Rizika parenterální výživy

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Historie parenterální výživy

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Tabulka 1. Klíčové objevy vztahující se k parenterální výživě (PV) – řazeno chronologicky (1,2)

Obecné	
1628	objev krevního oběhu Williamem Harweyem
1656–8	i.v. podání alkoholu, opia, tuků zvířatům
1831	úspěšné podání slaných roztoků i.v. při léčbě ztrát tekutin u cholery
1904	Freidreich – subkutánní podání vody, elektrolytů, tuků, glukó
1923	Seibrtovy práce o pyrogenitě, popsání metod k zajištění nep
1955–65	periferní a někdy centrální PV po omezenou dobu
1967	úspěšná prolongovaná i.v. výživa
1967	úspěšné zavedení prolongovaného centrálního žilního katét
1969	domácí PV v USA
od 1970	domácí PV v evropských zemích
Makronutrienty	
Cukry	
1843	C. Bernard ukázal parenterální podání roztoků cukru zvířatům
1887	Landner navrhnul podání glukózy jako součást režimu uměl
1896	úspěšné i.v. podání glukózy člověku (Biedl a Kraus, 1896)
1967	dlouhodobé humánní infuzní podání hypertonické glukózy
Proteiny/aminokyseliny	
1870–1900	infuzní podání mléka člověku, objevovaly se závažné systémové reakce
1913	úspěšné podání nealergizujících proteinových hydrolyzátů kozám (Henriques
1939	i.v. podání 2% kaseinového hydrolyzátu s 8% glukózou člověku, bez alergické
1940	i.v. podání kompletní směsi syntetických aminokyselin člověku
1964	představen první komerční roztok aminokyselin v Německu
od 1970	zavedení větvených aminokyselin do parenterální výživy
od 1980	zavedení ketoanalog do parenterální výživy
Tuky	
1678	i.v. podání tuku zvířatům (Ch. Vren)
1869	s.c. podání tuku psovi bez vedlejších účinků (Menzel a Perco)
1869	s.c. podání tuku člověku trpícího malnutricí
1915	první tuková emulze podána i.v. zvířeti (Mulrin a Riche)
1920	první tuková emulze podána i.v. v pediatrii v USA
1961	bezpečná a efektivní tuková emulze (Intralipid) vyvinutá Wretlindem ve Švéds používána ve většině evropských států od 1963, v severní Americe až od 1977
1964	FDA v USA zakazuje používání tukových olejů na bázi ricinového a bavlníkové vzhledem k nežádoucím účinkům
od 1980	zavedení nových typů olejů (triacylglyceroly se středním řetězcem (MCT), rybí rované lipidy)

Prakt. lékáren. 2009; 5(2): 97–98

Nekončící kritika/obhajoba parenterální výživy

- Lipman TO. Grains or veins: is enteral nutrition really better than parenteral nutrition? A look et the evidence. *JPEN 1998;22:167-182*
- Jeejeebboy KN. Total parenteral nutrition: potion or poison? *Am J Clin Nutr. 2001;74:160-163*
- Bistrian BR. Death by total parenteral nutrition. *Crit Care Med. 2011; 39(11):2586.*
- Marik PE. Death by total parenteral nutrition: the saga continues. *Crit Care Med. 2011;39:1536-1537*

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Early versus Late Parenteral Nutrition in Critically Ill Adults

Michael P. Casaer, M.D., Dieter Mesotten, M.D., Greet Hermans, M.D., Ph.D., Pieter J. Wouters, R.N Miet Schetz, M.D., Ph.D., Geert Meyfroidt, M.D., Sophie Van Cromphaut, M.D., Ph.D., Catherine Inge Philippe Meersseman, M.D., Jan Muller, M.D., Dirk Vlassela Yves Debaveye, M.D., Ph.D., Lars Desmet, M.D., Jasperina Aime Van Assche, M.D., Simon Vanderheyden, E Alexander Wilmer, M.D., Ph.D., and Greet Van den Berghe

ABSTRACT

BACKGROUND

Controversy exists about the timing of the initiation of parenter cally ill adults in whom caloric targets cannot be met by entera

METHODS

In this randomized, multicenter trial, we compared early initiati trition (European guidelines) with late initiation (American and C in adults in the intensive care unit (ICU) to supplement insufficie In 2312 patients, parenteral nutrition was initiated within 48 hou sion (early-initiation group), whereas in 2328 patients, parenter initiated before day 8 (late-initiation group). A protocol for th enteral nutrition was applied to both groups, and insulin was normoglycemia.

Table 2. Outcomes.*

Variable	Late-Initiation Group (N=2328)	Early-Initiation Group (N=2312)	P Value
Safety outcome			
Vital status — no. (%)			
Discharged live from ICU within 8 days	1750 (75.2)	1658 (71.7)	0.007
Death			
In ICU	141 (6.1)	146 (6.3)	0.76
In hospital	242 (10.4)	251 (10.9)	0.63
Within 90 days after enrollment†	257 (11.2)	255 (11.2)	1.00
Nutrition-related complication — no. (%)	423 (18.2)	434 (18.8)	0.62
Hypoglycemia during intervention — no. (%)‡	81 (3.5)	45 (1.9)	0.001
Primary outcome			
Duration of stay in ICU§			
Median (interquartile range) — days	3 (2–7)	4 (2–9)	0.02
Duration >3 days — no. (%)	1117 (48.0)	1185 (51.3)	0.02
Hazard ratio (95% CI) for time to discharge alive from ICU	1.06 (1.00–1.13)		0.04
Secondary outcome			
New infection — no. (%)			
Any	531 (22.8)	605 (26.2)	0.008
Airway or lung	381 (16.4)	447 (19.3)	0.009
Bloodstream	142 (6.1)	174 (7.5)	0.05
Wound	64 (2.7)	98 (4.2)	0.006
Urinary tract	60 (2.6)	72 (3.1)	0.28
Inflammation			
Median peak C-reactive protein level during ICU stay (interquartile range) — mg/liter	190.6 (100.8–263.2)	159.7 (84.3–243.5)	<0.001
Mechanical ventilation			
Median duration (interquartile range) — days	2 (1–5)	2 (1–5)	0.02
Duration >2 days — no. (%)	846 (36.3)	930 (40.2)	0.006

