

Energy expenditure and energy intake – Guidelines on Parenteral Nutrition, Chapter 3

Energieumsatz und Energiezufuhr – Leitlinie Parenterale Ernährung, Kapitel 3

Abstract

The energy expenditure (24h total energy expenditure, TEE) of a healthy individual or a patient is a vital reference point for nutritional therapy to maintain body mass. TEE is usually determined by measuring resting energy expenditure (REE) by indirect calorimetry or by estimation with the help of formulae like the formula of Harris and Benedict with an accuracy of $\pm 20\%$. Further components of TEE (PAL, DIT) are estimated afterwards. TEE in intensive care patients is generally only 0–7% higher than REE, due to a low PAL and lower DIT. While diseases, like particularly sepsis, trauma and burns, cause a clinically relevant increase in REE between 40–80%, in many diseases, TEE is not markedly different from REE. A standard formula should not be used in critically ill patients, since energy expenditure changes depending on the course and the severity of disease. A clinical deterioration due to shock, severe sepsis or septic shock may lead to a drop of REE to a level only slightly (20%) above the normal REE of a healthy subject. Predominantly immobile patients should receive an energy intake between 1.0–1.2 times the determined REE, while immobile malnourished patients should receive a stepwise increased intake of 1.1–1.3 times the REE over a longer period. Critically ill patients in the acute stage of disease should be supplied equal or lower to the current TEE, energy intake should be increased stepwise up to 1.2 times (or up to 1.5 times in malnourished patients) thereafter.

Keywords: energy requirements, sepsis, critically ill, intensive care

Zusammenfassung

Der aktuelle Energieumsatz (24-Stunden-Gesamtenergieumsatz, TEE) eines Gesunden oder eines Patienten ist für die Ernährungstherapie die entscheidende Bezugsgröße, um die Körpermasse zu erhalten. Die Ermittlung des TEE erfolgt entweder durch die Messung des Grundumsatzes (REE) mittels indirekter Kalorimetrie oder durch Schätzung mit Hilfe von Formeln (z.B. Harris und Benedict) mit einer Genauigkeit von $\pm 20\%$ bei Gesunden. Die anderen Komponenten (PAL, NIT) des TEE werden geschätzt. Der Gesamtenergieumsatz von Intensivpatienten liegt in der Regel nur 0% bis 7% über dem Grundumsatz, aufgrund eines niedrigen PAL und geringerer NIT. Während der Energieumsatz bei vielen Erkrankungen im Bereich des Standard-Grundumsatzes bleibt, führen einige Erkrankungen (besonders Sepsis, Trauma, Verbrennungen) zu einer klinisch relevanten Steigerung des REE von 40% bis 80%. Da die Steigerung des Energieumsatzes bei kritisch Kranken eine dynamische Größe ist, abhängig vom Verlauf und vom Schweregrad der Erkrankung, sollte keine fixe Formel angewandt werden. Eine klinische Verschlechterung (z.B. Schock, schwere Sepsis, septischer Schock) geht mit einer relativen Abnahme des Energieumsatzes bis auf ungefähr 20% des REE von Gesunden einher. Überwiegend immobile Patienten sollten eine Energiezufuhr zwischen dem 1,0–1,2-fachen des ermittelten REE erhal-

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ten, immobile Patienten nach Mangelernährung eine schrittweise Steigerung der Zufuhr auf das 1,1–1,3-fache des REE über einen längeren Zeitraum. Die zugeführte Energie sollte bei kritisch Kranken im Akutstadium im Bereich des aktuellen TEE oder leicht darunter liegen, danach sollte schrittweise auf das 1,2-fache (bei gleichzeitiger Mangelernährung bis 1,5-fach) gesteigert werden.

Schlüsselwörter: Energiebedarf, Sepsis, kritisch Kranke, Intensivtherapie

Energy expenditure in healthy people

An understanding of energy expenditure in healthy individuals is essential for planning energy supply for patients because:

1. it forms a basis for estimating the energy expenditure of the patient or
2. it may provide a reference when energy expenditure of a patient is measured to evaluate changes in energy expenditure caused by disease.

Components of energy expenditure

The energy expenditure of a healthy individual or a patient is a vital reference point for nutritional therapy. Under normal conditions this amount of energy must be provided to avoid a decrease or increase in body mass.

