displace parenteral nutrition, which dominated for decades the scene of nutrition in acute severe pancreatitis. The capacity of enteral nutrition to influence the acute phase response, the systemic inflammatory reaction and the infection rate recommend it as a therapeutical intervention promoting enhanced recovery.

In conclusion, enteral nutrition in severe acute pancreatitis is not an easy task and needs a lot of commitment and dedication. But, despite difficulties its early and "obstinate" application may favorably influence the evolution of the disease.

Bibliography:

1. MCCLAVE SA et al: Nutritional support în acute pancreatitis: a systematic review of the literature, *JPEN*, 2006, 30(2):143-156.
2. TIENGOU LE et al: Semi-elemental formula: is there a better chice for enteral nutrition în acute pancreatitis? Randomized comparative study, *JPEN*, 2006, 30(1):1-5.
3. WHITCOMB DC: Acute Pancreatitis, *N Engl J Med*, 2006, 354:2142-50.
4. MARIK PE, ZALOGA GP: Meta-analysis of parenteral nutrition versus enteral nutrition în patients with acute pancreatitis, *BMJ*, 2004, 328:1407-0.
5. KALE-PRADHAN PB et al: Enteral Nutrition în Patients with Pancreatitis, *Pharmacotherapy*, 1999, 19(9):1036-1041.
6. MEIER R et al: ESPEN guidelines on nutrition în acute pancreatitis, *Clinical Nutrition*, 2002, 21(2):173-183.
7. R MEIER et al: ESPEN guidelines on enteral nutrition: pancres; *Clinical Nutrition* 25,275-284, 2006.
8. CANO NJM: Nutrition in acute pancreatitis, *Crit Care & Shock,* 2004, 7(2):69-76.
9. ZHAO G et al: Clinical study on nutritional support în patients with severe acute pancreatitis, *World J Gastroenterol*, 2003, 9(9):2105-2108.
10. WINDSOR AC et al: Compared with parenteral nutrition, enteral feeding attenuates the acute phase response and improves disease severity în acute pancreatitis, *Gut*, 1998, 42:431-435.
11. GREENWOOD JK et al: Enteral nutrition in acute pancreatitis: A survey of practices în Canadian Intensive Care Units, *Nutrition in Clinical Practice*, 2004, 19(1):31-36.
12. CHANDRASEGARAM MD et al: The impact of parenteral nutrition on body composition of patients with acute pancreatitis, *Journal of Parenteral and Enteral Nutrition*, 2005, 29(2):65-73.
13. POWELL JJ et al: Randomized controlled trial of the effect of early enteral nutrition on markers of the inflammatory response in predicted severe acute pancreatitis; *Br J Surg*, 2000, 87:1375-1381.
14. RAIMONDO M, SCOLAPIO JS: What route to feed patients with severe acute pancreatitis: vein, jejunum, stomach?, *Am J Gastroenterol*, 2005, 100(2): 440-441.
15. ALSOLAIMAN MM et al: Should enteral feeding be the standard of care for acute pancreatitis?, *Am J Gastroenterol*, 2003, 98(11):2565.
16. ABOU-ASSI S et al: Hypocaloric jejunal feeding is better than total parenteral nutrition in acute pancreatitis: results of a randomized comparative study, *Am J Gastroenterol*, 2002, 97:2255-62.
17. EATOCK D et al: A Randomized Study of Early Nasogastric versus Nasojejunal Feeding in Acute Severe Pancreatitis, *Am J Gastroenterol*, 2005, 100:432-439.
18. GUPTA R et al : A randomised clinical trial to assess the effect of total enteral and total parenteral nutritional support on metabolic, inflammatory and oxidative markers in patients with predicted severe acute pancreatitis (APACHE II > or =6), *Pancreatology,*2003, 3:668.
19. AL-OMRAN M et al: Enteral versus parenteral nutrition for acute pancreatitis (review), http://www.thecochranelibrary.com, 2008, issue 3.
20. KUMAR A et al: Early Enteral Nutrition in Severe Acute Pancreatitis: A Prospective Randomized Controlled Trial Comparing Nasojejunal and Nasogastric Routes, *J Clin Gastroenterol,* 2006, 40(5):135.
21. GRIGORAS I et al: Is intragastric administration of enteral nutrition safe in severe acute pancreatitis?*, Crit Care,* 2009, 13(suppl 1):P140.