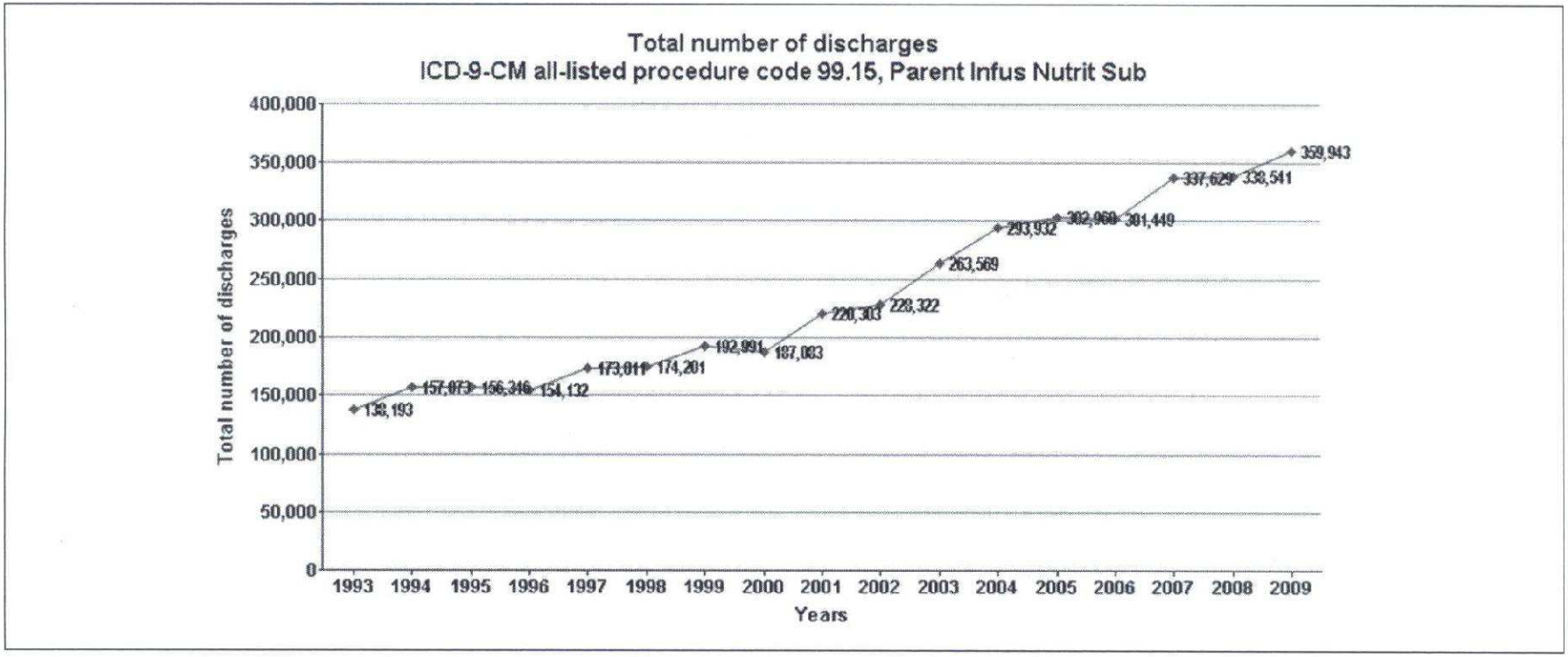
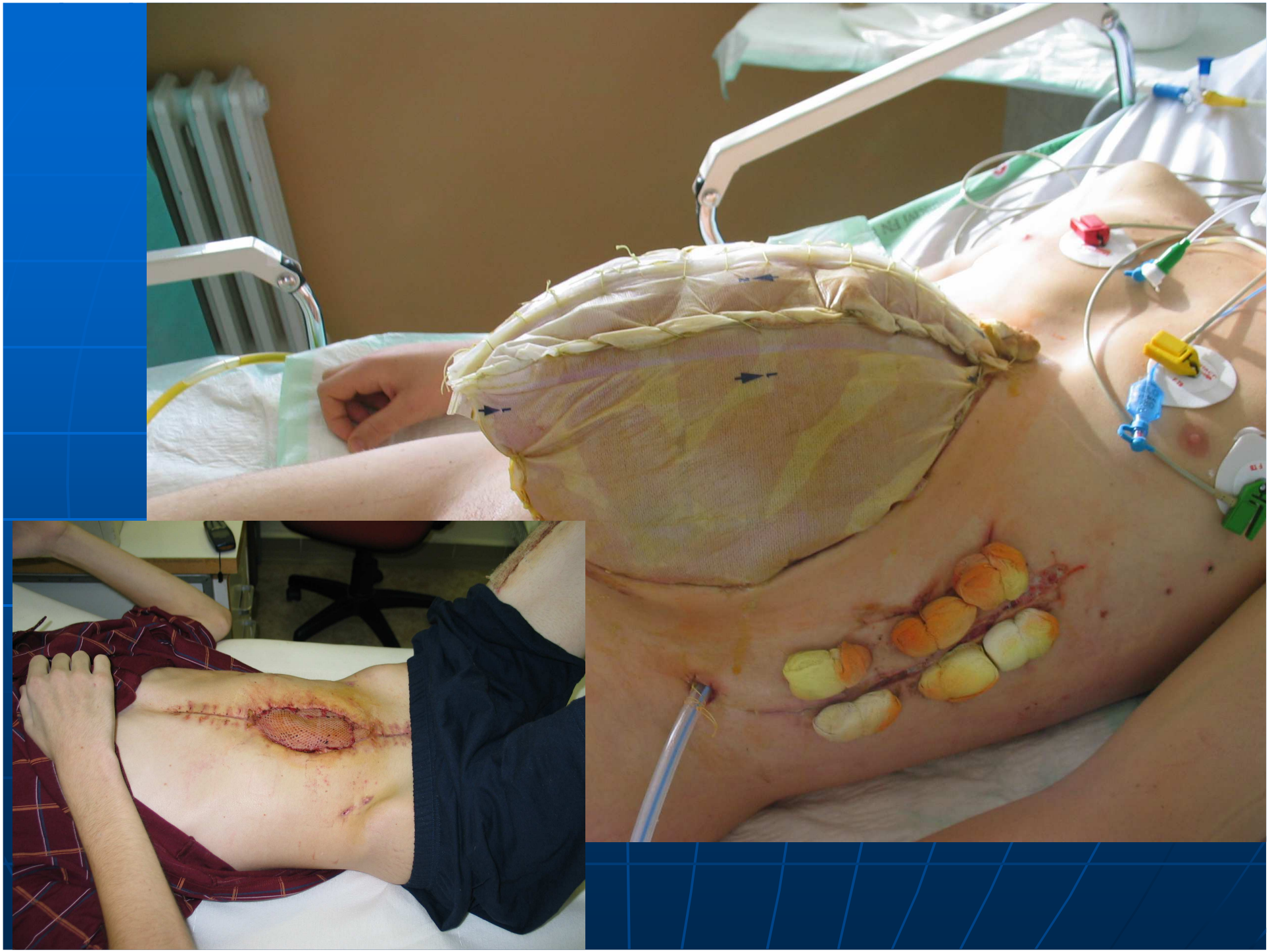


