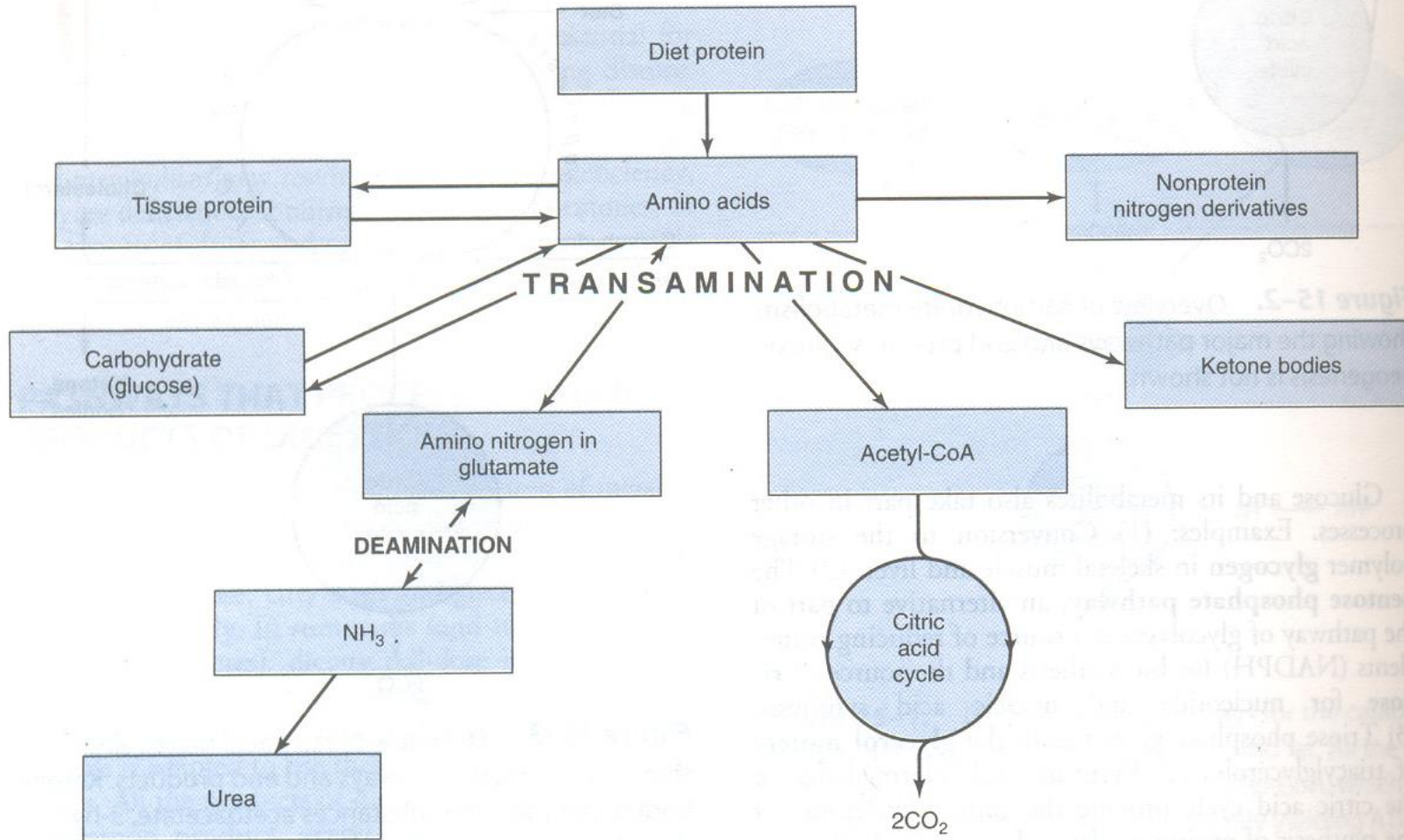


Figure 15-4. Overview of amino acid metabolism showing the major pathways and end products.

LIPIDS

Group of compounds that are not of uniform composition but are related by their relative insolubility in water & their solubility in organic solvents in either, alcohol & chloroform

FUNCTIONS:

- Phospholipids are an integral components of cells contribute to the proper functioning.
- True fats- excellent source of energy twice efficient as a source of energy- 9Cal/g
- Provide essential fatty acids . Arachodoinic acid, linoleic acid- indispensable for normal growth.

FUNCTIONS (contd)

- Carriers, - absorption of fat soluble vitamins
- Maintain body temp.
- cushioning mechanism against injury.
- pleasant flavor & consistency to food.
- Cause a sense of fullness & satisfaction.

SOURCES: 90% of fat:

- Salad, cooking oils, butter, margarine
- Meat poultry, fish
- Milk, cheese, yogurt
- Butter, margarine, vegetable oils- Vit A,E.

CLASSIFICATION:

1. SIMPLE LIPIDS: Neutral fats
Waxes
2. COMPOUND LIPIDS: Phospholipids
Glycolipids
Lipoprotein & others
3. DERIVED LIPIDS: Fatty lipids
Glycerol
Sterols and others

LIPID METABOLISM:

Mainly with the FA & cholesterol

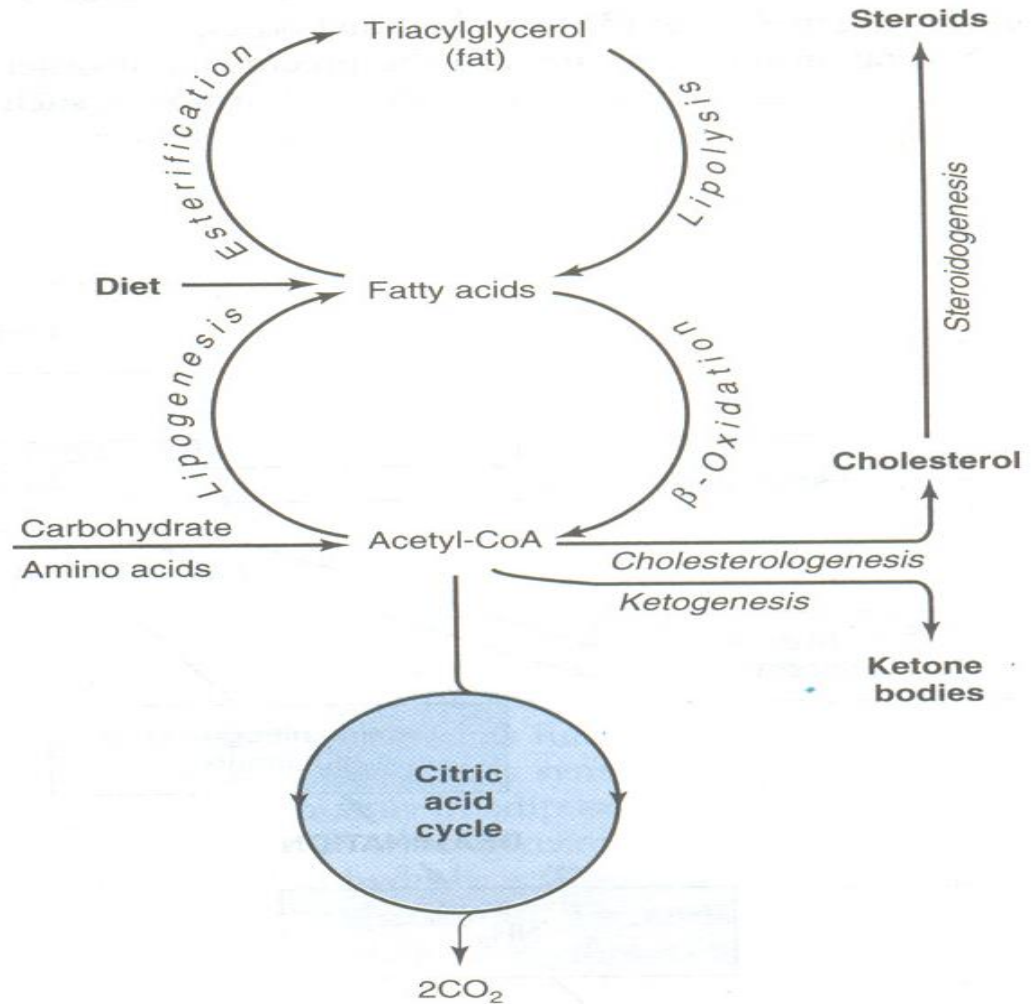


Figure 15-3. Overview of fatty acid metabolism showing the major pathways and end products. Ketone bodies comprise the substances acetoacetate, 3-hydroxybutyrate, and acetone.

Vitamins

An organic substance that occurs in foods in small amounts & is necessary for the normal metabolic functioning of the body.

