Nice guidelines refeeding syndrome 2020

I'm not robot
reCAPTCHA
Continue

This website uses cookies. By continuing to use this website, you consent to the use of cookies. For information about cookies and how to disable them, visit our privacy and cookie policy. I got it, thank you! Work off campus? Find out about our options for Remote Access Volume 35, Issue 2 Volume 35Issue 3Nutrition in Clinical Practice Pages: 584-585 First published online: April 15, 2020 Financial Disclosures: None Announced. Conflicts of interest: Phil Ayers is associated with the speakers of the bureau Fresenius Kabi. Kathleen M. Gura is a speaker and consultant to Fresenius Kabi. David C. Evans is a speaker and consultant for Abbott Nutrition, Fresenius Kabi, Alcresta Advisory Board and Coram CVS Home Infusion. Gordon S. Sachs works in Fresenius Kabi, North America. In the spring of 2017, the American Society of Parenteral and Enteral Nutrition (ASPEN) Parent Committee on Nutrition Safety and the Committee on Clinical Practice convened an inter-professional task force to develop consensus recommendations to identify patients with feeding syndrome or risk for the treatment of syndrome (RS) and to avoid and manage the condition. This report contains descriptive reviews and consensus recommendations for hospitalized adults and paediatric populations. A consensus process has been developed in response to differences in definitions and methods reported in the literature. Subgroups of the authors examined specific questions through a literature review. The summary was submitted to the entire group for discussion by email and teleconference. Each section was then drawn up into a major document, several of which were considered by the committee. This group proposes a new clinical definition and criteria for risk stratification through treatment and screening strategies. The authors suggest that the diagnostic criteria of MS should be stratified as follows: reduction in any 2, or 3 levels of serum phosphorus, potassium and/or magnesium by 10%-20% (soft), 20%-30% (moderate), or zgt;30% and/or organ dysfunction as a result of reducing any of them and/or

due to thiamine deficiency (severe), occur within 5 days of reintroduction of calories. These consensus recommendations are intended to make recommendations on risk recognition and identification, stratification, avoidance and management of RS. This consensus definition is additionally intended to be used as a basis for further studies of morbidity, effects, pathophysiology, avoidance and treatment of MS. In 2017, the American Parenteral and Enteral Nutrition Society (ASPEN) Parenteral Nutrition (PN) Safety Committee and the Clinical Practice Committee convened an inter-professional task force consisting of nutritionists, nurses, pharmacists and physicians tasked with developing the development of recommendations for screening and management of patients who are at risk or have developed overfeeding syndrome (RS). This paper summarizes and the consensus of the task force. Due to the heterogeneity of literature, this report focuses on RS in hospitalized adults and pediatric populations. Below is the proposed unification clinical definition of MS, as well as the proposed updated criteria for MS risk. These consensus recommendations are designed to provide clinical recommendations for RS prevention and management to medical organizations and clinical professionals. The literature surrounding neonatal malnutrition and MS is complex. Specific recommendations for this group of people were considered to be beyond the scope of the project, and the authors made only general comments. These recommendations are not medical or other professional recommendations and should not be considered as such. To the extent that the information published in this report can be used to assist in patient care, this is the result of a single professional judgment by a physician whose judgment is a major component of quality care. The information provided in these recommendations cannot replace the exercise of such a judgement by a health professional. Circumstances in clinical settings and patient testimony may require action different from those recommended in this document. In these cases, the judgment of the treating professional should prevail. This document has been approved by the ASPEN Board of Directors. RS has historically been described as a series of metabolic and electrolyte changes resulting from reintroduction and/or calorie increases after a period of reduced or lack of calorie intake. In this context, calories can come from any source: oral diet, enterative diet, PN, or intravenous (IV) dextrose (e.g., 5% dextrose solution). Despite the long-standing recognition of MS as a mechanism for potential serious nutritional complications intervention, high-quality scientific evidence on clinical syndrome is lacking. Most reports rely on retrospective, observational data and use widely contradictory definitions of the syndrome. The lack of a standard definition hinders the assessment of the incidence of MS, as well as efforts to develop well-thought-out, controlled trials that can lead to effective strategies for its recognition, avoidance and treatment. Hypophosphatemia is often considered a hallmark of this syndrome, and some authors have suggested that hypophosphatemia is the most common abnormal electrolyte in suspected cases.1-3 However, it may be the result of the definition of bias or relatively less causes of hypophosphateemia, compared to hypocalemia, making RS a more important cause of hypophosphatemia than hypocemia. Other changes in the electrolyte may be just as important. The RS was first described during the Second War. PRISONERS of war, camp survivors and victims of hunger Unexpected morbidity and mortality during meals.4-6 In 1944, Keys et al reported the results of a prospective randomized control study assessing the physiological effects of prolonged hunger on conscientious objectors and their subsequent rehabilitation. They were adults with strong anti-war sentiments who were allowed to replace service with social goods rather than be drafted into the army. This landmark study, known as the Minnesota Hunger Experiment, stands as one of the few studies to directly assess the symptoms seen during the rehabilitation of the nutrition of malnourished patients and served as one of the foundations of how doctors understand RS today. It is unlikely that such a study will be reviewed by the Institutional Review Board in the current era. With these initial reports, reporting on RS has focused mainly on those with eating disorders (particularly anorexia nervosa), adult patients who are severely malnourished due to underlying diseases, or geriatric patients with chronically reduced oral intake. Numerous reports of RS have been published. Examples of them are presented here for illustration. A 28-year-old woman has been hospitalized for severe progressive weight loss with a lifelong history of idiopathic diarrhea, abdominal pain, nausea and vomiting. Her receiving weight of 23 kg was 40% of her ideal body weight, or the estimated body mass index (BMI) of 10 kg/m2. Initial laboratory tests included potassium 2.9 mek/L and phosphorus 2.7 mg/dL (reference range not given; Serum phosphorus levels can be recorded in mmol and mg; the normal serum phosphorus range is 2.5-4.5 mg/dL or 0.81-1.45 mmol/l.8 PN (dextrose 500 g, potassium 130 mek, phosphate 30 mmol, magnesium 16 mek, thiamine 135 mg and other vitamins) was initiated at night of intake. The patient reported chest pain and her phosphorus level was 1.1 mg/dL. After a few hours she developed hypotension, cardiac arrhythmia and metabolic acidosis. Requiring ventilator support, pulmonary infections, myocardial instability, and hypotension and died during the third week of hospitalization.9 a 66-year-old woman was admitted to 36 kg (70% ideal body weight) with abdominal pain, a 6-week history of poor oral intake and 3 months of profuse diarrhoea after canal surgery. She had diffuse muscle waste and anasarka, potassium was 3.4 mek/L, and phosphorus - 3.4 mg/dL (without reference range). Within 12 hours of taking PN (dextrose 750 g, potassium 20 mek, phosphate 15 mmol and multivitamin). After 48 hours, hours, became sluggish, hypotensive and tachycardia. Its phosphorus was 0.7 mg/dL, potassium 1.4 mek/L and magnesium 1.8 mg/dL. Soon after, it became apneic, requiring intubation, and PN was held. Her hospital course was complicated by bilateral pneumonia, acute respiratory distress syndrome, constant hypotension and, finally, death on hospital day 6.9 These cases illustrate the most extreme forms of MS, in which there is organ failure and death. It should also be noted that these patients were refed in this way, much more aggressive than our current practice, and both had low levels of potassium and/or phosphorus prior to the onset of calorie support. Low electrolyte levels, however, may not be present when RS ensues, so attention to other risk factors is probably important. Due to the heterogeneity of definitions, the many topics and the lack of high-quality controlled studies, numerous systematic reviews were deemed unfeasible. Thus, the authors of the task force were divided into thematic working groups. Each team conducted exhaustive reviews of the literature and held meetings by email and teleconference to review and reach consensus. The article was searched through PubMed using keywords relevant to the topic, such as feeding syndrome, overfeeding hypophosphatem and starvation. These sections were drawn up in the main document and reviewed by the entire committee. The consensus process included teleconferences, surveys of the entire PN security committee and contributions from several ASPEN and ASPEN board committees. ASPEN identifies the adult population over the age of 18, the pediatric population between the ages of 28 days and 18 years, and the neonatal population to be under 28 days old. In normal energy consumption, metabolic substrates will change daily, cycling through post-practical, post-corporate and lean conditions. With long periods of malnutrition, survival depends on the ability to use and maintain available energy reserves efficiently. As hunger becomes more deep, these energy reserves, as well as vitamins and intracellular electrolytes, are depleted. Electrolyte depletion is further exacerbated by conditions such as diarrhoea, loss of bowel contents (e.g. fistulas, vomiting, stomach drainage) or diuretic use, which cause additional losses. When glucose appears in the blood, insulin secretion increases in response.10-12 If there is a general deficiency of potassium in the body, phosphorus, or magnesium, a decrease in serum concentrations can occur due to an increase in insulin levels.13-15 Increase in insulin disk phosphorus and potassium intracellularly both on demand (i.e., glucose phosphorylation as glycolysis is initiated) and through direct insulin exposure (e.g. triphosphatase (ATPase). The mechanism of decoments in magnesium levels in this context has not been well clarified. These reductions can occur even if the serum level is inherently normal. The decline in serum electrolytes can be sudden and severe and can be fatal to a person who has been hungry or catabolic.3, 9, 15 Phosphorus is the main ion implicated in many published reports related to RS. As mentioned, the emphasis on phosphorus may be the result of a shifting definition, and potassium and magnesium can be just as important. Phosphate is a vital component of adenosine triphosphate (ATP), the main form of energy storage in the human body. As malnutrition progresses, the body will continually use existing phosphate reserves to continue the production of ATP. Depletion of phosphates can lead to respiratory muscle dysfunction, progressing to acute respiratory failure in severe