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Recent advances in parenteral nutrition

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Carbohydrate, fat, and amino acid metabolism from the parenteral point of view are surveyed and reviewed. Differences between the habits in the United States and Europe is stressed. It is proposed that carbohydrates other than glucose can be used; especially fat emulsions as the source of both energy and essential fatty acids for the body. Variable compositions of amino acids for different conditions as well as for adults and infants are suggested. There is also a trend to include all vitamins and minerals.

In order to provide optimal nutritional effects with intravenous alimentation it is important to: 1) supply an adequate amount of amino acids; 2) supply adequate energy; 3) include at least 100 g of carbohydrate; and 4) supply adequate amounts of other nutrients (vitamins, minerals, and essential fatty acids).

The hyperalimentation technique to provide the necessary nutrients to patients who cannot take food orally was amply demonstrated by Dudrick[1,2] with the administration of hypertonic solutions by a central venous catheter. The primary reason for the use of high concentrations of a glucose solution probably was related to the unsuccessful experiences in giving fat emulsions as a caloric support many years ago. Although hyperalimentation has been highly popularized in the United States, it has not been widely used in Europe. There are problems involved with this technique. First, the complications associated with the central venous catheter. Second, nonketotic hyperglycemia and hypophosphotemia, as well as hypoglycemic reactions following hypertonic glucose infusions. After three to four weeks of hyperalimentation, there is usually a lack of essential fatty acids and lipid soluble vitamins.

In this report, our program with balanced carbohydrates, fat, and amino acids in parenteral nutrition, as well as their use in slightly catabolic and massively catabolic states, will be discussed. No special attention will be given to endocrinologic aspects of nutrition, nor to nutrition in renal and liver failure.

When all nutrients are given intravenously we call it "Complete Parenteral Nutrition". "Hyperalimentation" will be used here to indicate that fat is not included in the program. The term hyperalimentation can give the erroneous impression of an effort to overfeed the patient; instead,

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Table 1: Nutrients essential for complete intravenous nutrition.[3]

Fluid	
Water	
Sources for synthesis of body proteins and for energy	
Amino acids	
Carbohydrates	
Fat	
Minerals	
Sodium	Magnesium
Potassium	Copper
Calcium	Chlorine
Magnesium	Phosphorus
Iron	Fluorine
Zinc	Iodine
Water soluble vitamins	
Thiamine	Vitamin B_{12}
Riboflavin	Pantothenic acid
Niacin	Biotin
Vitamin B_6	Ascorbic acid
Folic acid	
Fat soluble vitamins	
Vitamin A	
Vitamin D	
Vitamin K_1	
Tocopherol	

it is an effort to meet the increased energy requirements of the traumatized patient. We also separate "Supplementary Parenteral Nutrition" which is used when a patient can take part but not all of the required food orally, or when he is expected to eat normally within a few days following an operation, as in most uncomplicated surgical cases. Table 1 shows the compartments which seem necessary for complete parenteral nutrition on a long-term basis (months) according to Wretlind.[3]

CARBOHYDRATES

Carbohydrates should always be included in nutritional protocols as they have a specific protein-saving effect not provided by fat. This is maximal with about 100 g of glucose;[4] little further protein-saving effect is seen with doses up to 300-400 g.[5] The brain, erythrocytes, leukocytes, renal medulla and intestinal mucosa, and skeletal muscle in severe exercise can only or mainly metabolize glucose. The human brain needs about 100 to 150 g/day of glucose. All caloric requirements beyond 100 g of glucose can be covered either by fat, amino acids, or other carbohydrates.

In the United States, the trend has been to use glucose as the choice of carbohydrates. When Elman[6] in the late thirties started with parenteral nutrition he found that the best effect on nitrogen balance was achieved when glucose and amino acids were given together, but later found that fructose solutions with amino acids produced a better nitrogen balance.[7] As the provision of total parenteral nutrition became possible and fat emulsions proved unsatisfactory, an interest in other carbohydrates developed, primarily glucose.

