

PRINCIPLES OF NUTRITION FOR THE INTENSIVE CARE UNIT PATIENTS

THESIS

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INTRODUCTION

Nutrition is basically the provision of biochemical substances in the quantities and ratios which are required for normal intracellular functions. Consideration must be given to the changes in requirements for nutrients due to pathophysiological conditions that we are called upon to treat or manage.

The intensive care unit "I.C.U." patient always has anorexia, gastro-intestinal dysfunction or other problems that make it difficult to maintain a normal food intake. In addition, many will have increased metabolic demands because of the illness or injury that precipitate their admission to the I.C.U. Surgery, trauma or serious illness are accompanied by endocrine and metabolic changes which, in general, reflect the severity of the illness. This is reflected by an increase in energy consumption, impaired glucose tolerance and abnormal responses to starvation as compared to healthy or mildly stressed patients.

Techniques of enteral and parenteral feeding now enable us to feed patients who might otherwise be unable to eat or assimilate enough normal food.

Parenteral feeding is capable of restoring the weight and body cell mass of chronically sick patients and maintaining

them at normal values over years if necessary.

The interest of the anesthesiologist being usually the intensive care physician in the nutritional therapy is mainly directed towards finding the answers for:

- a) How does the preoperative nutritional state of a patient influences the course of surgery and postoperative complications and accordingly when should preoperative restorative feeding be encouraged or insisted upon?
- b) How can nutritional support be used to benefit the acutely ill patients following trauma or during critical illness?

Using such treatment we aim to maintain organ function, promote healing, strengthen the patient and thereby reduce morbidity and hasten mobilisation and convalescence.

Nutritional treatment can also enhance immunological responses, particularly of cell mediated type, and therefore, may improve endogenous defences against infection.

Nutritional treatment inevitably modifies or enhances the metabolic responses to an illness such as energy consumption, protein turnover and hormonal output and much researches are aimed at delineating and understanding these changes.

HISTORICAL BACKGROUND

Progress in intravenous nutrition was slow until the end of nineteenth century. The work of Lister (1870) on asepsis and Pasteur (1877) on microbiological infection provided the basis of safe infusions. Kausch (1911) infused dextrose for nutrition after surgery, and Henriques and Anderson (1913) injected enzymatically hydrolysed protein into animals. Fat was infused experimentally by Mulin and Riche, (1916) and was used in man by Yamahawa in 1920.

Further progress initially centered on preparation of effective protein hydrolysates and Elman (1937) demonstrated an improvement in nitrogen balance in patients receiving casein hydrolysate. Early fat emulsions obtained from cotton seed oil had the valuable property of being energy rich yet isotonic, but they led to a variety of toxic reactions and were withdrawn from use in the U.S.A. in (1964).

A new type of fat emulsion based on soya bean oil with egg yolk phospholipids was developed in Scandinavia and proved to be free from toxic reactions.

It was also realised that many individuals could be fed enterally if a suitable delivery system and appropriate forms of tube feed were available. It has been amply proved that such enteral feeding systems can provide adequate nutrition for many patients who would previously have received intravenous nutrition. (Cuthbertson, 1980).

PHYSIOLOGICAL ASPECTS IN RELATION TO NUTRITION

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Optimal diet includes, in addition to sufficient water, adequate calories, protein, fat, vitamins and minerals.

CARBOHYDRATE METABOLISM

Dietary carbohydrates are mainly polymers of hexoses, of which the most important are glucose, fructose and galactose. -the principal product of carbohydrate digestion and the principal circulating sugar is glucose. Normal fasting level measured by highly specific glucose oxidase method is 60-80 mg/100 ml. Substrates of similar reducing reactions are responsible for higher values obtained with other methods. Glucose level is 15-30 mg/100 ml higher in the arterial blood. (Ganong, 1977).

The carbohydrate metabolism may be subdivided as follows:

I. **Glycolysis:** Oxidation of glucose or glycogen to pyruvate and lactate by Embden - Meyerhof pathway.

II. **The citric acid cycle:** the final common pathway of oxidation of carbohydrate, fat and protein through which acetyl-CoA is completely oxidized to CO_2 and H_2O .

III. **Hexose monophosphate shunt:** (HMP shunt) or direct oxidative pathway or pentose phosphate cycle : an alternative pathway to glycolysis and krebs cycle for glucose oxidation to CO_2 and H_2O .