The total energy expenditure (TEE) consists of three main components:

- Resting energy expenditure, REE, or basal metabolic rate
- Energy expended during physical activity,
- Energy required for metabolism of the infused substrates (diet-induced thermogenesis, DIT)

Some illnesses such as sepsis, trauma, burns, hyperthyroidism and hypothyroidism, may result in a change in energy metabolism and, therefore, in energy expenditure.

Measuring energy expenditure

Measurement of total energy expenditure over e.g. 24 hours or longer, is difficult to achieve and generally can only be carried out under experimental conditions, usually with the doubly-labelled water method [1].

It is common practice to first measure the resting energy expenditure (REE), and then to estimate the other components of total energy expenditure (TEE). In order to measure REE, a standardised protocol is followed and various conditions (frequency of feeding, time of day, room temperature etc.) are strictly observed. The method usually used for measurement of REE is indirect calorimetry [1]. In this method, the O₂ and CO₂ concentrations in the expired air are measured, and energy expenditure calculated, with the help of formulae, on the basis of oxygen consumption and carbon dioxide production. This

method can be used in both, spontaneously breathing and mechanically ventilated individuals.

Estimation of the basal metabolic rate in healthy individuals from formulae

• Harris and Benedict formula:

REE, men = $66.5 + 13.8 * \text{weight (kg)} + 5.0 * \text{height (cm)} - 6.8 * \text{age (years)}$

REE, women = $655 + 9.6 * \text{weight (kg)} + 1.8 * \text{height (cm)} - 4.7 * \text{age (years)}$

- The basal metabolic rate of healthy people can be estimated by formulae with an accuracy of $\pm 20\%$ (I)
- The following approximations can be used for estimating resting energy expenditure, when only weight is available (C):
 - 20–30 years: 25 kcal/kg body weight/day
 - 30–70 years: 22.5 kcal/kg body weight/day
 - >70 years: 20 kcal/kg body weight/day

Commentary

If REE is not measured, REE or basal metabolic rate (BMR) can be estimated with the help of formulae. Although many formulae have been developed to estimate the basal metabolic rate, the best known and used among these is the formula by Harris and Benedict [2]. In this formula, BMR is estimated based on the height, weight, age and gender of the individual. Although the formula was derived more than 90 years ago, using indirect calorimetry on a small group of 239 mostly young normal subjects, it is as accurate as more recently developed formulae, derived from larger groups. It is, therefore, still used as a guide to estimate energy requirements. In all the formulae used, the calculated values for women, with all other factors being same, are lower than for men. In addition, BMR decreases with increasing age.

The correlation coefficient squared (r^2) between calculated and measured BMR is <0.75 for all formulae, i.e. only 75% of the variance of BMR is predicted by weight, height, age and gender, with a 95% confidence interval of about $\pm 20\%$ in the samples where the respective formulae were developed.

Comparison of the basal metabolic rate values obtained by using the Harris and Benedict formula with measured values in a German reference database demonstrated that the estimated values of 42.6% of the subjects were more than 10% higher than the measured values, and for 18.0% the estimates were more than 20% higher [3].

In contrast, for 2.3% the estimated values were more than 10% lower than the measured values, and for 0.7% they were more than 20% lower. Another study with 130 healthy subjects, using Harris-Benedict equation overestimated the basal metabolic rate by more than 10% in 33% of the subjects [4]. Both studies show that the use of Harris-Benedict formula may lead to overestimation of basal metabolic rates in certain individuals. Estimation of BMR can be improved slightly by using formulae also considering the lean body mass and/or cell mass [4], which, however, must first be measured using an additional method. Use of body weight only is a poor predictor of BMR, and is not recommended.

When only body weight is available, BMR can be roughly estimated as follows: BMR in individuals aged 30 to 70 years is approximately 22.5 kcal/kg body weight/day. In younger adults, the value is about 25 kcal/kg body weight/day, and in older adults, 22 kcal/kg body weight/day. BMR of obese or malnourished subjects is estimated from actual, and not from ideal body weight.

Estimating total energy expenditure in healthy people

Diet-induced thermogenesis (DIT) contributes to approximately 10% of the estimated TEE. In healthy individuals TEE is markedly dependent on energy expended during physical activity, and it can be estimated from REE by multiplying the REE with a factor for physical activity level (PAL) [5]. If the TEE is not measured, the amount of the physical activity may be estimated according to the degree of physical activity. Published tables may be used for this [5]. For example, in an English study, the TEE of healthy women without any heavy physical work was on an average 138% of REE, i.e. the PAL was 1.38.