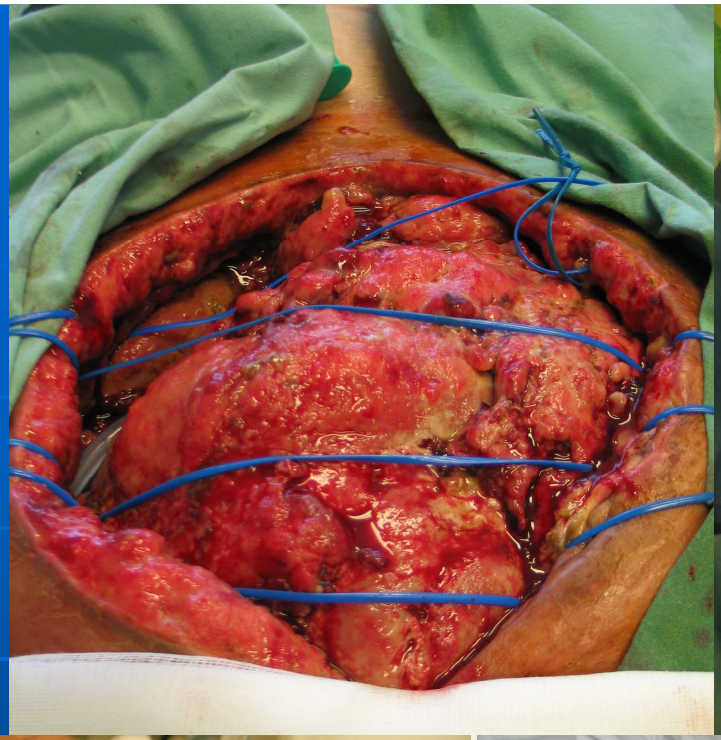
Figure 2. Annual parenteral nutrition use in hospitals: 1993–2009. Source: Data from HCUPnet: National Estimates on Characteristics of Various Types of Hospitalizations, the Agency for Healthcare Research and Statistics, 2011. *International Classification of Diseases, Ninth Revision, Clinical Modification* (*ICD-9-CM*) all-listed procedure code 99.15, Parent Infus Nutri Sub = code for parenteral nutrition.

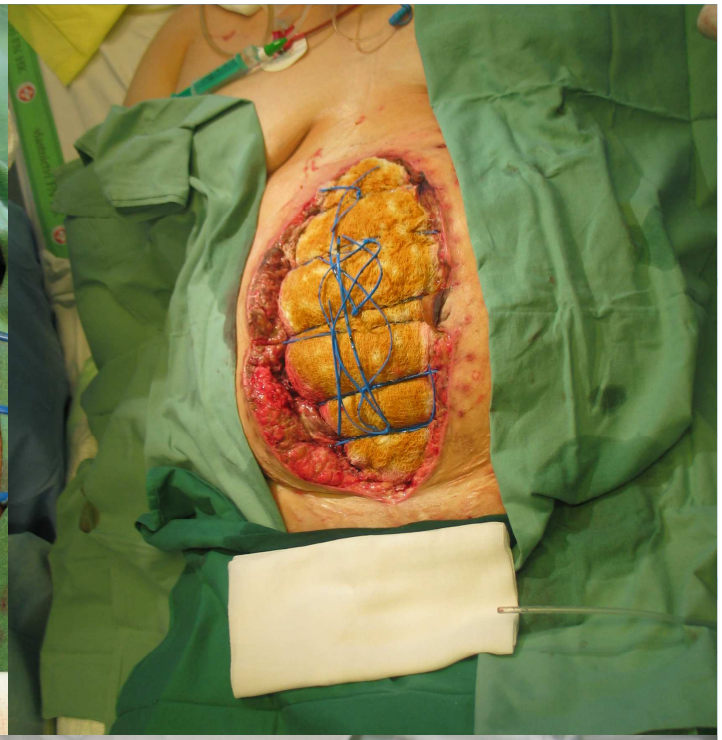
Mirtalo JM. Parenteral nutrition: Can outcomes be improved? JPEN 2013;37(2):181-189