FUNCTIONS:

- Acts as co-enzyme
- Regulate the metabolism by releasing energy from fats, carbohydrates
- Involved in AA metabolism, by releasing energy from fats, carbohydrates
- Acts as catalysts

Classification :

- Fat soluble-

1. Vit A,
2. Vit D
3. Vit E
4. Vit K

- water soluble:

1. Vit B
2. Vit C

TABLE 23-2. Fat Soluble Vitamins

Vitamin	Function	Sources	Deficiency Symptoms		
			General	Oral	Toxicity
Vitamin A (retinol) and Provitamin A (carotene)	Adaptation to dim light Promotes growth of bones Maintains epithelial cells of intestines and skin Maintains mucus and lacrimal secretions	Vit. A: Egg yolks, milk, butter, liver Provitamin A: Carrots, sweet potatoes, greens, winter squash, apricots, peaches, tomatoes	Night blindness Eye inflammation Bitot's spots Xerophthalmia, keratomalacia	Hyperkeratosis and hyperplasia of gingiva Dental abnormalities in animals	Anorexia Hyperirritability Skin lesions Bone decalcification
Vitamin D (calciferol)	Absorption of calcium and phosphorus	Vit. D: fortified milk, eggs, butter, cheese, fish (sunshine activates vit. D in our skin)	Rickets Osteomalacia		Calcification of the arteries Renal calculi Early symptoms: anorexia, lassitude, nausea, vomiting, diarrhea, weight loss

TABLE 23-2. Fat Soluble Vitamins

Vitamin	Function	Sources	Deficiency Symptoms		
			General	Oral	Toxicity
Vitamin E (tocopherol)	Antioxidant —thought to inhibit the oxidation of unsaturated fatty acids (constituents of cells and subcellular particles)	Vegetable oils Leafy green vegetables Germ portions of grain			
Vitamin K	Necessary for the synthesis of blood clot- ting proteins (prothrombin, proconvertin, Christmas and Stuart factors)	Green vegeta- bles Egg yolks Liver Soybean oil Synthesized by bacteria in in- testines	Hemorrhage (rare)		

TABLE 23-1. Water-Soluble Vitamins

Vitamin	Function	Sources	Deficiency Symptoms		
			General	Oral	Toxicity
Ascorbic acid (Vitamin C)	Collagen biosynthesis Converts folic acid to folinic acid (important in cell division) Elasticity and strength of capillaries	Citrus fruits Green peppers Tomatoes Strawberries Cantaloupe	Capillary fragility Scurvy	Gingivitis hemorrhagic enlarged, bluish-red gingiva	
Thiamine (Vitamin B ₁)	Forms thiamine pyrophosphate which assists in the breakdown and utilization of carbohydrates	Yeast Pork, liver, beef Dried peas, beans, nuts, whole grains	Beri-beri Peripheral neuritis Heart enlargement Leg cramps	Hyperesthesia of oral mucosa Burning tongue Loss of taste	
Riboflavin (Vitamin B ₂)	Catalyst in oxidation-reduction reactions in tissues Conversion of tryptophan to niacin	Milk, cheese, eggs Liver, heart, kidney Meat Leafy green vegetables	Ocular and skin changes	Angular cheilosis Glossitis	
Niacin (nicotinic acid)	Comprises enzymes DPN and TPN which function in cell respiration, carbohydrate metabolism, and fat	Meat, fish, poultry Liver Dry peas and beans Nuts, whole grain cereals	Pellagra: dermatitis in areas exposed to sun diarrhea dementia	Burning sensation of mouth, nausea, abdominal cramps	

TABLE 23-1. Water-Soluble Vitamins

vitamin	Function	Sources	Deficiency Symptoms		
			General	Oral	Toxic
Pyridoxine (B ₆)	Protein metabolism: for transamination and decarboxylation	Meat, liver Whole grains Bananas, cabbages	Deficiency is rare because of widespread distribution	Experimental cheilosis, stomatitis, glossitis	
Pantothenic acid	Part of coenzyme A: for the release of energy from carbohydrates	Meat, eggs, milk Whole grains Fruits, broccoli	Deficiency rare: Experimental deficiency: fatigue, malaise, nausea, abdominal distress		
Biotin	Catalyst: releases energy from carbohydrates	Liver, kidney, milk Egg yolk, yeast	Lassitude, anorexia, depression, dermatitis		
Folic acid (folacin)	Builds purines and pyrimidines for nucleic acid synthesis	Liver, kidney, yeast Leafy green vegetables	Macrocytic anemia		
B ₁₂ (cobalamin)	Maturation of red blood cells Carbohydrate metabolism Affects cells that are dividing rapidly	Animal foods—liver, meat, fish, eggs, milk, cheese	Pernicious anemia: large red blood cells, nervous disorders, weakness, fatigue		

Minerals

FUNCTIONS:

- ▶ Structural components for the body .
- ▶ Nerve, muscle function
- ▶ Blood clotting
- ▶ Tissue growth & repair
- ▶ Acid- base balance of body fluids
- ▶ Cofactors for enzymes in chemical reaction within the body

Classification:

- Major minerals: 100mg/ day.
- Trace minerals: lesser amounts

- Major minerals:

- | | | |
|------|-------|-----|
| 1.Ca | 4. K | 7.S |
| 2.Mg | 5. Na | |
| 3.P | 6. Cl | |

- Trace elements:

- | | | |
|-------|-------|------|
| 1.F | 5.Ch | 9.Mn |
| 2.Fe | 6. Cu | |
| 3.Zn | 7. I | |
| 4. Se | 8. Mb | |

Table 3
Minerals

Mineral	Actions	Sources	Deficiency
Calcium ^a	Bone/tooth formation; blood clotting; nerve/muscle function; CNS; blood pressure	Milk-based foods, sardines with bones, green leafy vegetables, legumes	Reduced bone density
Phosphorus ^a	Bone/tooth formation; metabolism; acid-base balance	Dairy foods, eggs, meat, fish, poultry, legumes, whole grains	Rare
Magnesium ^a	Bone/tooth formation; nerve and muscle function; blood clotting; cofactor in metabolism	Whole grains, green leafy vegetables, hard water, meats, dairy products, fish	Associated with FVD: weakness, muscle twitching, convulsions
Potassium ^a	Fluid/electrolyte balance; muscle and nerve function; hormone release	Whole grains, vegetables, meats, legumes, dairy foods, fruits, unprocessed foods	Associated with FVD: weakness, confusion, arrhythmias
Chloride	Fluid/electrolyte balance; gastric digestive acid	Table salt, processed foods	Associated with FVD
Sulfur	Component of body proteins (eg, hair, cartilage, nails)	Protein foods: eggs, meats, fish, poultry, legumes	Associated with protein deficiency
Sodium	Electrolyte/fluid balance; nerve function; blood pressure; acid/base balance	Table salt, processed foods	Associated with FVD: headache, cramps, weakness, confusion, decreased appetite

Fluoride ^a	Bone/tooth formation; increases resistance to caries	Fluoridated water, tea, seafood, seaweed	Increased dental caries
Zinc ^a	Required for digestion, metabolism, wound healing, tissue growth and repair, reproduction	Protein foods; meats, fish, poultry, eggs, legumes	Retarded growth; taste/smell alterations; decreased immune function and wound healing; slow physical/sexual maturity
Iron ^a	Growth; immune system health; hemoglobin and myoglobin formation; energy production	Liver and other meats, fish, eggs, poultry, green vegetables, legumes, enriched breads and cereals	Microcytic anemia (women and children at risk)
Copper ^a	Coenzyme in antioxidant reactions and energy metabolism; iron use; wound healing; blood and nerve fiber production	Organ meats, seafood, green leafy vegetables, nuts, seeds, water from copper pipes	Bone demineralization and anemia

Table 3 (continued)

Mineral	Actions	Sources	Deficiency
Iodine ^a	Thyroxin synthesis; regulates metabolism, growth, and development	Iodized salt, seafood	Goiter, tiredness, weight gain
Selenium ^a	Antioxidant; may be helpful in periodontal disease	Meats, fish, eggs, whole grains	Predisposition to heart disease
Chromium	Carbohydrate metabolism	Whole grains, cheese, meats, brewer's yeast	Possible cardiovascular disorders and insulin dysfunction
Molybdenum	Coenzyme	Whole grains, legumes, milk	Unknown
Manganese	Metabolic reaction participant	Whole grains, green leafy vegetables, legumes	Unknown

^a These minerals are associated with oral health promotion.
 CNS, central nervous system; FVD, fluid volume deficit.

Calcium metabolism

Body content, distribution :

- ▶ Avg sized man – 1kg of Ca in his body.
- ▶ 99% of body – bone
- ▶ Rest – cells, ECF.

Functions:

2 major functions.

- ▶ Functions due to Ca in bone & ECF.
- ▶ Functions due to presence of Ca^{++} in the cytosol.

Functions due to the Ca in the bone & ECF:

- Gives strength to the bone
- Takes part in the homeostasis
- Required in regulating neuromuscular activity
- Required in regulations of hormones-PTH, thyroid hormones,

Functions within the cell wall: ,

- For Muscular contraction- cardiac muscle contraction
skeletal muscle contraction
- Act as secondary messenger
- Responsible for release of Ach as neurotransmitter

Sources:

- Milk & milk products
- Eggs

Absorption:

principally – upper part jejunum
Other parts of the intestine as well.

Mechanism of absorption of Ca:

1. Chiefly by a carrier mediated mechanism.
 2. Simple diffusion
- Carrier mediated molecules – brush border of jejunal mucosa
 - Derivative of Vit D- 1,25 dihydrocholecalciferol-facilitates carrier mediated transport

Ca absorption in GIT--

- Presence of calcitriol
- Need of Ca in the body
- Both are interdependent

Water and Electrolytes

- ▶ Vital nutrient ,
- ▶ Varies from-50-70%.
- ▶ 2 main compartments- Extracellular , intracellular.

For a 70kg man,

Intracellular water-50% of body wt, - 35lts

Extracellular water 20% of body wt - 14lts

i) plasma 5% of body wt - 3.5lts

ii) interstitial fluid 15% of body wt - 10.5lts

FUNCTIONS:

- Maintains electrolyte balance.
- suitable medium for chemical reaction in the body.
- solvent & as a vehicle to transport inorganic solvents.
- contribute to the temp regulation.
- Transports heat from one part of the body to the other, thus stabilizing body temp.
- Helps in normal functioning of kidneys.
- Dissolves enzymes, hormones, co-enzymes.
- Lubricant in digestion
- Active role in hydrolysis of nutrient.

WATER REQUIREMENT:

- Based on body size
- Determined by- per kg body wt.

	Age in yrs	Water (ml/kg body wt
Infants	Birth- 1yr	100-120
Children	1-10	60-80
Adolescents	11-18	41-55
Adults	19-51	20-30

Special attention:

- Water needs of infants given protein formulas.
- Comatose pts
- Pts with fever, polyuria, vomiting , diarrhea
- Person on diuretic medication
- Pts consuming high – protein diet.
- Persons in hot environment.

