Therapeutic Phlebotomy
A Review of Diagnoses and Treatment Considerations

ABSTRACT

In the United States, phlebotomies are most often performed for reinfusion of blood to a designated or nondesignated recipient at a later time. These are known, respectively, as autologous or allogenic donations. For a few rare blood disorders, however, phlebotomy is performed as a medical intervention for disease management. These are referred to as therapeutic phlebotomies. Four classifications of blood disorders are discussed here for which symptoms and complications can be managed by the use of therapeutic phlebotomy. The article also offers considerations for setting up a therapeutic phlebotomy program.

ORIGINS OF THERAPEUTIC PHLEBOTOMY

The removal of blood for therapeutic purposes has been practiced for several thousand years and frequently is referred to as bloodletting. The ancient Egyptians, circa 1000 BC, believed that periodic self-sacrifice of blood maintained a strong spirituality and alleviated the body of all ills.1 Citizens were encouraged to practice bloodletting several times each month—taking care not to cause severe scarring since self-mutilation was frowned on. The Mayans, circa 1500 BC, also encouraged citizens to perform bloodletting privately in penitence for egregious sins. In addition, bloodletting on the royal Mayan family was done to prove their lineage to the gods. Important occasions, such as births, deaths, marriages, and the dedication of new buildings, were celebrated by public bloodletting of the queen and the king. To demonstrate their strength, the puncturing or cutting was done on sensitive body parts—the tongue of the queen and the penis of the king.2

It was not until the fifth century BC that the medical benefit of bloodletting was perceived. Although Hippocrates, circa 400 BC, is attributed with recognizing the potential benefits, it was an additional 200 years before bloodletting as a medical practice became widespread. The rise of the practice was led by Galen, a physician and follower of Hippocrates, who was a great orator of strong persuasion. He developed and touted the theory of the 4 humors—blood, phlegm, yellow bile, and black bile—and suggested that good health resulted when the humors were in perfect alignment with one another.3 An imbalance of any one of the humors was considered the cause of illness but could be corrected by bleeding and/or purging. Over the centuries, his philosophy was altered to incorporate new medical understanding, but the overall theory of the 4 humors was a basis for medical practice for 2000 years. Crude cutting instruments were devised and “enhanced” through the centuries,4 and the uses of cupping to prolong bleeding and leeches as an alternative to cutting waxed and waned in popularity.2 Throughout the 17th and 18th centuries, bloodletting reached the height of use. In 1789, George Washington was treated with bloodletting for a sore throat. After being bled of 9 pints in 24 hours, the Father of Our Country succumbed to the effects of anemia and dehydration.5,6

With the discovery of the circulatory system by Harvey in the early 1600s,7 the practice of bloodletting began to incorporate the use of venesection, describing the practice of removing blood directly from the vein. By the mid-1800s, the indiscriminate use of bleeding to treat all afflictions, large or small, was frowned on by the...
emerging medical society and deemed quackery. The social use of bleeding started to fade, but selected medical conditions were recognized as benefiting by the practice. By the turn of the century, about 35 conditions were recognized that were medically appropriate for blood removal.¹ Today, this number has dwindled down to a handful of rare blood disorders.

### DISORDERS BENEFITING FROM THERAPEUTIC PHLEBOTOMY

Therapeutic phlebotomy is the treatment of choice for blood disorders in which the removal of red blood cells or serum iron is the most efficient method of managing disease symptoms and complications. Few diseases are appropriate for such treatment, and generally, the need for phlebotomy can be predetermined and prearranged. Most nurses will rarely or never participate in the therapy. Phlebotomy patients may require services unexpectedly, however. Therefore, it is beneficial for all practitioners to familiarize themselves with these disorders.

**Polycythemia**

Polycythemia, which means “many cells in the blood,” is the direct antonym of anemia, which means “absence or lack of blood.” Polycythemia is a symptom, not a disease, and it is characteristic of several blood disorders. The term most commonly refers to a perceived or actual increase in red blood cell count; in certain conditions, an elevation in white blood cell and platelet counts may also be noted.