IV. **Glycogenesis :** The synthesis of glycogen from glucose

"Mayes, 1981."

V. **Glycogenolysis:** The conversion of glycogen to glucose, mainly in the liver.

VI. **Gluconeogenesis:** The formation of glucose or glycogen from non-carbohydrate sources.

I. Glycolysis "Anaerobic metabolism of glucose"

It is anaerobic breakdown of glucose or glycogen to pyruvic and lactic acid via "Embden Meyerhoff" Pathway, the enzymes of which are found in extramitochondrial fraction of the cell. The principal phases are represented in Figure (1).

1. Phosphorylation: by hexokinase and also by glucokinase only in the liver into glucose-6-P.

Unlike hexokinase, glucokinase is activated by insulin, and suppressed by starvation and diabetes.

2. Change of hexose diphosphate into phosphotrioses.
3. Oxidation of triose phosphate into pyruvate .
4. Reduction of pyruvate into lactate in the skeletal muscles working under oxygen lack.

The net yield of energy derived from glycolysis is 2 moles of A.T.P. when glucose is anaerobically oxidized, and 8 moles of A.T.P. when aerobically oxidized as 6 moles of A.T.P. will be given by respiratory chain oxidation of the 2 moles of reduced NADH.

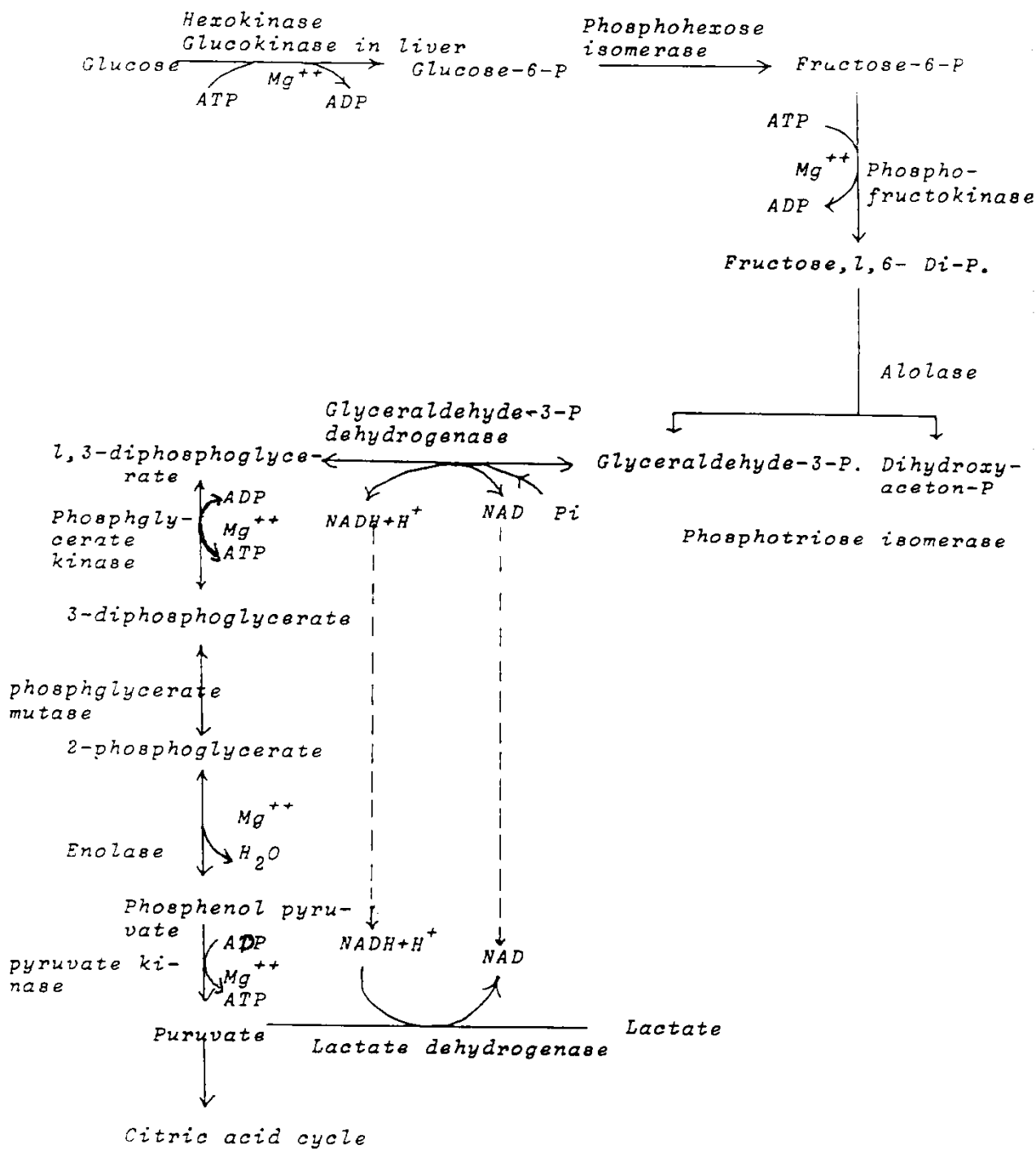


Fig.1 Embden-Meyerhof pathway of glycolysis.

II. Citric acid cycle:

This is the final common pathway in metabolism, the enzymes of which are found in the mitochondria. The net result is the oxidation of acetyl group to two molecules of CO_2 and four molecules of H_2O . Acetyl -CoA is derived by oxidative decarboxylation of pyruvate.

Steps are shown in figure (2), it includes 3 reactions in which NADH arises, and one reaction in which FAD arises, by oxidation via the respiratory chain NADH gives 3 moles of A.T.P., while FADH gives 2 moles only.

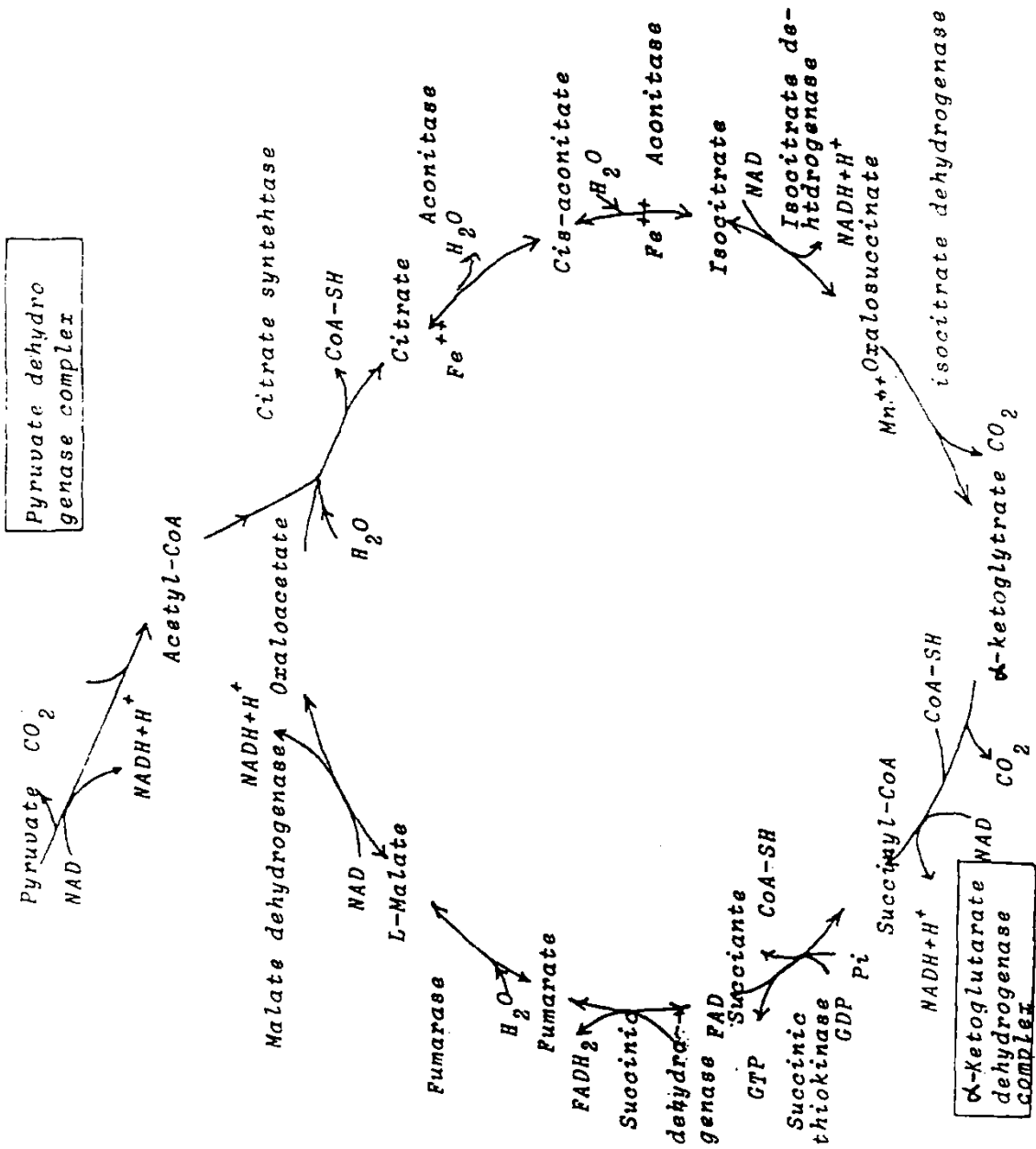
Reaction.	Conenzyme	net yield of A.T.P.
Isocitrate + CO_2 ===== ketoglutarate	NAD	3
Ketoglutarate CoA + CO_2 ---- succinyl,-	NAD	3
Succinyl - CoA + ADP + Pi --- succinate	GDP	1
Succinate ----- fumarate	F.A.D.	2
Malate ----- oxaloacetate	NAD	3