SOURCE:

- Drinking water
- Beverages
- Raw fruit & vegetables
- Cooked meat, eggs, bread

Body fluids:

- Intimately involved in the basic cellular process.
- consists – Water, protein, solutions of electrolytes, nonelectrolytes
- In healthy person - volume & composition of body fluids carefully regulated
- Illness, trauma, surgery – alteration of fluids takes place- dehydration- shock – even death

Water balance:

achieved when –

1. There is an osmotic equilibrium b/ n the diff body fluid

Compartments

2. Water intake equals the water output.

extra cellular fluid, intracellular fluid- permeable membrane

Water moves from the Extracellular fluid



Intracellular fluid(\uparrow solute concn) (and vice versa)

- Eg: dehydration,
- Changes in osmotic pr \rightarrow hypothalamus receptors \rightarrow ADH hormone \rightarrow reasorb water throug renal tubules \rightarrow \downarrow urine output

WATER METABOLISM IN INFANTS:

- 80% of water.
- ECF- 20%
- Infant of 7.0kg – ECF – 1.4lts.

- main diet – liquid

- input & output/ day - 0.7lts, ECF will be 50%.

- Adult ECF vol. - 14lts, input & output is 20-25lts, 14-16% of ECF vol.

Clinical implication:

- Fluid balance should be maintained ,
- Any deficit / excess intake- shrinkage of ECF vol.

NUTRITION AND DENTAL CARIES *

1. Interventional human studies
 - a) Vipeholm study
 - b) Hopewood study

2. Noninterventional human studies

3. Special population groups
 - a) hereditary fructose intolerance
 - b) hereditary intestinal sucrase deficiency

4. Occupational risk
 - a) confectionary workers
 - b) workers of fruit orchid.

VIPEHOLM DENTAL CARIES STUDY (1945-54) :

- ↑ sugar consumption- ↑ caries incidence.
- Consumption of retentive forms of sugar (8- 24 toffees/day) between meals - ↑ caries
- Consumption of sweet bread &/or sugar solun at meal times with max of 4 times- little effect.
- Quantity, frequency of sugar consumption with consistency

HOPEWOOD HOUSE STUDY: *

- Dental survey- age 5 & 13 – avg def, DMFT scores of 1.1 or about 10% of caries of gen populn of that age group.
- Same children were relocated and DMFT scores evaluated.
- Steep increase in the DMFT scores above 13 yrs of age.
- Didn't stick on to the original diet.

CARIES REDUCES ALONE WITH DIETARY MEANS INSPITE OF UNFAVOURABLE ORAL HYGIENE , FLUORIDE LEVELS.

Noninterventional human studies:

- Subjects- free to choose what ever they eat they please
- Longitudinal studies in England and US: 2-3 yrs
- Multiple questionnaire- data analyzed in computer

1. Children with high caries increments- more snack foods

2. Total calorie intake from snack carbohydrate & snack sugars- significantly high

Special population groups:

Hereditary fructose intolerance:

- Rare AR disorder of fructose metabolism.
- Intolerance of fructose- drastically reduced activity (2-5% of normal) of **Fructose 1- phosphate adolase** in liver, lower renal cortex, SI
- **Sweets, candies, cakes, most of the fruits- AVOIDED**
- **Milk dairy products, oats cereals, noodles- CONSUMED**

Studies-

- Caries incidence – low or free of caries
- If present – smooth surface caries.

Occupational risk:

confectionary workers:

Epidemiological survey –

- High caries prevalence DMFT 15.59 ± 5.97 than textile workers (DMFT 9.14 ± 4.75).

Epidemiological survey –

- Sugar cane cutters – raw sugar cane juice
- Cuban sugar cane worker with a similar age group of textile worker showed **higher caries incidence**.

RAW SUGAR CAN BE DELETERIOUS TO DENTAL HEALTH AS REFINED SUGAR

Dietary factors in caries promotion

Dietary factors in caries inhibition

Dietary factors in caries promotion

- Specific foods
- Unique forms
- Frequency intake

Dietary factors in caries inhibition

- Dairy products
- Sugars substitute & alternative sweeteners
- Plant foods
- Other foods

Specific foods:

Caries promoting potential ;

- **Simple sugars;** - dextrin , corn syrup , fruit sugar, levulose, table sugar, high fructose corn syrup, molasses,
- **Polysaccharide (starch)**- cooked potatoes, rice, legumes, corn starch, bananas.

• **Fiber – cellulose, pectin –NO CPP**

• **High intensity sweeteners- NO CPP**

aspartame

saccharin

sucralose

Burt BA et al - Regular exposure to fluoride reduces the risk of caries.

Lingstorm et al – whole grains , raw vegetables- lower CPP than heat processed breads, crackers, chips, dry cereals.

UNIQUE FORMS: *

BIBBY & MUNDROFF- 180 foods.

snacks with high sugar concn did not destroy much tooth enamel in a test solution as did low concn sugary foods in combn with starch.

GRENBLY et al, MORMAN et al – processed high starch produces as much as acid in dental plaque as sucrose alone but at slower rate .

BIBBY et al, KASHKET et al : high-sugar foods (caramels, chocolate,) clear the mouth more rapidly than high starch foods (crackers, potatoes, chips).

FIRESTONE AR et al*: raw vegetables < nuts < milk < corn < chips < fresh fruit < ice cream < French fries, < dried fruit

FREQUENCY OF DIETARY INTAKE:

- 4-6 times/day
- Frequent nibbling sipping – sugary/starch , highly processed foods, beverages >30 mins ↑ caries.
- ↑ frequency- ↑ total fermentable carbohydrate.

Frequency ,amount of sugar intake - α caries.

Dietary factors in caries prevention:

- Dairy products
- Sugar substitutes & other alternatives
- Plant foods
- other foods

Dairy products:

BOWEN WH et al –

- Milk – caries inhibiting potential when consumed after sugary foods.
- Ca, P which are bound casein protein in milk – protective effect on tooth enamel
- Cheese – anticariogenic, stimulates salivary flow- buffering effect & neutralization of plaque acids.

Sugar substitutes & alternatives sweeteners:

- Nutritive sugars- Sorbitol, mannitol, xylitol - 40-70% caloric content
sugars - low caries potential

- **MAKINEN et al** –

- Evaluated the effect of Sorbitol or xylitol on caries incidence-, caries rate - ↓ 30-60 %

- Xylitol chewing gums, mints - ↓ caries.

Nonnutritive intense sweeteners-Saccharin, aspartame, acesulfame K, sucralose cyclamate.

Plant foods:

- Grains, vegetables – anticariogenic agents.

- include – organic phosphate, inorganic phosphate, polyphenols, phytate etc

- Fibrous foods- stimulate salivary flow.

OTHER FOODS:

- Green, oolong, black tea-fluoride polyphenols / flavonoids- ↓oral bacterial growth.
- Oleic lenolic fatty acids in cocoa beans - streptococcus mutans.
- Licorice candies of glycyrrhizinic acid - ↑ plaque buffering capacity.
- Peanuts – mechanical stimulation , salivary flow.

Nutrition in growth and development of oral structures

ENAMEL:

- clinical studies – rickets during the time of tooth formation cause enamel hypoplasia
- Shelling & Anderson –in series of rachitic children 43% of teeth – enamel hypoplasia.
- Hypoplasia - pitting variety. Pits tend to stain.
- Involve those teeth that form within the 1st yr of birth.
- Central ,lateral incisors, 1st molars.
- Premolars, 2nd , 3rd molars – seldom affected .

- Vit A- primarily concerned with the process of differentiation of epithelial cells
- Def of Vit A, epithelial cells fail to proliferate.
- Studies – in developing tooth of a rat with def Vit A, odontogenic epithelium



fails

normal histodifferentiation & morphodifferentiation



increased cell proliferation.

- Young rats whose mothers are maintained on a diet def Vit A- 5 months ---
 - a) severe changes in enamel,
 - b) Distortion of shape of incisors, molars
- Harris& Navia --- ↑ caries susceptibility of rats molars of pups which were fed by Vit A def diet, indicating the pre-eruptive role for Vit A tooth development.
- Post eruptive Vit A def - ↑caries scores
- Sally co workers – ↑caries scores , due to salivary gland function than to dental changes.

DENTINE:

- Studies – Def Vit A causes changes in dentine
 - a) Tubular arrangement is disturbed
 - b) Contains cellular & vascular inclusions.

- Vit C def in guinea pigs – atrophy & disorganization of the odontoblasts --- irregularly laid down dentine with irregularly arranged tubules

- Dentin formation ceases,

- Predentin becomes hypercalcified producing heavy basophilic staining line b/ n dentin & pulp

PERIODONTAL LIGAMENT & ALVEOLAR BONE:

Goldman --Vit C def in scorbutic monkeys

- hypertrophy of the gingiva covering the entire crowns.
- Subperiosteal hemorrhages from the underlying bone.
- Focal areas of necrosis have been reported
- alveolar bone showed atrophic changes,
- Marrow spaces were replaced by fibroblasts growing in an edematous space

Protein calorie malnutrition –gestation neonatal period-

a)enamel hypoplasia

b)dental caries- primary dentition.

ORAL EPITHELIUM:**

Nutritional stress-critical period– impair the renewal of sulcular epithelium & compromise an important component of its defense mechanisms

BONY ORAL TISSUE:

- **Kuftinec** -bone development is similar to that of soft tissues
- Animal models-increased bone growth- period of weaning (period of low to high protein diet).
- Nutrient def, environmental factors- irreversible effect on growth.
- Prolonged Vit A def + PEM –
 - a) inadequate bone growth pattern
 - b) malalignment & malocclusion

ON CLEFT LIP & PALATE:

- Vit A - ↓ in folic acid supplements ↑ neural tube defects.
- Mills et al – prevalence of mutation in methylene tetrahydrofolate reductase and is 3 times > ICP, CLP
- Impairment of foliate metabolism - orofacial clefting
- Riboflavin - organogenesis.

PROTEIN NUTRITION IN ORAL HEALTH AND DISEASE

Effect of protein deficiency on the jaw and teeth:

- consumption of too little EAA –critical period of active growth – permanent structural damage.
- PEM- rotated teeth, crowded, retarded growth of jaw bone.
- BAVETTA et al-protein def diet to rats
- stringent def of AA lysine or tryptophan – irregular predentin layer & a no. of interglobular spaces in poorly calcified dentinal matrix.