An elevated hematocrit level is generally the first indication of possible polycythemia. The determining factor is whether erythrocytosis (increased manufacturing of red blood cells) is responsible for the increased hematocrit level or whether it is in relation to a decrease in plasma. If erythrocytosis is confirmed, the diagnosis is categorized as **absolute polycythemia**. The condition can then further be classified as **polycythemia vera**, or **secondary polycythemia**.² If erythrocytosis is not confirmed, the diagnosis is categorized as **apparent polycythemia**.

Apparent or relative polycythemia is a pathological condition in which the red blood cell count is within expected parameters and the cells themselves may be normocytic (ie, the size and shape of normal mature red blood cells). The increased hematocrit level is due to diminished fluid volume. Conditions associated with apparent polycythemia include, but are not limited to, dehydration, burns, stress, and some hypertensive conditions.³ Erythropoietin (the hormone that stimulates red blood cell production) levels are most often normal, indicating that the increased hematocrit level is not associated with the body’s attempt to increase red blood cell production. The hematocrit corrects itself as plasma levels return to normal. Patients with apparent polycythemia are not candidates for phlebotomy.

Secondary polycythemia, by comparison, refers to any disorder associated with an increase in the serum erythropoietin level in which the underlying cause of the erythrocytosis can be determined. Erythrocyte production is stimulated when erythropoietin is produced in response to the body’s perceived need for oxygen. In conditions associated with secondary polycythemia, oxygen exchange to the tissues is poor. The kidneys receive a message that additional red blood cells are needed to enhance oxygen availability. The body goes into overdrive to manufacture additional red blood cells; therefore, serum erythropoietin levels may be elevated. This condition is common among those who spend time at high altitudes, are heavy smokers, or suffer from severe lung disorders or heart failure.⁴

Altitude-induced erythrocytosis is generally self-limiting and does not present a problem as long as a period of acclimatization to the higher altitude is provided. The need for additional erythrocytes correlates with the barometric pressure in the atmosphere. Although oxygen saturation of the air remains at 21% even at high altitudes, the barometric pressure is lower, decreasing the rate of oxygen exchange into the tissues.⁵ Red blood cell mass is increased to enhance oxygen exchange. Phlebotomy for cellular reduction is not required or generally desired.

Smoking-related polycythemia, sometimes referred to as *smoker’s disease*, is an ongoing process. The alveoli are unable to provide the oxygen exchange required for red blood cell saturation. As tissue perfusion diminishes, the body attempts to compensate by increasing red blood cell mass.⁶ Erythropoietin levels are elevated to encourage red blood cell production. As oxygenation remains low, the process of erythrocytosis continues until, eventually, blood viscosity becomes an impeding factor; the reduction of red blood cell mass is required to improve oxygenation.

This same process occurs with severe lung disease. In heart failure, the mechanism that causes erythrocytosis is related to cardiac output, which may be too low to allow for proper oxygenation. Erythrocytes proliferate in an attempt to provide the needed oxygen and, as with smoker’s disease, hyperviscosity begins to impede oxygenation, requiring red cell mass reduction.

Polycythemia vera, also referred to as **polycythemia rubra vera**, is a primary blood disease. It is a myeloproliferative disorder in which erythrocytosis is of unknown origin. In the United States, it is estimated that 0.6 to 1.6 persons per million suffer from this condition.⁷ Most often, all blood cell levels (erythrocytes, leukocytes, and platelets) are elevated. The increased platelet count places the patient at risk for thrombotic episodes. The leukocyte count is also elevated with neutrophils being the predominant circulating white blood cell. The disease progresses through several
stages, beginning innocuously but terminating as acute leukemia. White blood cell production overrides red blood cells in end-stage disease, with anemia occurring either secondary to therapy or because of bone marrow exhaustion from red blood cell overproduction.

The mechanism driving cellular growth is not related to a perceived need for additional oxygen; indeed, it does not seem to be driven by any force. Erythropoietin levels remain normal or slightly subnormal. Pluripotential stem cells that normally determine the direction of blood cell development undergo an unexplained transition. The altered stem cells respond inappropriately to cytokines other than erythropoietin and other blood cell poietins and generate cells in a noncontrolled manner. Common with all neoplastic disorders, cell proliferation provides no advantage to the body. The actual process of red blood cell progenesis is fairly slow; essentially, 1 additional unit of blood is produced every 2 to 3 months.

