Nutrition support in liver disease

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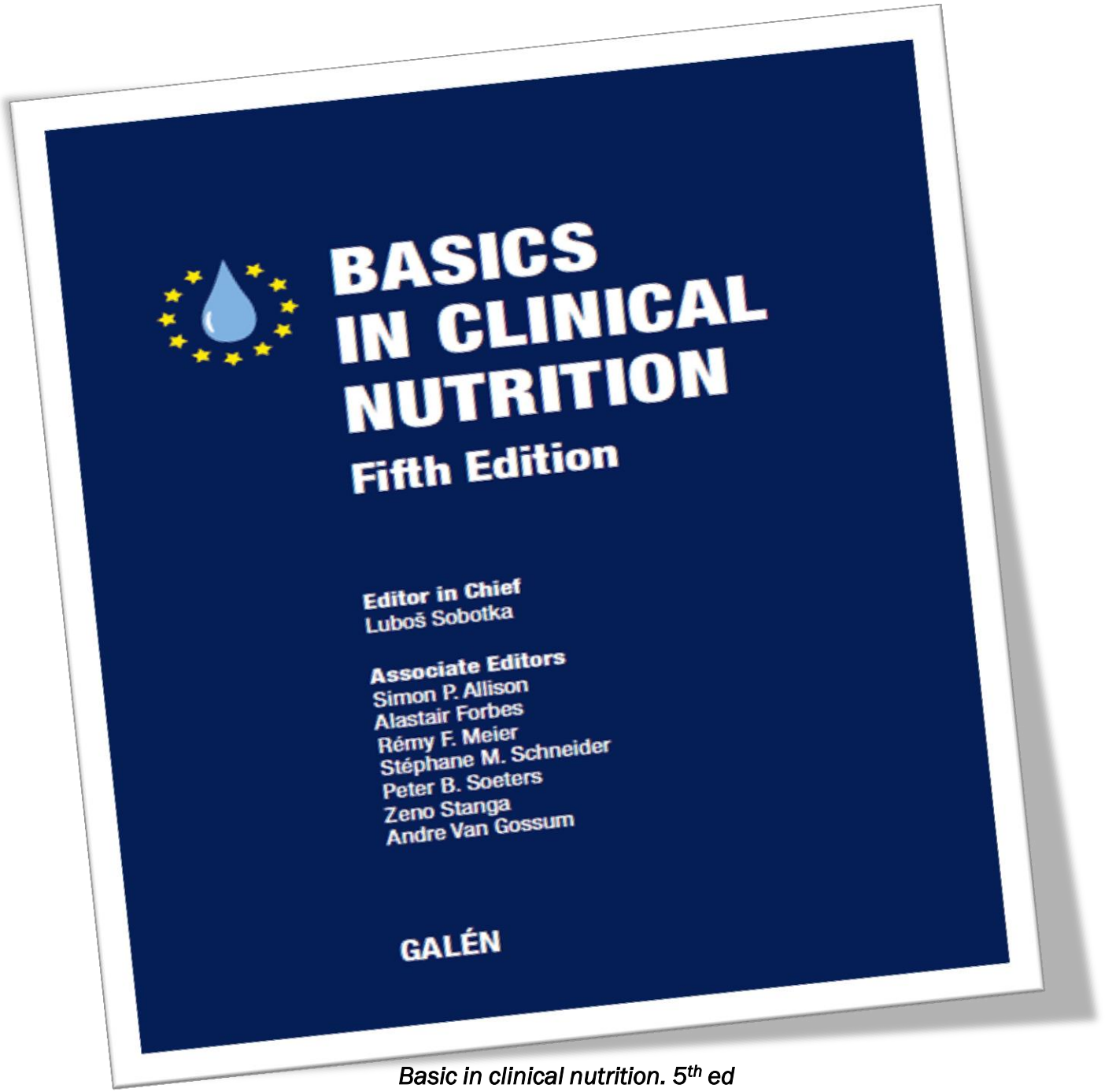