Nigerian children with kwashiorkor –
delayed eruption
hypoplasia of deciduous teeth.

Effect of protein deficiency on dental caries:

- Animal studies- lysine and glycine- prevent caries
- Quantity & quality of protein.

**IN HUMANS, NO DIRECT EVIDENCE-
B/N DENTAL EXPERIENCE & DIETARY
SUPPLEMENTS**

ROLE OF PROTEIN IN BIOCHEMISTRY OF PERIODONTAL TISSUE:

Collagen deficiency in CT is due to

1. Inability of fibroblasts to synthesize collagen due to lack of proline & lysine
2. Failure of soluble collagen to form insoluble fibers.
3. Degeneration of collagen.

Effect of protein on periodontal disease:

- Experimental animal studies - ↓ dietary protein intake affects fibroblasts, osteoblasts, cementoblasts.
- Histological- atrophic degenerative changes in CT of gingiva, pdl.
- **Human studies – inconclusive and controversial.**

FATS AND ORAL HEALTH

DENTAL CARRIES:

- Indirect effect – help in preventing caries
E.g.: Eskimo- diet of animal origin, 70-80% fat.
- Studies – diet containing fats - ↓ 25% - caries starts.

Mechanism:

- Coating of the oily substance on the tooth surface - ↓ retention of food.
- ▶ Fatty protected layer over plaque.-prevent fermentable sugar substrate from being reduced to caries
- ▶ Higher concn of fatty acids- interfere growth of bacteria
- ▶ ↑ amount of fat will ↓ amount of dietary fermentable carbohydrate for organic acid production.

PAROTID ENLARGEMENT:

- Studies – chronic swelling of parotid glands can be the result of disturbed lipid metabolism that accompanies alcoholism.

Alcoholism → fatty degeneration in liver , parotid glands,



enlargement of glands.

EFFECTS OF WATER BALANCE ON ORAL HEALTH

- ▶ Oral mucosa- very sensitive to fluid volume.
- ▶ Pts with xerostomia, dry, shrunken, fissured tongue or MM,- vol. deficit.
- ▶ Pts- rapid loss wt or whose denture suddenly feels loose – vol. deficit.
- ▶ Pts – edema ,feel their denture tight, presents with mucosal irritation .
- ▶ Enquire about the medication .
- ▶ Pts on high protein diet – increase amount of water to eliminate waste product.

Effects of Trace elements on oral health

Trace elements:

- ▶ 1.F
- ▶ 2.Fe
- ▶ 3.Zn
- ▶ 4. Se
- ▶ 9.Mn
- 5.Ch
- 6. Cu
- 7. I
- 8. Mb

Molybdenum

- In human tissues - principally in liver, kidney, fat and blood, concentrations are not high.

Effects on oral tissue:

more studies- the relationship of Mo to dental caries*

confusing results , no clear picture of the relationship, or role,

Several epidemiologic studies - possible anticariogenic element.

Selenium

Effects on oral tissue

effect dental caries - paradoxical

- some studies-Se is ingested during tooth development, caries promoted while other studies give equivocal results.
- nine studies - last 43 years , relationship b/ n Se and DC in man.
- Six -↑amounts of Se in the diet are associated with dental caries

Cadell and Cousins, 1960;Retief et al, 1976

conducted in areas of low Se - no relation between Se and caries.

Curzon and Crocker, 1978 -found a negative association between caries and the Se content of teeth

Kaqueles et al (1977)

paradoxical effect of selenate on caries development in the rat.

- Inj of high doses of selenate promoted caries
- ↓doses of Se may be required for optimal calcification.
- ↑doses may be detrimental to enamel development
may promote caries.

VANADIUM:

- 0.1% and 1% of a dose of V is absorbed from the intestinal tract in man of which 60% is excreted in the urine within 24 hours.
- An inverse relationship b/ n V concn in water supplies & DC
- Low frequencies of caries – V concn ↑ & unrelated to the F water concentrations., found to be related to ↑ Ca and Mg, as well as total water hardness.
- South American study - V ↑ in soils - low caries prevalence.

Zinc

- ▶ Relationship to caries in man is very thin, unlike Se, Sr, and Mo
- ▶ relate variations of Zn intake, via food or drinking water to caries.
- ▶ A daily admin of 3 mg ZnSO₄ for three months, gave a drop in increasing caries incidence.
- ▶ Zn concentration ↑ in the enamel in white population groups with higher caries incidence in comparison to black population groups with low caries incidence (**Retief et al, 1978**).
- ▶ limited number of human studies

Magnesium:

- The evidence relating Mg to dental caries is equivocal.
- In vivo epidemiological studies in man- inverse relationship to caries
- Animal studies - the opposite
- In vitro studies- Mg enter & effect the properties of the enamel crystallite but whether this results in any resistance or susceptibility to dissolution remains unclear.

MANGANESE

- ▶ The effects of Mn on dental caries in man are not well understood.
- ▶ positive relationship between Mn in saliva and caries is confused
- ▶ Acid and (IPS) production from glucose by seven *S. mutans* serotype strains - stimulated by the addition of Mn - growth of these organisms was unaffected.
- ▶ **Beighton (1982)** - Mn stimulated acid and IPS (iodophilic polysaccharide) production from sucrose and fructose by *S. mutans* strain Ingbritt

Sulfur:

- ▶ No direct evidence - between dietary amino acids & DC in humans,
- ▶ to date no studies - explored specific deficiencies of sulfur amino acids wrt dental development or caries in man.

Copper:

- The role of Cu in dental disease is not clear.
- difficulty of separating the effects of Cu from those of other elements, such as Zn, Pb, Cd or Fe, which often occur together in water or food.
- Cu and dental disease is negligible.
- Cu may have a role in influencing periodontal disease and alveolar bone resorption--more work is needed

Interaction of immunity, infection & nutritional status

- ▶ Nutrition - Critical determinant of immune responses
- ▶ even mild infection – adverse effects on the nutritional status

Several factors :

Concentration of the nutrient & its interaction with the other key nutrients.

Duration of the nutrient imbalance

Age of the host

Effects of infection on nutritional status:

- Imbalance b /n catabolic and anabolic substances – cytokines & glucocorticoids.
- 0.6 g of protein / kg / day
- Trauma induced surgery- ↑ excretion of N_2 .
- Mild stress – 0.8-1.0 g of protein /kg body wt /day.
- Anorexia- under nutrition, negative N_2 balance.

- GIT – negative impact.
Disruption GI mucosa - ↓ nutrient absorption.

protein absorption – ↓ 10-30%.

vitamin absorption - ↓ 30-70%
- Fever – for each 1° F - ↑ 9% calorie intake
- Hypermetabolism – 1.2 % - 100 % calorie intake.
Eg – burns.

Effects of nutrition on immune responses

Nutrient	Function	Deficiency impact on immune response ^a
Protein energy intake	Energy metabolism DNA/RNA synthesis	<ul style="list-style-type: none"> ↓ salivary antimicrobial properties ↓ immunoglobulin production ↓ lysozymes ↑ bacterial adhesion ↓ activation of lymphocytes ↓ production of antibodies
Vitamin A	Cellular differentiation and proliferation Integrity of the immune system	<ul style="list-style-type: none"> ↓ immune cell differentiation ↓ response to antigens ↓ antibody production ↑ bacterial adhesion ↓ immunoglobulin production ↓ production of lymphocytes
Vitamin E	Antioxidant protecting lipid membranes from oxidation	<ul style="list-style-type: none"> ↓ antibody synthesis ↓ response of lymphocytes ↓ phagocytic function
Vitamin C	Antioxidant that reduces free radicals that cause DNA damage to immune cells	<ul style="list-style-type: none"> ↓ phagocytic function of neutrophils and macrophages ↓ antibody response ↓ cytotoxic T-cell activity

Nutrient	Function	Deficiency impact on immune response ^a
Riboflavin, vitamin B ₆ , and panthothenic acid	Coenzymes in metabolic processes	<ul style="list-style-type: none"> ↓ antibody synthesis ↓ cytotoxic T-cell activity ↓ lymphocyte response
Folic acid and vitamin B ₁₂	Involved in DNA/RNA synthesis	<ul style="list-style-type: none"> ↓ production of lymphocytes ↓ cytotoxic T-cell activity ↓ phagocytic function of neutrophils
Zinc	<p>More than 100 enzymes associated with carbohydrate and energy metabolism</p> <p>Protein catabolism and synthesis</p> <p>Nucleic acid synthesis</p>	<ul style="list-style-type: none"> ↓ antibody response ↓ phagocytic function of macrophages ↓ B-cell and T-cell proliferation
Iron	Involved in hemoglobin, myoglobin, and cytochrome systems	<ul style="list-style-type: none"> ↓ lymphocyte proliferation ↓ neutrophil cytotoxic activity ↓ antibody response

- **Johnson et al** - vol. & antimicrobial properties of saliva (Ig A)- ↓protein intake < 5-8 % calories.
- **Chandra et al** – lysosomal concn of saliva - ↓ due to ↓neutrophils & monocytes.
- Bacterial adhesion to epithelial cells- ↑ in PEM.
- ↓ antimicrobial properties of saliva- ↑ overgrowth of anaerobic micro flora

Micronutrient deficiencies & immune responses:

1. The extent of impairment depends:
 - a) the type of nutrient involved
 - b) its interaction with other essential nutrients
 - c) the severity of the deficiency.
 - d) presence of concomitant infection
 - e) the age of the pt.
2. Extent of the abnormalities in the immune response which predict the risk of infection and the mortality.
3. Excessive intake is associated with impaired immunity.
4. Tests of immunocompetence may be useful in assessing the appropriate levels of micronutrient intakes to optimize the immune response.

Nutritional assessment

Christakis: “ Health condition of individual as influenced by his intake & utilization of nutrients determined from the correlation of informed from physical, biochemical, clinical & dietary studies.”