Phlebotomy is the treatment of choice for most conditions of absolute polycythemia, although the treatment goals are different for polycythemia vera and secondary polycythemia. In all cases, no improvement of the disease state is anticipated, although phlebotomy may reduce symptoms. Patients in respiratory distress may experience improved breathing by reducing the sluggishness of the blood and/or relieving fluid overload. Frequency of phlebotomy for these patients is determined by oxygen saturation and hematocrit levels.

Not all conditions resulting in secondary polycythemia are appropriate for treatment with phlebotomy. As previously stated, altitude-induced polycythemia does not generally require red blood cell reduction. The use of phlebotomy for the treatment of cor pulmonale is also controversial. Cor pulmonale is a lung disorder that results in right ventricular enlargement. The use of phlebotomy to reduce hypoxia has been suggested as therapy, but the reduction of oxygen-carrying red blood cells may have a negative effect. Therapeutic phlebotomy for this and other heart-associated polycythemias is sometimes considered, however, and is covered by many insurance companies.

In polycythemia vera, the overall goal is the reduction of red blood cells and serum iron, although the therapeutic reduction of white blood cells and platelets may be beneficial. By keeping iron levels low, red blood cells that form tend to be smaller and, therefore, less invasive as they multiply. Iron stores may be nearly depleted by phlebotomy and the repletion of iron is rec-

Hemochromatosis

Iron is essential to most living organisms and plays a major role in cellular functions; however, excessive amounts of iron can be toxic, leading to multiple organ failure and death. The toxicity of iron comes from its propensity to form oxygen radicals that damage cells. When iron overload occurs, the body has no way of intentionally modifying excretion to increase removal. Excretion occurs naturally, but only to a small degree, through sloughing of the cells of the interstitial lining and skin cells, through sweat, and from the urinary tract. Women lose additional iron as a result of menstruation and pregnancy.

Iron levels are controlled more by absorption than through excretion. A small amount is absorbed directly from the stomach; higher amounts are absorbed from the duodenum or jejunum. Although the intestines can generally regulate absorption, increased dietary iron may result in iron overload. Parenteral administration or transfusion of red blood cells may also lead to iron excess.

When iron enters the system, it is initially stored intracellularly and intramucosally as free iron. This unused iron forms protein complexes known as ferritin that act as storage tanks. Iron is mobilized from ferritin into cells by the protein transferrin. When iron-rich cells, such as red blood cells, die, the newly freed iron forms an insoluble protein complex known as hemosiderin, which is stored in tissues, especially in the liver. As long as it is available, hemosiderin is the source of iron used when new cells are manufactured, again using transferrin as the mobilizer. At any one time, 65% of the body’s iron stores are bound to hemoglobin, 4% is bound to myoglobin (muscle heme), and 30% is stored as ferritin or hemosiderin in the spleen, bone marrow, and liver. The remaining 1% is distributed throughout various tissues.

The normal value of serum iron in adult men is 65 to 177 μg/dL and is slightly less for women. The measurement of serum iron indicates the amount of iron that is immediately available for cellular uptake, that is, the amount that is bound to transferrin. Total iron-binding capacity provides an indication of the maximum amount of iron that transferrin is able to carry. Transferrin can accommodate up to 200 to 450 μg/dL of iron but generally carries only about 20% to 55% of this maximum capacity. This percentage is termed iron saturation. Serum ferritin levels provide the best indirect measurement of total body iron stores and usually are between 15 to 200 ng/mL. Hemosiderin levels are
not tested by blood examination because the complex is found within cells only.19

When the amount of iron stored as hemosiderin becomes excessive, a condition known as hemosiderosis is diagnosed. Hemosiderosis is a nonpathological condition; it does not cause disease. The condition may be symptomatic of disease, however. Hemosiderosis occurs when iron stores are higher than normal and iron is deposited excessively in tissues.