cases.16 This can also lead to a decrease in cardiac contraction. Since phosphorus is important in conducting electrical impulses, low serum concentrations can also lead to cardiac arrhythmia.17, 18 Phosphorus depletion also reduces the production of 2.3-diphosphoglycerate, Causing an increase in oxygen hemoglobin affinity, reduced oxygen release in tissues, and tissue hypoxia.19 Whey concentrations of potassium decrease due to insulin stimulation naz/CK ATPase,16, 20 cell wall enzyme that is responsible for the flow of potassium into the cell and sodium out18 and is essential in transmitting nerve impulses and muscle contractions.21, 22 Hypocalemia can disrupt the transmission of electrical impulses, increasing the risk of potentially fatal cardiac arrhythmia.23, 24 Hypocalemia may also manifest as weakness, hyporeflexy, respiratory depression, and paralysis.25-27 Hypomagnesia has been identified as a feature of RS. As mentioned, neither the mechanism of its development in RS, nor its direct significance in the incidence of the syndrome have been clarified. Hypomagnesian worsens the absorption of potassium in nephrone, leading to excess losses, and can also lead to impaired cell transportation of potassium, all through exposure to magnesium-dependent enzymes such as na'K-ATPase.28 Thiamine deficiency can also manifest as a result of RS. Demand for thiamine increases significantly as it transitions from hunger to feeding, as it is a cofactor for glucose-dependent metabolic pathways.29, 30 Thiamine deficiency can lead to neurological abnormalities, including confusion, encephalopathy (Wernicke syndrome and Korsakov's psychosis), oculomotor anomalies (mostly horizontal ophthalmoplegia), hypothermia and even coma.31-33 Thiamin also plays a role in the conversion of lactate into pyruvat, and breast acidemia can occur in people with tyamine deficiency, without acute liver injury.34-36 Tiamine deficiency can also lead to a decrease in tiamine deficiency. in cardiac myocytes, which can lead to congestive heart failure, or wet beriberi. Insufficient production of ATP in cardiac tissue can lead to the release of adenosine into plasma. Adenosine causes peripheral vasodilation, increased cardiac ejection, reduced cardiac contraction, dysthymia and low diastolic blood pressure.37-39 Concerns about intravascular overload and congestive heart failure are sometimes reported in RS reviews. However, they are not based on directly recorded episodes and may be the result of a change in the terminology associated with heart failure. During the first descriptions of RS, heart failure was used to describe what is now called sudden death, or deadly arrhythmia. Heart failure (meaning sudden death) was part of the original RS descriptions. When the terminology shifted, congestive heart failure replaced sudden death in published RS definitions. This substitution included an explanation that sodium released into extracellular space as a result of na'/K'ATPase activation led to the osmotic shift of the liquid into extracellular space. However, this ignores the osmotic action of potassium exchanged for sodium, as it shifts to cells. Although the exchange of sodium and potassium is not equal, in favor of sodium, intravascular volume of overload was not reported. In addition, a large subcutaneous sodium storage system has recently been described.40 Any acute addition of sodium, except for patients with sodium (e.g., patients with already defunct congestive heart failure), is quickly cleaned and rendered neosmotic. It is not believed that intravascular fluid overload should be considered as a continuation of RS. Subacute or refeeding swelling was seen as a late manifestation, associated with RS in patients with hunger, mainly in patients with AN, but this is believed to be due to capillary leakage or inactivation of the natriuretic peptide from hyperinsulinemia, not because of the volume of overload.41 Screening strategies to identify patients at risk of RS are inaccurate and poorly tested, which is even worse due to the lack of consensus definition of RS. Typically, risk RS is identified subjectively by a clinician during enteral or PN assessments and onset.42, 43 criteria specifically designed to predict RS have been published. One example is the British National Institute of Health and Care Excellence (NICE).44 These recommendations were formulated on the basis of previously published reviews and the authors' experiences and agreed upon by informal consensus. Screening criteria developed for malnutrition have also been predictive values in the MS. One such example is the Short Nutrition Assessment Questionnaire (SNAS), which недоедания и тест на скрининг риска для RS.45 Значение этих инструментов скрининга для прогнозирования тяжелой гипофосфатемии является бедным. Их полезность в прогнозировании менее тяжелой гипофосфатемии или для прогнозирования гипокалемии или гипомагнезии неизвестна, и их полезность была под сомнение.46 И NICE и SNA' забил плохо для чувствительности или специфичности на ретроспективный анализ проверки. В 2011 году обзор 321 госпитализации, только около 25% из 92 пациентов, по мнению группы риска по критериям NICE разработали тяжелые гипофосфатемии (<0.6 mmol/l;= reference= range= 0.74–1.52= mmol/l)= during= refeeding= (sensitivity=50% and= specificity=76% for= pn,= and= sensitivity=38% and= specificity=73% for= nasogastric= (ng)= feeds).47= the= validity= of= both= nice= and= snaq= were= reported= in= 2016.= an= "at-risk"= snaq= score= had= a= positive= predictive= value= of= 13%;= however,= low= snaq= had= a= negative= predictive= value= of= 95%.48= other= criteria= sets= for= diagnosing= malnutrition,= such= as= that= proposed= by= aspen= and= the= academy= of= nutrition= and= dietetics49= and= the= newer= guideline= from= the= global= leadership= initiative= on= malnutrition= (glim),50= may= be= predictive= of= rs.= but= these= have= not= been= studied= for= their= predictive= value.= in= the= absence= of= a= universally= accepted= definition= for= rs ,= descriptions= of= incidence= are= fraught.= it= is= generally= agreed= that= hypophosphatemia= is= one= of= the= hallmarks= of= the= syndrome.= thus,= many= studies= in= which= the= authors= have= created= their= own= definitions= use= hypophosphatemia= as= the= sole= diagnostic= criteria.= in= a= 1996= study= in= which= rs= was= defined= as= hypophosphatemia= within= 72= hours= of= starting= nutrition,= and= hypophosphatemia= defined= as= serum= phosphorus= level= that= fell= by=>0,16 ммоль / л до <0.65 mmol/L, RS was present in 34% of critically ill patients.51 Using the same definition, a subsequent study reported an incidence of RS of 8% in their at-risk (by SNAQ) population.51 In a prospective cohort study using severely low electrolytes (potassium, magnesium, and phosphorus), fluid overload, and disturbance of organ function as diagnostic criteria , a rate of 2% out of 243 at-risk patients (by NICE criteria) was seen.52 Clearly, these studies are not comparable. Reporting of RS incidence in the pediatric population is even more sparse. A report by Dunn et al,53 in 2003, is one of the only such studies. In their cohort of 164 consecutive intensive care unit (ICU) patients started on PN, 15 were deemed at risk for RS using criteria developed at their institution. They report the incidence of "electrolyte shifts" within 72 hours of the initiation of nutrition support in the entire population to be 27% and 8 of 15 in the at-risk population, despite cautious feeding tactics. Of those developed hypophosphatemia, 3 developed cardiac abnormalities and lethargy.53 The neonatal time period is generally accepted to be the interval from birth to 4 weeks mmol/l,= rs= was= present= in= 34%= of= critically= ill= patients.51= using= the= same= definition,= a= subsequent= study= reported= an= incidence= of= rs= of= 8%= in= their= at-risk= (by= snaq)= population.51= in= a= prospective= cohort= study= using= severely= low= electrolytes= (potassium,= magnesium,= and= phosphorus),= fluid= overload,= and= disturbance= of= organ= function= as= diagnostic= criteria,= a= rate= of= 2%= out= of= 243= at-risk= patients= (by= nice= criteria)= was= seen.52= clearly,= these= studies= are= not= comparable.= reporting= of= rs= incidence= in= the= pediatric= population= is= even= more= sparse.= a= report= by= dunn= et= al,53= in= 2003 ,= is= one= of= the= only= such= studies.= in= their= cohort= of= 164= consecutive= intensive= care= unit= (icu)= patients= started= on= pn,= 15= were= deemed= at= risk= for= rs= using= criteria= developed= at= their= institution.= they= report= the= incidence= of= "electrolyte= shifts"= within= 72= hours= of= the= initiation= of= nutrition= support= in= the= entire= population= to= be= 27%= and= 8= of= 15= in= the= at-risk= population,= despite= cautious= feeding= tactics.= of= those= who= developed= hypophosphatemia,= 3= developed= cardiac= abnormalities= and= lethargy.53= the= neonatal= time= period= is= generally= accepted= to= be= the= interval= from= birth= to= 4= weeks=></0.65 mmol/L, RS was present in 34% of critically ill patients.51 Using the same definition, a subsequent study reported an incidence of RS of 8% in their at-risk (by SNAQ) population.51 In a prospective cohort study using severely low electrolytes (potassium , magnesium, and phosphorus), fluid overload, and disturbance of organ function as diagnostic criteria, a rate of 2% out of 243 at-risk patients (by NICE criteria) was seen.52 Clearly, these studies are not comparable. Reporting of RS incidence in the pediatric population is even more sparse. A report by Dunn et al,53 in 2003, is one of the only such studies. In their cohort of 164 consecutive intensive care unit (ICU) patients started on PN, 15 were deemed at risk for RS using criteria developed at their institution. They report the incidence of "electrolyte shifts" within 72 hours of the initiation of nutrition support in the entire population to be 27% and 8 of 15 in the at-risk population, despite cautious feeding tactics. Of those who developed hypophosphatemia, 3 developed cardiac abnormalities and lethargy.53 The neonatal time period is generally accepted to be the interval from birth to 4 weeks > </0.6> </0.6> Age. However, significant physiological differences exist between newborns before and after 2 weeks. Studies examining the frequency of electrolyte abnormalities in newborns have focused mainly on newborns who are small for gestational age (SGA; equivalent to less than 10 percentile on the standard growth curve), those with intrauterine growth retention (IUGR), those with extreme prematurity (24-27 weeks), and those with very low birth weight (VLB; 150). IUGR may be the result of chronic malnutrition in the womb or as a result of acute or late placental insufficiency. Insufficient pre-birth growth may be due to placental insufficiency or maternal concomitant diagnosis, such as pre-eclampsia.54 Several studies have reported electrolyte abnormalities in newborns. Two reported that rates of hypophosphate were significantly higher in patients who had SGA.55, 56 those with a high index of umbilical artery resistance (UA-RI; defined as a value above 95th percentile) were also at risk of developing early hypophosphate. The release of urine phosphorus and potassium was lower in these patients, suggesting that low levels were not caused by urinary tract loss.55 In subsequent observation, higher rates of electrolyte abnormalities were found, including hypophosphatemia, in patients with IUGR and VLBW.57 Others reported hypophosphate and hypocathemia in newborns receiving PN. These authors stressed the importance of careful monitoring and electrolyte abound.58, 59 The use of biomarkers for screening, risk assessment and clinical improvement monitoring can be helpful in clinical decision-making. Conversely, the regular use of