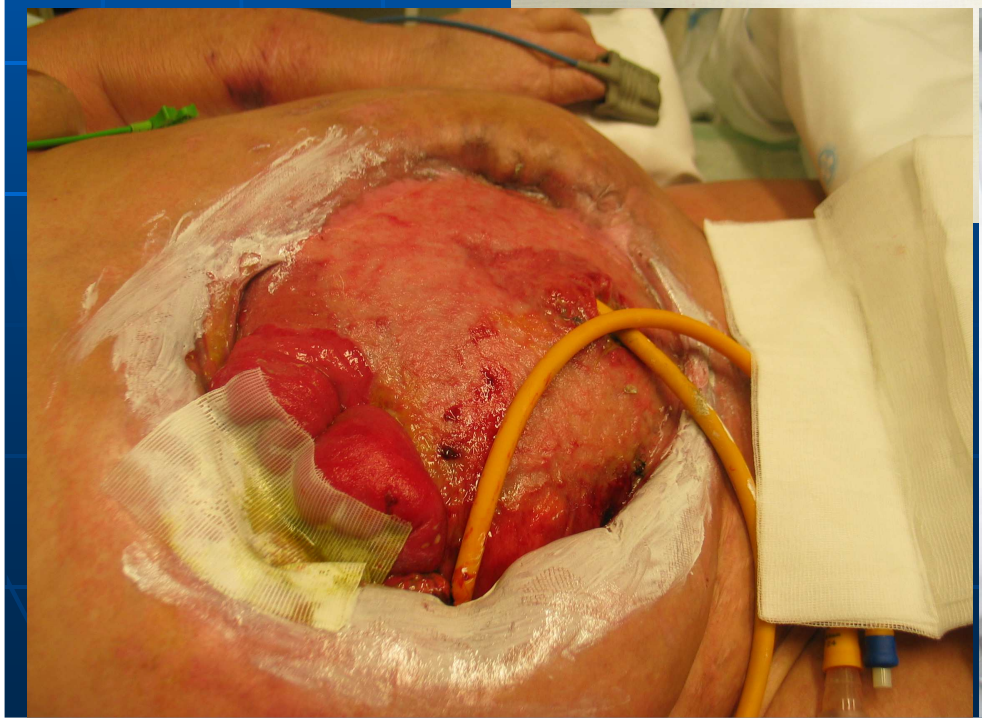


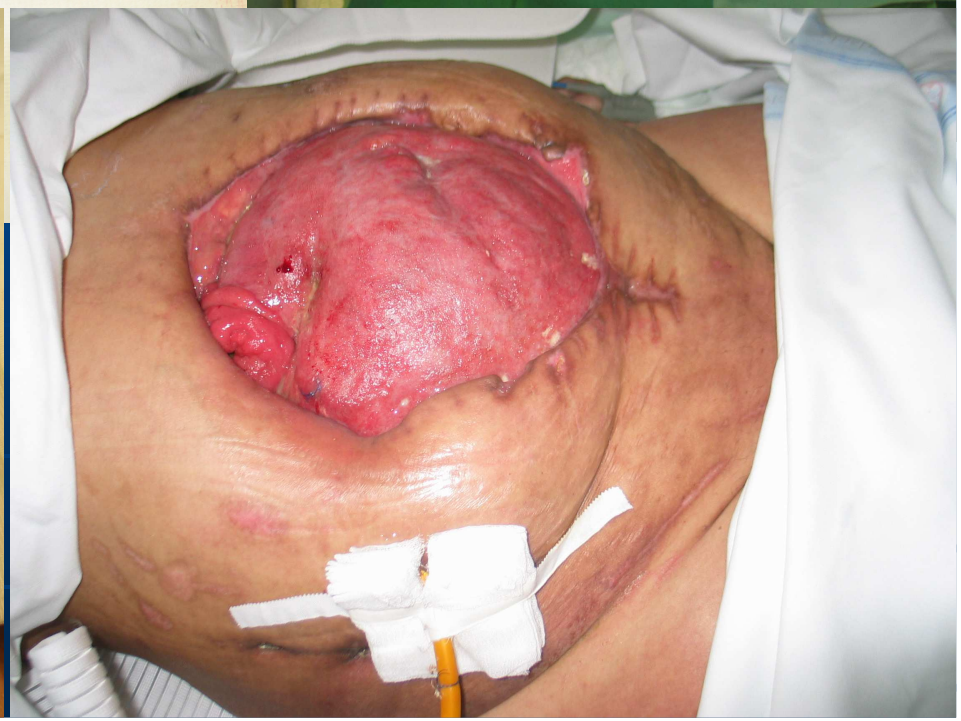
Parenterální výživa - zachraňuje v kritickém stavu

PV x EV









Rizika parenterální výživy

1. Infekce
2. Hepatopatie
3. Technické chyby

Infekční komplikace parenterální výživy

- Stará informace při hyperkalorickém (56kcal/kg) režimu a velké dodávce glukózy (>300g/den)

Rosmarin DK 1996, Kortez RL 2001, Muller TF 1995

- 1643/56 pacientů s nozokomiální infekcí,
  5 let, retrospektivní analýza rizikových faktorů

Göçmez C et al. Evaluation of risk factors affecting hospital-acquired infections in the neurosurgery intensive care unit. Int J Neurosci. 2013 Dec 10

- V rameni standardní parenterální výživy více infekcí proti vodě

Xian-Li H et al. Effect of total parenteral nutrition (TPN) with and without glutamine dipeptide supplementation on outcome in severe acute pancreatitis (SAP). Clinical Nutrition Supplements 2004;1:43.

J Vasc Access 2012; 00 (00): 000-000
DOI: 10.5301/jva.5000133

ORIGINAL ARTICLE

Catheter-related complications in long-term home parenteral nutrition patients with chronic intestinal failure

Francesco William Guglielmi[1], Nunzia Regano[1], Silvia Mazzuoli[1], Massimiliano Rizzi[1], Simona Fregnan[1], Giuseppina Leogrande[1], Irene Addante[1], Altomarino Guglielmi[2]

[1]Department of Gastroenterology & Artificial Nutrition, "San Nicola Pellegrino" Hospital - Trani, (BT) - Italy
[2]General Surgery Department II University 'Francesco Paccione', D.E.T.O. - University of Bari - Italy

TABLE II - CENTRAL VENOUS CATHETER-RELATED COMPLICATIONS IN CANCER AND NONCANCER PATIENTS

	With cancer	Without cancer	P<
Patients	139	131	
Patients with complications	26	45	0.05
Overall CVC-related complications	46	171	0.005
Mean number of CVC-related complications/patient	0.33	1.30	0.005
Incidence rate/1000 days-catheter CVC-related complications:			
Total	0.89	0.51	0.005
Septic	0.71	0.46	0.001
Mechanical	0.82	0.91	
Local skin infection	0.01	0.03	0.05

CVC, central venous catheter

TABLE III - SEPSIS OF CENTRAL VENOUS CATHETER IN CANCER AND NON-CANCER PATIENTS

	Total patients		Cancer patients		Noncancer Patients	
	Number of episodes	Percent	Number of episodes	Percent	Number of episodes	Percent
Sepsis	75	16	39	16	36	17
Gram+	41	55	20	51	21	58*
Gram-	24	32	11	28	13	36*
Candida	10	13	8	21	2	6^

^ $p<0.001$, * $p<0.05$

Clinical Nutrition 30 (2011) 730–737

Contents lists available at ScienceDirect

Clinical Nutrition

journal homepage: http://www.elsevier.com/locate/clnu

Original article

Efficacy of hypocaloric parenteral nutrition for surgical patients: A systematic review and meta-analysis☆

Hua Jiang [a,b], Ming-Wei Sun [a,*], Brook Hefright [c], Wei Chen [d], Charles Damien Lu [a], Jun Zeng [a]

Table 4
Outcomes of included studies.