**Hemochromatosis** refers to an inherited disease state in which iron absorption from the duodenum is not regulated, resulting in higher than desirable serum iron levels. Hemochromatosis is a characteristic symptom of hemochromatosis. There are 4 variations of hemochromatosis, each of which is related to an alteration of a specific cytokine required to regulate iron uptake. These are distinguished by age at onset, genetic cause, and various factors of inheritance.20

As iron progressively builds up in tissues and organs, organ dysfunction and failure occurs, notably affecting the heart, liver, and endocrine portion of the pancreas. Although some types of hemochromatosis are asymptomatic, complications of iron saturation may include arthritis, diabetes, liver cirrhosis, heart arrhythmias and failure, and an increase in skin pigmentation termed *bronzing*. Laboratory values indicate that iron saturation is approaching 100%, meaning that there is no transferrin available to mobilize unused iron. Consequently, serum ferritin levels also rise.17

The estimated prevalence of hereditary hemochromatosis is 1 in 200 persons to 1 in 250 persons in the general population, making it one of the most common genetic disorders.20 The disorder was first recognized in 1889, but no known therapy was available until 1952, when Davis and Arrowsmith21 reported the success of therapeutic phlebotomy to reduce the iron load. Today, phlebotomy is the most common treatment of iron reduction in this disorder. Initially, 1 unit of blood (usually 500 mL) is removed weekly until iron stores have been reduced and a state of hypoferritinemia exists. Time between treatments is then dependent on laboratory values; treatment is usually performed 4 to 6 times annually to keep serum ferritin levels between 25 and 50 ng/mL and saturation below 50%. Interestingly, despite the morbidity associated with hemochromatosis, a 2002 study indicated that patient compliance with therapy diminishes over time.22

**Porphyria**

Porphyria is a generic term referring to a group of 7 diseases related to the disruption of heme biosynthesis. Heme is the iron-carrying portion of hemoglobin. Porphyria is an organic compound that is essential to the development of heme.

Porphyria is synthesized in nature by all living matter and is present in virtually every tissue in the human body.

A significant trait is its ability to bind to metal; metal-bound porphyrins are also referred to as metalloporphyrins. In the human body, synthesized porphyrin binds with iron to form the compound ferroporphyrin, otherwise known as heme, and with cobalt to form vitamin B12. Unbound, it is present in minute amounts in many cells, but the “free” form serves no biological purpose.23

The development of heme follows a precise pathway.24 Porphyrin enters the developing heme molecule in stage 2 of this pathway and binds with iron in the final stage. During the 8-stage process, the porphyrin molecule is altered by the presence of enzymes. These stage-specific enzymes, secreted from either the liver or the bone marrow, are required for each stage of the pathway to progress (Table 1). An absence or deficiency of any one of these enzymes causes the heme pathway to be disrupted. The initial enzyme in the pathway, δ-aminolevulinic acid synthase, does not involve porphyrin; the resulting disease is termed *sideroblastic anemia*. The remaining 7 enzymes cause progressive changes of a porphyrin compound; associated diseases are categorized as porphyria. As the body perceives the decrease in heme production, it begins to overproduce the precursor porphyrin molecule in an attempt to advance heme biosynthesis. This results in overaccumulation of that particular porphyrin molecule. Each porphyria has unique symptoms that are related to the increased production of the precursor molecule. The severity of these symptoms varies even within the same classification.25

Porphyrias are classified as acute or nonacute according to the site of synthesis of the deficient enzyme. Four of the enzymes are synthesized within the liver; 3 are synthesized within the bone marrow. When the liver is the origin of the triggering enzyme, attacks tend to be acute, with periods of total remission. Those porphyrias arising from erythropoietic enzymes have no particular pattern of remission and are therefore considered chronic.

Erythropoietic porphyrias are also known as *cutaneous* because a common feature is the development of skin lesions with exposure to sunlight. Some acute (hepatic) porphyrias also exhibit this trait. The excessive porphyrins in the body are deposited in all cells; the amount varies with the disease type. When the porphyrin is photoactivated, it generates free radicals in the presence of sunlight. These oxygen-free radicals cause oxidization, which compromises cellular integrity. Extremely sensitive patients may develop severe lesions after even minimal exposure to sunlight as cells are destroyed. At times, this destruction may be severe enough to trigger a fatal reaction.

