The Art and Science of Infusion Nursing

Niels D. Martin, MD, FACS
Dane Scantling, DO, MPH

Hospital-Acquired Anemia

A Contemporary Review of Etiologies and Prevention Strategies

ABSTRACT

Advances in medicine have significantly improved lives and life spans globally. However, these practices have come with their own set of secondary consequences. Hospital-acquired anemia is one such consequence and is conferred by new medicines, operations, procedures, and tests. In this review, the authors will explore the data on this poorly considered phenomenon and discuss the etiologies, outcomes, and prevention strategies for some of the more prolific causes of hospital-acquired anemia. This study also will review the risks and benefits of treating hospital-acquired anemia.

Key words: anemia, complications, hemoglobinopathies, intensive care, traumatology

Medical technology has advanced rapidly in the past century. Invasive monitoring, complex surgeries, and the advent of advanced blood tests have all contributed a great deal to improved patient outcomes. However, with new technology comes exposure to new secondary consequences. Most contemporary attention to secondary consequences has centered on the prominent nature of nosocomial infections and drug reactions, but more occult and morbid consequences also exist. Hospital-acquired anemia, a danger of modern medical care, is one such entity.

Author Affiliations: Division of Traumatology and Surgical Critical Care, Perelman School of Medicine, University of Pennsylvania, Philadelphia, Pennsylvania (Dr Martin); Drexel University/Hahnemann University Hospital, Philadelphia, Pennsylvania (Dr Scantling).

Niels D. Martin, MD, FACS, is an assistant professor of surgery at the University of Pennsylvania's Perelman School of Medicine and the medical director of the surgical intensive care unit at the Hospital of the University of Pennsylvania.

Dane Scantling, DO, MPH, is a general surgery resident at Drexel University/Hahnemann University Hospital in Philadelphia, Pennsylvania.

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Corresponding Author: Niels D. Martin, MD, FACS, Division of Traumatology, Surgical Critical Care, and Emergency Surgery, Perelman School of Medicine at the University of Pennsylvania, 3400 Spruce St, 5 Maloney Building, Philadelphia, PA 19104 (Niels.Martin@uphs.upenn.edu).

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DEFINITION

Hospital-acquired anemia is anemia that is directly attributable to hospitalization. It is a reduction of hemoglobin during hospitalization, as compared with the hemoglobin on admission, regardless of the numeric starting point. It should be noted that no universally accepted definition of anemia exists.

Three commonly used definitions of anemia exist. The most widely used definition was developed by the World Health Organization (WHO). It is the simplest but also the most conservative. It defines anemia as a hemoglobin level of less than 13 g/dL in men and less than 12 g/dL in women[1] (Table 1). The definition, however, fails to account for gradations of anemia or the normal variations that exist across different ethnicities and ages.

With criticisms of WHO's definition in mind, Beutler and Waalen[2] developed a hemoglobin scale of anemia accounting for variances by age, gender, and race (Table 2). Differences for men were noted by both age and race, while women were differentiated by race alone. This definition is still a global definition but without gradations for severity or clinical significance of anemia.

Kosiborod et al[3] built on the WHO definition and added severity indices of mild, moderate, and severe (Table 3). Many contemporary studies of hospital-acquired anemia use Kosiborod and colleagues' definition.[4-6] Although it fails to account for age, gender, or race, it does provide a standardized means of stratifying the degree of hemoglobin changes in hospitalized patients.

TABLE 1

World Health Organization Definition of Anemia[1]

Men	< 13 g/dL
Women	< 12 g/dL

SIGNS AND SYMPTOMS

Hospital-acquired anemia is often insidious in onset. Drops in hemoglobin from many iatrogenic causes can take several days to become clinically evident, and the incremental diminishments are often written off by most clinicians. When symptoms are present, they are often vague with a slow progression of pallor, fatigue, malaise, shortness of breath, headaches, tachycardia, or dizziness. In clinical practice, especially during acute hospitalizations, most anemias do not garner clinical attention until thresholds of transfusion are approached.