Total		12

Thus, per each mole of acetyl-CoA consumed - 12 A.T.P. moles are generated and as one mole of pyruvate oxidized to acetyl - CoA yields 3 A.T.P. (oxidative decarboxylation).

Thus complete oxidation of pyruvic acid to $\text{CO}_2 + \text{H}_2\text{O}$ yields 15 high -energy A.T.P. moles.

Fig. (2) Citric Acid Cycle



Since 1 mole of glucose gives 2 moles of pyruvic and 8 moles of A.T.P. by glycolysis, thus the complete oxidation of 1 mole of glucose to CO_2 and H_2O yields 38 A.T.P.

Metabolic significance of citric acid cycle:

1. Provides A.T.P. the major source of energy.
2. Complete oxidation of carbohydrates, fatty acids and amino acid.
3. Production of precursors important for other pathways as active succinate for haemoglobin synthesis, ketone body metabolism and detoxication (Goodwin, 1968).

III. Hexose monophosphate shunt "HMP. shunt: "

This is an alternative pathway for glucose oxidation in the liver, lactating breast and adipose tissues, the enzymes of which are located in the extramitochondrial fraction.

It is a multicyclic process in which 3 molecules of G.6.P. give rise to 3 moles of CO_2 and 3 (5- carbon residues) . Then, the latter are rearranged to regenerate 2 molecules of G-6.P one molecule of glyceraldehyde -3-P as shown in figure 3. 2 molecules of the latter can regenerate a molecule of G-6-P by reversal of glycolysis. Thus, the pathway is a complete oxidation of glucose.

Metabolic significance of HMP shunt:

1. Generation of NADPH in tissues that carry synthesis of

fatty acids and steroids.

2. Production of pentoses particularly D-ribose used in synthesis of nucleic acids.

3. Deficiency of enzyme system of H.P.M. results in Favism.

IV. Glycogenolysis:

It is glycogen breakdown into glucose by action of phosphorylase a "active form". Phosphorylase is present in two forms; one active phosphorylase and other inactive phosphorylase. Phosphorylase a_1 breaks 1,4 linkages, while debranching enzyme hydrolyses 1,6- linkages to produce free glucose. Only in the liver, specific G-6 phosphatase removes phosphate group, enables free glucose to diffuse in extracellular fluids including the blood, while in the muscle G-6-P proceeds into glycolysis.

V. Gluconeogenesis:

This provides glucose when carbohydrates are not available, from glyceric amino acids, glycerol and lactate. As brain, red cells and contracting skeletal muscle demand a continual supply of glucose, there is always a certain basal requirements for glucose provided by metabolic pathway including gluconeogenesis. In addition gluconeogenesis clears the products of other tissue metabolism as lactate produced by muscles and glycerol by adipose tissues (Krebs, 1964).