Because porphyrins are excreted in the urine, an interesting manifestation of some of the porphyrias is that collected urine left standing in sunlight will turn red. This is a characteristic of the pigment by which porphyrin gets its name (Greek for purple pigment). Increased hair growth (hypertrichosis) is also a symptom.
of the porphyrias, leading to the appearance of fine hairs over the entire face, including the forehead and on the extremities. It is likely that legend of werewolves developed as a result of this characteristic. Diet control is important for some types of porphyria. The need to avoid foods high in sulfur, such as garlic, and the need to avoid sunlight helped develop the folklore of vampires. Porphyria has been deemed the disease of royalty because of hypothetical “proof” that King George III and Mary Queen of Scots both were sufferers. Porphyria is associated with so many nonspecific symptoms, however, that these retrospective diagnoses are not respected by all researchers.

Porphyria cutanea tarda is caused by a deficiency of the fifth enzyme in heme production, uroporphyrinogen decarboxylase. Because this is a hepatic enzyme, the disease is classified as acute. Characteristic of acute porphyria, the disease is manifested by seizures and psychosis. The severe back and abdominal pain associated with other acute porphyrias is uncommon in this disease, however. This is the only acute (hepatic) porphyria that shares the cutaneous manifestation of the erythropoietic porphyrias; skin blistering is a significant feature of the disease. Full-thickness lesions may lead to scarring with lifelong disfigurement. While porphyria is most commonly a genetic disorder, porphyria cutanea tarda is unique because it may develop in response to certain stimuli without genetic predisposition. Risk factors for disease development include excessive hepatic iron, alcohol consumption, hepatitis C infection, estrogen administration, human immunodeficiency virus infection, and induction of cytochrome P450 enzymes such as are typically seen in smokers. These triggers may also exacerbate symptoms of patients with diagnosed disease.

In porphyria cutanea tarda, for reasons that are not clear, the production of the enzyme uroporphyrinogen decarboxylase is inhibited by body iron, even if iron stores are normal. The preferred therapy is to remove iron by phlebotomy until the enzyme activity is able to resume. Chelation therapy with deferoxamine will achieve the same results, but the expense makes it undesirable as the treatment of choice.

**PROCEDURAL CONSIDERATIONS FOR PHLEBOTOMY**

Phlebotomies fall into 3 categories. Allogenic phlebotomy, the most commonly performed, refers to blood drawn through a public or private donor program (such as the American Red Cross) for storage in hospital blood banks. These programs are credentialed by the AABB (formerly the American Association of Blood Banks), which sets forth stringent guidelines for the donation and storage process. The donor is not the designated recipient in these volunteer donor programs.

Autologous phlebotomy refers to a medical program in which the donor is always the designated recipient. AABB guidelines are followed to ensure patient safety and appropriate storage; however, no predonation screening is required.

Therapeutic phlebotomy refers to a medical intervention in which blood is drawn for the therapeutic reduction
of iron or blood cells. Although AABB guidelines for patient safety are followed, no special treatment of the blood pre- or postwithdrawal is required except to ensure proper disposal.

With many of the disorders that have been discussed, therapeutic phlebotomy not only provides symptom relief but also reduces the potential organ damage caused by high serum iron levels. Phlebotomies for these patients are most commonly performed at the physician’s office or an outpatient clinic and incur some expense to the patient. Depending on the underlying disorder, some patients may be eligible to donate through a volunteer donor program. This not only decreases costs to the patient but also produces a unit that can cross over for public use. Patients with smoker’s disease, which is classified as a secondary polycythemia, and some patients with hemachromatosis may be eligible for this consideration. Patients with other disorders are often not eligible for allogenic donation, because of the disease itself, medications being used for the treatment, or instability of the patient at the time of blood withdrawal.