However, in Germany and also Australia, balanced carbohydrate solutions containing equal parts of glucose, fructose, sorbitol and xylitol were utilized. Fructose appears to be more rapidly metabolized than glucose. The rapid disappearance of fructose from the blood results from its rapid distribution in the total body fluid,[8] its transformation into glucose, and its rapid conversion to glycogen. Indeed, fructose builds up the liver glycogen stores more quickly than does glucose.[9] Some fructose seems to be metabolized in the cells independent of insulin action, but the glucose transformed from fructose requires insulin for its further metabolism.[10] Another advantage is that the urinary losses of fructose are smaller than those of glucose. However, rapid infusion of fructose may deplete liver ATP levels. The rapid metabolism of fructose increases the concentration of lactic acid in the liver and blood;[11] however, fructose infusions at rates less than 0.5 g/kg/hr cause only small changes in the lactate concentration. Bergström et al[13] showed that the infusion of 20% fructose at 0.5 to 3.5 g/kg/hr to healthy volunteers increased lactate by 0.8 to 8 mM/L. Anderson et al[12] reported a death in a dehydrated child who was given 20% fructose at 3 g/kg/hr for seven hours. The increased lactate concentration may influence intra- and extra-cellular osmolality, and fructose may be contraindicated in acidosis and dehydration.

When amino acids and fructose are given together the lactate production from fructose decreases.[14] The infusion of 1 g fructose/kg/hr results in hepatic lactate production of 0.5 to 1 mM/min, but when amino acids (Intramin Forte*) are added, the lactate production falls to nearly zero. In muscle biopsy studies we

* Intramin Forte® manufactured by Astra Pharmaceutical, Sodertalje, Sweden.

found increased levels of intracellular fructose-6-phosphate, fructose-1-phosphate, and fructose-1,6 diphosphate levels, indicating either increased gluconeogenesis or a metabolic block. We believe this represents induced gluconeogenesis.

When glucose and fructose are given together, it is possible to infuse more total carbohydrate at lower concentrations of each sugar (with less risk of thrombophlebitis) than would be possible if the total carbohydrate were supplied by only one sugar. Therefore, there are definite advantages in including the proper level of fructose in a parenteral nutritional program.

The addition of sorbitol and xylitol rather than glucose, to amino acid solutions solves the problem of carmelization (browning) which occurs when the solution is autoclaved. If a clear, stable autoclaved solution with all the needed carbohydrate ingredients can be made possible, the risks of contamination during the mixing of the various components at the time of administration would be avoided. Sorbitol is readily excreted in the urine and has an osmotic diuretic effect. It is oxidized through the fructose pathway fairly slowly. Its effect on liver glycogen is negligible.[9] In animal experiments, xylitol rapidly increases liver glycogen, but this has not been confirmed in man. Rapid infusion in rats deplete the hepatic ATP content. Studies in Australia indicated that xylitol has toxic effects in man;[15] however, there is reason to believe that this reaction was due to impurities in the reagents employed rather than to the xylitol itself. Studies from Germany suggest that xylitol is not toxic to man.[16] In essence, there is still an open question as to the advantages of sorbitol and xylitol as compared with glucose and fructose. I have not discussed ethanol, as it has no place in parenteral nutrition today for several reasons.

FAT

Intravenous administration of fat emulsions can easily supply nutritional needs with several advantages. The solutions are isotonic and therefore do not produce hyperosmolar reactions or hyperglycemia. The essential fatty acids and phosphorus requirements are easily supplied, the risk of thrombophlebitis is small since they can be given by a peripheral vein, and they do not have a diuretic effect.

Fatty acid utilization was studied with ^{14}C labeled fatty acids in fat emulsions.[17] About 30% of the administered triglycerides were completely oxidized to CO_2 within 24 hours. It is reasonable to conclude that triglycerides from parenteral administration are utilized and metabolized in the same manner as fat in the normal diet.

The tolerance to fat emulsions is an important question. Comparative studies of chronic toxicity of the four commercially available fat emulsions have been made in many laboratories. These studies indicate that the egg-yolk phospholipid, soybean oil emulsion (Intralipid®) is the least toxic of the fat emulsions on the market today. Håkansson[18] infused fat emulsions in dogs at 9 g fat/kg/day for 4 weeks. There were no adverse reactions to Intralipid, while there were serious toxic reactions to the other fat emulsions.

Fraser and Håkansson[19] showed pronounced physical and clinical similarities between natural chylomicrons and the fat particles of Intralipid. The 20% solutions were stable for months, but showed small changes after two years, which may not increase toxicity. The 10% solutions were stable for years. Boberg and Carlson[20] studied the effect of induced serum lipoprotein lipase on different fat emulsions and found that chylomicrons and Intralipid have similar effects; this was not the case with the other fat emulsions.