- a) Pts complaints & medical & social histories.
- b) Dietary history & evaluations
- c) Physical examination- anthropometric measurements
- d) Pertinent lab test.

COMPLAINTS:

- general weakness
- Chronic fatigue
- Loss of appetite
- Painful bleeding gums
- Loss of wt
- Sore lips, OMM, tongue
- Irritability
- Loss of ability to concentrate.
- Loss of manual dexterity

Medical and social histories:

- Chronic debilitating diseases
- Alcoholism
- Digestive disturbances
- Ulcerative colitis
- Diarrhea

Emotional problems:

- Excessive wt loss – anorexia nervosa
- Food fads
- medically unsupervised quick wt reduction diets

DIETARY HISTORY & EVALUATION:

- 24 hr recall
- 3day record
- 5 day record
- 7 day record

•Adequacy of food =
$$\frac{\text{Nutrients consumed (nutritive values of food)}}{\text{RDA}}$$

Physical signs of malnutrition

TABLE 22-3. PHYSICAL SIGNS AND CAUSES OF MALNUTRITION

BODY AREA	SIGNS ASSOCIATED WITH MALNUTRITION	NUTRITION-RELATED CAUSES
Hair	Lack of natural shine; dull, dry, sparse, straight, color changes (flag sign); easily plucked	Protein-calorie deficiency; often multiple coexistent nutrient deficiencies
Face	Dark skin over cheeks and under eyes (malar and supraorbital pigmentation), scaling of skin around nostrils (nasolabial seborrhea) Edematous (moon face) Color loss (pallor)	Inadequate caloric intake; lack of B complex vitamins, particularly niacin, riboflavin, pyridoxine Protein deficiency Iron deficiency, general undernutrition
Eyes	Pale conjunctivae Bitot's spots ^o , conjunctival and corneal xerosis ^o , soft cornea (keratomalacia) Redness and fissuring of eyelid corners (angular palpebritis)	Iron deficiency Vitamin A deficiency Niacin, riboflavin, pyridoxine deficiency
Skin	Follicular hyperkeratosis ^o , dryness (xerosis) with flaking Hyperpigmentation ^o Petechiae ^o Pellagrous dermatitis ^o Scrotal and vulval dermatosis ^o	Vitamin A deficiency; insufficient unsaturated and essential fatty acids B ₁₂ , folic acid, niacin deficiency Ascorbic acid deficiency Niacin or tryptophan deficiency Riboflavin deficiency
Nails	Spoon nails (koilonychia), brittle or ridged	Iron deficiency

Effects of nutrition on oral cavity:

3 MAJOR CHANGES ;

- Functional
 - Anatomical
 - Color
-
- Functional changes- burning sensation of mouth
nonspecific*
resolve within weeks

Anatomic changes: chronic disturbances
weeks – months

Color changes-

<i>Lips</i>	Redness and swelling of mouth or lips (cheilosis)*, angular fissure and scars	Niacin or riboflavin deficiency
<i>Tongue</i>	Red, raw and fissured, swollen (glossitis)* Magenta color Pale, atrophic Filiform papillary atrophy Fungiform papillary hypertrophy	Folic acid, niacin, B ₁₂ , pyridoxine deficiency Riboflavin deficiency Iron deficiency Niacin, folic acid, B ₁₂ , iron deficiency General undernutrition
<i>Teeth</i>	Cariious or missing Mottled enamel (fluorosis)	Excess sugar (and poor dental hygiene) Excess fluoride
<i>Gums</i>	Spongy, bleeding, may be receding	Ascorbic acid deficiency
<i>Glands</i>	Thyroid enlargement (goiter) Parotid enlargement	Iodine deficiency General undernutrition, particularly insufficient protein

Anthropometric measurements:

- Valuable index for measurements for nutritional status'
- Weight
- Weight for height
- Mid upper arm circumference
- Dugdale index
- QUAC- stick method
- Shakir tape method
- Bangle method

Weight:

Simplest, most widely accepted, most reliable, provided recorded correctly to the corrected age.

Difficult- underprivileged, ignored sections of the society.

Weight eight for height ratio:

Partially age independent,

$$\% \text{ weight for height} = \frac{\text{actual weight} \times 100}{\text{expected weight for actual height}}$$

Mid-upper- arm-circumference:

- Mid-way between the point of the shoulder (acromian) & olecranon process
- Left arm with hanging down by the side of the body
- 2-3 yrs-----16.25-16.75cm.
< 80% i,e 12.5—malnutrition

Pediatric nutrition disorders

PROTEIN ENERGY MALNUTRITION:

class of clinical conditions that may result from varying degree of protein lack and energy inadequacy.

Classification :

1. Syndromal classification
2. Gomez classification
3. Wellcome or international classification
4. Classification of IAP
5. Jelliffe classification
6. McLaren classification
7. Water classification

CLASSIFICATION

I. Syndromal Classification

- Kwashiorkor
- Nutritional marasmus
- Marasmic kwashiorkor
- Prekwashiorkor
- Nutritional dwarfing

II. Gomez Classification

According to this classification, PEM is graded with reference to the weight for age as percentage of the expected weight (Harvard standard) as below:

- | | |
|---------------|---------------------------------------|
| First degree | weight between 90 and 75% of expected |
| Second degree | weight between 75 and 60% of expected |
| Third degree | weight below 60% of expected. |

III. Wellcome or International Classification

Weight between 80 and 60% of expected
with edema kwashiorkor ·
without edema undernutrition

Weight below 60% of expected
with edema marasmic kwashiorkor
without edema nutritional marasmus

Classification:

IV. Classification of Indian Academy of Pediatrics

First degree	weight between 80 and 70% of expected
Second degree	weight between 70 and 60% of expected
Third degree	weight between 60 and 50% of expected
Fourth degree	weight below 50% of expected

In case the patient has demonstrable edema, the letter "K" is placed in front of the evaluated grade.

V. Jelliffe Classification

First degree	weight between 90 and 80% of expected
Second degree	weight between 80 and 70% of expected
Third degree	weight between 70 and 60% of expected
Fourth degree	weight below 60% of expected

VI. McLaren Classification

Mild	weight between 90 and 80% of expected
Moderate	weight between 80 and 70% of expected
Severe	weight below 70% of expected

VII. Waterlow Classification

Acute (wasted but not stunted)	weight for height low height for age normal
Acute/chronic (wasted and stunted)	weight for height low height for age low
Nutritional dwarfing (stunted but not wasted)	weight for height normal weight for age low height for age low

As per recommendations of the Indian Academy of Pediatrics, until acceptable Indian data become available, we should continue referring to

KWASHIORKOR

Infants, preschool children – 1- 4 yrs

Clinical features:

➤ Diagnostic features:

- Growth retardation- low wt and height
- Muscle wasting with retention of some subcutaneous fat.
- Psychomotor changes – mental apathy, lack of interest in surroundings
- Hypoalbuminemic pitting edema- legs and feet

Other features :

- Hair changes – hypochromotrichia, texture, alternate bands of light & dark color hair (flag sign)
- Skin changes-areas of pigmentation intervened by areas of raw red skin caused by shedding of superficial skin- **flaky paint dermatosis.**
- GI- diarrhea, vomiting, anorexia.
- Mineral deficiencies
- Hepatomegaly
- Clubbing.
- Super added infections

- Hypoalbuminemic pitting edema- legs and feet .



Fig. 8.9: Kwashiorkor, showing massive edema over feet and legs also mosaic dermatosis. This is the same child as in Fig. 8.8

Hair changes –
hypochromotrichia, texture,
alternate bands of light &
dark color hair (flag sign)

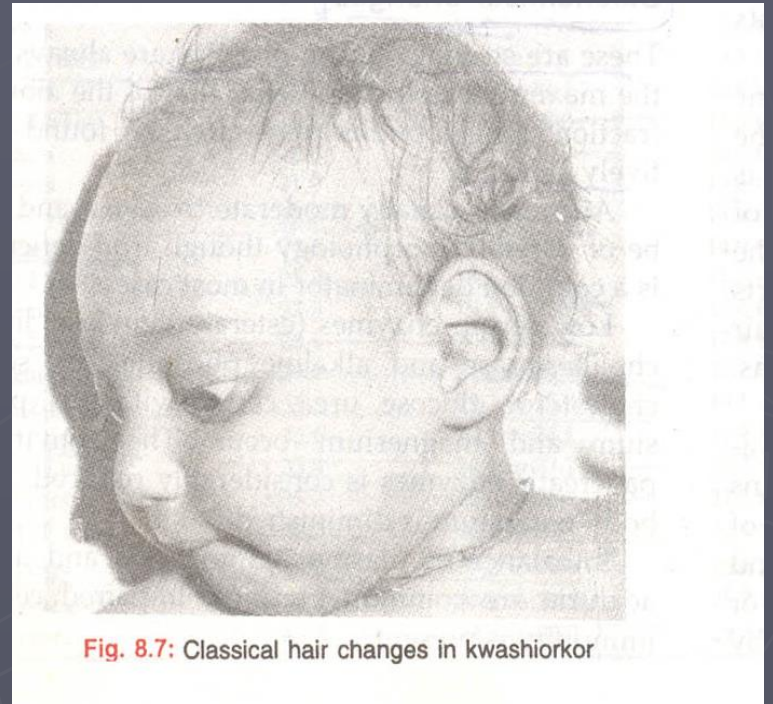


Fig. 8.7: Classical hair changes in kwashiorkor

Psychomotor changes – mental apathy, lack of interest in surroundings, typical moon shape face



Fig. 8.8: Kwashiorkor, showing characteristic hair changes, mental apathy and moon facies with periorbital edema

Oral findings:

- Bright reddening of the tongue.- loss of papillae
- Bilateral angular cheilosis
- Fissuring of lips
- Loss of circumoral pigmentation
- Growth of jaws- ↓
- **VAN WYK** – dry dirty, caries free, easily traumatized
- Epithelium – readily detachable from underlying mucosa- raw bleeding surface.
- Cytologic smears – perinuclear vacuolization / halo around nucleus

Biochemical changes:

- serum proteins- low albumin, globulins- elevated
- anemia – iron def anemia
- Pancreatic enzymes activity- ↓
- impaired cellular immunity
- imbalance - plasma amino acid amino aciduria.