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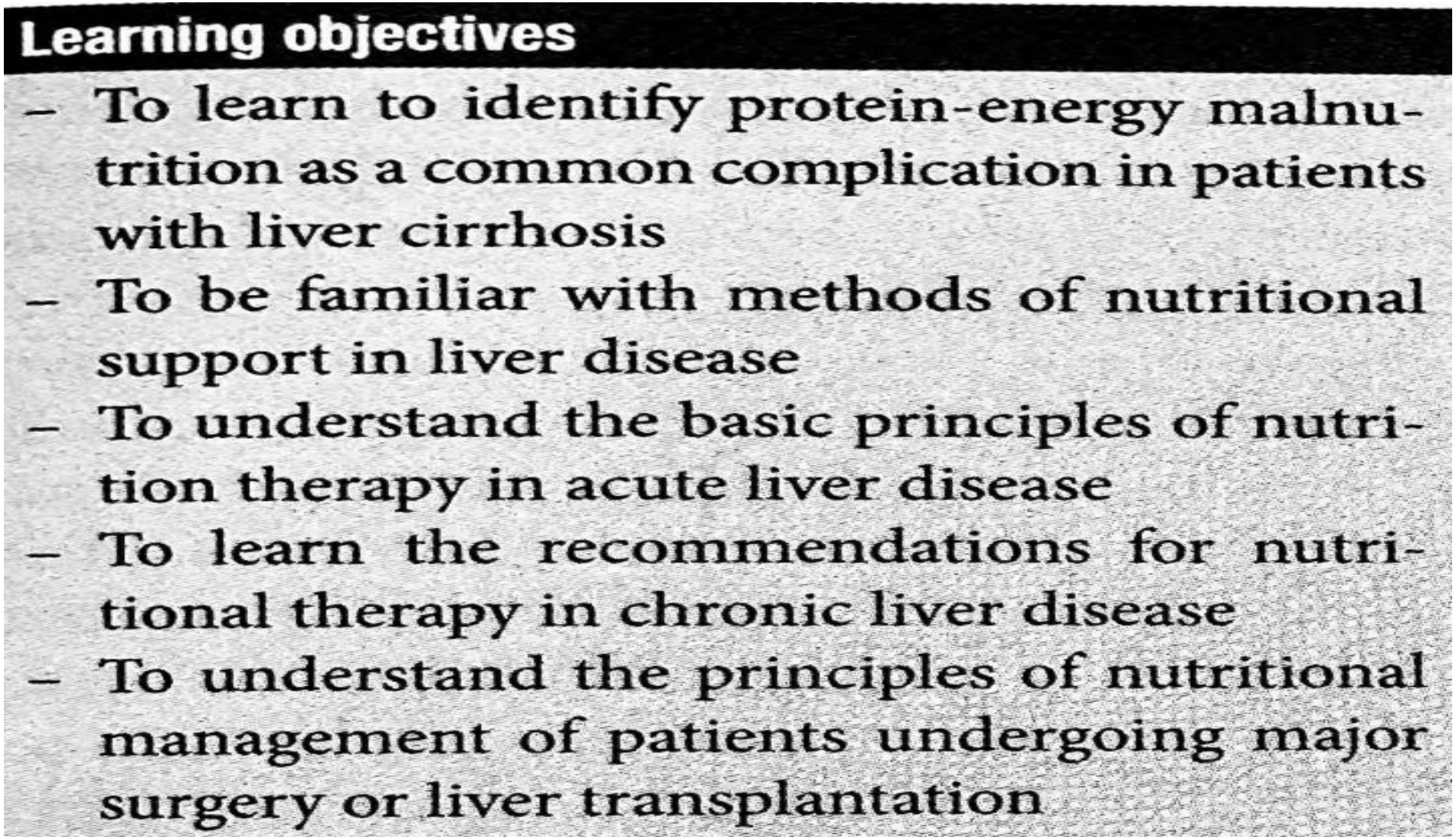
Learning Objectives