Hallberg[21,22] investigated the kinetics of Intralipid clearance and found that both chylomicrons and Intralipid removal from the blood stream were similar. The clearance process is maximal at high concentrations of the infused lipids. The clearance continues at a high constant rate until a "critical concentration" is reached. Below this critical concentration the rate of elimination is dependent on the lipid concentration; the fractional removal rate is proportional to the concentration. Controlled adult subjects, who had fasted overnight, cleared 3.8 g fat/kg/24 hr; this amount corresponds to 35 cal/kg/24 hr. After fasting for 38 hours, the clearance capacity increased to 52 cal/kg/24 hr. After about two days fasting in the postoperative period, the clearance capacity was equivalent to 100 cal/kg/24 hr. Wilmore et al[23] showed an accelerated plasma disappearance of the fat emulsion in acutely burned patients.

Heparin strongly increases the disappearance rate of fat, since it releases lipase from the walls of the vessels and therefore increases hydrolysis of the triglycerides.[24]

Scholler[25] found fat accumulation in the reticuloendothelial cells after administration of some fat emulsions; these fat particles are treated as foreign bodies. Accumulation of fat particles in the Kupffer's cells after administration of Intralipid was not observed and there was no signifi-

cant reduction in the formation of antibodies. After long-term infusions, a yellow pigment in the liver was seen, but no functional liver alterations could be found; no effect on either coagulation or the fibrinolytic system were demonstrated. Although no effects on the liver function or blood coagulation were demonstrated, our experience in patients with liver damage or coagulation abnormalities is limited. Therefore, no recommendations can be made at this time, but caution is used in the administration of fat emulsion in these situations as well as in cases of lipoprotein lipase deficiency and hyperlipemia.

It has been reported that fat infusions influence pulmonary diffusion capacity.[26] With a ^{133}Xenon perfusion, Wilmore et al[23] found a normal capacity, using a diffusion and carbon monoxide rebreathing technique. Blood gases did not change following infusion of single or multiple units of the fat emulsion. No studies have been done in patients with the shock lung syndrome. Finally, febrile reactions were observed in a small percentage of cases; the fever disappeared when the fat infusion was stopped.

Metabolism of infused triglycerides is dependent on the lipase activity. There appear to be more lipid receptor sites in adipose tissue, liver, and muscle than in other organs. The distribution of blood flow to various organs in the body and the activity of the receptor sites are thought to be under CNS and hormonal control. During starvation, the triglycerides stored in the adipose tissue are mobilized as free fatty acids. Normally, these are transported in the blood as free fatty acids (FFA) bound to albumin. As FFA they can be taken up by most of the cells of the body and directly metabolized. Some FFA are transported to the liver and transformed into triglycerides to be recycled by the blood in the form of lipoprotein triglycerides. The only organ that can metabolize the triglycerides directly without hydrolysis may be the heart muscle.

During pathophysiological states such as trauma this mechanism is disturbed. The FFA mobilized from the fat tissue may have difficulty reaching the plasma albumin with fatty acids accumulating in adipose tissue, and fat necrosis may develop. More fatty acids will be provided to the liver than it can handle, and fatty liver with an enormous amount of triglycerides will develop. It is well known that severe trauma will cause heavy fat infiltration in the liver. The high plasma fatty acid concentrations during trauma may be responsible for fat infiltration into other organs. Norepinephine infusions in dogs produce similar effects that can be prevented with nicotinic acid administration.[27,28] In stress, infused triglycerides are not handled in the same manner as endogenous triglycerides. Infused triglycerides are acted on by the lipase activity of blood vessels with a lesser increase of free fatty acids in the blood than in trauma per se. The triglycerides cannot infiltrate peripheral cells without being hydrolyzed.

The administration of heparin with fat emulsions increases the rate of hydrolysis of the triglycerides. In these conditions the fatty acid levels and the risks of secondary fat infiltration increase. Therefore, one must consider all aspects of the problem when heparin is to be given during triglyceride infusions.

A certain quantity of fat is necessary to provide the body with the essential fatty acid requirements. Collins et al[29] observed a linoleic acid deficiency dermatitis in an adult on total parenteral nutrition for more than three months; the symptoms disappeared when Intralipid was given. In the treatment of a deficiency, 0.1 g of linoleic acid/kg/day is deemed to be the required dose, this corresponds to 15 g of soybean oil; infants have higher requirements which correspond to 3 to 4% of their total energy intake, or about 0.4 g of linoleic acid/kg/day.