Before being admitted to the volunteer donor program, a patient should be evaluated by a physician to ensure appropriateness of the decision. Only blood that has been drawn from a Red Cross volunteer donor can be considered for the public blood supply. Blood that is drawn at any other site cannot be used by a blood bank because (1) the donor does not qualify as a volunteer and (2) the process of collection lacks the required quality control.

When setting up a therapeutic phlebotomy program, it is strongly recommended that the facility outline policies and procedures to address patient safety, withdrawal technique, and blood disposal. The AABB sets forth practice criteria as it relates to blood banking, but this information may be useful when developing a therapeutic phlebotomy program.

Another useful document is the Infusion Nurses Society’s (INS) Infusion Nursing Standards of Practice (hereafter referred to as “Standards”), which addresses phlebotomy in Standard 66, Practice Criteria III. Staff education should include, but not be limited to, potential adverse reactions, information on infection control, vascular access options, venipuncture technique, and homeostasis of the patient. Personnel should also be proficient in basic cardiopulmonary resuscitation. Patient education and consent for treatment are emphasized by the INS Standards. Patient education should include a review of the potential symptoms for which the patient should alert the nurse during or immediately after blood removal.

Therapeutic phlebotomy is a medical intervention and requires a physician’s order. Proper orders will include the frequency of phlebotomy and the amount of blood to withdraw. An order to “draw 1 unit of blood” is not appropriate because the term unit is arbitrary with no defined amount. If parameters for laboratory values are ordered, laboratory results need to be available before starting the phlebotomy. Fluids may be ordered for patients in compromised states who may not tolerate the rapid loss of plasma. Fluid orders should include the type, amount, and rate of infusion. Orders to “administer fluid as a bolus” are not appropriate because selecting a rate makes the nurse an unauthorized prescriber. Orders may also include the timing of fluid administration: prior to phlebotomy, at completion, or during a midpoint.

Unless otherwise ordered or specified by practice criteria, the rate of blood removal and selection of equipment are at the discretion of the practitioner. Phlebotomy through peripherally inserted catheters, midlines, tunneled catheters, and implanted ports is discouraged largely because of the risk of clotting or otherwise damaging the catheter. If a central catheter must be used for blood withdrawal, the catheter should be flushed periodically to maintain patency and a fresh injection cap or dead-ender applied at the completion of therapy.

Patients receiving multiple phlebotomies may benefit from placement of an apheresis catheter; otherwise, peripheral access is preferred. The practitioner may determine the appropriate catheter size and venipuncture location for peripheral draws. It is not necessary to limit vascular access to the medial cubital vein, nor is a 16-gauge needle appropriate for all patients. Adequate blood flows can be attained through 20- or 18-gauge catheters, and some practitioners successfully use 22-gauge catheters. Guidelines for general venipuncture technique should be observed.

One caveat to blood removal is that the rate flow cannot be adjusted if blood needs to be removed incrementally to improve tolerance. It may be necessary to stop the procedure and resume with fresh equipment as the patient’s tolerance permits.

The amount of blood withdrawn may be estimated by gradual increments indicated on the collection bag or, for more accurate assessment, a scale may be used. The use of syringes may also be considered to increase accuracy. Use of vacuum bottles to facilitate blood flow is controversial. Potentially fatal air embolisms have been detected when blood has been evacuated using suction; however, many phlebotomy sites are using bottles without this repercussion. The most common problem cited in the literature is that the suction collapses the vein, decreasing the rate of flow. There is no literature to support or reject the use of Vacutainer® tubes to withdraw blood; however, expense may be a consideration because up to 40 to 60 tubes may be needed for a therapeutic draw.

The same equipment used for volunteer donation may also be used for therapeutic phlebotomy. Keep in mind that AABB-approved bags may be expensive because they contain the preservatives necessary for storage.
These bags also have a 16-gauge needle permanently attached that may not be appropriate for all patients. A benefit of vacuum bottles is the ability to select a smaller catheter gauge. A standard collection bag specifically for therapeutic phlebotomy is also available that contains no preservatives. This bag also has an adapter for catheter connection so the phlebotomist is able to select the catheter size of choice. For patients who are being phlebotomized weekly or more often, this consideration is important to reducing vessel damage.