MECHANISM

In general, anemia is caused in the same manner as many other bodily deficiencies; the supply of red blood cells (RBCs) is outpaced by their loss. Typically, this is caused by a profound loss of RBCs, a diminishment in the ability to replace them, increased destruction, or a decrease in normal RBC life span.[7] In a healthy adult, 15 mL of blood is produced each day, but when necessary, a maximum of 200 mL per day may be achieved in the ideal patient after sudden extreme hemorrhage.[8]

In a hospitalized patient with a number of comorbidities, the cause of hospital-acquired anemia may well be extraordinarily complex and encompass a combination of different factors, only some of which may be controllable by the health care team. Patients may be suffering from marrow suppression by inflammatory cytokines, nutritional deficiencies, medical suppression of RBC production, surgical and procedural losses, actual bleeding, and more occult losses by phlebotomy.[7] Other causes of anemia may not involve real loss of RBCs, and instead it may a result of dilutional factors. These factors will be discussed in more detail in the following sections.

TABLE 2

Beutler and Waalen Definition of Anemia Accounting for Differences in Sex, Race, and Age[2]

	Age 20-59 Years	Age ≥ 60 Years
White males	< 13.7 g/dL	< 13.2 g/dL
Black males	< 12.9 g/dL	< 12.7 g/dL
White females	< 12.2 g/dL	
Black females	< 11.5 g/dL	

TABLE 3

Expanded WHO Definition With Gradations Based on Severity[3]

Mild anemia	Men: < 13 g/dL but > 11 g/dL Women: < 12 g/dL but > 11 g/dL
Moderate anemia	9.1 to 11 g/dL
Severe anemia	< 9.1 g/dL

Abbreviation: WHO, World Health Organization.

INCIDENCE

Hospital-acquired anemia has not benefited from mainstream attention, but some work has been done secondarily in relation to the dangers of transfusion.[7] Cardiac-related hospital admissions and outcomes have lent themselves to a more simplistic study of the impact of anemia. In separate studies, Salisbury et al[4-6] examined anemia in patients admitted for acute myocardial infarction.

The first study to identify blood loss from diagnostic phlebotomy as independently predictive of hospital-acquired anemia was a 2011 study by Salisbury et al[6] of 17 676 patients from 57 hospitals; 3551 patients (20%) without anemia on admission developed moderate or severe anemia.[6] The mean phlebotomy volume was higher in patients with hospital-acquired anemia than in those without, but there was substantial variance in the amount of phlebotomy at various hospitals.[6] For each 50 mL of blood drawn, the risk of moderate to severe anemia rose by 18%, suggesting that phlebotomy volume could alter the incidence of hospital-acquired anemia.[6]

An additional publication by Salisbury et al[5] in 2011 on the 17 676 patients found that 57.5% of patients admitted for acute myocardial infarction suffered from some level of hospital-acquired anemia: 37.4% of these patients had mild hospital-acquired anemia, 15.5% had moderate hospital-acquired anemia, and 4.6% had severe hospital-acquired anemia. In all groups, including those not suffering from clinical anemia, hemoglobin declined during admission.[5] Importantly, moderate and severe hospital-acquired anemia were independently associated with higher in-hospital death when compared with no hospital-acquired anemia.[5] It was not determined whether treatment for hospital-acquired anemia would alter outcomes or whether the disease process was preventable.[5]

When looking specifically at a population with no known risk factors for anemia and a normal hemoglobin level on admission, 20% of patients requiring an intensive care unit (ICU) stay still developed anemia.[9] This finding is the closest in the literature to the incidence of purely iatrogenic hospital-acquired anemia.

RISK FACTORS

Patients with chronic anemia are at increased risk of additional hospital-acquired anemia. In a 2005 study by Nissenson et al,[10] 81 000 members of a specific health plan were reviewed for the incidence of anemia. They noted chronic anemia in 34.5% of chronic kidney disease patients, 21% of cancer patients, 18% of chronic heart disease patients, 13% of inflammatory bowel disease sufferers, 10% of those with rheumatoid arthritis, and 10% of those with infection with human immunodeficiency virus.[10] These patients may be at risk of excessive losses during their hospitalization secondary to their comorbidities, but they are also more at risk of severe anemia secondary to their lower starting point.