Learning objectives

- To learn to identify protein-energy malnutrition as a common complication in patients with liver cirrhosis
- To be familiar with methods of nutritional support in liver disease
- To understand the basic principles of nutrition therapy in acute liver disease
- To learn the recommendations for nutritional therapy in chronic liver disease
- To understand the principles of nutritional management of patients undergoing major surgery or liver transplantation

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Outline

Introduction

8.5.1. Oral diet

8.5.2. Dietary supplements

Supplemental sip feeding

Branched-chain amino acids (BCAA)

Micronutrients

8.5.3. Enteral feeding

8.5.4. Parenteral nutrition

8.5.5. Conclusions for diagnosis related nutritional therapy

Alcoholic Steatohepatitis (ASH)

Cirrhosis

Cirrhosis with encephalopathy

perioperative parenteral nutrition in chronic liver disease

Nutrition in acute liver failure

Nutrition and liver transplantation

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INTRODUCTION

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Introduction

Acute liver disease *without* *fulminant hepatic failure* induces the same metabolic effects as any disease associated with an acute phase response.

Effects on nutritional status depend on the **duration** of the disease and on the presence of any **underlying chronic liver disease** that may have already compromised the patient's nutritional status.

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Introduction

Patients with chronic liver disease are ***at risk for malnutrition***

Advanced cirrhosis

High prevalence of mixed **protein energy malnutrition**

Significant **loss of total body protein**

Such as :

Impaired skeletal muscle

Or immunologic performance

Concomitant loss of organ function

Sarcopenia

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Introduction

Subjects **at risk for malnutrition** can reliably be identified by

Medical and ***nutritional history*** and ***clinical examination***

Performed in a standardized fashion by

Subjective global assessment (SGA)

Or ***anthropometry*** (***arm circumference, skin fold thickness***).

At the bedside, ***bioimpedance analysis*** can be used to

Quantitate the ***loss*** of **body cell mass** (**phase angle**).

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Introduction

Total body protein is reduced to a greater degree in

alcoholic cirrhosis

than in that from other origins

Body cell mass can be **restored** under

stable metabolic conditions

and

adequate nutrition

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ORAL DIET

Oral diet

In general, patients with **liver disease** tolerate a **normal diet**.

The majority of patients do not need any dietary restrictions and they may even be harmed by them.

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Oral diet

A **decrease in dietary fat *may be useful***

for **reducing symptoms of steatorrhea**

in patients with *moderate to severe cholestasis*,

but

is associated with a **risk of inadequate energy intake**

and *is not supported by appropriate clinical trials*.

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Oral diet

A **modified eating pattern** with

Four to **seven small meals**,

Including **at least one late evening meal**,

Improves **nitrogen economy**

And **substrate utilisation**

In **stable cirrhotic patients**

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Oral diet

In general, patients with liver disease tolerate a normal diet.

Decrease in dietary fat

- **Reducing symptoms of steatorrhea in patients with moderate to severe cholestasis**
- **But is associated with a risk of inadequate energy intake**

Is not supported by appropriate clinical trials.

Modified eating pattern with :

- **Four to seven small meals**
- **At least one late evening meal**

improves nitrogen economy and substrate utilization in stable cirrhotic patients.

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Oral diet

If patients are able to eat **1.2 g / kg· d protein** or more *without deterioration of mental status*, no modification of their diet is necessary or effective.

In patients with **borderline protein intolerance**

(< 1.0 g/ kg · d protein),

a **vegetable diet** or a **diet rich in fibre** *may help to prevent hepatic encephalopathy.*

It should be kept in mind, however, that such diets *do not consistently improve nitrogen economy.*

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Oral diet

No modification of their diet is necessary or effective

Able to eat

≥1.2 g/ kg · d protein without deterioration of mental status

Borderline protein intolerance

(< 1.0 g / kg · d protein),

A **vegetable** diet

Or a diet rich in **fiber**

May help to prevent **hepatic encephalopathy**.

Such diets do not consistently

Improve nitrogen economy.