Many reports have been published which show that fat can support 60 to 80% of the total energy needs without side effects.[3]

AMINO ACIDS

Elman and Weiner[30] were the first to give protein hydrolysate intravenously to man. Subsequently, many different hydrolysates have been used with good effects. The amino acid patterns of the hydrolysates are dependent on the original protein. Because of incomplete hydrolysis, these preparations contain between 30% and 50% of the amino acids as peptides and are utilized less efficiently than the free amino acids, especially in posttraumatic catabolic states.[31] More recently, solutions of synthetic crystalline amino acids have been made for parenteral use. Shohl and Blackfan[32] were the first to use solutions of crystalline amino acids; they administered d-l amino acids to children. As the d-isomer is poorly utilized and can be toxic, these trials were mainly experimental. When l-amino acids became commercially available, Bansi et al[33] were first to prepare solutions for parenteral use; a number of other preparations soon followed. The ideal composition of amino acid solutions for parenteral use is still open to discussion.

Total requirement of amino acids

Total protein needs vary with age. Based on figures from literature, Munro[34] estimated that infants require 1,700 mg/kg/day, children of 10 to 12 years 700 mg/kg/day and adults 425 mg/kg/day. The metabolism of amino acids is dependent upon the route and rate of administration. Intravenous administration leads to rapid distribution to all tissues for use in protein synthesis. Oral feeding requires absorption by the intestine and subsequent transport to the liver where they may be absorbed and used for protein synthesis. However, a large proportion, especially after rapid intake, may be directly converted into urea and thus become unavailable for protein synthesis in the peripheral tissues. Elwyn[35] has estimated that during the absorptive period of amino acid nitrogen, 57% was excreted by the liver as urea, 6% was secreted from the liver as plasma proteins, 14% was retained in the liver as proteins, and only 23% passed into the systemic circulation as free amino acids.

Fürst et al[36] found that liver production of urea in healthy subjects was higher when essential amino acids were supplied by means of stomach tube than when the same quantity of amino acids was infused intravenously at the same rate. However, Bergström et al[37] reported no significant difference in nitrogen balance following either oral or intravenous administration of essential amino acids to uremic patients when the oral administration was spread out over the day. On the other hand, intravenous administration of amino acids was followed by a higher incorporation of ^{15}N into muscle protein than into proteins synthetized in the liver; a reversed incorporation pattern was obtained after oral administration of amino acids.[37] Bergström et al[38] obtained nitrogen equilibrium with the intravenous administration of 4 g of nitrogen per day during long-term parenteral nutrition to an unconscious noncatabolic patient with cerebral damage. This is in accord with earlier studies that reported the nitrogen requirement upon intravenous administration as identical with that of the oral route.

The clinical status has a great influence on the nitrogen requirements. Many reports document increased nitrogen requirements after trauma. Johnston et al[39] stated that 15 g of nitrogen is the daily postoperative requirement of a 70 kg male. Protein synthesis occurs even in severely catabolic patients.[40] We administered ^{15}N (ammonium chloride) to a patient who had a fractured femur in both legs and recovered the isotope in proteins of the muscle and plasma.

Requirement of essential amino acids

The amount of essential amino acids in the total amino acid supply for maximal nutritional effect, is 43% in infants, 36% in children 10 to 12 years and 19% in adults.[34] Fürst et al[41] obtained roughly the same nitrogen balance when 25, 30, 36, or 46% of the nitrogen (total dosage 5 g I.V.) was supplied as essential amino acids in healthy, adult women. The nitrogen balances were negative when only nonessential amino acids were given under similar experimental conditions. This study suggests that a supply of essential amino acids exceeding one-quarter of the total nitrogen is of no benefit in adults and that the basic requirements of essential amino acids are the same when oral or intravenous administration is employed.

The requirements of essential amino acids may be increased during catabolic states, but there are also large increases in the total nitrogen requirements during these conditions. Thus, it seems rather unlikely that the essential amino acids will be a limiting factor in clinical situations with the use of the recently developed amino acid solutions.

The oral "minimum requirement" of each essential amino acid, determined in the extensive investigations by Rose,[42] has been used as a basis for the formulation of the majority of amino acid solutions intended for intravenous administration. The normal pattern of plasma essential amino acids or egg protein have also been used.