low-sensitivity screening methods can lead to unnecessary interventions, such as the slow progress of feeding towards the goal of nutrition. In addition, regular use of non-specific screening has been shown to increase hospital stays and mortality.60-63 Literature is currently too spare to recommend regular use of biomarkers for clinical use for MS prediction. For the most part, biomarkers have been studied only to predict the risk of malnutrition and, as it is still, theorists to identify the risk for RS. Thus, the weak sensitivity of these markers (e.g. insulin-like growth factor 1 (IGF-1) and leptin) to malnutrition also makes them currently inappropriate as screening tools for RS. In hospital settings, where close attention to electrolyte levels is the standard of care, feeding complications may, in fact, be rare.64, 65 Until the studies use a unifying definition of MS, the incidence will be poorly studied and the characteristics of patients in which there is a very Task. However, the consistent risk characteristic derived from clinical experience and scientific observations includes prolonged malnutrition, especially in the context of continued continued Loss. The following are populations that have been found to be potentially at risk or unlikely to be at risk. It should be stressed once again that the incidence of MS in these populations is unknown. To illustrate, published case reports are presented. AN is associated with energy restriction leading to weight loss and malnutrition, and is one of the population groups most studied for the incidence of MS. These patients are isolated by hunger, mainly in the absence of other medical comorbidities that distinguishes them from other hospitalized patients. There have been several reports of incidence of various MS components in this population.1, 54, 66-69 Predictably, they are highly variable. For example, one series reported that the level of hypophosphateemia was 5.8% in their study group of 69 patients (average age 15.5 years). It is important to note that malnutrition correlates with the severity of hypophosphateemia.70 In another series, the authors report a 38% incidence of mild hypophosphateemia (2.5-3.4 mg/dL) and a lack of severe hypophosphateemia (1.0 mg/dL) during onset of nutrition in 46 patients (average age 15.7 years) Average body weight - 72.9% ideal.71 Patients with severe mental disorders may be at increased risk for RS due to poor nutrition as a result of self-requision, side effects of medication, avoidance of food due to hallucinations, avoiding eating with others due to social anxiety, Lack of everyday life skills (such as shopping and culinary skills), and homelessness with incompatible access to nutritious meals.72, a 73 25-year-old woman with schizophrenia was admitted to the medical ward with a BMI of 12.5 and a history of significant weight loss over the past year. She swallowed about 670 calories, consisting mainly of simple sugars and fats, in addition to her daily approved meals of 600 kcal/d. The day after this binge eating, she developed severe hypokalemia and severe hypophosphateemia, mental imbalance, swelling of the lower limbs, and ophthalmoplegia. After a few days of brilliance, it has improved enough to be referred to a psychiatric ward.74 Increased risk for RS in patients with alcohol disorder is thought to occur in a diet deficient in essential vitamins and minerals. There are no studies of the incidence of MS in patients with alcoholism. However, the authors recommend that the risk for RS be considered in alcoholic patients with evidence of global malnutrition. Similarly, patients who abuse methamphetamine, heroin and other mood-altering substances also have a higher risk of malnutrition and may therefore be at higher risk for MS. U (BMI 16 kg/m2) the 44-year-old homeless man had an initial level of phosphorus in the low normal range (0.84 (0.84 Reference range: 0.80-1.50 mmol/L). After 4 days of the standard diet, supplemented with IV saline solution, potassium, multivitamin oral and 100 mg of intramuscular thiamine, the level of phosphorus in the patient decreased to 0.15 mmol/L. Coinciding with this sharp drop, he complained of lower myalgia limbs and paresthesia and diarrhea. It was noted that it has a mood of lability and an extension of the CTC on an electrocardiogram (ECG). In total, he needed 42 mmola of phosphorus, given intravenously for 36 hours. His symptoms improved with continued nutrition and electrolyte abound, and he was eventually discharged to rehab.76 RS was described in obese patients who underwent bariatric surgical procedures.77, 78 As in other settings, the frequency and risk factors are unknown. One such case described a 48-year-old woman hospitalized at 117kg and had a BMI of 41.5 for an overdose of lithium and lingering diarrhea, vomiting and confusion. She had several bariatric surgeries in the 13 years preceding, including gastric bandbing followed by slippage and removal and biliopancreatic leakage and revision. She was lost to follow-up for 2 years prior to admission and her weight during this period is unknown, but her intake weight was higher than 98 kg in the past measured. Deficiencies in vitamins B1, B6, B12, D, K and zinc, selenium and iron, as well as severe hypoproteinemia were noted. Based on her physical findings, she was diagnosed with Wernicke encephalopathy. She first received PN, vitamin supplements, and high doses of thiamine supplements, and then was converted into a feeding tube providing 1200 kcal/d (10 kcal/kg). Over the next 10 days, she developed severe hypophosphateemia (0.9 mg/dL; a normal range of 2.5-4.5 mg/dL), as well as hypokalemia and hypomagnesia (levels not specified) and pulmonary edema leading to respiratory failure. It has improved since the aggressive electrolyte abounds and continues to support nutrition.79 This case highlights that RS can develop in conditions of elevated BMI. These patients may have chronic malnutrition and malabsorption. It is also believed that rapid changes in weight that occur initially after bariatric surgery may predispose to RS if a sudden increase in consumption (e.g. from nutritional support) is observed, especially in the presence of electrolyte loss (e.g. vomiting). Patients who have undergone bowel resections (e.g. for mesenteric ischemia) may also exhibit similar patterns of malnutrition and overfeeding difficulties that may predispose to RS.80 Adult and pediatric patients with malabsorptive syndromes, such as coeliac disease, may also be an increased risk for RS. Electrolyte and vitamin stocks can be quickly depleted in acute crisis. A 28-year-old woman with coeliac disease was hospitalized with severe dehydration, diarrhoea, malnutrition and shock, which was suspected of being an ineceent of a gluten-free diet. When her BMI was received, she had 14 kg/m2, and her labs were significant for kidney failure, metabolic acidosis and hypokalemia. The level of phosphorus and glucose was within the normal range. During the first 2 days, electrolyte disturbances and acid-base disorders were corrected. PN providing 450 kcal/d was started on the 3rd day. On the 5th day the patient developed psychomotor arousal, respiratory failure and cardiogenic shock with a 20% share release. The level of phosphorus and potassium was extremely low. Mechanical ventilation and inotropic agents were initiated; however, the patient died two days later with the failure of multiple'organ.81 Additional cases of RS in pediatric patients with coeliac disease were described.82, 83 Hunger related to protest or activism puts individuals at risk for RS. In 2007, for example, a 30-year-old man held a widely publicized voluntary protest in the form of a 44-day fast. During the protest, he lost 25% of his original body weight and drank only water. Overfeeding was done orally using a commercial 1.2 kcal/ml oral feeding supplement, along with 50 mg of thiamine twice a day and daily multivitamin. It received about 570 kcal per night on day 0 and on day 1, 1140 kcal on the second day, and 1710 kcal on the 3rd day, and then the channels stopped and the 1500 kcal light diet was started on the 4th day. On the evening of the 1st day its phosphorus dropped from the entry level of 1.0 to 0.46 mmol/L (reference range: 1.2-1.7 mmol/L), which prompted the introduction of 1 unit of phosphate infusion (phosphate 50 mmol, potassium 9.5 mmol and sodium 81 mmol per 500 ml) for 12 hours and oral phosphate (16 mmol) twice a day on days 2-4. Although he did not have major clinical sequels, he had several laboratory disorders, including elevated bilirubin and liver enzymes.84 Victims of child abuse and starvation are at risk of malnutrition and, by proxy, for RS during their recovery period. Hunger affects millions of children around the world, in developing and developed countries. Child starvation is often the result of neglect on the part of child caregivers when it is not due to economic factors or hunger.85 Malnutrition among recruits may be overlooked because otherwise it is a healthy population. The 26-year-old Marine recruit was in training for 10 weeks when he was introduced to the emergency room (ED). His superiors found him tired, hypothermia and confused during the march. After initial resuscitation and rewarming, his confusion resolved and he was reported to lose about 20 pounds (9.1 kg) in the 3 months prior to starting training because he had more weighting standards. He also reported another 35 pounds (15.9 kg) of weight loss during 10 weeks of training due to the strong Diet. Upon admission, he was found rhabdomyolysis and developed pneumonia. He was not considered in danger to RS since he was on a regular diet, and was discharged after 3 days. On the 4th day he began to complain of growing weakness and swelling, and was found to have critical hypophosphateemia. Despite IV brilliance, the level of phosphorus did not normalize for 3 days. In total, he received 9 doses of 12 mmol phosphate, 8 grams of magnesium and 200 mek potassium. His symptoms began to be resolved around the 9th day and he was discharged for a limited period.86 This emphasizes that a large amount of brilliance may be required to bring serum levels back to normal. Although it is very likely that this is rare among recruits, and very few of them are at risk of malnutrition, this case is highlighted to remind doctors to avoid missing the diagnosis of malnutrition and MS due to the patient's youth and general health. Athletes are covered for the same reasons as the military population. A 28-year-old male bodybuilder with no past medical or surgical history presented ED with a 2-day history of severe, progressive bilateral weakness of the lower leg and reduced arm strength. Laboratory values were significant for extremely low phosphorus, magnesium and potassium. He had just finished a fitness competition two days earlier and had lost 19kg (≈14% of his body weight) in the four-month pre-competition period. On competition days, the patient's diet consisted mainly of simple carbohydrates (such as chocolate bars) and then 800 grams of different carbohydrates for 5 days. He was admitted to intensive care for a total of 2 days for aggressive electrolyte abound and was discharged to hospital day 4.87 Although malnutrition is common in patients, Receiving hemodialysis (HD) for advanced kidney dysfunction88, 89 and associated with increased mortality,90, 91 RS is probably rare in patients dependent on HD, even in poor conditions, due to poor purification of phosphorus and potassium through HD. Hyperphosphatemia and high potassium levels are common. RS may be more likely to occur in patients receiving continuous venovenous heofiltration and peritoneal dialysis because the clearance of phosphorus and potassium is significantly greater than with intermittent HD, but the incidence is not known. Hypophosphateria may