TRANSFUSION POLICY IN CRITICALLY ILL PATIENTS

Grigoraş Ioana, MD, PhD

University of Medicine and Pharmacy "Gr.T. Popa"
Anesthesia and Intensive Care Department
University Hospital "Sf. Spiridon", Iaşi, Romania

Anemia is a common and early condition in critically ill patients. 29% patients have Hb < 10g% at ICU admission (1). 95% patients have anemia in the 3rd ICU day (2). We studied the incidence of anemia in 132 critically ill patients with long ICU stay (>7 days) and found it presence in 110 (83.33%) patients at ICU admission and in 126 (95.45%) patients at ICU discharge (3).

Causes of acquired anemia in intensive care patients are blood loss due to surgery, trauma, gastrointestinal bleeding or medical procedures (mainly phlebotomy), hemodilution, decreased red blood cell production or increased distruction and others (4). Phlebothomy may result in 30-70ml blood withdrawal/day (1,2). In critically ill patients an increase of 3,5ml/day withdrawn blood doubles the risk of transfusion (5). The amount of blood withdrawn by phlebotomy decreases over time during ICU stay, mainly due to decreased number of ABG (5).

The critical illness is frequently associated with inflammatory status. The proinflammatory cytokines (TNFα, IL-1, IL-6) result in failure of circulating EPO to appropriately increase in response to Hb reduction (relative EPO deficiency), inhibition of erythrocyte production (relative EPO resistance) and decreased iron availability (6,7,8). Corwin terms this status as *anemia of critical illness*.

Iron metabolism is severely altered in during critical illness. The main features are decreased iron availability for erythropoesis and increased iron storage (9). Markers of iron metabolism alterations during inflammation are decreased serum Fe, decreased

serum transferrin, decreased transferrin saturation and increased serum ferritin. This status is termed *functional iron deficiency* (FID) (10). Critically ill patients with FID have a longer ICU stay compared with those without FID (11).

Taking into account that severity of iron metabolism disturbances parallel the inflammatory status (12), can ferritin be used as a prognostic marker in critical illness? In ICU patients with long ICU stay higher the SOFA score, higher the maximal ferritin level and nonsurvivors have higher ferritin level compared with survivors (13). Mortality of ICU patients significantly corelates ($p<0.01$) with an increase of serum ferritin >300ng/ml during ICU stay (13).

During ICU stay Hb has a steady tendency to decrease over time. Despite the fact that Hb values on ICU admission may vary widely, after 14 days of ICU stay the Hb values tend to converge (14).

Blood transfusion is a common occurrence in ICU patients. Three landmark studies are dedicated to transfusion policy in critically ill patients: TRICC study (Canada, 838 patients, 25 ICUs, over 3 years) (15), ABC study (Europe, 3534 patients, 146 ICUs) (1) and CRIT study (SUA, 4892 patients, 284 ICUs) (2). The incidence of blood transfusion in critically ill patients is as high as about 40-65% (1,2,5). In our ICU the incidence is about 60% in patients with long ICU stay (14). In our study over 50% of transfusion are given in the first ICU day, the incidence decreases along ICU stay and a transfusional event increases Hb level with an average of 0,89g% (3). The reported indications for blood transfusion vary widely: active bleeding, low Hb level, postoperative status, need to increase oxygen delivery and others. Reported Hb transfusion trigger vary between 7-8,6g% (1,2,5,15). In our study Hb transfusion trigger was 7.83 ± 2.30g% (3). However the actual transfusion policy relies not on a specific Hb figure, but on the need to increase tissue oxygen delivery.