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DIETARY SUPPLEMENTS

SUPPLEMENTAL SIP FEEDING
BRANCHED-CHAIN AMINO ACIDS (BCAA)
MICRONUTRIENTS

Dietary supplements

Oral supplementation offers the opportunity to

Provide the patient with the

Desired amount of a particular substrate

While permitting the continuation of an **oral diet**.

Supplemental sip feeding

In malnourished cirrhotic patients

sip feeding

Two 200 ml drinks of a standard polymeric formula

Containing 300 kcal and 19 g protein

Advised to consume these supplements after 8 p.M. (20:00 h).

Nocturnal sip .feeding: improves *total body protein status* significantly better than daytime sip feeding

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Branched-chain amino acids (BCAA)

In the very **rare cirrhotic patient** intolerant of a daily protein intake of≥ 1.0 g/kg.d, dietary protein may need to be reduced to an intake of **0.5g/kg· d,** but this should be avoided if at all possible.

In this situation, positive nitrogen balance and improvement in nitrogen intake can be achieved by oral supplementation of ***BCAA at 0.25 g/kg·d*** without undue risk of encephalopathy.

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Branched-chain amino acids (BCAA)

It should be recognized, however, that **protein intolerance** may be a **transient** phenomenon and that patients may later be tolerant of a higher protein intake when the **increment in daily dietary protein has been slow.**

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Branched-chain amino acids (BCAA)

It has been shown that **long term BCAA supplementation** is associated with **better nitrogen accretion** and **liver function**.

Also,

Supplementation of a normal diet with bcaa (0.25 g/kg.D)

Has yielded positive effects on

Mental state in patients with stable cirrhosis.

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Micronutrients

Deficiency of **fat-soluble vitamins** is observed in patients with

steatorrhea due to cholestasis and bile salt deficiency

and

in alcohol abusers.

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Micronutrients

Both, **vitamin A** and **zinc** supplementation

may *indirectly* alter nutritional state by

improving gustatory function and,

thereby, probably also volitional food intake.

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Micronutrients

A depletion in **water soluble vitamins** is *not uncommon* in

Cirrhosis, particularly in alcohol- induced cirrhosis

Alcohol abusers are at particular risk for vitamin B1 deficiency

Which may be unmasked during

Carbohydrate refeeding and lead to

Wernickes encephalopathy or ***lactic acidosis*** unless

Vitamin B1 has been given preemptively.

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Micronutrients

Zinc and **selenium** deficiency have been observed in both

Alcoholic and non-alcoholic liver disease.

Although an association between *encephalopathy and zinc deficiency* could be demonstrated,

three randomized controlled trials of *oral supplementation* produced *conflicting results* in patients

with *subclinical encephalopathy.*

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Micronutrients

Supplementation with **calcium** and **vitamin D**
has been recommended for treatment of patients
with **osteopenia**,
although these measures
failed to improve ***bone mineral density***
in patients with primary biliary cirrhosis;
estrogen replacement was ***more effective.***

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Micronutrients

Since vitamin and trace element deficiency *may be difficult to diagnose*, **oral supplementation may be instituted liberally**.

Malnourished cirrhotic patients are **at risk**

for the development of

refeeding syndrome and,

thus,

serum levels of

potassium, **magnesium**, and **phosphate**

must be monitored.

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ENTERAL FEEDING

Enteral feeding

Many malnourished cirrhotic patients are anorexic and cannot meet their nutrient requirements by "ad lib" oral intake.

This has been demonstrated in intervention trials when **artificial feeding using liquid formulae** proved to be *more effective in providing adequate amounts of nutrients* than just volitional oral nutrition.

Moreover, in patients with **alcoholic liver disease**,

The ***magnitude of daily caloric intake in general***

Is ***positively correlated with*** **survival**.

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Enteral feeding

The decision of **when** to initiate tube feeding is a subject of *debate*. While tube feeding yields superior results over ad lib oral feeding due to inadequate voluntary intake, some are hesitant because of the *risk of variceal bleeding*.

Slow or intermittent GI bleeding

is not an absolute contraindication to enteral feeding.