Generous fluid resuscitation, especially in young patients, can result in significant drops in hemoglobin concentration.[9] This type of pseudoanemia should not prompt transfusion, because equilibrium commonly will return with correction of the physiologic insult. However, actual blood loss in patients with a higher presenting hemoglobin level results in more RBCs per milliliter of blood lost and can hasten actual anemia development.[9]

Other patient factors that increase hospital-acquired anemia include nutritional deficiency, iron deficiency, and impaired systemic iron transport, as well as impaired erythropoietin production.[7] Coagulopathies and gastric bleeding caused by stress also play a role in hospital-acquired anemia (Table 4).[7]

Duration of hospitalization and severity of illness are other risk factors. These are likely as a result of the cumulative effects of daily phlebotomy and the increased likelihood of more procedures with a longer time spent in the hospital, especially when critically ill.[6,8,11-13]

TABLE 4

Causes and Respective Risk Factors of Hospital-Acquired Anemia

Cause	Risk Factors	Prevention/ Treatment
Surgical losses	Type and urgency of surgery Coagulopathies	Hemostatic agents
Bedside procedures	Critical illness Coagulopathy	Meticulous technique
Phlebotomy	Critical illness Order sets	Staff education
Hemodilution	Massive resuscitation	Staff education
Drug-induced	Antibiotics and chemotherapy	Attentive care
Coagulopathies	Shock, sepsis, chronic, iatrogenic	Early recognition and correction
Bone marrow suppression	Sepsis, drug-induced	Correction of underlying issues

SPECIFIC ETIOLOGIES OF HOSPITAL-ACQUIRED ANEMIA

Surgical Loss

Etiology

Surgery-related blood loss varies greatly according to the type of surgery, patient comorbidities, and technical factors. Surgical advances of technology and technique, including vessel-sealing energy devices and minimally invasive surgical techniques, have stemmed losses in some frequently performed operations. Many surgeries, however, still confer high amounts of blood loss. Highly invasive operations, such as those involving the liver, pancreas, or major bony structures, frequently require intraoperative or postoperative transfusions to maintain adequate hemoglobin levels.[14]

Incidence and magnitude

Surgery is the most frequent cause of a medical blood loss of 20% or more of a patient's total blood volume.[15] Such extreme losses can certainly lead not only to anemia but also to other morbidities and even death.[15]

In fact, more than half of the 20 million units of blood and blood products transfused annually are perioperative in nature.[16] Patients undergoing high-risk procedures—such as cardiac, hepatic, and certain orthopedic surgeries—are especially prone to hemorrhage.[15,16] Trauma patients are also at extreme risk for bleeding from injuries; hemorrhage is the leading cause of death in trauma patients. Surgical hemostasis is therefore paramount during all types of surgery.

Prevention

Some surgical blood losses cannot be predicted or prevented. However, diligent surgical technique concentrating on meticulous hemostasis and the use of advanced modalities, such as laparoscopy, can aid in reducing intraoperative blood losses.[17] Conscientious anesthesia care with optimal fluid and volume management also plays a strong role.[15] Careful patient selection is equally important, recognizing patient factors that may lead to coagulopathy from comorbidities or technical challenges due to body habitus. Screening for abnormal hemostasis and coagulopathies preoperatively may spare a vulnerable patient from hemorrhage and the need for rapid correction of missing factors intraoperatively.[15] Prudent discontinuation of anticoagulants preoperatively should be performed whenever possible. In patients undergoing coronary artery bypass grafts, cessation of aspirin has been linked with a lesser requirement for transfusion of blood products after surgery, and continued use of tirofiban (Aggrastat [Medicure Pharma]) and clopidogrel (Plavix [Bristol-Myers Squibb and Sanofi]) preoperatively has been linked with higher transfusion requirements.[18-21]

Correction of surgically significant coagulopathy also can be promoted pharmaceutically. Aprotinin (Bayer), aminocaproic (Amicar [Xanodyne]), and tranexamic acid (Lystenda [Ferring]), and antifibrinolytics, have been studied for their perceived ability to achieve reduced surgical blood loss and, therefore, decreased transfusion requirements.[15] Recombinant activated factor VIIa and desmopressin also have been studied for this purpose with variable results.[15]

Aprotinin, a plasmin inhibitor, has been studied especially to this end in randomized controlled trials of cardiac surgery patients.[15] Results consistently have indicated a lower operative blood loss, ranging from 50 to 1350 mL, and a reduction in the need for blood product transfusion, ranging from 1.5 times to 3 times reduction, even in patient populations predisposed to bleeding.[15,22-25]

Antifibrinolytics are less well studied but may hold some promise. In a study of 210 cardiac surgery patients, a nearly 70% reduction in RBC transfusion was noted when tranexamic acid was used, and less than half as many patients required any transfusion compared with the placebo.[26] Henry et al[27] found that the use of tranexamic acid or aprotinin saved approximately 1 unit of blood for each surgery performed.