Author, year	Average calorie Administrated (kcal/kg/d)		Nitrogen Administrated (g/kg/d)		Mortality n/ N(ITT)		Infectious Morbidity n/ N(ITT)		ICU–LOS mean ± xs or (median)		LOS mean ± xs or (median)	
	Hypo	Control	Hypo	Control	Hypo	Control	Hypo	Control	Hypo	Control	Hypo	Control
Battistella FD, 1997	21	30	0.24	0.24	2/27	0/30	13/27[a]	22/30[a]	18 ± 12	29 ± 22	27 ± 16	39 ± 24
Jiang ZM 2003	18	30	0.2	0.1	n/a	n/a	1/50	4/50	n/a	n/a	13 ± 5	16 ± 6
Ahren CL 2005	20	30	0.26	0.24	n/a	n/a	5/20	2/20	14	24	15	25
Mao YL 2005	18	28	0.2	0.2	n/a	n/a	1/22	1/20	n/a	n/a	10	11
Zhan WH 2007	18	30	0.1	0.2	n/a	n/a	2/60	10/58(10/60)	n/a	n/a	12.35 ± 4.04	14.19 ± 5.89

[a] Pneumonia n/a: not applicable.

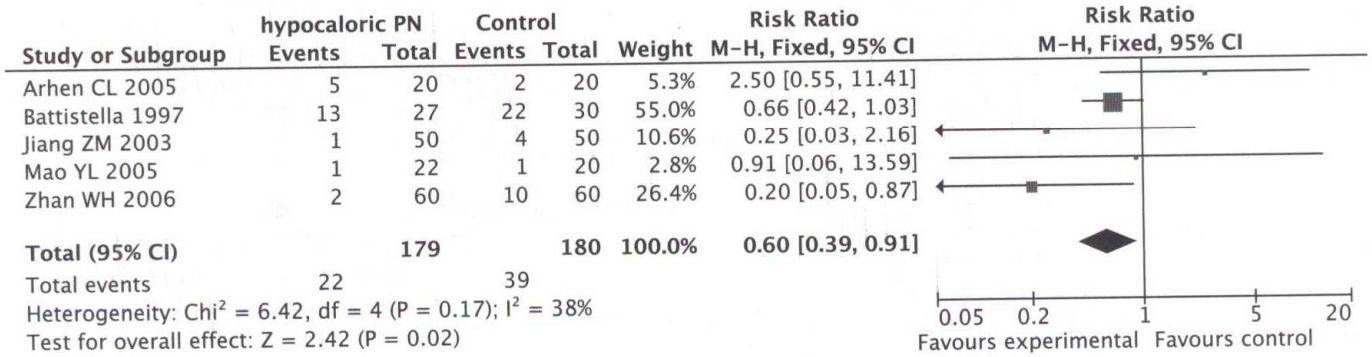


Fig. 2. Effect of hypocaloric PN on infectious complications: overall results of aggregated data from all included trials. Legend: 95%CI, 95% confidence interval; M-H, Mantel-Haenszel model.

The New England
Journal of Medicine

VOLUME 345 NOVEMBER 8, 2001 NUMBER 19

INTENSIVE INSULIN THERAPY IN CRITICALLY ILL PATIENTS

GREET VAN DEN BERGHE, M.D., PH.D., PIETER WOUTERS, M.SC., FRANK WEEKERS, M.D., CHARLES VERWAEST, M.D., FRANS BRUYNINCKX, M.D., MIET SCHETZ, M.D., PH.D., DIRK VLASSELAERS, M.D., PATRICK FERDINANDE, M.D., PH.D., PETER LAUWERS, M.D., AND ROGER BOUILLON, M.D., PH.D.

TABLE 2. INSULIN THERAPY AND CONTROL OF BLOOD GLUCOSE LEVELS.*

VARIABLE	CONVENTIONAL TREATMENT (N=783)	INTENSIVE TREATMENT (N=765)	P VALUE†
Administration of insulin — no. (%)	307 (39.2)	755 (98.7)	<0.001
Insulin dose — IU/day‡			
Median	33	71	
Interquartile range	17–56	48–100	<0.001
Duration of insulin use — % of ICU stay			
Median	67	100	<0.001
Interquartile range	40–100		
Morning blood glucose ?/dl§			
nts	153±33	103±19	<0.001
receiving insulin	173±33	103±18	<0.001

TABLE 3. MORTALITY.

VARIABLE	CONVENTIONAL TREATMENT (N=783)	INTENSIVE TREATMENT (N=765)	P VALU
Death during intensive care — no./total no. (%)	63/783 (8.0)	35/765 (4.6)	<0.04 (ad
During first 5 days of intensive care	14/783 (1.8)	13/765 (1.7)	0.9
Among patients receiving intensive care for >5 days	49/243 (20.2)	22/208 (10.6)	0.005
Reason for intensive care			
Cardiac surgery	25/493 (5.1)	10/477 (2.1)	
Neurologic disease, cerebral trauma, or brain surgery	7/30 (23.3)	6/33 (18.2)	
Thoracic surgery, respiratory insufficiency, or both	10/56 (17.9)	5/66 (7.6)	
Abdominal surgery or peritonitis	9/58 (15.5)	6/45 (13.3)	
Vascular surgery	2/32 (6.2)	2/30 (6.7)	
Multiple trauma or severe burns	3/35 (8.6)	4/33 (12.1)	
Transplantation	1/44 (2.3)	2/46 (4.4)	
Other	6/35 (17.1)	0/35	
No history of diabetes	57/680 (8.4)	31/664 (4.7)	
No history of diabetes and >5 days of intensive care	45/218 (20.6)	20/187 (10.7)	
History of diabetes	6/103 (5.8)	4/101 (4.0)	
History of diabetes and >5 days of intensive care	4/25 (16.0)	2/21 (9.5)	
Cause of death — no.			0.02
Multiple-organ failure with proven septic focus	33	8	
Multiple-organ failure without detectable septic focus	18	14	
Severe brain damage	5	3	
Acute cardiovascular collapse	7	10	
In-hospital death — no./total no. (%)			
All patients	85/783 (10.9)	55/765 (7.2)	0.01
Patients receiving intensive care for >5 days	64/243 (26.3)	35/208 (16.8)	0.01

On admission, all patients were fed continuously with intravenous glucose (200 to 300 g per 24 hours). The next day, total parenteral, combined parenteral and enteral, or total enteral feeding was instituted according to a standardized schedule, with 20 to 30 nonprotein kilocalories per kilogram of body weight per 24 hours and a balanced composition (including 0.13 to 0.26 g of nitrogen per kilogram per 24 hours and 20 to 40 percent of nonprotein calories in the form of lipids).[23] Total enteral feeding was attempted as early as possible.