Energy metabolism in patients

Components of energy metabolism in patients

- The total energy expenditure in patients is generally only 100–107% of resting energy expenditure (REE) (I).

Commentary

Most patients, especially those who are bedridden, have a low level of physical activity that has little effect on TEE (except for patients undergoing intensive rehabilitation program). Furthermore, parenteral infusion of amino acids or substrate administration by means of enteral feeding induces DIT to a much lesser extent than an oral protein intake. Therefore, TEE of many parenterally fed patients is not significantly higher than REE.

Several studies studied the ratio of TEE to REE in intensive care patients and concluded that TEE is only 0–7%

higher than REE [6], [7], [8], [9], [10]. In contrast, other studies reported a substantial increase of TEE up to 80% in critically ill patients [11], [12], [13]. However, in these latter studies, TEE was not *measured* but *calculated*, using energy intake and energy balance, i.e. the increase or decrease in individual body compartments.

In severely ill patients, energy expenditure may be altered due to several factors. Diseases that induce metabolic changes by increasing or decreasing the release of hormones or cytokines, may cause a corresponding increase as well as decrease in REE. Severe pain, psychological stress, shivering, and fever may all increase REE.

Since the usual circadian variation in REE tends to be absent in critically ill patients, a valid estimate of their TEE per day can be made from measuring energy expenditure during a 20 to 30 minute period at any time of the day. REE determined in this way can be compared to normal REE for a healthy person of the same weight, height, gender and age to assess the disease-induced change in REE.

Changes in the energy expenditure caused by illness

- In many disease states, the TEE is not markedly different from REE (I).
- Some illnesses, particularly sepsis, trauma and burns, cause a clinically relevant increase in REE between 40–80% (I).

Commentary

Not all diseases induce changes in REE. In most disorders, particularly in acute medicine, elective operations etc., measured REE is within $\pm 10\%$ of the values predicted from formulae [14], [15], [16], which is within the range of measurement error. Therefore, one of the formulae used for estimating REE can be applied in most patients, e.g. the Harris und Benedict formula. TEE can be calculated as the estimated REE \times 1.0–1.2 to accommodate low levels of physical activity.

Some diseases, however, may cause a clinically significant change in REE, for example hypo- or hyperthyroidism, sepsis, trauma and burns, that often result in a marked increase in REE of 40 to 80% or more [14], [15], [16].

These disease-induced changes in REE are difficult to estimate, because they depend both on the severity and the duration of illness. A number of published studies show considerable differences between the estimated and measured REE in critically ill patients, and the ratio between the two values can change very rapidly.

Time dependent changes in the course of disease

- Energy expenditure in critically ill patients changes depending on the duration and the severity of disease.

Therefore, a standard formula should not be used for critically ill patients (I).

- A clinical deterioration due to shock, severe sepsis or septic shock decreases energy expenditure of the patient (I).

Commentary

The difficulty in assessing energy expenditure was highlighted by one study of 47 intensive care patients in which the predictive values of five different formulae for estimating REE were investigated by measuring energy expenditure [4]. The authors showed that REE could be estimated with one formula with an accuracy of $\pm 10\%$ in 72% of 130 intensive care patients, and concluded that this formula was preferable to the other four tested. However, this result was based on assessments on day 19 of treatment on average, and different results might have been obtained at other time points.

Studies assessing energy expenditure of critically ill patients on several consecutive days found considerable day to day variation [15], [17], [18]: a continuous decrease from a peak value [19] or, in most cases, a pattern with a rise, a peak and then a slow decrease [11], [12], [13], [20], [21], [22], [23]. These studies indicate that no single formula can reliably predict the energy expenditure for the total course of illness for a critically ill patient. It seems sensible to assume that for most patients with sepsis, trauma or major operations, the course of energy expenditure increases during the first days of disease, then rises continually, reaches a peak usually between day 4 and 10, and then drops gradually over a period of weeks or even months. The peak value, the time at which the peak is reached, and the total duration of metabolic changes, depends on the severity and course of the illness. Peak values usually are about 40–80% above normal REE.

In burns patients, a very rapid rise of REE to up to 90% above normal, followed by a gradual continual drop returning to normal values only after more than 100 days was observed [19].