NUTRITIONAL MARASMUS: (athrepsia, infantile atrophy)

North India

< 3yrs of age , \uparrow 1st yr .

Diagnostic feature:

- Growth retardation
- Gross muscle wasting as well as subcutaneous fat wasting.
- **ABSENCE OF EDEMA**



Fig. 8.16: Marasmic kwashiorkor in a 9-year-old girl. Note the wasting of the highest order. She also had multiple worm infestations and tuberculosis

Other features:

- Hair change – absent
- Classical dermatosis- absent
- GI- vomiting , diarrhea.
- Mineral , Vit def - common
- Psychomotor changes- irritability.
- Clubbing – not common

Management of PEM:

Domiciliary management:

- Children - mild to moderate nutrition
- Best managed in homes.
- Educate parents

Hospital management:

Advance management:

- 100 kcal/ kg of expected wt, 3g/kg of proteins – actual wt.
- Feed in small amounts
- Frequent intervals
- Diet therapy – tube feeding
- Associated anemia – treated
- Superadded infection – chemotherapy.

Avitaminoses

Vitamin A:

OCCURRENCE:

malnutrition, ch GI disorders,
pancreatic diseases-cystic fibrosis of pancreas,
hepatic insufficiency.

DIARRHEA , SEVERE MEASLES- RISK FACTORS

CLINICAL FEATURES:

1. Night blindness
2. Xerophthalmia
3. Keratomalacia

Infants- keratinizing metaplasia – trachea, bronchi, kidney,
pelvis, conjunctiva, sal glands, GUT

Oral findings:

<6 yrs of life.

Failure of odontogenic epi - histodifferentiation,
morphodifferentiation



↑ cell proliferation

Keratomalacia

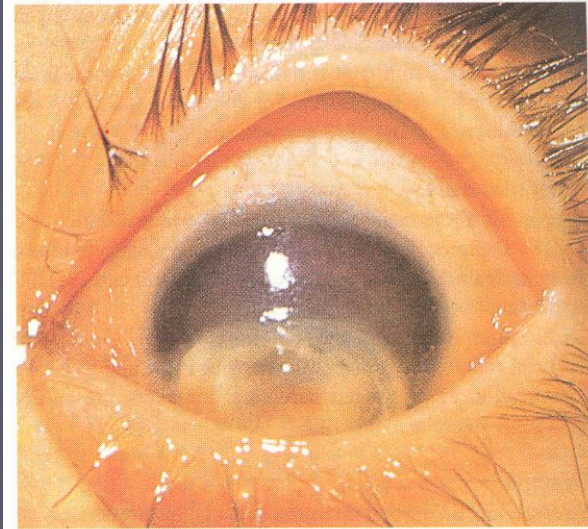


Fig. 7.26 Keratomalacia secondary to vitamin A deficiency in a 14-month-old child. There is colliquative necrosis affecting the greater part of the cornea. The relative sparing of the superior aspect of the cornea is typical.

Management: WHO schedule for treating Xerophthalmia:

Table 8.3: WHO/UNICEF treatment schedule of xerophthalmia

<u>Children 1 to 6 years and above</u>	
Immediately on diagnosis	200,000 IU vitamin A (Q)
The following day	200,000 IU vitamin A(Q)
Four weeks later	200,000 IU vitamin A(Q)
Children under 1 year and under 8 kg weight at any age	
Half the doses as indicated for children 1 to 6 years and above	
For night blindness or Bitot Spot	
Treat with a daily dose of	10,000 IU of vitamin A(Q) for 2 weeks

Note: If there is a persistent vomiting or profuse diarrhea, an intramuscular injection of 100,000 IU of water-miscible vitamin A (but not an oil-based preparation) may be substituted for the first dose.

VITAMIN D

VITAMIN D def RICKETS

- Disorder in the Vit D – Ca- P axis – results in the hypomineralized bone matrix, failure of endocranial ossification.
- Reflected on bones and teeth.
- bone changes – epiphyseal plates, metaphysis, shaft

Oral manifestation:

- Mellanby- 1st effects of Vit D on teeth
- Developmental abnormalities on enamel & dentin
- Delayed eruption
- Malign alignment of teeth & jaws

- Infantile rickets do not result in hypoplastic enamel

Vitamin resistant rickets (familial hypophosphatemia, Refractory Rickets, phosphate diabetes)

- Albright & coworkers- 1937
- Characterized by:
 1. Hypophosphatemia & hyperphosphaturia associated with ↓ renal tubular reabsorption of inorganic phosphates
 2. Familial occurrence, being inherited as an X linked dominant trait.
 3. Rickets or Osteomalacia which does not respond to the usual doses of Vit D.

1. Normocalcemia with high- normal parathyroid hormone levels
2. Diminished intestinal Ca phosphate absorption
3. Decreased growth with short stature
4. Normal Vit D metabolism
5. The absence of other related abnormalities

Clinical features:

Mildest form- simple hypophosphatemia with slight ↓ in the height

In hypophosphatemic adults- varying degrees of deformities due to rickets in childhood with more serious disturbance like;

- a) Bowing of legs
- b) Shortening of stature
- c) Presence of pseudo fractures

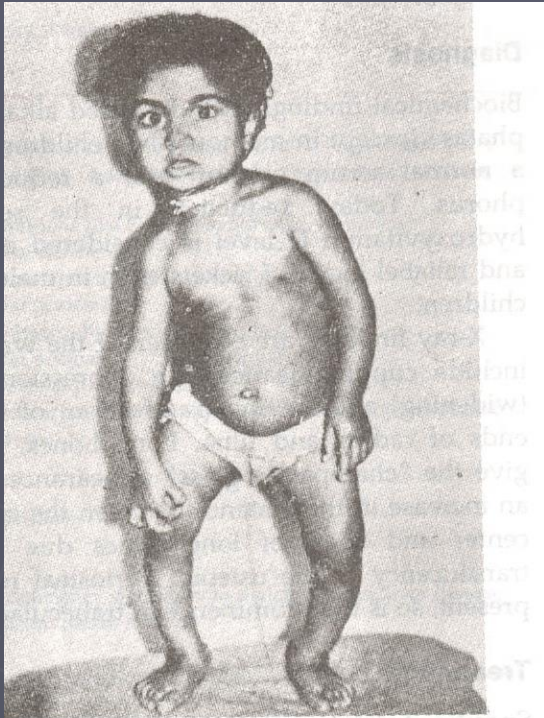
Children:

Recognized when the child starts to walk

X ray findings

Skull deformities

Sitting deformities of the legs.



7: Gross bowleg deformity of rickets. Note widening of ankles and "double malleoli"

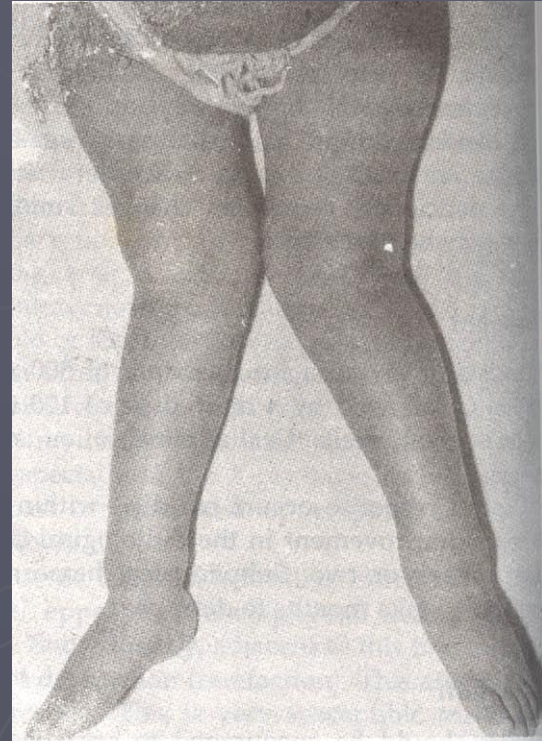


Fig. 8.26: Gross knock-knee deformity of rickets

Oral manifestations:

- Widespread of globular hypocalcified dentin
- Clefs & tubular defects occurring in the region of pulp horns
- Pulp horns are elongated extended high often reaching DEJ
- Periapical involvement of grossly normal- appearing deciduous / permanent teeth followed by the development of gingival tissue.
- Aalong with the abnormal cementum, lamina dura around the teeth is- frequently absent , alv bone patterns abnormal.

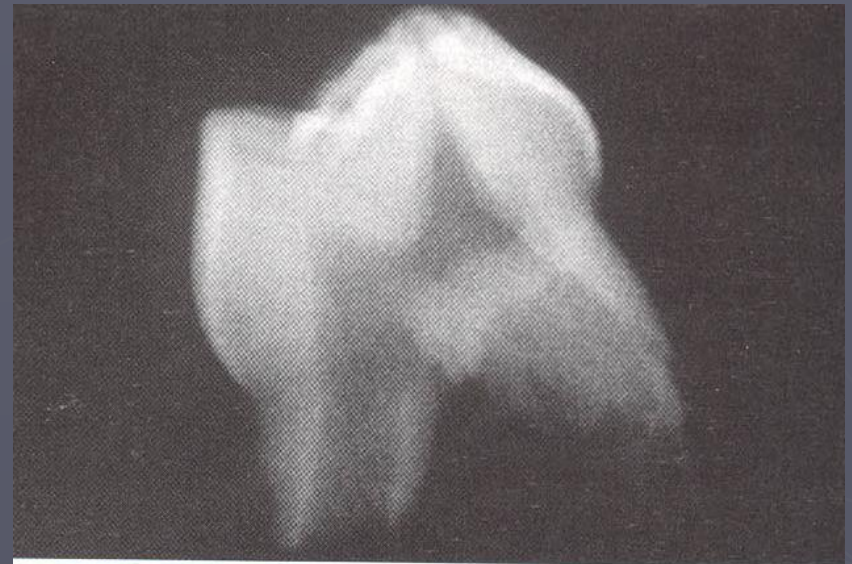


Figure 17-34 ♦ **Vitamin D-resistant rickets.** This radiograph of an extracted tooth shows a prominent pulp chamber with pulp horns extending out toward the dentinoenamel junction.