Treatment

While patient selection and surgical technique are critical components of preventing surgical losses, a number of techniques can be employed to combat hemorrhage. Mechanical hemorrhage control remains the gold standard, but, as in prevention, pharmaceutical intervention has a role in ceasing hemorrhage. Recombinant activated factor VII has been studied with success in severe blunt trauma. In a controlled trial of 143 patients, transfusion requirements were reduced by an average of 2.6 times with a more substantial reduction in the need for massive transfusion of more than 20 units (14% versus 33% of patients).[15,28]

There are a host of topical hemostatic agents also available commercially for use during operative procedures. In general, these agents promote clot formation in the region of injury. Application techniques vary greatly, and a comprehensive understanding of application techniques is imperative to being an attentive surgeon or proceduralist.

Special populations in surgery

Surgical losses can be further mitigated with specific interventions. Much can be learned from centers that offer "bloodless" surgical techniques, aimed at upholding the religious beliefs of groups like Jehovah's Witnesses. A recent study by Konstantinidis et al[14] focused on the community of Jehovah's Witnesses and reviewed several strategies to avoid transfusion needs in operations with historically significant blood loss.

These include anesthesia protocols of maintaining a central venous pressure of less than 5 cm H_2O, clamping the porta hepatis in hepatic operations, and using cell-saver devices to return RBCs as they are suctioned out of the surgical field.[14] Conservative use of clotting factors and antifibrinolytics, as well as the use of preoperative erythropoietin, also may aid the surgeon in avoiding transfusion.[14] Finally, Konstantinidis et al[14] also discussed the concept of good surgical judgment, knowing when to abort an operation before subjecting the patient to profound risk based on real-time factors.

Bedside Procedures

Etiology

Bedside procedures include placement of central venous catheters, arterial catheters, and chest tubes; thoracentesis; paracentesis; dialysis fistula access; wound care; and

other procedures. These procedures occur with increasing frequency in the ICU and can be associated with profound and sometimes unnecessary blood loss.[9] This is in part due to their bedside nature and the tendency to downplay actual losses. Further, these procedures are often performed independently by house staff, and the importance of meticulous technique may not always be realized. This includes clean access without wasted motions that allow for catheter bleeding or excessive back bleeding from a newly placed catheter. Often, losses are not recognized because the bleeding occurs into the bed sheets and direct quantitation may not be possible.

A secondary consequence of central venous access or arterial access is the ease of subsequent phlebotomy. The catheters can be accessed and can prevent the need for a separate needlestick. Practitioners must be cognizant of not using these catheters to draw more blood tests than they otherwise would under similar circumstances.[29] In one study, Low et al[29] noted a 33% increase in blood testing and a 44% increase in blood loss in the ICU setting when patients had a central or an arterial catheter versus peripheral access only.

Prevention and treatment

The most potent antidote for blood loss resulting from bedside procedures and invasive monitoring is a cautious approach to their use. If such interventions prove necessary, they should be removed as soon as they are no longer needed. In addition, catheters that are placed should be used only for the purpose for which they were placed initially.[29] Curiosity and excessive medical care can be dangerous to susceptible patients.

Blood Draws

Etiology

Blood sampling for laboratory testing is the greatest contributor to hospital-acquired anemia; this is especially true for critically ill patients in the ICU. A single milliliter of phlebotomy can decrease hemoglobin by an average of 0.007 g/dL and blood cultures an average of 0.14 g/dL.[13] Clinically significant losses are believed to be in the range of 0.66 to 1.0 g/dL, or only 100 mL of phlebotomy.[13] This can be accomplished within only 5 days on average of basic lab testing each day.[13] Blood loss may be in excess of 40 to 70 mL per patient day in the ICU for lab work alone and is highest in the first 48 hours of admission.[6,8,11,12] The presence of central venous access further perpetuates unnecessary blood testing secondary to ease of phlebotomy.