Transfusion policy in critically ill patients changed over time. Former recommendations encouraged blood transfusion in ICU patients with Hb <10g%. Due to several risks and side effects, mostly due to immunodisreggulation, blood transfusion was placed under scrutiny. Starting with the reported results of TRICC study, which documented that transfused ICU patients had a higher mortality compared with non-transfused patients, despite the fact that mean Hb level was much lower in this later group (15), restrictive transfusion policy was promoted and several studies were dedicated to investigate the relation between different blood transfusion policies and mortality. In our study mortality was significantly higher in ever transfused (65%) compared with never transfused (20%) critically ill patients with long ICU stay (3). Multivariate analysis identified SOFA scores and transfusion status as independent risk factors for mortality (3). Despite the fact that a lot of papers demonstrated the increased mortality rate in transfused critically ill patients, a recent paper published by JL Vincent from the European SOAP study showed no correlation between mortality and transfusion status (16).

In conclusion, when is blood transfusion indicated in ICU? The indication according to a specific Hb transfusion trigger is no longer accepted. The decision to transfuse should be made according a case-to-case judgment, which should take in account the specific features of each patient (actual status of tissue oxygen delivery and its match with oxygen consumption, coexisting diseases, risk of ischemic events, transfusion risk/benefit ratio, s.o.).

General recommendations for clinical practice: if Hb is <6g%, blood transfusion is probably indicated, but not mandatory; if Hb is >10g%, transfusion is usually contraindicated; when Hb is 6-10g%, perform an evaluation of tissue oxygenation, take into account other means to increase oxygen delivery and indicate blood transfusion only after weighting risk/benefit ratio.

Bibliography:

1. VINCENT JL et al: Anemia and Blood Transfusion in Critically Ill Pacients, JAMA, 2002, 288:1499-1507.
2. CORWIN HL et al: The CRIT Study: anemia and blood transfusion in the critically ill: current clinical practice in the United States. Crit Care Med 2004, 32:39-52.
3. GRIGORAS I et al: Transfusion policy in critically ill patients with long ICU stay, Crit Care, 2008,16(suppl 2):P235.
4. HAJJAR LA et al: Blood tranfusion in critically ill patients: state of the art, Clinics, 2007, 62(4):507-24.
5. CHANT C et al: Anemia, transfusion, and phlebotomy practices in critically ill
patients with prolonged ICU length of stay: a cohort study, Crit Care, 2006, 10:R140.
6. JELKMANN WE et al: Inhibition of erythropoietin production by cytokines. Implications for the anaemia involved in inflammatory states, Ann NY Acad Sci, 1994, 718:300–311.
7. JOHNSON CS et al: In vivo hematopoietic effects of recombinant interleukin-1 alpha in mice: stimulation of granulocytic, monocytic, megakaryocytic, and early erythroid progenitors, suppression of late-stage erythropoiesis, and reversal of erythroid suppression with erythropoietin, Blood, 1998, 73:678-683.
8. POJDA Z et al: In vivo administration of interleukin-6 delays haematopoietic regeneration in sublethally irradiated mice, Exp Hematol, 1992, 20:862-867.
9. DARVEAU M et al : Bench-to-bedside review: Iron metabolism in critically ill patients, Critical Care, 2004, 8: 356–362.
10. MEANS RT, Recent developments in the anemia of chronic disease, Curr Hematol Rep, 2003, 2:116-121.
11. PATTERIL MV et al: Functional iron deficiency, infection and systemic inflammatory response syndrome in critical illness, Anaesth Intensive Care, 2001, 29:473–478.
12. ELLIOT JM et al: Erythropoietin mimics the acute phase response in critical illness, Critical Care, 2003, R35-R40.
13. GRIGORAS I et al: Is ferritin level a predictor of mortality in critically ill patients with long ICU stay, Intensive Care Medicine, 2008, 34(suppl1):P115.
14. GRIGORAS I et al: Hemoglobin trend in critically ill patients with long ICU stay, European Journal of Anaesthesiology, 2008, 25(suppl 44):12AP4-8.
15. HERBERT PC et al: A multicenter, randomized, controlled clinical trial of transfusion requirements in critical care. Transfusion Requirements in Critical Care Investigators, Canadian Critical Care Trials Group. N Engl J Med. 1999; 340:409-417.
16. VINCENT JL et al: Are Blood Transfusions Associated with Greater Mortality Rates? Results of the Sepsis Occurrence in Acutely Ill Patients Study Anesthesiology 2008; 108:31–9.