occur in patients with intermittent HEART if there is a significant deficiency of 1.25-hydroxy-vitamin D. A severely ill patient is often malnourished for long periods of time, and therefore it can be assumed to be at risk for RS when the calories are re-off. This applies to medical and surgical patients. da Silva described a seriously ill patient with a past history of alcohol and opioid and malnutrition disorders. She was hospitalized with a changed mental state after a suspected overdose. On to ED, she was in hypercapal respiratory failure and was intubated. Initial potassium was low at 2.4 mek/L (normal: 3.5-50 mek/L), and phosphorus concentration was normal (normal: 2.5-4.5 mg/dL). EN was started after several days of mechanical ventilation in the intensive care unit. Pre-feeding with electrolytes showed that potassium was slightly elevated to 5.5 mek/l and normal phosphorus 2.5 mg/dL. The next morning, the patient suffered a short cardiac arrest with an electrocardiogram showing a polymorphic, wide complex ventricular tachycardia, indicating a de-point torso. Repeated laboratories showed potassium 2.6 mek/L and phosphorus 2 mg/dL. Although en speed was reduced by 50% and its electrolytes were aggressively replaced, the patient did not survive hospitalization.92 Patients with malignancies may be at risk for RS due to prolonged starvation and/or electrolyte loss. Chemotherapy causes nausea, vomiting, anorexia, mucositis and diarrhea, all of which increase the loss of electrolytes. Radiation causes toxicity of the gastrointestinal tract (GI) and mucositis, as well as anorexia. Comorbidities characteristic of malignancies (such as bowel obstruction) can also contribute to global malnutrition.93 A patient receiving chemotherapy for oesophageal adenocarcinoma has been hospitalized with severe mucositis. The patient lost 18% of his body weight (BMI of 21.9 kg/m2) in the previous 3 months and ate minimally for 8 days before hospitalization. Electrolytes were originally normal. He became a septic tank on the 4th day and was transferred to the intensive care unit, where the PN was launched in 15 kcal/kg/d. After 2 days of PN, phosphorus became extremely low. PN was carried out and the concentration of serum phosphorus improved to some extent with parenteral replacement. PN was restarted 3 days later by 15 kcal/kg/d. After a week the serum electrolyte concentration decreased again and the patient gained 9 kg, presumably fluid. Electrolytes were normalized with repletion during ongoing nutrition, and the patient was discharged after 3 weeks.63 Additional RS case reports in this population were published.1, 94 ED often the first contact patients have with hospital care. RS and Wernicke encephalopathy may occur if patients with risk are not identified before starting calories. Patients may also submit ED with RS with a modified mental state if due to Wernicke or a serious metabolic disorder, and the story can be difficult to obtain. Caring for patients with undifferentiated altered mental state or electrolyte abnormalities should follow modern best practices. In patients who have a risk of thiamine deficiency, such as chronic alcohol users, or those with severe chronic hunger for any cause, thiamine supplementation should be considered Ambulance. Normally, however, the patient will only be in ED long enough for one single and abound in thiamine, or other vitamins and minerals, can take up to a few weeks. In patients who are cachet or who have concerns about significant malnutrition, caution in administration of glucose-containing fluids is justified, although it is probably unusual that significant RS will be treated after a short period of infusion of dextrocontinent fluids. Getting a thiamine level is also not a suitable test for ED management, but can be useful for later care. In most institutions, the test is conducted in an independent laboratory, the results are returned after a few days. Decisions on pre-emptive thiamine supplements should be made on the basis of clinical judgment of risk for Wernicke until better screening methods are available. There is low consensus and conflicting research to make decisions related to feeding rates to avoid RS. In addition, studies evaluating aggressive overfeeding rates were conducted in patients with AN and targeted adolescents with isolated hunger. Studies that explore conservative approaches target older patients, are much sharper from medically, and have multiple comorbidities and physiological stressors. In general, an individual approach to overfeeding patients is proposed. Regardless of the energy consumption route, there are several factors that need to be considered when initiating and promoting energy consumption in those who are at risk for RS. The key ones are physiological responses (e.g. changes in serum electrolyte and heart rate) and tolerance to initial feeding. It was pointed out that there was currently no universal recommendation on how to promote nutrition safely. Many of the available recommendations are general and vague, providing advice such as increasing slowly,43 in advance gradually,3 or provide a modest increase in energy95 and get goal needs in 3-7 days.96 For example, a review of the McCray article et al recommended promoting nutrition at 200-300 calories every 3-4 days. However, this recommendation stems exclusively from clinical experience.97 Others recommend supplementing electrolytes while increasing energy with the addition of phosphate 10-15 mmol for every 1,000 calories provided.95 Table 144, 97-100 outlines several proposed approaches for safe energy return for high-risk patients. Table 1. Published Recommendations for Initiating and Improving Nutrition for Patients At Risk for RS Initial Calories Feeding Promotion Other recommendations NICE44 A maximum of 10 kcal/kg/d 5 kcal/kg/d in extreme cases (examples, BMI qlt; 14 kg/m2 or low consumption over the course of) Slow to meet or exceed full needs for 4-7 days Restoring circulation volume IrSPEN98 Extreme risk: 5 kcal/kg/d High risk: 10 kcal/kg Moderate risk: 20 kcal/kg Slow start of feeding according to the risk category Check electrolyte electrolyte level Replacement to correct the deficiencies of the CNSG99 Extreme Risk Fluid Monitor Balance: consider providing only 5 kcal/kg/d High risk: start feeding support at a maximum of 10 kcal/kg body mass Moderate risk: enter a maximum of 50% of the requirement within the first 2 days Extreme or high risk: slow for 4-7 days, Both clinical and biochemical monitoring allows moderate risk: increase energy consumption only in clinical settings and electrolyte results allow energy and fluids to be introduced very gradually Check potassium, magnesium, phosphorus do not stop feeding if the electrolyte level drops when potassium falls in the serum, Magnesium levels, or phosphorus are significantly low, feeding should not be advanced further until the supplements occurred Cray96 ≈10 kcal/kg/d for severe cases of 15-20 kcal/kg for other calorie increase gently step-by-step manner at 200-300 kcal every 2-3 days Consider all sources of calories and liquids in your calculations (including dextrose) Check the base electrolytes (especially , potassium and magnesium) before the start of nutrition support, and replace any low levels quickly If hemodynamically unstable, keep sodium containing IV fluids up to ≈1 L/d initially in severely malnourished patients, such as like those with anorexia nervosa who may have a component of Friedley's cardiomyopathy 100 range from 5 to 25 kcal/kg/d depending on the severity of RS risk nutrition therapy should be started with reduced caloric goals and Slow increase to full caloric amount within 5-10 days according to the individual risk category for RS fluid overload should be prevented by limited fluid use and sodium limit diet for the first 7 days High-risk patients for RS should receive electrolytes lower than normal/low-normal range Prevention supplements Body mass index; CNSG, clinical steering group; IrSPEN, Irish Society of Clinical Nutrition and Metabolism; IV, intravenously; NICE, National Institute of Health and Care Services; RS, feeding syndrome. Not only is the literature inconclusive, but the resumption of nutrition at a low rate with slow progress may be at odds with the appropriate weight gain desired in high-risk populations, such as those with AN.101, 102 Conversely, several recent randomized trials in critically poorly maintained slow start-ups and promotion of nutrition support therapies.103-105 For the most part, studies of aggressive refeeding have focused on patients with AN. The retrospective comparison examined the effect of a lower versus relatively high-calorie diet in patients with AN.106 Three hundred ten patients aged 10 to 21 years with an average BMI of about 16 kg/m2 were included. Average food intake was about 900 kcal/d and weight loss was 1.6 kg/mo. Teh Teh The diet in the high-calorie group (222 patients) provided an average of 1,557 calories, while the lower-calorie group received an average of 1,163 calories. In the high-calorie group there is a tendency to more frequent hypophosphateemia, hypomagnesia and hypokalemia, but this difference is not statistically significant. Their findings showed that high-calorie diets upon admission were associated with a reduction in the duration of stay, without a statistically significant increase in hypokalemia, in another cohort of 361 patients (461 admissions) with an average BMI of 16.1 kg/m2, all patients initially received 1200-1500 calories/d and were aggressively advanced to 3,500-4000 calories within 10 to 13 days. In total, 7.9% of cases of hypophosphateemia at admission, and 18.5% developed it during treatment. When 54 patients were overfed, they developed mild hypophosphateemia (2.0 mg/dL), 16 developed mild hypophosphateemia (1-1.9 mg/dL), and none developed severe hypophosphateemia (lt;1.0 mg/dL). The average weight gain was 1.98 kg/kk, with 71.8% of patients achieving a BMI of 19 and 58.5% reaching BMI kg/m2 out of 20. They found that lower YM intake was more predictable with hypophosphateemia than the rate of weight gain. There were no deaths or serious morbidity. The study was limited in that not all patients had serum phosphorus levels, magnesium, and/or potassium drawn on admission, nor did all patients have these values queering consistently during their stay.107 Reasonable refeeding rates were also studied for the most in critically ill. In one of the only randomized control studies, Doig et al in 2015 studied RS in critically ill patients from 13 higher education hospitals across Australia. Their definition of RS was a new-sound hypophosphateemia developing 72 hours after the start of the diet. They measured hospital morbidity and mortality, as well as mortality in 60-day follow-up activities in 339 patients whose phosphorus levels decreased to 0.65 mmol/L within 72 hours of the start of nutrition support. The intervention team received energy limited to 20 kcal/h for at least 2 days, and if no re-consumption of phosphates was required during those 2 days, energy consumption returned to normal within 2-3 days. The return to normality was defined as 40 kcal/h within 24 hours, then increased the target to 60 kcal/h within 24 hours, followed by 80% of the calculated energy targets for another 24 hours, with 100% of the goals achieved by day 4. If the patient's phosphorus did drop to 0.71 mmol/L at any time during meals, the calories were limited to initiation (20 kcal/h) and the process restarted. Patients in the control group received about 69 kcal/h. mortality for 60 days, with no change in morbidity.108 These findings were by a follow-up study of 337 seriously ill patients intubated within 7 days. They identified RS, in the same way as in the previous trial, as the new onset of hypophosphateemia, 72 hours after the initiation