Miller[43] showed that the branched-chain amino acids (isoleucine, leucine, valine) pass through the liver unchanged after intestinal absorption and are metabolized mainly in the muscles and kidneys. Munro[34] has suggested that the amino acid composition in solutions for parenteral infusions should have higher concentrations of branched-chain amino acids.

Experimental results indicate that the currently available commercial solutions of crystalline l-amino acids have patterns of essential amino acids which give the same nitrogen retention.[44] Some nonessential amino acids seem to be required during certain conditions; eg, histidine is essential during infancy[45] and severe uremia.[46,47] Snyderman[48] and Sturman et al[49] have suggested that cysteine is an essential amino acid for the fetus and prematures. Liver cystathionase, catalyzing one of the intermediate reactions in the sequence from methionine to cysteine, was not

measurable in human fetuses, but increased levels of cystathionine were found in the liver.[49] On the other hand, Jürgens and Dolif[44] could not demonstrate a decrease in the plasma level of cysteine, when an amino acid solution without cysteine was given in prematures. Börresen et al[50] achieved nitrogen balance in newborns with the same amino acid solution. When Stegink and Den Besten[51] gave a cysteine-free amino acid solution to eight healthy men they found that cysteine plasma concentration dropped markedly, and returned to normal concentration only when a cysteine-containing diet was fed. On the other hand, cysteine is synthesized from methionine in a wide variety of tissues,[52] but there is a question as to extent of the synthesis, especially in children.

Tyrosine also seems to be an essential amino acid for prematures; however, a certain phenylalanine intolerance may occur.[48] In malnutrition, the ratio between phenylalanine and tyrosine is increased in plasma and erythrocytes.[53] Jürgens and Dolif[44] demonstrated a decrease in the tyrosine concentration in plasma when prematures were given a tyrosine-free but phenylalanine-containing amino acid solution; at the same time, phenylalanine rose sharply in plasma. They postulated that prematures have a block in tyrosine synthesis. This is in agreement with the concept that the phenylalanine hydroxylase system is not fully developed in prematures. It seems that tyrosine synthesis is also blocked in severe uremia.[54] However, it is not possible to make adequate substitution of phenylalanine for tyrosine in concentrated solutions due to the low solubility of tyrosine (approximately 0.5 g/L).

Requirement of nonessential amino acids

Efficient utilization of the essential amino acids for protein synthesis also requires an external supply of nonessential amino acids sources for their endogenous synthesis. However, the external supply of nonessential amino acids should be minimized in situations were specialized amino acid solutions are required; ie, acute and chronic renal insufficiency, and hepatic coma.

The pattern of nonessential amino acids has been extensively discussed in recent years. Most of the early commercially-available solutions made of crystalline amino acids contained only a few nonessential amino acids. However, several studies have clearly indicated that nitrogen balance is improved when several but not necessarily all of the nonessential amino acids are administered. Of the individual nonessential amino acids, alanine, arginine and proline give the best nitrogen balance, at least in healthy subjects.[44,55] Solutions without cysteine should contain serine to prevent serine from becoming rate-limiting for the endogenous synthesis of cysteine.

Aspartic and glutamic acid, both of which produce very good nitrogen retention,[44,55] can be given intravenously only in limited amounts because of nausea, vomiting and other side effects. In oral experiments, glutamic acid has been the individual nonessential amino acid shown to give the best nitrogen retention when administered in combination with essential amino acids.[56] After oral intake, a large proportion of the absorbed aspartic and glutamic acid in the intestinal mucosa immediately take part in transaminations yielding alanine. Furthermore, glutamic acid is synthetized in most cells either from the corresponding alpha-keto acid, the ubiquitous alpha-ketoglutaric acid by reactions catalyzed by glutamate dehydrogenase or transminases, or by deamination of glutamine. Aspartic acid or acetic acid can be used instead of hydrochloric acid to neutralize the basic amino acids, thereby avoiding the danger of precipitating metabolic acidosis.[55]

The amides, asparagine and glutamine, are present in relatively large amounts in proteins. However, they are unstable in solutions for parenteral use. To the best of our knowledge, no commercially-available solution prepared of crystalline amino acids contains either asparagine or glutamine.