4,4 – 6,1 mmol/l

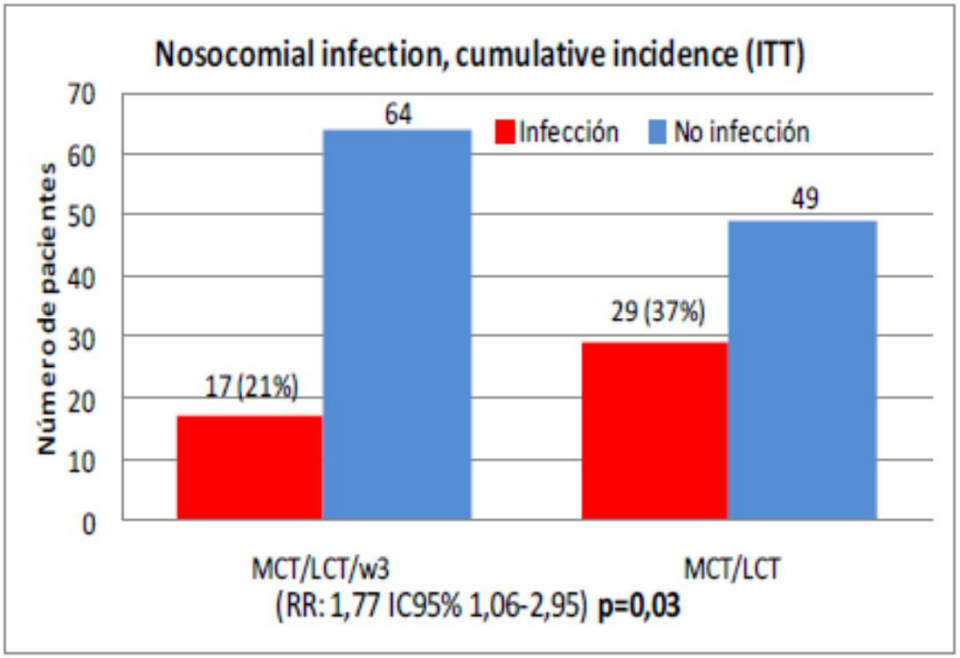
Effect of two different lipid emulsions on the incidence of nosocomial infections in critically ill patients treated with TPN: A multicenter, prospective, double blind randomized trial. ICULIP study.

A. Bonet, T. Grau, A. García de Lorenzo, C. Sánchez, A. Rodríguez, E. Miñambres, A. Robles, J. Acosta, J.I. Herrero, A. Mesejo
Fo the Working Group of Nutrition and metabolism ofSEMYCIUC

Methods

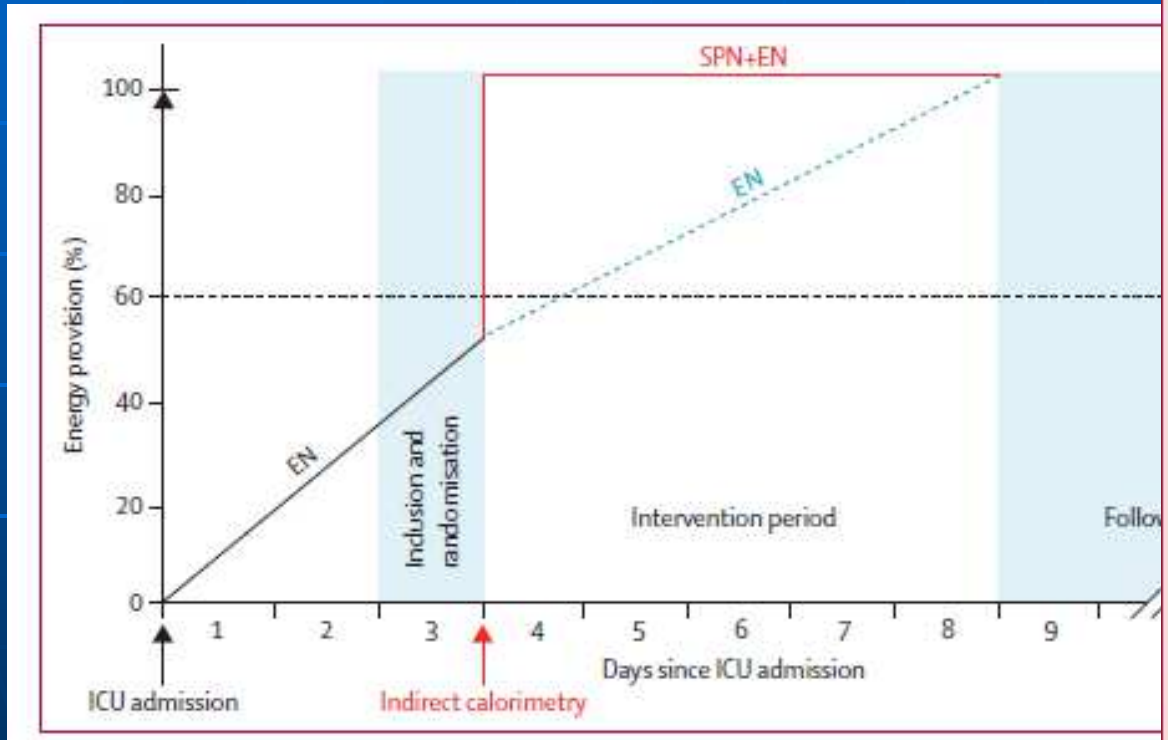
- **Prospective, multicenter, double blind, randomized trial**
- **Phase IV Clinical Trail performed in 17 Spanish ICUs.**
- **Automatic Randomization at two levels: APACHE II (two groups, minor and major 20) and the presence of severe sepsis and / or septic shock at baseline.**