Figure 17-35 ♦ **Vitamin D-resistant rickets.** Ground section of the same tooth depicted in Figure 17-34. A pulp horn extends to the dentinoenamel junction. (Courtesy of Dr. Carl Witkop.)

Treatment

- highly individualized
- Massive doses results in repair
- Success-
- 25 hydroxycholecalciferol in lower dosages -10000-25000 IU/day
- Healing – initiated by administering massive doses of Vit D
- Include immobilization & administering large doses of phosphate.

VITAMIN E :

Infants: **Hassan & co workers:** – low level of Vit E

- Edema
- Desquamating erythematous papular dermatitis
- Thrombocytosis
- Anemia

Nitowsky co workers: 2^{dary} Vit E

- Muscular dystrophy symptoms
- ↑ serum creatinine phosphokinase activity
- creatinuria

Management:

Infants – formulas rich in linoleic acid, fortified iron .

- 0.7 IU of Vit E /100 kcal or
- 1 IU/ g linoleic acid

VITAMIN K :

occurs —

- New born- before adequate colonization of the intestine by intestinal bacterial flora.
- Newborn fed on unsupplemented breast milk.(human milk-1.5 mcg/ 100 of 12 mcg of required).
- Chronic intestinal parasitosis
- Malsorption states
- Biliary obstruction
- Oral antibiotics.

In Adults :

Impaired Fat absorption accompanied - obstructive jaundice, sprue, ulcerative colitis.

Oral manifestation:

- Gingival bleeding
- Prothrombin < 35% - bleeding after tooth brushing.
- < 20% - spontaneous gingival hemorrhage.

Management:

- 1-2mcg /kg depending on gut bacterial production.

VITAMIN C:

Occurs:

- Primary PEM
- exclusively artificially infants.
- Newborns of Vit C def mothers.
- Infections.

Infantile scurvy:

- 6mons- 2yrs
- Irritability
- Excessive crying
- Tenderness to touch
- Hemorrhages on skin
- Scorbutic rosary- post displacement of sternum

Childhood scurvy:

- Follicular hyperkeratosis , hemorrhages on the root of hair follicles
- Bleeding , petechiae, purpuric swellings

Oral manifestations;

- Interdental & marginal gingiva- red, swollen, smooth, shiny surface.
- Fully dev scurvy- Boggy ulcerate and bleeds
- Infants –a) Enlarged tissue covers crowns.
b) Foul breath
c) Severe- swelling of periodontal mem → bone loss, loosening of teeth- exfoliate

Management:

- 500mg of Vit,
- 100-300 mg – weeks
- oral route- good
- Respond- 24-48 hrs.



Figure 17-9 • Scurvy. The gingival inflammation and ulceration (scurbutic gingivitis) are due to severe vitamin C deficiency.

Vitamin B complex :

Vitamin B₁: (Thiamine):

Occurs:

- Infants –wet beriberi
- Children –dry beriberi
- Meningitic form

Earliest symptoms:

- Restlessness
- Bouts of crying
- Vomiting
- Abdominal pain
- Constipation
- Insomnia

Wet beri- beri :

Infant –

Congestive cardiac failure- tachycardia, gallop rhythm, dyspnea, cyanosis Hepatomegaly, cardiomegaly, edema, pulmonary edema.

Dry beri-beri:

- Anorexia
- Wt loss
- Weakness
- Diarrhea
- Drowsy apathetic
- Ataxia

Oral findings:

No convincing findings

Management:

- 10 mg thiamine IV
- 3 days- 10 mg IM bd
- 6 weeks- 10mg orally

RIBOFLAVIN B₂

CHILDREN – no milk

- Glossitis
- Filiform papillae- - atrophic
- Fungiform papillae- normal / engorged , mushroom shaped, - granular appearance.
- Paleness of lips
- Nasolabial fold- scaly greasy dermatitis
- Ocular changes- corneal vascularization, photophobia, superficial & interstitial keratitis.

Management:

3-10 mg orally / 2mg IM – few days

10mg orally – 3 weeks

Niacin

Etiology:

children – maize staple diet.

chronic diarrhea

Malsorption states

Anorexic state.

Clinical features of pellagra:

Occurs –school children

- Diarrhea
- Dermatitis
- Dementia
- Loss of appetite
- Vague GI symptoms.

Characteristic skin lesion- exposed areas of the skin

- limbs
- neck – **casal necklace**
- cheeks

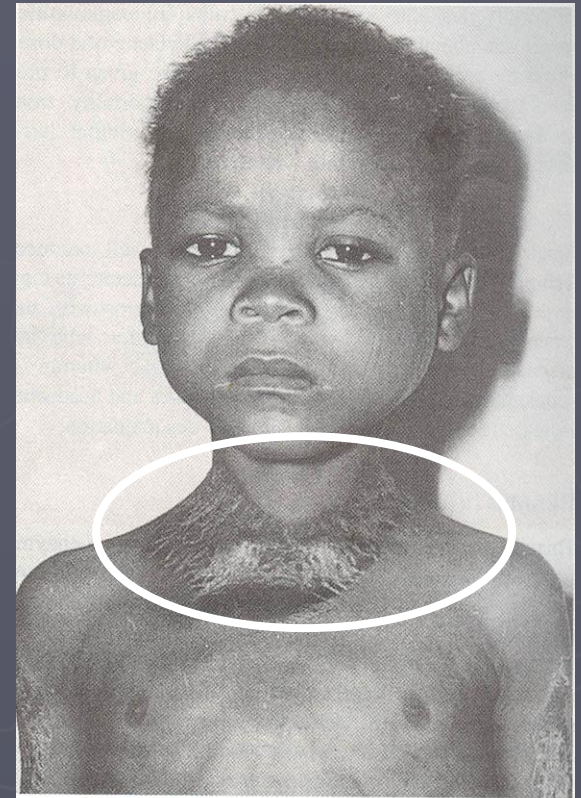


Fig. 7.27 Pellagra in a girl of 5 years. This skin lesion on the neck (Casal's collar) is pathognomonic of niacin deficiency.

oral findings:

- Lesion - tongue
- Oral mucosa- fiery red, painful
- Profuse salivation
- Desquamated tongue
- Tenderness pain ,
- redness ulceration –Interdental gingiva
- ANUG – squeal

Management:

50-300mg daily orally- 2weeks.

Adequate supply of Vit B complex.

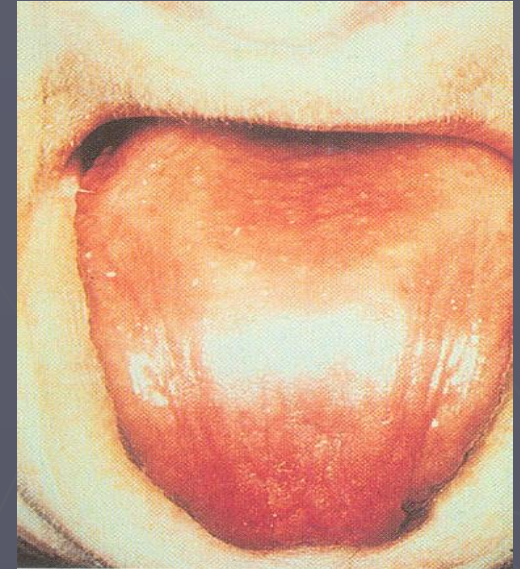


Figure 5.4 The red, smooth tongue of a patient deficient in B vitamins.

Pyridoxine (Vitamin B₆)

rare in children

Clinical features:

- Microcytic hypochromic anemia
- Growth retardation
- GI – diarrhea
- Seborrheic dermatitis- nose, eyes
- Sensory neuropathy
- Cheilosis
- Glossitis

Management:

- 5mg IM
- 0.5mg daily orally -2 weeks.

Folic acid

Characterized by,

- Glossitis
- Diarrhea
- Macrocytic anemia
- Oral manifestation:**
- Glossitis- initial appears as swelling & redness of tip Lateral margins of dorsum.
- Filiform papillae disappear
- Fungiform remain as prominent spots.

Advanced (toxic symptoms following aminopterin therapy for leukemia)-Fungiform lost

- Tongue- slick, smooth & either pallid or fiery red in color

Management:

30 mcg – infants

400mcg -adults

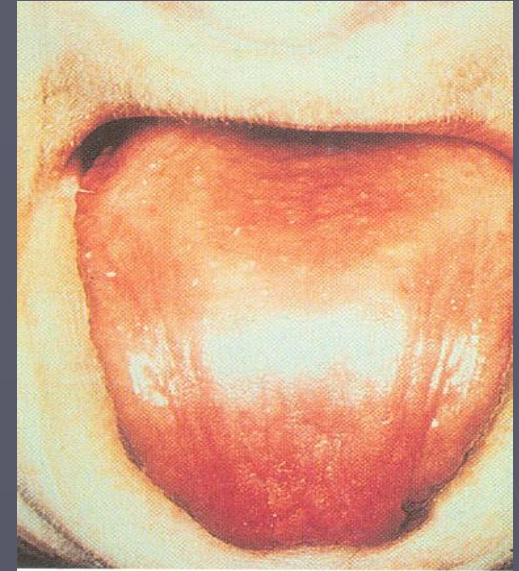


Figure 5.4 The red, smooth tongue of a patient deficient in B vitamins.