Healthy individuals produce 15 mL of red cells per day with a maximum of 200 mL per day.[8] It is likely that critically ill patients cannot match such an output for a variety of reasons. As a direct result, development of hospital-acquired anemia has been independently correlated with the summative amount of blood drawn during admission.[6]

Incidence and magnitude

Salisbury et al[6] found that for every 50 mL of diagnostic phlebotomy performed in the hospitalized patient, the risk of moderate to severe anemia rose by 18%. In those patients sustaining moderate to severe hospital-acquired anemia, approximately 100 mL more blood was drawn during the admission on average compared with those who did not develop anemia.[6]

While normal iron intake in a typical diet is 1 to 2 mg/d, routine phlebotomy alone in the ICU can extract 64 mg of iron in a single day.[11] This deficit is even more pronounced in patients not taking a normal oral diet.

Prevention

An effort should be made to be aware of the often slow and occult blood loss suffered by patients through blood draws and procedures. Efforts to reduce blood losses from phlebotomy must focus on minimizing unnecessary testing. Protocols for acceptable indications for specific tests, avoidance of order sets, staff education, integrated prompts, and reminders incorporated into the electronic medical documentation system should all be employed to reduce unnecessary testing.[30]

Point-of-care hemoglobin monitoring has been found to be safe and accurate while still diminishing the volume of blood drawn and thus avoiding transfusions.[7,8]

Historically, when sampling from a catheter, the initial aspirate was discarded because it was not representative of the circulating blood. This led to rapidly diminishing hemoglobin levels. In contemporary practice, after both the initial pull and the actual lab sample are drawn, the initial pull should then be returned to the patient. There are even closed commercial vamp systems that allow for this in an unintruded way and have been shown to reduce transfusion needs and in-hospital mortality.[7]

When blood is drawn by traditional methods, several studies have shown that using pediatric blood sample tubes decreases the total volume of blood drawn and may contribute to reducing the need for eventual development of hospital-acquired anemia.[6] In a study by Smoller et al[30] in 1989, average phlebotomy with adult blood tube sets in the ICU was 226.1 mL, or 55.6 mL/d. The same testing conducted with pediatric blood tube sets averaged 120.2 mL total or 32.2 mL/d, a decrease in blood loss of nearly 50%.

Finally, if noninvasive testing such as end-tidal CO_2, pulse oximetry, and other oximeters are capable of

inferred hemoglobin monitoring, they should be employed instead of blood testing wherever possible.

Hemodilution

Etiology and magnitude

Unlike the other etiologies in this review, hemodilution does not actually involve a true loss of RBCs. Instead, hemodilution is the dilution of available RBCs and typically is the result of substantial fluid resuscitation.[31] Importantly, the measured hemoglobin levels are not indicative of true anemia, and so should not be treated as such. However, it is imperative to rule out actual bleeding, as hemodilution is a diagnosis of exclusion. Following hemodilution, the body ultimately will return to equilibrium with removal of the initial stressor. However, when taken to extremes, hemodilution can compromise oxygen delivery.

Risk factors

Aggressive fluid resuscitation is necessary at times. However, hemodilution can occur if there is no clear understanding of the patient's intravascular volume status and volume infusion continues beyond euvolemia. Patients with severe trauma, hypovolemic shock, or sepsis are at particular risk for over-resuscitation and hemodilution.[31] Postoperative patients also are at high risk because of large volumes given based on presumed intraoperative evaporative losses. An inability to accurately assess volume status is not uncommon, as routine volume-status markers—such as heart rate, blood pressure, central venous pressure, pulmonary capillary wedge pressure, and physical findings—all have limitations and individually are only reliable about 50% of the time, compared with the patient's hemoglobin status.[31]

Prevention and treatment

Although fluid resuscitation will remain the cornerstone of medical treatment for many maladies, provider recognition of its consequences is also vitally important. Careful assessment of volume status must be employed to avoid overly aggressive fluid resuscitation and thus dilution of red cell concentration resulting in a lower measured hemoglobin level. Further, if over-resuscitation does occur, recognizing it is even more important because overzealous correction with blood products can lead to further morbidities.