Eudra-CT: 2005-003542-33
ClinicalTrials.gov Identifier: NCT00396461



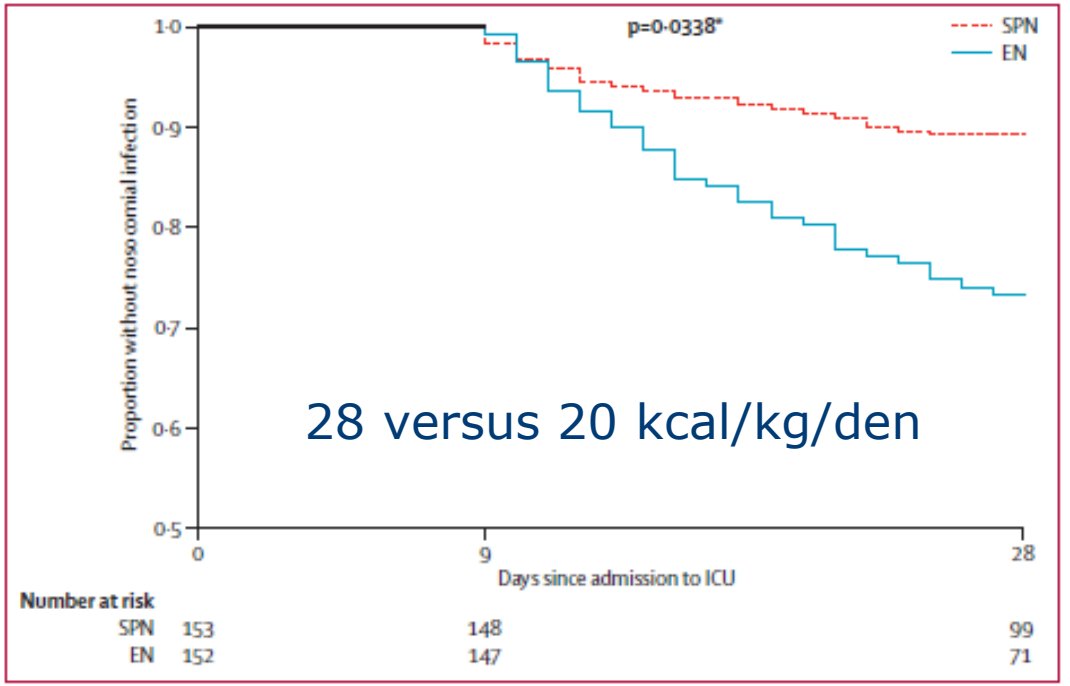
Optimisation of energy provision with supplemental parenteral nutrition in critically ill patients: a randomised controlled clinical trial

Claudia Paula Heidegger, Mette M Berger, Séverine Graf, Walter Zingg, Patrice Darmon, Michael C Costanza, Ronan Thibault, Claude Pichard



	SPN (n=153)	EN (n=152)
Age (year)	61 (16)	60 (16)
Weight (kg)	74·8 (12·9)	77·3 (15·3)
Body-mass index (kg/m²)	25·4 (3·9)	26·4 (4·6)
SAPS II score	49 (17)	47 (15)
APACHE II score	22 (7)	23 (7)
Hospital		
Geneva	99 (65%)	101 (66%)
Lausanne	54 (35%)	51 (34%)
Surgery	70 (46%)	69 (45%)
Sex (male)	110 (72%)	105 (69%)
Primary diagnosis		
Shock (all)	30 (20%)	29 (19%)
Neurological	23 (15%)	23 (15%)
Cardiac surgery	21 (14%)	18 (12%)
Polytrauma	19 (12%)	20 (13%)
Pneumonia	16 (10%)	8 (5%)
Cardiac arrest	11 (7%)	11 (7%)
Respiratory failure	8 (5%)	13 (9%)
Myocardial infarction	6 (4%)	9 (6%)
Acute pancreatitis	4 (3%)	2 (1%)
Liver failure	0	2 (1%)
Other	15 (10%)	17 (12%)
Infection at ICU admission	77 (50%)	65 (43%)
Energy target* (kcal/day)	1892 (365)	1836 (388)
Energy target per ideal bodyweight* (kcal/kg/day)	28 (4)	27 (5)
Protein target† (g/day)	81 (7)	80 (6)

Heidegger CP. Lancet 2013;381: 385 - 393



28 versus 20 kcal/kg/den

	Intervention period (days 4–8)		Follow-up (days 9–28)	
	SPN	EN	SPN	EN
Pneumonia	35 (67%)	28 (65%)	22 (46%)	32 (45%)
Bloodstream infection	10 (19%)	6 (14%)	9 (19%)	13 (18%)
Urogenital infection	4 (8%)	2 (5%)	7 (15%)	5 (7%)
Abdominal infection	1 (2%)	4 (9%)	8 (17%)	8 (11%)
Other infection*	2 (4%)	3 (7%)	2 (4%)	13 (18%)

Data are number of events (%). Patients can have one or more infections. Comparisons by type of infections were not significant for the intervention period (p=0·4866) or follow-up period (p=0·1476). SPN=supplemental parenteral nutrition. EN=enteral nutrition. *Skin, bone, soft tissue, ear, nose, throat, upper respiratory, and non-pulmonary intrathoracic infections.

Table 3: Distribution of nosocomial infections during intervention and follow-up

Parenterální výživa a infekční komplikace

1. Hyperglykémie a vysoká dávka glukózy
2. Katétr
3. Střevní atrofie

Jsou to vedlejší účinky parenterální výživy?