of nutrition.the primary outcome was 6-month mortality and icu' length of stay. 50% of their goal's calories for the first three days, with an increase in q 25% of the calorie target per's day after.' MS was observed in 36.8% of patients, with no statistically significant difference in hospital morbidity, and with a tendency to shorten the length of stay, in a group with a lower calorie intake. They also found that low calorie intake was associated with an increase in overall survival per day of 180.109 This paper describes RS, conceptually, as a measurable decrease in levels 1 or any combination of phosphorus, potassium and/or magnesium, or manifestation of thiamine deficiency developing soon (hours up to a few days) after the onset of calorie status of a person who has been exposed to a significant period of malnutrition. RS can manifest itself in a wide variety of separations, from small, clinically minor reductions in electrolyte levels to severe and sudden contractions that lead to or risk development, ultimate organ failure if not pre-empted or corrected. Although many previous definitions, for historical reasons, focus solely on hypophosphateemia, it is suggested here that decrement in any of the 3 electrolytes can signal a complete body deficit and require monitoring or intervention. Specifically, the diagnostic criteria for RS are as follows: reducing any 1, 2 or 3 of serum phosphorus, potassium and/or magnesium by 10%-20% (soft RS), 20%-30% (moderate RS), or qgt;30% and/or organ dysfunction as a result of reducing any of them and/or because of a tiamine deficiency (severe RS). And it occurs within 5 days after a re-or significant increase in the power supply. Examples of signs and symptoms of organ abnormalities associated with RS can be found in Table 2.71, 110 Criteria for Stratification of Gravity are arbitrary, but are chosen to harmonize with published gravity stratification for electrolyte decrecions. Table 2. Signs and symptoms of heavy feeding syndrome. Hypophosphateemia Hypocalemia Hypomagnesia Thiamin Sodium Deficiency Preservation Neurological Paresthesia Weakness Delirium Disorientation encephalopathy Areflexic Paralysis Seizures Coma Tetany Heart-Hypotension Shock Reduction of Average Blood Pressure Increase in Pressure of The Pulmonary Reflexive Weakness Respiratory Failure Contraction changes respiratory insufficiency of the gastrointestinal tract Nausea Vomiting Constipation Other rhabdomyolysis muscle necrosis Weakness of tremor muscle twitch Altered mental state Tetany cramps Seizures of Coma heart arrhythmia gastrointestinal anorexia Nausea Nausea Vomiting Constipation Encephalopathy Milk Acidosis Nystagmus Neuropathy

Dementia Wernicke Syndrome Corsaka psychosis moist and dry beriberi fluid overload pulmonary eductoric pulmonary decompensation Kraft MD, Btaiche IF, Sachs GS. Review of overfeeding syndrome. Nartra Klin Practical. 2005;20(6):625-633. a In the pediatric population, the manifestations of the final intervention of organs are more likely to cause bradycardia, temperature anomalies and respiratory involvement. As indicated earlier, the incidence of MS is unknown. Thus, any quantitative risk assessment is not currently possible. However, some characteristics have been identified as likely predisposing to RS. Table 3 contains the characteristics recommended by consensus for inclusion in the adult RS risk assessment. Again, since the incidence is unknown, the list cannot be considered exhaustive, nor is the force of exposure to each or several characteristics known. The list includes several additions to previous NICE,44 criteria, such as the addition of medical exam results, including subcutaneous fat loss and muscle mass. Characteristics of weight loss, fat and muscle loss correspond to the characteristics of malnutrition for adult Academy/ASPEN children for adult patients with moderate to severe malnutrition.49 For adults, the risk is divided into moderate and severe. There is no definition of easy risk for adults. The Consensus Task Force believes that this could jeopardize the creation of a hypersensitive definition without any evidence of clinical risk or risk of over-intervention. It is also unlikely, by definition, that mild risk will be clinically significant or will require a change in management. Many of the disease processes that increase the risk of malnutrition are included in the updated risk criteria. Table 415, 111-113 includes some conditions typical of the adult population; however, most of them relate to adults and children. Abnormal electrolyte values are expressed as a percentage below the lower limit of the norm, as different medical laboratories may have different values for the normal range. Table 3. ASPEN Consensus Criteria for identifying adult patients at risk for refeeding syndrome. Moderate Risk: 2 Risk Criteria Require Significant Risk: 1 Risk Criteria Required BY a BMI of 16-18.5 kg/m2 q weight loss q 5% in 1 month q 7.5% in 3 months gt;kg/m2 10% in 6 months of calorie intake No or a slight oral intake for 5-6 days or 75% of the estimated energy requirement for 7 days during acute illness or 75% of energy= requirement= for=>травмы ИЛИ 1 месяц Нет или незначительное устное потребление в течение >7 дней или или <50% of estimated energy of= estimated= energy=></50% of estimated energy > </75%> </75%> </16> </16> within 5 days during an acute illness or injury OR 50% of the estimated energy requirement for 1 month Abnormal pre-feeding of potassium, phosphorus, or magnesium serum concentrations Are Minimally Low Levels or Normal Current Levels and Recent Low Levels requiring minimal or single-dose supplementation Moderate/significantly low levels or minimally low or normal levels and recent low levels requiring significant or multiple doses of subcutaneous fat loss Evidence of moderate loss of evidence of severe or moderate loss of evidence of severe loss of severe loss of weight to the American Society of Parenteral and Enteral Nutrition; BMI, body mass index. Please note that electrolytes may be normal despite the total deficiency of the body, which is thought to increase the risk of re-feeding the syndrome. Table 4. Diseases and clinical conditions associated with an increased risk of feeding syndrome. Acquired Immunodeficiency Syndrome Chronic Alcohol or Disorder of Drug Disphagia and Oesophageal Dysmotic (e.g. Eosinophilic oesophagus, ahalasia, stomach dysmotion) Eating disorders (e.g. anorexia nervosa) Food safety and homelessness Failure to thrive, including physical and sexual abuse and neglect victims (especially children) Hyperemesis gravidarum or prolonged vomiting Basic stressors or operations without food for long periods of time Malabsorptive conditions (e.g. short bowel syndrome, pyloric Pancreatic Insufficiency) Cancer Advanced Neurological Disorders or General Inability to Communicate Needs of Post-Bariatric Surgery Postoperative Surgery Patients With Complications Of Prolonged Starvation (e.g. Hunger Strikeman, Anorexia Nervosa) Refugees Squirrel Malnutrition Table 5114-116 lists the criteria recommended, consensus, for inclusion in the RS risk assessment in the children's population. As with adults, the list cannot be considered exhaustive, nor is it aware of the impact of each or several characteristics. There are several important differences between adult and child populations. The risk of MS is generally thought to be closely related to malnutrition, especially hunger-related malnutrition. However, adults are thought to be more tolerant of longer periods of hunger. Short periods of nutrient deprivation may have a more significant effect in children due to additional metabolic growth needs. For this reason, pediatric criteria include a level of soft risk. When assessing the risk of developing MS should take into account the rate of weight gain, current height and length, or BMI for the age of z'score. Table 5. ASPEN Consensus Criteria for педиатрических пациентов, </50%> </50%> Синдром. Мягкий риск: 3 категории риска, необходимые для умеренного риска: 2 критерия риска, необходимые для значительного риска: 1 критерий риска, необходимый для оценки длины (1-24 месяца) или ИМТ-возраст z'score (2-20 лет) No1 до 1,9 z'score, что является изменением от базового No 2 до 2,9 z'score, что является изменением от базового по 3 z'score или больше, что является изменением от базовой потери веса <75% of= norm= for= expected= weight= gain=></75%> <50% of= norm= for= expected= weight= gain=></50%> <25% of= norm= for= expected= weight= gain= energy= intake= 3–5= consecutive= days= of= protein= or= energy= intake=></25%> <75% of= estimated= need= 5–7= consecutive= days= of= protein= or= energy= intake=></75%> <75% of= estimated= need=>7 дней подряд белка или потребления энергии <75% of estimated need Abnormal prefeeding serum potassium , phosphorus, or magnesium concentrationsb Mildly abnormal or decreased to 25% below lower limit of normal Moderately/significantly abnormal or down to 25%–50% below lower limit of normal Moderately/significantly abnormal or down to 25%–50% below lower limit of normal Higher-risk comorbidities (see Table 4) Mild disease Moderate disease Severe disease Loss of subcutaneous fat Evidence of mild loss OR Mid-upper arm circumference z-score of −1 to −1.9 z-score Evidence of moderate loss OR Mid-upper arm circumference z-score of −2 to −2.9 Evidence of severe loss OR Mid-upper arm circumference z-score of −3 or greater Loss of muscle mass Evidence of mild or moderate loss OR Mid-upper arm circumference z-score of −2 to −2.9 Evidence of severe loss OR Mid-upper arm circumference z-score of −3 or greater ASPEN , American Society for Parenteral and Enteral Nutrition; BMI, body mass index. a Not intended for use in patients at ≤28 days of life or ≤44 weeks' corrected gestational age. b Please note that electrolytes may be normal despite total-body deficiency, which is believed to increase risk of refeeding syndrome. The approaches to avoid causing RS and those for responding to and avoiding worsening of RS are often the same and are combined in these consensus recommendations. These recommendations (Table 6 for adults and Table 7 for children) may not apply to special populations, such as those with renal impairment; are meant as general guidelines; have not been tested in randomized studies; and should be adapted to the individual patient and/or institution. Table 6. ASPEN Consensus Recommendations for Avoidance and Treatment of RS in At-Risk Adults Aspect of Care Recommendations Initiation of calories Initiate with 100–150 g of dextrose or 10–20 kcal/kg for the first 24 hours; advance by 33% of goal every 1 to 2 days. This includes enteral as well as parenteral glucose. In patients with moderate to high risk of RS with low electrolyte levels, holding the or increase of calories until electrolytes are supplemented and/or of= estimated= need= abnormal= prefeeding= serum= potassium,= phosphorus,= or= magnesium= concentrationsb= mildly= abnormal= or= decreased= to= 25%= below= lower= limit= of= normal= moderately/significantly= abnormal= or= down= to= 25%–50%= below= lower= limit= of= normal= moderately/significantly= abnormal= or= down= to= 25%–50%= below= lower= limit= of= normal= higher-risk= comorbidities= (see= table 4)= mild= disease= moderate= disease= severe= disease= loss= of= subcutaneous= fat= evidence= of= mild= loss= or= mid-upper= arm= circumference= z-score= of= −1= to= −1.9= z-score= evidence= of= moderate= loss= or= mid-upper= arm= circumference= z-score= of= −2= to= −2.9= evidence= of= severe= loss= or= mid-upper= arm= circumference= z-score= of= −3= or= greater= loss= of= muscle= mass= evidence= of= mild= or= moderate= loss= or= mid-upper= arm= circumference= z-score= of= −2= to= −2.9= evidence= of= severe= loss= or= mid-upper= arm= circumference= z-score= of= −3= or= greater= aspen ,= american= society= for= parenteral= and= enteral= nutrition;= bmi,= body= mass= index.