Fortunately, recent surgical practice patterns have resulted in restrictive intravenous fluid resuscitation regimens that have statistically significantly reduced postoperative complications.[32] Statistically significant reductions in cardiopulmonary and tissue healing complications also have been noted.[32]

Drug-Induced Anemia

Etiology

Bone marrow suppression can occur as a complication of drugs that are often given in an inpatient setting. In particular, certain chemotherapeutics and antibiotics such as nitrofurantoin (Macrobid [Almatica Pharma]), phenazopyridine (Pyridium [Actavis]), primaquine, and sulfa drugs can cause a non–immune-related decrease in red cell production.[7] Cephalosporins, beta lactam antibiotics, nonsteroidal anti-inflammatory drugs, chemotherapeutics, methyldopa, and quinine/quinidine can also cause hemolytic anemia through immunologic mediation in select patients.[7]

Prevention

Avoidance of commonly associated medications should be undertaken whenever possible, especially when common culprit drugs such as piperacillin, cefotetan, and ceftriaxone (Rocephin [Genentech]) are used.[7]

Treatment

Early recognition is paramount. Contributory medications should be rapidly discontinued. In the setting of drug-independent hemolytic anemia, corticosteroids may have a role in treatment.[7]

Coagulopathic Populations

Etiology

Hospitalized patients frequently suffer from some sort of acute coagulopathy during their course. In addition, patients with inherited coagulopathies, such as von Willebrand's disease and hemophilia, may also find themselves hospitalized and at risk of an acute exacerbation of their disease.

Patients with sepsis, major trauma, disseminated intravascular coagulation, hepatic disease, viral infections, and splenic dysfunctions are also at particular risk. Cancer patients are also prone, as are those on medications such as aspirin, warfarin, clopidogrel, and other anticoagulants.[7,33]

Magnitude

Because of the vast contributing etiologies of coagulopathy in hospitalized patients, risk is significant. Up to 45% of patients in an intensive care setting suffer from thrombocytopenia, and a full half of severe sepsis patients will experience a clinically significant coagulopathy.[34] Hemodilution, blood loss, platelet consumption, and marrow disease can all exacerbate the effects of coagulopathy.

Prevention

Prevention of anemia due to coagulopathy often involves identifying and, whenever possible, alleviating the cause of the underlying coagulopathy. Events inciting bleeding should be avoided until corrective measures have been instituted.

Bone Marrow Suppression

Etiology

Under normal conditions, endogenous erythropoietin stimulates bone marrow to produce RBCs. Critically ill patients, as well as those with renal disease, have diminished levels of circulating erythropoietin. These patients also suffer from a diminished responsiveness to erythropoietin. Lower iron levels, common in such patients, further diminish the marrow's ability to produce RBCs even in the face of acute anemia. This is further exacerbated by medications, low B12 levels, and inadequate folate intake. While piperacillin, cefotetan, and ceftriaxone act to induce hemolysis, angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, calcium channel blockers, theophylline, and beta blockers may suppress renal release of erythropoietin, impairing marrow function further.[35,36]

Prevention

Prevention of bone marrow suppression may prove difficult. Proper nutrition and avoidance of inciting drugs are crucial. All efforts to avoid blood loss in the first place should be undertaken.

Treatment

Although erythropoietin supplementation would be a logical choice to manage low erythropoietin levels, it is a controversial treatment modality. Benefits remain questionable, and risks for supplementation include thrombotic complications.[36] Careful review of the risks and benefits on a case-by-case basis should be undertaken.

GENERAL TREATMENT STRATEGIES FOR HOSPITAL-ACQUIRED ANEMIA

Although the most obvious and rapid response to an insufficient hemoglobin level is to offer transfusion of red cells, it is a treatment modality that recent research increasingly has associated with negative patient outcomes and complications. About 30% of all ICU patients receive a transfusion during their stay, and more than 60% receive one if they spent a week or more in intensive care.[7]