A deterioration in the patient's condition may reduce REE [24], [25] and thus may lead to an even more difficult estimation of energy expenditure. Severe sepsis or septic shock may lead to a drop of REE to a level only slightly (20%) above the normal REE or close to normal REE of a healthy subject. In case of clinical deterioration, development of multi-organ failure and/or shock, if it is not feasible to measure REE, one should assume that REE is either slightly above or close to normal REE.

Determination of parental energy supply based on total energy expenditure

Energy metabolism and infusion

- The measured or estimated energy expenditure is not necessarily the target value for parenteral energy supply (C).

Commentary

An energy intake corresponding to the energy expended (isocaloric nutrition) results in energy balance, and the preservation of status quo. In contrast, an energy intake *below* the energy expenditure (hypocaloric nutrition, underfeeding) leads to a loss of body mass and weight. An energy intake *above* the energy expenditure (hypercaloric nutrition, hyperalimentation) can compensate for preceding malnutrition.

It is often assumed that energy intake of patients, particularly of critically ill patients, should match the current energy expenditure (measured or estimated), in order to maintain balance. This is not recommended, but rather the amount of energy provided relative to energy expenditure should be a conscious therapeutic decision, taking into account the objective of nutrition therapy in each case.

Determining energy intake

- Patients who are largely immobile, with diseases not resulting in a significant change of REE, and who show no signs of malnutrition, should initially receive an energy intake similar to their current measured or estimated REE. The energy intake can be increased to about 1.2 times REE depending on individual tolerance (C).

Commentary

In patients, as in healthy people, isocaloric nutrition leads to preservation of body mass. No controlled studies are available on this issue; therefore, the above recommendation represents only an expert opinion (C).

Energy intake in malnourished patients

- In predominantly immobile malnourished patients, energy intake should be increased stepwise over a longer period to a target value of 1.1–1.3 times the REE (C).

Commentary

In malnourished patients, the lost endogenous stores have to be replenished. Therefore, energy intake should be higher than current energy expenditure. To avoid a

refeeding syndrome (cf. chapter “Complications and Monitoring” <http://www.egms.de/en/gms/2009-7/000076.shtml>), the energy intake should be gradually increased from a value close to REE to a higher value of about 1.1–1.3 x REE. Thus in acute medical care, energy intake should be close to the current value of REE for these patients.

Energy intake in critically ill patients

Assessment of energy intake in critically ill patients

- In critically ill patients, hyperalimentation is not recommended in the acute stage of the disease (A).

Commentary

For a long time, nutrition therapy in critically ill patients was dominated by the concept of hyperalimentation, i.e. the provision of a parenteral energy intake of 40–60 kcal/kg/day corresponding to 50–100% above the increased energy expenditure (even 33.5 kcal/kg/day corresponds to an energy intake of 50% above the normal REE). The aim of hyperalimentation was to achieve a positive nitrogen balance.

There are no controlled trials comparing parenteral hyperalimentation with a nutritional regime providing a lower energy intake. However, many studies comparing parenteral hyperalimentation with simple fluid therapy or enteral nutrition showed no advantages or disadvantages between the two groups. A meta-analysis of 27 studies on PN patients revealed a higher mortality (risk ratio 1.78, 95% confidence interval 1.11–2.85) in a sub-group of critically ill patients receiving PN [26]. This higher mortality might have resulted from the hyperalimentation used in many of these studies. A considerable proportion of energy supply in these studies was provided as intravenous glucose, resulting in the risk of pronounced and lasting hyperglycaemia, which could not be corrected even by large quantities of insulin. Hyperglycaemia may lead to adverse effects on the immune system, and thereby affect the survival of critically ill patients [27] (I). In view of these adverse effects of hyperglycemia and the associated higher mortality [26], it is recommended that hyperalimentation not be used in critically ill patients.

Energy intake in acute stages of disease

- In critically ill patients in the acute stage of disease, parenteral energy supply should be either lower than or equal to the current TEE (B).
- In critically ill patients who have survived the acute stage, energy intake should be increased stepwise to 1.2 times the current REE (or to 1.5 times REE in malnourished patients) (C).

Commentary

Animal studies revealed negative consequences of hyperalimentation, but also found that in experimental sepsis, lower parenteral energy provision (hypocaloric nutrition) resulted in improved survival [28], [29]. No controlled clinical trials have assessed the impact of hypocaloric nutrition in critically ill patients. An observational study [30] in patients treated for more than four days in intensive care showed that patients suffering from SIRS who cumulatively received only 33–65% of the planned energy intake had a significantly higher survival (odds ratio 1.22, 95% confidence interval 1.15–1.29). A sub-group of these patients, who received only 25% of planned energy intake, had a significantly lower incidence of positive blood cultures [31].