Hepatopatie a parenterální výživa

- Ne zcela objasněna
- 40-60% dětí, 15-40% dospělých na dlouhodobé parenterální výživě
- Rizikové faktory:
  střevní klid
  dlouhodobá parenterální výživa
  podvýživa nebo overfeeding
  velký objem glukózy
  fytosteroly kontaminující sojový olej
  rekurentní sepse

Léčba hepatopatie

- Preventivní opatření a léčba rizikových faktorů
- Tuková emulze s rybím tukem
- Ursodeoxycholová kyselina
- Enterální stimulace, vláknina a probiotika
- Glutamin ?
- Transplantace

Xu ZW, Li YS. Hepatobiliary Pancreat Dis Int 2012;11:586-593)

Technické chyby

- Riziko chyby 1,6% (indikace, dávka, složení nutrientů)
- 8% z chyb bylo závažných, většinou spojených s podáváním

  Sacks GS. Pharmacotherapy 2009;29:966-974
- 2/3 respondentů observační studie zaznamenalo 1-5 chyb/měsíc, polovina respondentů zaznamenala nebezpečnou příhodu /měsíc

  Seres D. JPEN 2006;30:259-265

Renal consequences of parenteral nutrition

Jan Dudley · Rebekah Rogers · Laura Sealy

Table 4 Metabolic and electrolyte complications of parenteral nutrition (PN)

Acid-base disorders	Hypokalemia	Hypomagnesemia	Hypophosphatemia	Hyponatremia	Hyperglycemia
Metabolic acidosis; Causes:	Causes:	Causes:	Causes:	Causes:	Causes:
1. Phosphate deficiency	1. Secondary hyperinsulinism due to glucose & amino acid load	1. Inadequate intake	1. Inadequate intake	1. Inadequate intake	1. Excessive glucose in PN solution
2. Chloride Vs acetate PN	2. Accelerated renal excretion of potassium: • Metabolic alkalosis	2. Increased renal excretion of Mg: • Resolving ATN • Diuretics	2. Chronic acidosis	2. Concurrent administration of large volumes of intravenous sodium chloride	2. Concurrent medications e.g. glucocorticoids
3. Loss of alkali from gastro-intestinal tract	• Glycosuria	• Amino-glycosides	3. Anabolic response to amino acids/glucose		3. Systemic response to inflammation
4. Sepsis	• Glucocorticoids • Diuretics				
		3. Malnutrition/Malabsorption			
Metabolic alkalosis Causes:	3. Loss of potassium from GI tract				
1. 'refeeding alkalosis'					
2. Vomiting					
3. Potassium deficiency					

ATN, acute tubular necrosis

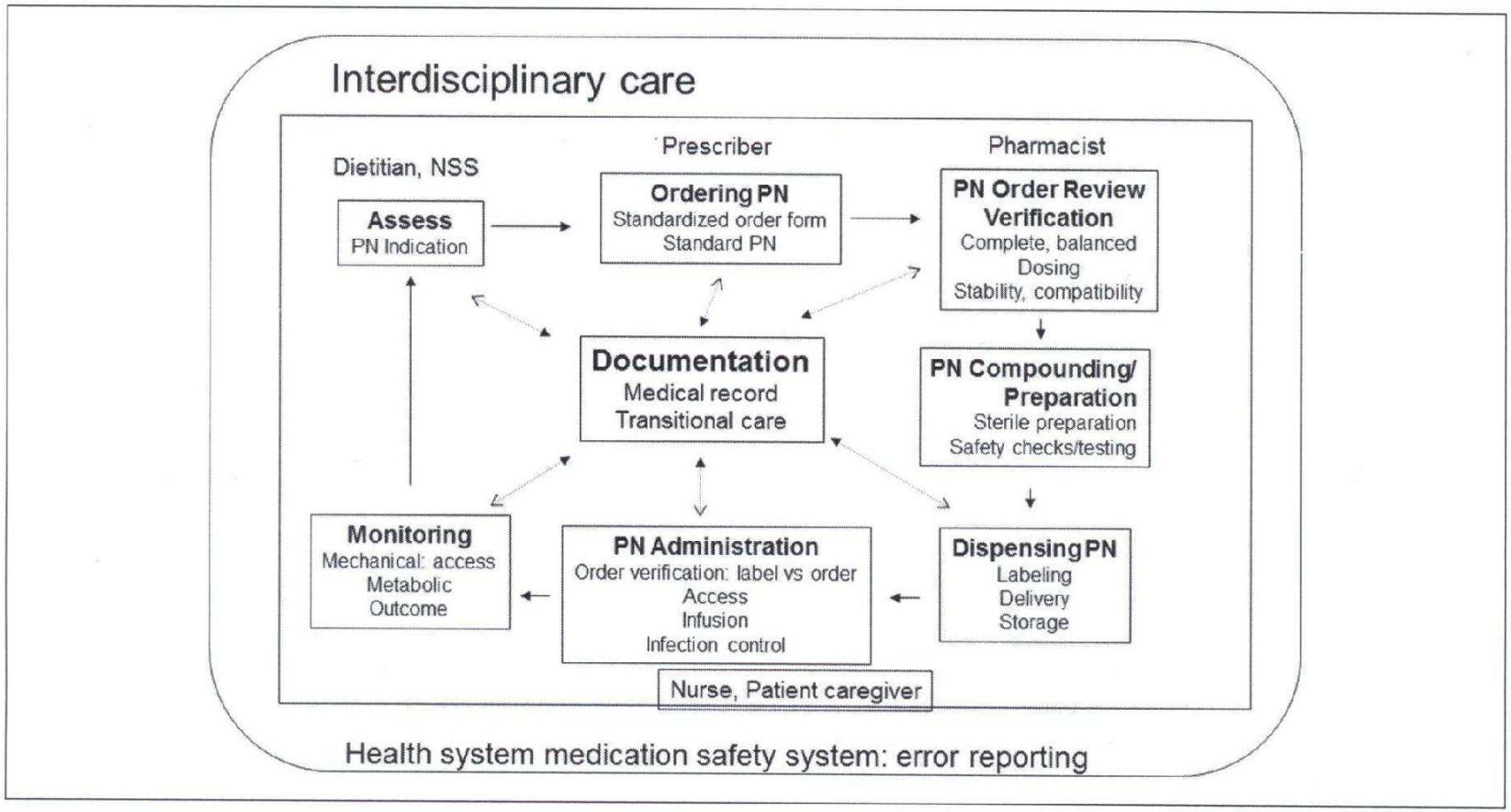


Figure 6. Parenteral nutrition: a system of care. NSS, nutrition support service; PN, parenteral nutrition.

Mirtalo JM. Parenteral nutrition: Can outcomes be improved? JPEN 2013;37(2):181-189

Závěr:

1. Bezpečnost parenterální výživy je podmíněna hlavně kvalitou péče
2. Na zvýšeném riziku infekce se podílí hlavně hyperglykémie
3. Moderní složení akcentuje dodávku aminokyselin s redukovanou dávkou glukózy podle hmotnosti, tukové emulze s dodávkou ω-3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