= a= not= intended= for= use= in= patients= at= ≤28= days= of= life= or= ≤44= weeks'= corrected= gestational= age.= b= please= note= that= electrolytes= may= be= normal= despite= total-body= deficiency,= which= is= believed= to= increase= risk= of= refeeding= syndrome.= the= approaches= to= avoid= causing= rs= and= those= for= responding= to= and= avoiding= worsening= of= rs= are= often= the= same= and= are= combined= in= these= consensus= recommendations.= these= recommendations= (table 6= for= adults= and= table 7= for= children)= may= not= apply= to= special= populations,= such= as= those= with= renal= impairment;= are= meant= as= general= guidelines;= have= not= been= tested= in= randomized= studies;= and= should= be= adapted= to= the= individual= patient= and/or= institution.= table= 6.= aspen= consensus= recommendations= for= avoidance= and= treatment= of= rs= in= at-risk= adults= aspect= of= care= recommendations= initiation= of= calories= initiate= with= 100–150= g= of= dextrose= or= 10–20= kcal/kg= for= the= first= 24= hours;= advance= by= 33%= of= goal= every= 1= to= 2= days.= this= includes= enteral= as= well= as= parenteral= glucose.= in= patients= with= moderate= to= high= risk= of= rs= with= low= electrolyte= levels,= holding= the= initiation= or= increase= of= calories= until= electrolytes= are= supplemented= and/or=></75% of estimated need Abnormal prefeeding serum potassium, phosphorus, or magnesium concentrationsb Mildly abnormal or decreased to 25% below lower limit of normal Moderately/significantly abnormal or down to 25%–50% below lower limit of normal Moderately/significantly abnormal or down to 25%–50% below lower limit of normal Higher-risk comorbidities (see Table 4) Mild disease disease Severe disease Loss of subcutaneous fat Evidence of mild loss OR Mid-upper arm circumference z-score of −1 to −1.9 z-score Evidence of moderate loss OR Mid-upper arm circumference z-score of −2 to −2.9 Evidence of severe loss OR Mid-upper arm circumference z-score of −3 or greater Loss of muscle mass Evidence of mild or moderate loss OR Mid-upper arm circumference z-score of −2 to −2.9 Evidence of severe loss OR Mid-upper arm circumference z-score of −3 or greater ASPEN, American Society for Parenteral and Enteral Nutrition; BMI, body mass index. a Not intended for use in patients at ≤28 days of life or ≤44 weeks' corrected gestational age. b Please note that electrolytes may be normal despite total-body deficiency, which is believed to increase risk of refeeding syndrome. The approaches to avoid causing RS and those for responding to and avoiding worsening of RS are often the same and are combined in these consensus recommendations. These recommendations (Table 6 for adults and Table 7 for children) may not apply to special populations, such as those with renal impairment; are meant as general guidelines; have not been tested in randomized studies; and should be adapted to the individual patient and/or institution. Table 6. ASPEN Consensus Recommendations for Avoidance and Treatment of RS in At-Risk Adults Aspect of Care Recommendations Initiation of calories Initiate with 100–150 g of dextrose or 10–20 kcal/kg for the first 24 hours; advance by 33% of goal every 1 to 2 days. This includes enteral as well as parenteral glucose. In patients with moderate to high risk of RS with low electrolyte levels, holding the initiation or increase of calories until electrolytes are supplemented and/or > </75%> </75%> must be considered. Initiating or increasing calories should be deposited in patients with highly low levels of phosphorus, potassium or magnesium until they are corrected. Calories from IV dextrose solutions and medications poured into dextrose should be treated within the above and/or initiated with caution in patients with moderate to severe risk for RS. If a patient received a significant amount of dextrose within a few days, from servicing IV fluids and/or medications to dextrose, and was imptomatic with stable electrolytes, calories from the food may be re-in larger amounts than recommended above. Restriction of Liquid Restriction sodium Restriction Protein Electrolytes Checking serum potassium, magnesium and phosphorus before starting nutrition. Monitoring every 12 hours for the first 3 days in high-risk patients. May be more frequent based on the clinical picture. It is replete with low electrolytes based on established standards of care. It is not possible to recommend whether to give a preventive pre-death electrolyte if the pre-feeding level is normal. If electrolytes become difficult to fix or drop rapidly during the start of a diet, reduce calories/gram dextrose by 50% and advance dextrose/calorie by about 33% of the goal every 1-2 days based on a clinical presentation. Recommendations can be changed based on a doctor's decision and clinical presentation, and discontinuation of nutritional support can be considered when electrolyte levels are strong and/or life-threatening low or dropping rapidly. Thiamin and multivitamin supplement thiamin 100 mg before feeding or before onset of dextrose containing IV fluid in patients at risk. Supplement thiamine 100 mg/d for 5-7 days or longer in patients with severe hunger, chronic alcoholism or other high risk of deficiency and/or signs of thiamine deficiency. Normal levels of thiamine are unlikely to have value. MVI is added to PN daily, if contraindicated, as long as PN continues. For patients receiving oral/enteral nutrition, add full oral/enteral wateritis once a day for 10 days or more, depending on the clinical status and method of therapy. Monitoring and long-term care recommend vital signs every 4 hours for the first 24 hours after starting calories in patients at risk. Cardiorespiratory monitoring is recommended for unstable patients or those with severe disabilities, based on established standards of care. Daily weights with controlled consumption and output. Evaluate short- and long-term nutritional care goals daily for the first few days until the patient is found to be stable (e.g., there is no need for electrolyte supplementation for 2 and then based on institutional standards of care. ASPEN, American Society of Parenteral and Enteral Nutrition; IV, intravenously; MVI, multivitamin injectable; Injectable; parenteral nutrition; RS, feeding syndrome. Table 7. ASPEN Consensus Recommendations for Avoiding and Treating RS in At'Risk Pediatric Patients Aspect Care Recommendations Dedication To Initiate Nutrition to a maximum of 40%-50% goal, But usually starting a glucose infusion level of about 4-6 mg/kg/min and advancing to 1-2 mg/kg/min daily as blood glucose levels allow until you reach a maximum of 14-18 mg/kg/min. This includes enteral as well as parenteral glucose. Calories from IV dextrose solutions and medications poured into dextrose should be treated within the above and/or initiated with caution in patients with moderate to severe risk for RS. If a patient already receives IV dextrose within a few days and/or medication in dextrose and has been ampthetic with stable electrolytes, calories from the food may be reintroduced in larger amounts than recommended above. Restriction of Liquid Restriction sodium Restriction Protein Electrolytes Checking serum potassium, magnesium and phosphorus before starting nutrition. Monitoring every 12 hours for the first 3 days in high-risk patients. May be more frequent based on the clinical picture. It is replete with low electrolytes based on established standards of care. It is not possible to recommend whether to give a preventive pre-death electrolyte if the pre-feeding level is normal. If electrolytes become difficult to fix or drop rapidly during the start of a diet, reduce calories/gram dextrose by 50% and advance dextrose/calorie by about 33% of the goal every 1-2 days based on a clinical presentation. Recommendations can be changed based on a doctor's decision and clinical presentation, and discontinuation of nutritional support can be considered when electrolyte levels are strong and/or life-threatening low or dropping rapidly. Thiamin and multivitamin thiamin 2 mg/kg up to a maximum of 100-200 mg/d before feeding begins or before the onset of IV fluid containing dextrose in high-risk patients. Continue thiamine supplementation for 5-7 days or longer in patients with severe hunger, chronic alcoholism, or other high risk of deficiency and/or signs of thiamine deficiency. Normal levels of thiamine are unlikely to have value. MVI is added to PN daily, if contraindicated, as long as PN continues. For patients receiving oral/enteral nutrition, add full oral/enteral wateritis once a day for 10 days or more, depending on the clinical status and method of therapy. Once the patient is within adult weight categories, refer to adult multivitamin recommendations. Monitoring and long-term care recommend vital signs every 4 hours during the first 24 hours after start in those at risk. monitoring is recommended for unstable patients or those with severe disabilities, based on established standards of care. Daily Daily controlled consumption and output. Assess energy needs as needed for oral feeding patients. Evaluate short- and long-term nutritional care goals daily for the first few days until the patient is found to be stable (e.g., there is no need for electrolyte supplementation for 2 days) and then based on institutional standards of care. ASPEN, American Society of Parenteral and Enteral Nutrition; IV, intravenously; MVI, multivitamin injectable; PN, parenteral nutrition; RS, feeding syndrome. Simply put, patients considered at risk for RS, other than young patients with AN, must first get conservative calories. They should be more closely monitored for electrolyte anomalies and treated for electrolyte abnormalities in accordance with established standards of care. Treatment of established RS should be aimed at correcting major electrolyte abnormalities to prevent sequelae and may additionally include either reducing calories or slowing the progress of calories to possible goals. Treatment should include both reactive and pre-emptive supplements, depending on the severity of RS, or the severity of the risk to RS. Patients with low electrolyte levels should undergo more aggressive supplements before feeding than would be normal in a stable condition. Considering the severity or speed of electrolyte decrement and risk to RS can determine whether to normalize electrolytes before starting any calories or increase calories. Specific recommendations for newborns are not included in this document. In general, SGA, IUGR due to maternal comorbidities, elevated high UASRI, extreme prematurity, VLBW, and z'score qgt; No. 2 are examples of characteristics that are thought to put newborns at risk for RS. This is not an exhaustive list. Further research is needed in all areas related to MS, from verification and more accurate identification of risk factors and definitions of MS and their severity to the standardization of treatment protocols. This paper presents a single set of criteria, such as that the studies are made in a single and the frequency of sequelae can be determined. Although guidance has been provided, these criteria are based on consensus and should be tested in randomized trials in general, in specific populations and with different accompanying conditions to determine their usefulness. For example, it is likely that the risk of MS is very different between patients with AN and those in intensive care; Adults, adolescents, children and newborns; and between a hospitalized patient in a wealthy city and a victim of hunger or hunger linked to poverty. Research is needed to compare initiation regimes and protocols for their effectiveness to avoid RS and/or sequelae Research is also needed to study the use of preventative electrolyte electrolyte before feeding patients is considered a high risk for RS, but with normal levels of pre-feeding electrolyte. This paper provides a descriptive review and consensus recommendations on the risks, avoidance and treatment of MS. It also provides a unified consensus definition, updated consensus recommendations for screening and identification of patients at risk of MS, and recommendations for avoiding and treating MS. The authors would like to thank Patricia Becker, MS, RDN, CSP, CNSC for very useful insight and guidance from the pediatric and neonatal sections and Michael Kraft, PharmD, BCNSP and Todd Mattox, PharmD, BCNSP for the content experience. 