Enteral feeding

In any case, patients **must not be fasted**

And, thus,

The introduction of ***tube feeding should not be delayed***

Because **hepatic glycogen stores** are **reduced in cirrhotic patients**

To the extent that,

After an **overnight fast**, ***protein catabolism*** is **increased** for provision of substrates for **gluconeogenesis**.

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Enteral feeding

There is **no general agreement** as to whether enteral feeding should be

intermittent (common clinical practice) or **continuous**

Liquid enteral formulae for **cirrhotic patients**

should preferably be of

high energetic density (1.5 kcal/ ml)

with a **low sodium content (40 mmol /d)**

so that they can be used in patients with **fluid retention**

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PARENTERAL NUTRITION

Parenteral nutrition

Parenteral nutrition should be reserved for those who are

not capable or willing to participate in

oral nutrition or **enteral tube feeding**.

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Parenteral nutrition

Solutions with an

Increased content of **BCAA (40%-45%)**

And *reduced* amounts of

Aromatic amino acids and **methionine**

Are beneficial with regard to improvement of

Mental state in patients with ***hepatic encephalopathy***,

But it is ***uncertain*** whether they are superior to standard amino acid solutions

With regard to **nutritional state**

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Parenteral nutrition

The *efficacy of BCAA in the treatment of hepatic encephalopathy* has been studied in a number of controlled but very heterogeneous trials yielding contradictory findings.

A meta-analysis of these studies showed an improvement in mental state with BCAA-enriched solutions, but no improvement in survival.

Parenteral nutrition

In **cirrhotic patients**, encephalopathy is caused by *serious and life-threatening complications* such as

Infection or haemorrhage,

Which are overriding determinants of **survival** and,

Therefore, it is not surprising that

Parenteral nutrition using BCAA-enriched solutions

Failed *to improve short-term survival.*

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Parenteral nutrition

Also, a cochrane analysis of seven randomised controlled trials studying 397 patients with acute HE found that

The **parenteral BCAA administration** had a significant,

Positive effect on the course of *HE*,

But **not on survival**.

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Parenteral nutrition

Only a ***few trials*** have addressed the question of the *optimal composition of energy-yielding substrates,* fat and carbohydrate, and **no data from systematic studies** are available.

Plasma *clearance* and ***oxidation*** of **infused lipids** are

Normal in ***cirrhosis patients***.

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Parenteral nutrition

Glucose and lipids have been used as metabolic fuels in a caloric ratio of ***40-50: 50-60 (glucose:lipid)*** in two trials.

One study reported that substrate and metabolite concentrations are more favourable when both *glucose and lipids are infused simultaneously* compared to glucose alone.

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Parenteral nutrition

Preoperative parenteral nutrition is not recommended on a *routine* basis in patients with ***stable cirrhosis***.

In **cirrhotic** patients undergoing **non-shunt laparotomy**, ***malnutrition*** *increases the risk of* ***postoperative complications***, including ***death***.

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CONCLUSIONS FOR DIAGNOSIS RELATED NUTRITIONAL THERAPY

ALCOHOLIC STEATOHEPATITIS (ASH)

CIRRHOSIS

CIRRHOSIS WITH ENCEPHALOPATHY

PERIOPERATIVE PARENTERAL NUTRITION IN CHRONIC LIVER DISEASE

NUTRITION IN ACUTE LIVER FAILURE

NUTRITION AND LIVER TRANSPLANTATION

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Alcoholic Steatohepatitis (ASH)

Supplemental enteral nutrition using

Polymeric enteral formulae with a **high energy density**

Should be used when

ASH patients *cannot meet their caloric requirements*

Through **oral intake**

And when there are no ***contraindications*** like

Ileus or *advanced encephalopathy*.

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Alcoholic Steatohepatitis (ASH)

If patients are ***not able to maintain an adequate oral diet***, then delivery via

nasogastric tubes *(even in case of oesophageal varices)*

is recommended.