Oral manifestations of anemia:

Pernicious anemia: >30 yrs of age

- Tongue is generally inflamed
- beefy red in color
- some cases- small shallow ulcers, resembling aphthous ulcers

Characteristic –

- glossitis,
- glossodynia
- Glossopyrosis
- Gradual atrophy of papillae of the tongue – hunter's tongue / moller's glossitis
- Loss of distortion of the tongue

Management:

- Administration of Vit B12

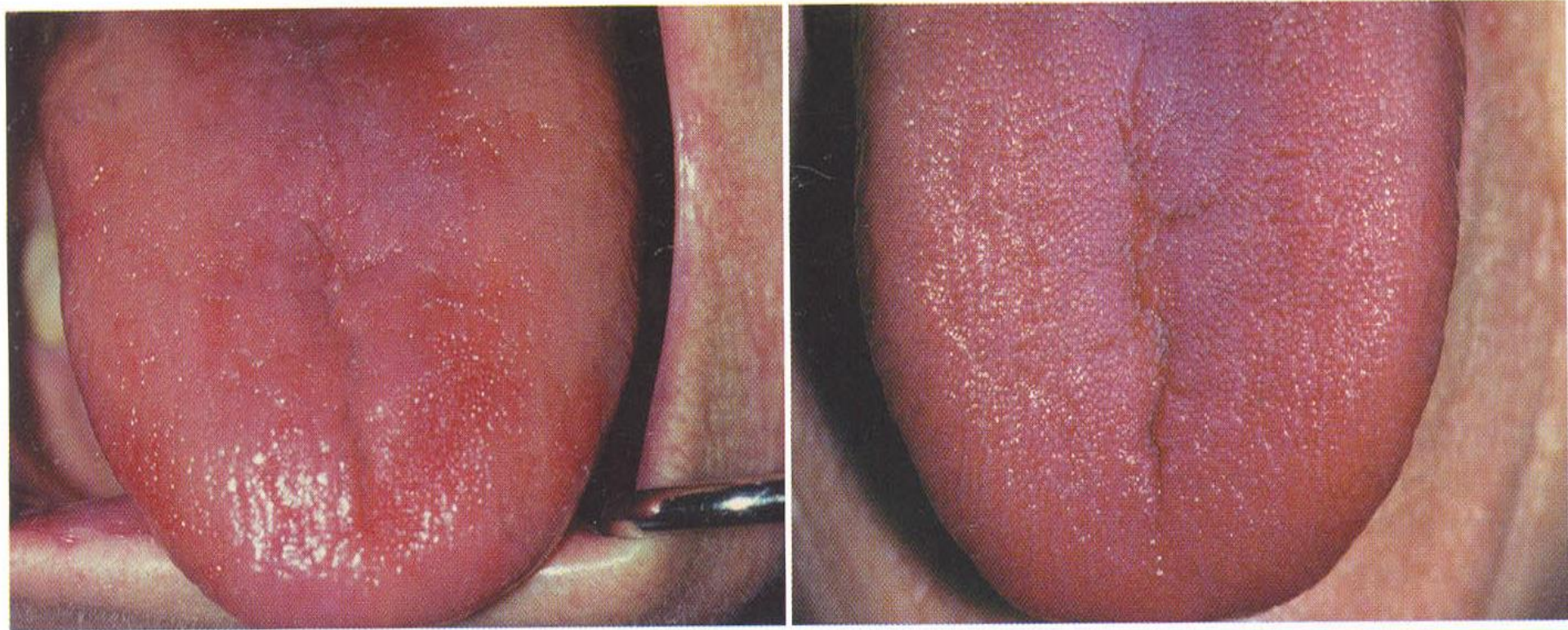


Figure 17-12 ♦ Pernicious anemia. **A**, The dorsal tongue shows erythema and atrophy. **B**, After therapy with vitamin B₁₂, the mucosal alteration resolved.

Iron deficiency anemia (Plummer- Vinson syndrome):

women

arise through

- a) Chronic blood loss
- b) Inadequate dietary intake
- c) Faulty iron absorption
- d) Increased requirements- infancy, childhood, adolescence,
- e) during pregnancy

Oral manifestations:

- Cracks in the corners of the mouth
- Smooth, red, painful tongue, with atrophy of the
- Filiform papillae- Fungiform papillae
- Dysphagia
- mucous membrane shows atrophic



Figure 17-10 ♦ **Plummer-Vinson syndrome.** Patients often show angular cheilitis.

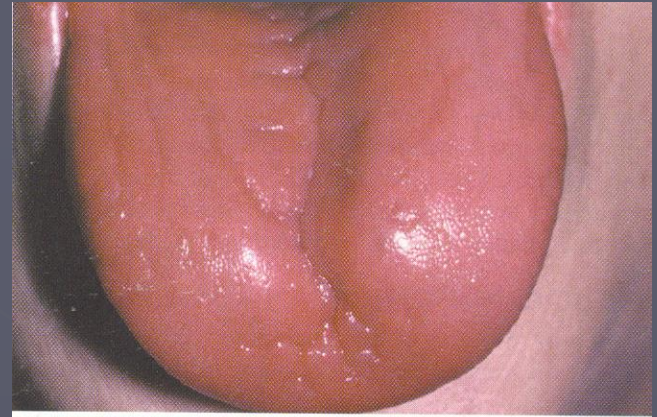


Figure 17-11 ♦ **Plummer-Vinson syndrome.** The diffuse papillary atrophy of the dorsal tongue is characteristic of the oral changes. (From Neville BW, Damm DD, White DK: *Color atlas of clinical oral pathology*, ed 2, Philadelphia, 1999, Lippincott, Williams & Wilkins.)

Management ;
iron therapy
high protein diet

FRUCTOSE MALABSORPTION

Absorption in SI – 1 of 2 mechanisms,

- a) In presence glucose – active transport.
- b) consumed alone- facilitated diffusion.

Free absorption in children – limited, results in osmotic diarrhea
severe cases- toddler's diarrhea- ↑ fructose consumption

Causes:

- Healthy beverages – infancy , early childhood
- Consumption- high fructose corn syrup, present in soft drinks

Management:

- limit intake of fructose
- limited intake of less processed food

DISORDERED EATING:

Psychosocial dwarfism:

18-48 months

- Deceleration of linear growth
- Characteristic behavior patterns- bizarre eating patterns, sleep habits.
- No expected growth pattern in relation to appropriate food intake
- Severely dysfunctional care giver- child interaction.

C/F: Severe food insecurity –
polyphagia ,
polydispia,
gorging with vomiting,
Stealing and hoarding of food
Eating garbage / pet food.

Rumination:

- 3-12 months, later-mental retardation
- voluntary regurgitation, chewing, & reswallowing of stomach contents.
- Self stimulatory behavior
- associated- psychosocial issues/mental retardation.
- Risk of enamel erosion.

Pica:

- Pathologic craving of food item or substance not commonly regarded as food
- Classic ex; starch, ice paint chips, dirt, paper,
- Risk of direct toxicity- ↑

- Pb poisoning - incidental exposure.

MANAGEMENT:

Treatment – 2 fold :

1. Management of underlying social situation / psychological disorder.
2. Environmental control of the feeding situation

Interdisciplinary approach:

1. Physician
2. Psychologist
3. Nurse
4. Dietician

Nutritional management of acute mucosal problems

- ▶ adequate , well balanced diet- physical form
- ▶ Bland nutritious foods – soups, fruit juices.
- ▶ Liquid diet – 1st day , soft diet later
- ▶ painful mucosal lesion- Corticosteriod topical application.
- ▶ Mouth rinses dyclone solution prior to eating.
- ▶ Vitamin supplements

Types of vitamin therapy:

2 major types

- Supportive therapy
- Therapeutic therapy.

- **Supportive therapy:**

1. protect the individual against Vit def- illness & pregnancy
2. supportive care –maintaining state of well being.

- **Therapeutic therapy:**

Treatment of specific Vit def

Maintaining good nutritional status- debilitating diseases

**TABLE 22-5. FORMULAS OF SUPPORTIVE
MULTIVITAMIN PREPARATIONS**

VITAMIN	DAILY DOSAGE (RANGE)
Basic formula*	
Vitamin A (synthetic or natural)	1500-5000 U.S.P. units
Vitamin D	400 U.S.P. units
Thiamin (hydrochloride or mononitrate)	1.0-2.0 mg.
Riboflavin	1.0-3.0 mg.
Niacinamide	5.0-20.0 mg.
Ascorbic acid	50.0-100.0 mg.
Expanded formula (add to basic formula)	
Pyridoxine hydrochloride	0.5-2.0 mg.
Calcium pantothenate	5.0-15.0 mg.
Folic acid	0.05-0.10 mg.
Vitamin B ₁₂	1.0-2.0 μ g.
Vitamin E	5.0-10.0 mg.
Preparations of water-soluble vitamins	
Remove vitamins A, D and E from the expanded formula	

**TABLE 22-6. FORMULAS OF THERAPEUTIC
MULTIVITAMIN PREPARATIONS**

<i>VITAMIN</i>	<i>DAILY DOSAGE (RANGE)</i>
Basic therapeutic formula*	
Vitamin A	10,000–25,000 U.S.P. units
Vitamin D	400 U.S.P. units
Thiamin	5.0–10.0 mg.
Riboflavin	7.0–15.0 mg.
Niacinamide	50.0–150.0 mg.
Ascorbic acid	150.0–400.0 mg.
Water-soluble vitamins (stress formula)	
Thiamin	5.0–10.0 mg.
Riboflavin	7.0–15.0 mg.
Niacinamide	50.0–100.0 mg.
Pyridoxine	5.0–10.0 mg.
Calcium pantothenate	20.0–50.0 mg.
Folic acid	0.05–0.10 mg.
Vitamin B ₁₂	2.0–5.0 μg.
Ascorbic acid	50.0–400.0 mg.
Expanded therapeutic formula† (add to stress formula)	
Vitamin A	10,000–25,000 U.S.P. units
Vitamin D	400–1000 U.S.P. units
Vitamin E	30.0–100.0 mg.

Conclusion

WE ARE WHAT WE EAT



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