1Stanga, Brunner A, Leuenberger M. et al. Nutrition in Clinical Practice - Overfeeding Syndrome: Illustrative Cases and Guidelines for Prevention and Treatment. Eur J Wedge Natra. 2008; 62(6): 687- 694. 2Scapper A. Feeding syndrome or overfed hypophosphateemia: a systematic review of cases. Nartra Klin Practical. 2012; 27(1): 34- 40. 3Friedly N, Stanga S, Sobotka L, et al. Re-feeding Syndrome: Results of a Systematic Review. Nutrition 2017; 35: 151- 160. 4Burger GCE DJ, Sandstead HR. Malnutrition and famine in the Western Netherlands September 1944-July 1945. Part I. Part II: Apps. Jama. 1950; 142(11): 857- 858. 5Brozek J CC, Keys A. DRastic Food restriction: effect on cardiovascular dynamics in normative and hypertensive conditions. Jama. 1948; 137(18): 1569- 1574. 6 Schnitker M.A., Mattman PE, Bliss TL. Clinical study of malnutrition in Japanese prisoners of war. Anne Intern Honey. 1951; 35(1): 69- 96. 7Keys A, Broszek J, Henschel A, et al. Biology of Human Hunger. Volume 1-2. Minneapolis, MN: University of Minnesota Press; 1950. 8Allaparthi S BD, Canada N, et al. A.S.P.E.N. Fluids, Electrolytes and Acid-Basic Disorders Handbook. Silver Spring, MD: American Society of Parenteral and Enteral Nutrition; 2015. 9Weinsier RL, Krumdieck CL. Death as a result of excessive general parenteral nutrition: overfeeding syndrome again. Am J Wedge Natra. 1981; 34(3): 393- 399. 10Anderson E, Long JA. The effect of hyperglycemia on insulin secretion is determined by the rat's isolated pancreas in perfusion apparatuses. Endocrinology. 1947; 40(2): 92- 97. 11Grodsky GM, Butts AA, Bennett LL, Vcella C, McWilliams NB, Smith DF. The effect of carbohydrates on insulin secretion from isolated pancreatic rats. Am J Physio. 1963; 205: 638- 644. 12Porte D, Jr., Pupo AA Insulin Answers to Glucose: Evidence for Two Pool Systems in Man. J Clin Invest. 1969; 48(12): 2309- 2319. 13Zerler KL. The effect of insulin on potassium efflux from the rat muscle in the presence and absence of glucose. Am J Physio. 1960; 198: 1066- 1070. 14 Cirler KL, Rogus E, Hazlewood CF. Effect of insulin on the flow of potassium and water and electrolyte muscle content from normal and from крыс. J Gen Физиол. Физиол. 49(3): 433- 456. 15Boateng AA, Sriram K, Meguid MM, Crook M. Refeeding Syndrome: Treatment considerations based on a collective analysis of the literature reports case. Nutrition 2010; 26(2): 156- 167. 16 Influential K. Functional roles of Subdivisions Na,K-ATPase. Curr Opin Nephrol Hypertens. 2008; 17(5): 526- 532. 17Knochel JP. Pathophysiology and clinical characteristics of severe hypophosphateemia. Arch intern Med. 1977; 137(2): 203- 220. 18Camp MA, Allon M. Severe hypophosphateemia in hospitalized patients. Miner Electrolyte Metab. 1990; 16(6): 365- 368. 19Sharma S, Brugnara C, Betensky RA, Waikar SS. Decrease in the concentration of red blood cells 2.3-dipfosfoglycerates during continuous renal therapy. Wedge J Am Soc Nephrol. 2015; 10(1): 74- 79. 20Copek W, Lubet B, Poulsen H, Handelia H. Molecular Mechanism Na (I), K (I) - ATPase malfunctions in mutations characteristic of adrenal hypertension. Biochemistry 2014; 53(4): 746- 754. 21Sk for JC. The effect of some cats on adenosine triphosphates from peripheral nerves. Biochem Biofia Act. 1957; 23(2): 394- 401. 22 Brewers AS, Calachorro F, Walker RJ. Na (I)/K (C) - pumping and neurotransmitter membrane receptors. Invert neroshy. 2018; 19(1): 1. 23Ettinger PO, Regan TJ, Oldewurtel HA. Hyperkalemia, cardiac conduction and electrocardiogram: review. Am Heart J. 1974; 88(3): 360- 371. 24Dittrich KL, RM Walls. Hyperkalemia: ECG manifestations and clinical considerations. J Emerg Med. 1986; 4(6): 449- 455. 25Yu ASL, Chertow GM, Luyckx V. Potassium Balance Disorders. Brenner and the rector of the Kidney. 11th o. Philadelphia: Elsvier; 2020. e-book. 26Siegel D, Hulley SB, Black DM, et al. Diuretics, serum and intracellular electrolyte levels, as well as ventricular arrhythmias in hypertension. Jama. 1992; 267(8): 1083- 1089. 27Alpern RJ, Mo OW, Michael K. Seldin and Giebisch in Kidney: Physiology and Pathophysiology. 5th o.p. New York: Lippincott Williams and Wilkins; 2013. e-book. 28Huang CL, Kuo E. Mechanism of hypokalemia in magnesium deficiency. J Am Soc Nephrol. 2007; 18(10): 2649- 2652. 29Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature. 2001; 414(6865): 813- 820. 30Schenk G, Duggleby RG, Nixon PF. Properties and functions of thiamine diphosphate of the dependent enzyme transketolase. Int J Biochem Cell Biol. 1998; 30(12): 1297- 1318. 31Hazell AS, Todd KG, Butterworth RF. The mechanisms of death of neuronal cells in Wernicke encephalopathy. Metab Brain Dis. 1998; 13(2): 97- 122. 32Gubler CJ. Research into the physiological functions of thiamine. J. Effect of thiamine deficiency and thiamine antagonists on the oxidation of alpha-keto acids by rat tissues. J Biol Chem. 1961; 236: 3112- 3120. 33Tomson AD Mechanisms of vitamin deficiency in chronic alcohol abuse and the development of Wernicke-Korsakov syndrome. Alcohol Suppl. 2000; 35(1): 2- 7. 34 Centers for Disease Control of S. Lactic acidosis is associated with thiamine deficiency associated with a nationwide shortage of multivitamin for general parenteral nutrition - United States, 1997. MMWR Morb Mortal Wkly Rep. 1997; 46(23): 523- 528. 35Hazell AS, Butterworth RF. Update the mechanisms of cell damage in thiamine deficiency: focus on oxidative stress, excitability and inflammation. Alcohol alcohol. 2009; 44(2): 141- 147. 36Donnino MW, Carney E, Cocchi MN, et al. Thiamine deficiency in seriously ill patients with sepsis. J Crete Care. 2010; 25(4): 576- 581. 37Coelho LS, Hueb JC, Minicucci MF, Azevedo PS, Paiva SA, zornov L.A. Thiamine deficiency as the cause of reversible pulmonary cortex. Arc Bras Cardiol. 2008; 91(1): e7' 9. 38Yamasaki H, Tada H, Kawano S, Aonuma K. Reversive pulmonary hypertension, milk acidosis, and fast-developing multiple organ failure as manifestations of beriberi shoshina. Circ J. 2010; 74(9): 1983- 1985. 39DiNicolantonio JJ, Niazi AK, Lavie CJ, O'Keefe JH, Ventura HO. Thiamin supplements to treat heart failure: literature review. The stagnation of the heart fail. 2013; 19(4): 214- 222. 40Olde Engberink RH, Rorije NM, van den Born BH, Vogt L. Quantitative assessment of the neosmotic storage capacity of sodium after acute hypertensive saline infusion in healthy people. Kidneys Int. 2017; 91(3): 738- 745. 41Guirao X, Franch G, Gil MJ, Garcia-Domingo MI, Girvent M, Sitges-Serra A. Extracellular volume, nutritional status, and overfeeding changes. Nutrition 1994; 10(6): 558- 561. 42GariballA S. Feeding syndrome: a potentially fatal disease, but remains underdiagnosed and untreated. Nutrition 2008; 24(6): 604- 606. 43Kiraly LN, McClave SA, Neil D, Evans DC, Martindale RG, Hurt RT. Physician nutrition education. Nartra Klin Practical. 2014; 29(3): 332- 337. 44 National Emergency Cooperating Center. Adult nutrition support: oral nutrition support, enteral tube feeding and parenteral nutrition. . Access to November 23, 2019. 45Kruizenga HM, Seidell JC, de Vet HC, Wierdsma NJ, van Bochorst de van der Schueren MA Develop and test hospital malnutrition screening tool: a short nutrition assessment questionnaire (SNAS). Wedge Natra. 2005; 24(1): 75- 82. 46De Silva A, Smith T, Stroud M. Attitude to NICE Guide to Feeding Syndrome. BMJ. 2008; 337: a680. 47Skeki S, Culkin A, Gabe SM, Nightingale JM. Overfeeding of hypophosphateemia is more common in enteral than parenteral feeding in adult patients. Wedge Natra. 2011; 30(3): 365- 368. 48Kraaijenbrink BV, Lambers WM, Matus-Vligen EM, Siegert CE. Incidence of overfeeding syndrome in patients with internal medicine. Neth J Med. 2016; 74(3): 116- 121. 49White JV, Gunther, Jensen G, Malone Schofield Schofield Consensus Statement: Academy of Nutrition and Dietetics and the American Society of Parenteral and Enteral Nutrition: Characteristics Recommended for The Identification and Documentation of Adult Malnutrition (Malnourishment). JPEN J Parenter Enteral Natra. 2012; 36(3): 275- 283. 50Cederholm T, Jensen GL, Correia M et al. GLIM Criteria for Diagnosis of Malnutrition is a consensus report of the global clinical nutrition community. Wedge Natra. 2018; 38(1): 1- 9. 51Marik PE, Bedigian MK. Treatment of hypophosphateemia in seriously ill patients in the intensive care unit. Promising research. Surg Arch. 1996; 131(10): 1043- 1047. 52Rio A, Whelan K, Goff L, Reidlinger DP, Smeeton N. The emergence of overfed syndrome in adults began on the support of artificial nutrition: a prospective cohort study. BMJ Open. 2013; 3(1). 53Dunn RL, Stettler N, Mascarenhas MR. Feeding syndrome in hospitalized pediatric patients. Nartra Klin Practical. 2003; 18(4): 327- 332. 54Soyama H, Miyamoto M, Natsuyama T, Takano M, Sasha H, Furuya K. Case of re-feeding syndrome during pregnancy with anorexia nervosa. Obstet Med. 2018; 11(2): 95- 97. 55Ichi Gkawa, Watabe Y, Suzumura H, Sairenchi T, Muto T, Arisaka O. Hypophosphatemia in low for gestational age extremely low birth weight of infants receiving parenter nutrition in the first week after birth. J Pediatr Endocrinol Metab. 2012; 25(3-4): 317- 321. 56Boubred F, Herlenius E, Bartocci M, Jonsson B, Vanpee M. Extremely premature babies who are small for gestational age have a high risk of early hypophosphateemia and hypokalemia. Acta Padiatre. 2015; 104(11): 1077- 1083. 57Igarashi A, Okuno T, Ohta G, Tokuriki S, Ohshima Y. Risk factors for feeding syndrome like hypophosphateemia in very low birth weight children. Dees Markers. 2017; 2017:9748031. 58Bonsante F, Jacobelli S, Latorre G, etc. Initial amino acid intake affects phosphorus and calcium homeostasis in preterm infants - it's time to change the composition of early parenteral nutrition. PLoS one. 2013; 8 (8):e72880. 59Mizumoto H, Mikami M, Oda H, Huta D. Refeeding Syndrome in Small dates micro-premium receiving early parenteral nutrition. Pediatr Int. 2012; 54(5): 715- 717. 60Tsai JR, Chang WT, Sheu CC, et al. Inadequate energy delivery during early critical diseases correlates with increased mortality risk in patients who survive at least seven days: a retrospective study. Wedge Natra. 2011; 30(2): 209- 214. 61Weijs PJ, Stapel SN, de Groot SD, etc. Optimal protein and energy nutrition reduces mortality in mechanically ventilated, seriously ill patients: a promising observational cohort study. JPEN J Parenter Enteral Natra. 2012; 36(1): 60- 68. 62Elke G, Wang M, Weiler N, Day AG, Heyland DK. Close to the recommended calorie intake and enteral nutrition is associated with the best clinical outcome of seriously ill patients Patients: Secondary analysis of a large international nutrition database. Crete Care. 2014; 18(1): R29. 63Windpessl M, Mayrbaeurl B, Baldinger C, et al. Overfeeding Syndrome in Oncology: Report on four cases. World J Oncol. 2017; 8(1): 25- 29. 64Pantoja F, Fragkos KC, Patel PS, et al. Overfeed syndrome in adults receiving general parenteral nutrition: audit practices in the TERC uk. Wedge Natra. 2019; 38(3): 1457- 1463. 65Matthews KL, Capra SM, Palmer MA. Throw caution to the wind: is feeding syndrome really the cause of death in the emergency room? Eur J Wedge Natra. 2018; 72(1): 93- 98. 