Based on these data, it is concluded by the expert group that energy intake of critically ill patients, in the acute stage of disease, should be less than or equal to the current TEE. In the initial phase, energy intake could be even lower than energy expenditure, and then increased gradually over the course of several days until it matches the current energy expenditure.

Once the maximum value of energy expenditure is reached, energy intake should be gradually reduced, over a longer period of time. At this time, patients should reenter an anabolic state, in which the loss from the preceding catabolic state can be corrected. This can be achieved by a stepwise increase of energy and substrate supply. Due to a lack of controlled trials, this recommendation represents only an expert opinion (C).

Notes

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References

1. Müller MJ, Selberg O, Süttmann U, Weimann A, Kruse ER. Schätzung und Messung des Energieverbrauchs: Methoden und Stellenwert in der klinischen Diagnostik. *Intensivmed Notfallmed.* 1992;29(8):411-26.
2. Harris JA, Benedict FG. A biometric study of basal metabolism in man. Washington: Carnegie Institution of Washington; 1919. (Carnegie Institution of Washington publication; 279)

3. Müller MJ, Bosity-Westphal A, Klaus S, Kreymann G, Lührmann PM, Neuhäuser-Berthold M, Noack R, Pirke KM, Platte P, Selberg O, Steiniger J. World Health Organization equations have shortcomings for predicting resting energy expenditure in persons from a modern, affluent population: generation of a new reference standard from a retrospective analysis of a German database of resting energy expenditure. *Am J Clin Nutr.* 2004;80(5):1379-90.
4. Frankenfield DC, Rowe WA, Smith JS, Cooney RN. Validation of several established equations for resting metabolic rate in obese and nonobese people. *J Am Diet Assoc.* 2003;103(9):1152-9. DOI: 10.1016/S0002-8223(03)00982-9
5. Shetty PS, Henry CJ, Black AE, Prentice AM. Energy requirements of adults: an update on basal metabolic rates (BMRs) and physical activity levels (PALs). *Eur J Clin Nutr.* 1996;50 Suppl 1:S11-23.
6. Behrendt W, Surmann M, Raumanns J, Giani G. How reliable are short-term measurements of oxygen uptake in polytraumatized and long-term ventilated patients? *Infusionstherapie.* 1991;18(1):20-4.
7. Frankenfield DC, Wiles CE 3rd, Bagley S, Siegel JH. Relationships between resting and total energy expenditure in injured and septic patients. *Crit Care Med.* 1994;22(11):1796-804.
8. Smyrniotis NA, Curley FJ, Shaker KG. Accuracy of 30-minute indirect calorimetry studies in predicting 24-hour energy expenditure in mechanically ventilated, critically ill patients. *JPEN J Parenter Enteral Nutr.* 1997;21(3):168-74. DOI: 10.1177/0148607197021003168
9. Swinamer DL, Phang PT, Jones RL, Grace M, King EG. Twenty-four hour energy expenditure in critically ill patients. *Crit Care Med.* 1987;15(7):637-43. DOI: 10.1097/00003246-198707000-00002
10. Weissman C, Kemper M, Elwyn DH, Askanazi J, Hyman AI, Kinney JM. The energy expenditure of the mechanically ventilated critically ill patient. An analysis. *Chest.* 1986;89(2):254-9. DOI: 10.1378/chest.89.2.254
11. Monk DN, Plank LD, Franch-Arcas G, Finn PJ, Streat SJ, Hill GL. Sequential changes in the metabolic response in critically injured patients during the first 25 days after blunt trauma. *Ann Surg.* 1996;223(4):395-405. DOI: 10.1097/0000658-199604000-00008
12. Plank LD, Connolly AB, Hill GL. Sequential changes in the metabolic response in severely septic patients during the first 23 days after the onset of peritonitis. *Ann Surg.* 1998;228(2):146-58. DOI: 10.1097/0000658-199808000-00002
13. Uehara M, Plank LD, Hill GL. Components of energy expenditure in patients with severe sepsis and major trauma: a basis for clinical care. *Crit Care Med.* 1999;27(7):1295-302. DOI: 10.1097/00003246-199907000-00015