When preparing a site for catheter or needle insertion, the same considerations for asepsis should be practiced that are indicated for standard use of a peripheral catheter. Because the blood will not be reinfused, the stringent cleansing process used by the American Red Cross is not required. For best success, the catheter should be inserted immediately prior to the phlebotomy and discontinued at completion to reduce vein trauma.

The incidence of adverse reactions with phlebotomy is rare. Thrombosis is seldom a problem postphlebotomy; however, be aware that patients with polycythemia vera have an increased risk of postphlebotomy thrombosis and be sure to address relative issues with the donor for at-home monitoring.15

The most common adverse event from blood donation is the development of hematoma on catheter removal.30 Although generally mild, hematomas can become significant enough to cause damage to surrounding tissues, nerves, or vessels. Holding pressure at the site after removal of the needle will help reduce the incidence of hematoma formation. If hematoma occurs despite this precaution, reapply pressure or use ice to slow the bleed. Monitor the site frequently and document interventions.

Syncope is another common side effect and may be recognized by weakness, sweating, dizziness, or pallor. It may progress to loss of consciousness and/or convulsions. The pulse is more likely to decrease than to increase, and systolic blood pressure may fall precipitously. If syncope occurs during the procedure, remove the tourniquet immediately and either cap the catheter or remove the needle. Supportive treatment will be for symptom management.

Nausea and vomiting may be associated with the rapid decrease in blood volume. Phlebotomy should be held and appropriate measures taken if vomiting is severe. Pretreatment with antiemetics is not generally recommended.

Convulsions are rarely associated with phlebotomy. They may be preceded by muscular twitching or spasms. Provide patient protection to avoid injury and notify the physician immediately. In the event of serious cardiac difficulty, call emergency services for assistance.

Postphlebotomy, patients should remain quietly seated in a reclined position for several minutes and be allowed to slowly return to upright position. Once the patients are able to stand and walk, they may go to an observation area and be given something light to eat or drink. Encourage patients to increase fluid intake over the next few hours (avoiding alcohol and caffeinated products), if not contraindicated by the disease process. Smoking should be avoided for at least 30 minutes postphlebotomy. Provide the donor with information on how to cope with dizziness and when to resume normal activities. These recommendations for donor postphlebotomy care should be a part of the policy and procedure manual, and all observations should be noted on the donor’s chart.30

**NONPHLEBOTOMY Cellular AND IRON REDUCTION**

Although phlebotomy remains the treatment of choice for porphyria, polycythemia, and hemochromatosis, other options for red blood cell and iron depletion are available. A new study in Scandinavia is looking at the potential of performing erythrocyte apheresis in lieu of standard phlebotomy.35 The hypothesis of the study is that 2- to 3-fold the amount of iron may be removed per treatment, which would require less frequent phlebotomies for the patient. Limitations for this type of therapy are likely to be cost, increased time per treatment, and certain requirements in determining who is an appropriate candidate.

The use of chelating medication to reduce iron in lieu of phlebotomy has been studied for years and has been found to have merit in some cases. Deferoxamine is a chelating agent that binds with iron to remove it from the body. It is capable of binding up to 85 mg iron per 1 g medication versus phlebotomy, which can reduce the iron load by 250 mg/500 mL.36 Deferoxamine may be beneficial when iron loads need to be reduced but the patient cannot tolerate phlebotomy. The expense of therapy is one of the limiting factors to use. Deferoxamine is the treatment of choice for the removal of iron in anemias such as sickle cell and thalassemia when anemia is already present.

Other pharmaceutical interventions are available to help reduce bone marrow stimulation. The use of interferon as a myelosuppressive agent has been found to have some advantages for patients with polycythemia vera. Other medications aimed at reducing the platelet count are associated with decreased risk of postphlebotomy thrombosis but may increase the risk of developing leukemia.15

Because the diseases requiring therapeutic phlebotomy are rare, many nurses may never have an opportunity to perform the procedure. However, patients requiring this treatment walk among us in every town across America and may show up at your facility unexpectedly.
REFERENCES