RATIO BETWEEN AMINO ACIDS, CARBOHYDRATES, AND FAT

In Western Europe, the ordinary diet contains about 10 to 13 energy per cent of protein, about 50 energy per cent of carbohydrates and about 40 energy per cent of fat; and the recommendations for complete parenteral nutrition include about 2 g carbohydrate, 2 g fat and 0.7 g amino acids/kg/day to achieve an adequate energy supply of not less than 30 cal/kg/day. Neonates and infants require 90 to 120 cal/kg/day as 2.5 g of amino acids, 12 to 18 g of carbohydrate and up to 4 g of fat/kg/day.[3] In patients with severe catabolism, the recommended amounts are revised because the total energy supply can increase from 30 to 50-70 cal/kg/day and the nitrogen supply from 4-5 to 15-20 g/day. During noncatabolic states the ideal ratio is approximately 200 cal/g of nitrogen, while 75-150 cal/g of nitrogen is preferred in severe catabolic states.[57] The reason for this is probably accelerated gluconeogenesis.

Table 2: Tentatively recommended daily supply of energy and nutrients for patients on complete intravenous nutrition. The supply will cover resting metabolism, moderate physical activity of a patient, and specific dynamic action; but no increased need because of trauma, burns etc.[3]

	ADULT *Daily allowance per kg body weight*	*NEONATE* and *INFANTS* *Daily allowance per kg body weight*
Water	30 ml	120-150 ml
Energy	30 kcal = 0.13 MJ	90-120 kcal = 0.38-0.50 MJ
Amino acid nitrogen	90 mg (0.7 g amino acids)	330 mg (2.5 g amino acids)
Glucose or fructose	2 g	12-18 g
Fat	2 g	4 g
Sodium	1-1.4 mmol	1-2.5 mmol
Potassium	0.7-0.9 "	2 "
Calcium	0.11 "	0.5-1 "
Magnesium	0.04 "	0.15 "
Iron	1 μmol	2 μmol
Manganese	0.6 "	1 "
Zinc	0.3 "	0.6 "
Copper	0.07 "	0.3 "
Chlorine	1.3-1.9 mmol	1.8-4.3 mmol
Phosphorus	0.15 "	0.4-0.8 "
Fluorine	0.7 μmol	3 μmol
Iodine	0.015 "	0.04 "
Thiamine (B_1)	0.02 mg	0.05 mg
Riboflavin (B_2)	0.03 "	0.1 "
Nicotinamide	0.2 "	1 "
Pyridoxine (B_6)	0.03 "	0.1 "
Folic acid	3 μg	20 μg
Cyanocobalamine (B_{12})	0.03 "	0.2 "
Pantothenic acid	0.2 mg	1 mg
Biotin	5 μg	30 μg
Ascorbic acid (C)	0.5 mg	3 mg
Retinol (A)	10 μg	0.1 mg
Ergocalciferol or cholecalciferol (D)	0.04 "	2.5 μg
Phytylmenadione (K_1)	2 "	50 "
α-Tocopherol (E)	1.5 mg	3 mg

VITAMINS AND ELECTROLYTES

There appears to be good reason to include all of the vitamins in parenteral nutrition programs, even though not much research has been done in this field. Special attention must be given to vitamin K, although it is not included in most programs. In a patient treated with antibiotics on parenteral nutrition without exogenous supply of vitamin K, signs of vitamin K deficiency were observed after nine days.[5,8] The change of the intestinal flora and a loss of intestinal vitamin K production may occur occasionally in the critically ill patient.

Table 2 summarizes the tentative recommendations for adults according to Wretlind.[3] Sodium, potassium, magnesium, calcium, and chloride have generally been included in parenteral nutrition. The addition of phosphorus and zinc becomes more important after one week of parenteral nutrition. It is very difficult to supply the phosphorus requirements when fat emulsions are not used. When the fat is included, the phospholipids in the emulsion provide adequate phosphorous supplies. Lack of phosphorus will influence all of the phosphorylation processes in the body.

Zinc is present in several enzymes and a deficiency will, among other things, cause anemia.

Tissue repairs make substantial demands on the body reserves of zinc.

WHEN SHOULD TPN BE USED?

Finally, the question frequently arises as to when total parenteral nutrition should be started. There are no general recommendations, but we must remember that starvation is a very important and the most common reason for catabolism. The administration of 100 g of glucose is not enough to prevent catabolism. We usually begin TPN when we expect that a patient will not start a fairly normal oral intake of food within five or seven days postoperatively. Several investigators have shown that it is possible to supply all essential nutrients in adequate amounts intravenously for long periods of time (months).

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