14. Adolph M, Eckart J. Der Energiebedarf operierter, verletzter und septischer Patienten [Energy requirements of surgically treated, injured and infected patients]. *Infusionstherapie.* 1990;17(1):5-16.
15. Behrendt W, Kuhlen R. Der Energieverbrauch des kritisch-kranken Patienten. *Intensiv Notfallbehandlung.* 2000;25(1):20-4.
16. Chioléro R, Revelly JP, Tappy L. Energy metabolism in sepsis and injury. *Nutrition.* 1997;13(9 Suppl):45-51.
17. de Klerk G, Hop WC, de Hoog M, Joosten KF. Serial measurements of energy expenditure in critically ill children: useful in optimizing nutritional therapy? *Intensive Care Med.* 2002;28(12):1781-5. DOI: 10.1007/s00134-002-1523-z
18. Khorram-Sefat R, Behrendt W, Heiden A, Hettich R. Long-term measurements of energy expenditure in severe burn injury. *World J Surg.* 1999;23(2):115-22. DOI: 10.1007/PL00013172
19. Milner EA, Cioffi WG, Mason AD, McManus WF, Pruitt BA Jr. A longitudinal study of resting energy expenditure in thermally injured patients. *J Trauma.* 1994;37(2):167-70. DOI: 10.1097/00005373-199408000-00001
20. Ishibashi N, Plank LD, Sando K, Hill GL. Optimal protein requirements during the first 2 weeks after the onset of critical illness. *Crit Care Med.* 1998;26(9):1529-35. DOI: 10.1097/00003246-199809000-00020
21. Long CL, Schaffel N, Geiger JW, Schiller WR, Blakemore WS. Metabolic response to injury and illness: estimation of energy and protein needs from indirect calorimetry and nitrogen balance. *JPEN J Parenter Enteral Nutr.* 1979;3(6):452-6.
22. Plank LD, Hill GL. Sequential metabolic changes following induction of systemic inflammatory response in patients with severe sepsis or major blunt trauma. *World J Surg.* 2000;24(6):630-8. DOI: 10.1007/s002689910104
23. Plank LD, Metzger DJ, McCall JL, Barclay KL, Gane EJ, Streat SJ, Munn SR, Hill GL. Sequential changes in the metabolic response to orthotopic liver transplantation during the first year after surgery. *Ann Surg.* 2001;234(2):245-55. DOI: 10.1097/0000658-200108000-00015
24. Forsberg E, Soop M, Thorne A. Energy expenditure and outcome in patients with multiple organ failure following abdominal surgery. *Intensive Care Med.* 1991;17(7):403-9. DOI: 10.1007/BF01720678
25. Kreymann G, Grosser S, Buggisch P, Gottschall C, Matthaei S, Gretten H. Oxygen consumption and resting metabolic rate in sepsis, sepsis syndrome, and septic shock. *Crit Care Med.* 1993;21(7): 1012-9. DOI: 10.1097/00003246-199307000-00015
26. Heyland DK, MacDonald S, Keefe L, Drover JW. Total parenteral nutrition in the critically ill patient: a meta-analysis. *JAMA.* 1998;280(23):2013-9. DOI: 10.1001/jama.280.23.2013
27. van den Berghe G, Wouters P, Weekers F, Verwaest C, Bruyninckx F, Schetz M, Vlasselaers D, Ferdinande P, Lauwers P, Bouillon R. Intensive insulin therapy in the critically ill patients. *N Engl J Med.* 2001;345(19):1359-67. DOI: 10.1056/NEJMoa011300
28. Alexander JW, Gonce SJ, Miskell PW, Peck MD, Sax H. A new model for studying nutrition in peritonitis. The adverse effect of overfeeding. *Ann Surg.* 1989;209(3):334-40.
29. Peck MD, Alexander JW, Gonce SJ, Miskell PW. Low protein diets improve survival from peritonitis in guinea pigs. *Ann Surg.* 1989;209(4):448-54. DOI: 10.1097/0000658-198904000-00010
30. Krishnan JA, Parce PB, Martinez A, Diette GB, Brower RG. Caloric intake in medical ICU patients: consistency of care with guidelines and relationship to clinical outcomes. *Chest.* 2003;124(1):297-305. DOI: 10.1097/01.CCM.0000089641.06306.68
31. Rubinson L, Diette GB, Song X, Brower RG, Krishnan JA. Low caloric intake is associated with nosocomial bloodstream infections in patients in the medical intensive care unit. *Crit Care Med.* 2004;32(2):350-7. DOI: 10.1097/01.CCM.0000089641.06306.68

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