Parenteral nutrition provides a useful therapeutic option for

The treatment of ***malnutrition***

In alcoholic hepatitis patients

Not suitable for or not tolerating ***enteral nutrition***

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Cirrhosis

If patients with **compensated cirrhosis** require *parenteral* or *enteral* nutrition, then this can be supplied by

Standard solutions *preferably* of a **high nutrient density**

The **therapeutic goal** is to provide

Energy and *protein* or *amino acids*

In amounts **adequate** to ensure

Positive energy and nutrient balance

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Cirrhosis

In **cirrhosis**, ***hepatic glycogen stores are depleted***

→cirrhotic patients who can be *fed sufficiently*,

either by the *oral* or *enteral* route,

but who *have to abstain from food*

temporarily (including nocturnal fasting!) for

more than 12 hours, **should be given** ***IV glucose at 2-3 g /kg· d***

When this fasting period lasts ***longer than 72 h*** total,

PN is required

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Cirrhosis

In patients with **clinically stable cirrhosis**,

An **energy intake** of *1.3 x REE* or *25-30 kcal/kg·d*

And the provision of *1.2 g /kg·d* of **protein**

Is recommended for **maintaining body composition**

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Cirrhosis

In **malnourished patients** requiring repletion,

Protein intake should be <u>higher</u>

And such patients should receive protein of

Up to 1.5 g/kg.d

In these patients,

Low grade encephalopathy (I-II°)

Is *<u>not a contraindication to an adequate supply of protein</u>*

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Cirrhosis with encephalopathy

Adequate nutrition counteracts hepatic encephalopathy

And **parenteral** nutrition is only indicated

When *oral or enteral nutrition are not possible.*

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Cirrhosis with encephalopathy

BCAAs may improve mental state

in patients with *hepatic encephalopathy*, provided that

liver function does not further deteriorate

and *major clinical complications are absent*

The improvement of encephalopathy by BCAAs

is *not necessarily a result of better nutrition alone*

The use of BCAA-enriched solutions has **no effect on survival**

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Cirrhosis with encephalopathy

In patients with **encephalopathy** *as their main problem,*

Other precipitating causes *should be excluded*

Before considering the patient protein-intolerant

Apart from this *very, very rare condition*,

Even transient protein restriction is not beneficial

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Cirrhosis with encephalopathy

In proven protein-intolerant patients,

Oral BCAA-supplementation may be helpful

In achieving an ***adequate nitrogen intake***

Patients in coma (encephalopathy iii-iv°)

Can *safely* be given **TPN** regimens providing

25-30 kcal/ kg.d energy plus **amino acids 1.0 g/kg.d**

Using *BCAA-enriched amino acid solutions*

Perioperative parenteral nutrition in Chronic liver disease

Cirrhotic patients benefit from

Immediate postoperative nutrition

And, in the ***absence of encephalopathy***,

There is ***no need to use BCAA-enriched***

Rather than conventional amino acid solutions

Most likely,

Early enteral nutrition may be *at least as effective as*

Parenteral nutrition

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Nutrition in acute liver failure

There are no controlled studies in these patients, but there are studies giving more insight into the prevailing metabolic changes.

Parenteral glucose (2.0 g/kg·d) administration is ***mandatory***

To *prevent or treat* ***hypoglycaemia***.

Nutrition in acute liver failure

In principle, patients with **acute liver failure**

Should be *fed like other critically ill patients*,

Giving priority to *enteral nutrition over parenteral whenever possible*.

Nutrition and liver transplantation

It has been shown, that

Sarcopenic, **hypermetabolic**, and **malnourished** patients

have a **higher mortality risk** when *undergoing liver transplantation*,

but

malnutrition per se is not a general contraindication to transplantation.

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Nutrition and liver transplantation

After transplantation,

Grafted patients do not differ from *general surgical patients*

With regard to substrate requirements or utilisation

Postoperatively, *early enteral nutrition* is well tolerated and *may reduce complication rates and cost*.

Long-term survivors are at risk of developing sarcopenic obesity and cardiovascular morbidity due to metabolic syndrome

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Thank you…