Transfusions are not only expensive, they also contribute to early mortality and even longer hospital stays.[37,38] Transfusions have been associated with as much as a 40% increase in 30-day mortality and with up to 67% mortality in 6 months following hospital discharge.[37] Moreover, a study by Marik and Corwin[38] found that of 45 cohort studies involving RBC transfusion, 42 studies (consisting of almost 300 000 patients) showed a negative risk-to-benefit ratio. RBC transfusions were even an independent predictor of death in 17 of 18 studies examining mortality and an independent predictor of nosocomial infection in every study with it as an outcome.[38] In particular, RBC transfusions have been associated with multiple organ dysfunction syndrome, adult respiratory distress syndrome, transfusion-related lung injury, transfusion-related circulatory overload, and transfusion-related immunomodulation.[38]

The risks of viral and bacterial transmission and immune reactions in patients receiving blood products are more well known.[18] Cancer patients receiving transfusions at the time of surgical resection endure a higher recurrence of cancer and higher death rates from the disease, although this is variable based on cancer and patient characteristics.[14,39]

Defining a cutoff for when transfusion becomes necessary is important in balancing the risks and benefits of such treatment. In the Transfusion Requirements in Critical Care trial, 838 normovolemic patients who were not actively bleeding and had not had cardiac surgery were studied.[40] Approximately half received a transfusion when their hemoglobin was less than 7.0 g/dL.[40] The other half received a transfusion when their hemoglobin dropped below 10 g/dL. In-hospital mortality was 22% versus 28%, respectively, showing statistically significant improved outcomes with the restrictive strategy.[40]

In the Transfusion Requirements after Cardiac Surgery trial, liberal and conservative transfusion strategies were also compared.[41] Transfusions were given either at a hematocrit of less than or equal to 30% (liberal) or less than or equal to 24% (restrictive). As a noninferiority study, no differences were found in terms of 30-day all-cause mortality or severe morbidity when transfusions were restricted.[41]

In a similar study performed on critically ill children, a restrictive transfusion strategy was again compared with a liberal one.[42] In the restrictive strategy, 7 g/dL was again used as a transfusion trigger with a goal of 8.5 to 9.5 g/dL. In the liberal strategy, 9.5 g/dL was a trigger for transfusion, and a target of 11 to 12 g/dL was achieved. Patients in the restrictive transfusion strategy group safely received 44% fewer transfusions but had no difference in their rate of multiple organ dysfunction or mortality.[42]

As a result of these studies, it has become the standard to transfuse very conservatively in critically ill patients.[40-42]

Although it has repeatedly been shown that restrictive transfusion strategies can be employed to lessen the morbidity associated with blood transfusion without exposing patients to increased morbidity and mortality, some situations may lend themselves to more liberal transfusion strategy.[36] These include acute myocardial ischemia, difficulty with ventilator weaning, respiratory muscle weakness or high minute ventilation, and the early phases of septic shock where the volume status by central venous pressure and blood pressure are normal, but oxygen delivery by mixed venous gas is low.[36]

INFUSION NURSE'S ROLE

Infusion nurses are the first-line care providers for patients at risk for and suffering from hospital-acquired anemia. It is their duty to be aware of the risks for inducing hospital-acquired anemia and to be a vocal patient advocate. As a hands-on member of the care team, infusion nurses are the most likely to first recognize excessive blood loss from medical processes. Likewise, they hold a duty to educate staff and all members of the care team about hospital-acquired anemia whenever relevant. Posters, unit-based initiatives, and other educational outreach endeavors can help alleviate the burden of hospital-acquired anemia.

SUMMARY AND CONCLUSION

Hospital-acquired anemia is an established, documented, and potentially deadly complication of medical care that was not well studied until recently. Exacerbated by the same advanced tests and procedures that so often are credited for saving countless lives, the severity of hospital-acquired anemia is just now beginning to be studied and understood. Caregivers must recognize the magnitude and importance of this morbidity and strive to find the appropriate balance between prevention and the need for various medical interventions with the idea that we may often do substantially more harm than good. Clearly, while the timing, risks, and benefits of treatment for the various causes of hospital-acquired anemia can sometimes be in doubt, a keen understanding of the etiology of this complication and a consistent attention to its prevention, whenever possible, is absolutely essential. It is the responsibility for all members of the medical team to be aware of the risk factors for hospital-acquired anemia and to work dutifully to avoid the morbidity and mortality of both the anemia itself and its treatment.

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