66Azumagawa K, Kambara Y, Kawamura N, et al. Anorexia nervos and Feeding Syndrome. The case report. Scienceworld. 2007; 7: 400- 403. 67Kasai M, Okajima Y, Takano E, Kato S. Anorexia nervosa with Feeding Syndrome: Prevention and Treatment of RS. Seyshin Sinkeigaku Sashi. 2009; 111(4): 388- 397. 68Parkash O, Ayub A. Abid S. Feeding syndrome in a young girl with anorexia nervos. J Coll Phys Surg Pak. 2014; 24 (suppl 2): S78' S80. 69Bargiacki A, Clark J, Paulsen A, Leger J. Refeeding in anorexia nervosa. Eur J Pediatrician. 2019; 178(3): 413- 422. 70Rnstein RM, Golden NH, Jacobson MS, Schenker IR. Hypophosphateemia during food rehabilitation in anorexia nervosa: effects on feeding and monitoring. J Adolesc Health. 2003; 32(1): 83- 88. 71White M., Gilbertson H, Lam PY, Sayyer S.M. Does aggressive overfeeding of hospitalized adolescents with anorexia nervosa lead to increased hypophosphateemia? J Adolesc Health. 2010; 46(6): 577- 582. 72Armstrong E. Primary Psychiatric Care Toolkit. London: HMSO; 1997. 73Macdougall S. The impact of nutrition education on the shopping and eating habits of a small group of chronic schizophrenia patients living in the community. Br J Occupy Tr. 1992; 55(2): 62- 68. 74Hershkowitz E, Reshef A, Munich O, Yosefi B, Markel A. Thiamin deficiency in self-indicaced feeding syndrome, an undetected and potentially fatal condition. Rep Med case. 2014; 2014:605707. 75Santholaria-Fernandez FJ, Gomez-Sirvent JL, Gonzalez-Reimers CE, et al. Food Evaluation addicts. Drug Alcohol depends. 1995; 38(1): 11- 18. 76Fung AT, Rimmer J. Hypophosphatevia secondary oral overfeeding syndrome in a patient with long-term alcohol abuse. Med J Aust. 2005; 183(6): 324- 326. 77Baltasar A, Del Rio J, Escriva C, Arlandis F, Martinez R, Serra C. Preliminary results of the duodenum. Obes Surg. 1997; 7(6): 500- 504. 78Silk, Jones L, Heath D. Feeding Syndrome: An important complication after bariatric surgery. Surg Obes Relat Dis. 2011; 7(5): e21- 23. 79Chiappetta S, Stein J. Feeding Syndrome: An Important Complication After Obesity Surgery. Obs Facts. 2016; 9(1): 12- 16. 80Machado JD, Suen VM, Chueire FB, Marchini JF, Marchini JS. Overfeeding syndrome, and forgot the potentially fatal condition. BMJ Case Republic 2009; 2009. 81Hammami S, Aref HL, Halfa M, Kohtalli I, Hammami M. Refeeding Syndrome in Adults With Coeliac Crisis: Report on The Case. J Med Case Republic 2018; 12(1): 22. 82Agarwal J, Poddar U, Yachha SC, Srivastava A. Feeding syndrome in children in developing countries who have coeliac disease. J Pediatr Gastroenterol Natra. 2012; 54(4): 521- 524. 83Lenicek Kleza J., Misak S., Jadresin O., Skorik I. Feeding syndrome in children with different clinical etiology. Eur J Wedge Natra. 2013; 67(8): 883- 886. 84Korbonits M, Blaine D, Elia M, Powell-Tuck J. Metabolic and hormonal changes during long-term fasting. Eur J Endocrinol. 2007; 157(2): 157- 166. 85Kellogg ND, Lukefahr JL. A criminal case has been opened on the fact of child starvation. Pediatrics. 2005; 116(6): 1309- 1316. 86Bunge PD, Frank LL. A case of overfage syndrome in a marine recruit. Mil Med. 2013; 178(4): e511- 515. 87Lapinskiene I, Mikulevichen G, Laubner G, Badaras R. Consequences of extreme diet in professional sports: bodybuilder feeding syndrome. Wedge Natra ESPEN. 2018; 23: 253- 255. 88Schoenfeld PY, Henry RR, Laird NM, Roxe DM. Assessment of the nutritional status of the population of the National Cooperative Dialysis. Kidneys Int Suppl. 1983 (13): S80 and 88. 89Tunberg BJ, Swami AP, Cestero R.V. Transverse and Longitudinal Nutrition Measurements in the Care of Hemodialysis Patients. Am J Wedge Natra. 1981; 34(10): 2005- 2012. 90Markmann. Nutritional status of patients with hemodialysis and peritoneal dialysis. Wedge Nefrehl. 1988; 29(2): 75- 78. 91Lowrie EG, Lew NL. Risk of death in patients with hemodialysis: the prognostic value of commonly measured variables and the assessment of mortality differences between institutions. Am J Kidney Dis. 1990; 15(5): 458- 482. 92da Silva J. Torsades is precipitated by overfeeding syndrome: the case report is abstract. Crit Care Med. 2016; 44(12): 484. 93Mariella M.A. Overfeeding Syndrome: An Important Aspect of Supporting Oncology. J Support Oncol. 2009; 7(1): 11- 16. 94Dolman RC, Conradie C, Lombard MJ, et al. SASPEN Case Study: Managing the nutrition of a patient at high risk of overficking syndrome. S Afr Klein Natra. 2015; 28(3): 140- 145. 95 Soolomon SM, Kirby DF. Overfed syndrome: review. JPEN J Parenter Enteral Natra. 1990; 14(1): 90- 97. 96Craft MD, Btaiche IF, Sachs GS. Review of overfeeding syndrome. Nartra Klin Practical. 2005; 20(6): 625- 633. 97McCray S, Parrish CR. Feeding malnourished patients: lessons learned. Pract Gastroenterol 2016; 155: 56- 66. 98Boland K, Solanki D, O'Hanlon C. Prevention and treatment of overfed syndrome in emergency rooms. . Published in 2014. Access to August 7, 2018. 99 Hamilton AK. CNSG East Cheshire NHS Trust to prevent and treat feeding syndrome in adults. 20-%20Refeeding%20Syndrome%20Guidelines%20ECT2366.pdf. Access to August 7, 2018. 100Fridley N, Stanga S., Culkin A. et al. Management and Prevention of Overfed Syndrome in Medical Hospitals: An algorithm based on evidence and consensus. Nutrition 2018; 47: 13- 20. 101Baran SA, Weltzin TE, Kaye WH. Low weight discharge and result in anorexia nervosa. Am J Psychiatry. 1995; 152(7): 1070- 1072. 102Rigaud D, Pennacchio H, Bizeul C, Reveillard V, Verges B. Result in adult AN patients: 13-year follow-up in 484 patients. Diabetes Metab. 2011; 37(4): 305- 311. 103 National Heart L, Blood Acute Respiratory Syndrome Clinical Trials N, Rice TW, Wheeler AP, et al. Initial Trophic vs. Full Enteral Feeding in Patients With Acute Lung Injury: Randomized EDEN Study. Jama. 2012; 307(8): 795- 803. 104Doig GS, Simpson F, Sweetman EA, and others Early parenter nutrition in seriously ill patients with short-term relative conditions to early enteral nutrition: a randomized controlled trial. Jama. 2013; 309(20): 2130- 2138. 105Casaer MP, Mesoten D, HermanS G, et al. Early and later parenteral food in seriously ill adults. N Engl J Med. 2011; 365(6): 506- 517. 106Golden NH, Keen-Miller C, Sainani KL, Kapphahn CJ. Higher calorie intake in hospitalized adolescents with anorexia nervosa is associated with a reduction in stay and a lack of increased rate of overfeeding syndrome. J Adolesc Health. 2013; 53(5): 573- 578. 107Redgrave GW, Coughlin JW, Schreier CC, et al. Recovery and Weight Recovery Results in Anorexia Nervosa: Complex Current Guidelines. Int J Eat Disord. 2015; 48(7): 866- 873. 108Doig GS, Simpson F, Heighes PT, et al. Limited compared to the continuation of standard calorie intake during the use of overfeeding syndrome in seriously ill adults: randomized, parallel, multicenter, single-blind controlled study. Lancet Respir Med. 2015; 3(12): 943- 952. 109Olthof LE, Koekkoek W, van Setten C, Kars JCN, van Blockland D, van ernten ARH. Effect of calorie intake in seriously ill patients with and without overfalation syndrome: retrospective study. Wedge Natra. 2018; 37(5): 1609- 1617. 110 World Health Organization. Managing Severe Malnutrition: A Guide for Physicians and Other Senior Health Care Workers. Switzerland: WHO Publications Bureau; 1999. ISBN 92 4 154511 9 (NLM CLASSIFICATION: WD 101). 111Kruk M.A. Overfeeding Syndrome: Problems with Definition and Management. Nutrition 2014; 30(11-12): 1448- 1455. 112Khan LU, Ahmed J, Khan S, Macfie J. Feeding Syndrome: Literature review. Gastroenterol Res Pract. 2011:pii: 410971. 113Mehanna H, Nankivell PC, Moledina J, Travis J. Feeding Syndrome - Awareness, Prevention and and Oncol's neck head. 2009; 1: 4. 114Becker P, Carney LN, Corkins MR, et al. Consensus Statement of the Academy of Nutrition and Dietetics/American Society of Parenteral and Enteral Nutrition: Indicators Recommended for Identification and Documents on Child Malnutrition (Malnourishment). Nartra Klin Practical. 2015; 30(1): 147- 161. 115Secker DJ, Gigibhoy K.N. How to perform a subjective global assessment of nutrition in children. J Akade Natra Diet. 2012; 112(3): 424-431 e426. 116Green Corkins K. Nutrition is focused on physical examination in pediatric patients. Nartra Klin Practical. 2015; 30(2): 203- 209. Luis Eduardo Gonzalez-Salazar, Marta Guevara-Cruz, Carla Guadalupe Hernandez-Gomez, Aurora Elizabeth Serralde Sieg, Nutrition Management of Seriously Ill Inpatients with COVID-19. Storytelling Review, Nutrici'n Hospitalaria, 10.20960/nh.03180, (2020). Verena Haas, Michael Cohn, Torsten Koerner, Ulrich Kunz, Andrea K. Garber, Daniel Le Grange, Ulrich Voderholzer, Christoph W. Correll, practical evidence and clinical guide to support the accelerated re-evaluation of patients with anorexia nervosa, Journal of the American Academy of Pediatric and Adolescent Psychiatry, 10.1016/j.jaac.2020.09.010, (2020). Robert Martindale, Jayshil J. Patel, Beth Taylor, Yasin M. Arabi, Malissa Warren, Stephen A. McClave, Nutrition Therapy in Critical Patients With Coronavirus Disease 2019, Journal of Parenteral and Enteral Nutrition, 10.1002/jpen.1930, 44, 7, (1174-1184), (202). Annika Reintam Blazer, Jan Ganst, Carol Ichai, Michael. Kasaer, Karina Benstom, Guillaume Besh, Stefan Dauger, Sonia M. Fruhwald, Michael Hismayr, Olivier Joanns-Boyau, Manu L.N.G. Malbrein, Maria-Elena Perez, Stefan Schaller, Angelica de Man, Joel Starkopf, Kadri Tamme, Jan Wernerman, Mette M. Berger, Hypophosphatemy in seriously ill adults and children - Systematic Review, Clinical Nutrition, 10.1016/j.clnu.2020.045, (2020). Gabriel J. Y. Wong, Janelle G. T. Pang, Yuan Y. Lee, Charles Chin Han Liu, Refeeding hypophosphateemia in patients receiving parenteral nutrition: Prevalence, risk factors, and prediction of its occurrence, nutrition in clinical practice, 10.1002/ncp.10559, 0, 0, (2020). Torsten Kerner, Verena Haas, Julia Hize, Matislava Karacic, Elmar Ngo, Christophe W. Correll, Ulrich Voderholzer, Ulrich Kunz, Results of accelerated inpatient feeding protocol in 103 Extremely underweight adults with anorexia nervosa at a specialized clinic in Prien, Germany, Journal of Clinical Medicine, 10.3390/jcm9051535, 9, 5, (1535), (2020). Patrick F van Renen, Marina Aloi, Amit Assa, Jiri Bronski, Johanna S. Escher, Ulrika L. Fagerberg, Marco Gasparetto, Konstantinos Gerasimidis, Ann Griffiths, Paul Henderson, Sybille Kolejo, Caia-Lina Kolho, Arie Levine, Johann van Limbergen, Francisco Javier Martin Lissie de Ridder, Richard K Russell, Dror Schuval, Antonino Spinelli, Dan Turner, David Wilson, Eitan Wine, Frank M Ruemmele, Medical Management of Pediatric Crohn's Disease: ECCO-ESPGHAN Guide Update, Crohn's Journal and Colitis, 10.1093/ecco-jcc/jjaa161, (2020). Kerry C. McMillen, Tara Coglin-Dixon, Peter A. Adintori, Optimization of nutrition support practices in the early stages after hematopoetic cell transplantation, bone marrow transplantation, 10.1038/s41409-020-01078-9, (2020). Kylie Matthews-Rensh, Sandra Capra, Michelle Palmer, Systematic Energy Review Initiation Stakes and Refeeding Syndrome Results, Nutrition in Clinical Practice, 10.1002/ncp.10549, 0, 0, (indefinite). The full text of this article, posted on the iucr.org is unavailable due to technical difficulties. Difficulties.

normal_5f89bafcc8870.pdf
normal_5f872d73dc61c.pdf
normal_5f8953be982e7.pdf
normal_5f8996203bdf6.pdf

video downloader link apk
récupérer sms effacés samsung android
frigidaire gallery heavy duty dryer manual
виктор суворов контроль pdf
manual do moto e5 play
fikir eske mekabir pdf free download
macmillan skillful reading and writing pdf
properties of alkanes chemguide
pendule de foucault exercice
juegos de presentacion para adolesce
blank map of europe before ww1
letters to live by vol. 1 download
bridge engineering book pdf
400 wrf fiche technique
download nintendo ds roms
normal_5f8719a28b346.pdf
normal_5f8734aa65ed8.pdf
normal_5f89594d516fd.pdf
normal_5f872e607c8e5.pdf
normal_5f873094a03a2.pdf