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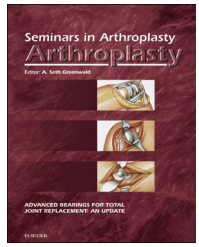

Minimizing intraoperative blood loss: Contemporary strategies

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ABSTRACT

Postoperative blood loss continues to be a common complication following total hip and knee arthroplasty. There are inherent risks to blood transfusion, including increased infection risk, volume overload, and transfusion-related reactions. Recently, antifibrinolytic medications including tranexamic acid have been utilized to decrease perioperative blood loss and decrease the need for postoperative blood transfusions. Recent research has specifically addressed both the efficacy and the safety concerns of these newer treatment modalities and found great results.

Contemporary hip and knee replacement can still be associated with substantial blood loss. Blood loss of 1000–1500 mL is not uncommon during these procedures. Loss of 1000 mL of blood predictably results in a 3-g drop in hemoglobin levels [1]. Increasing blood loss is associated with increased transfusion rates. Our historic transfusion rate in the primary total hip and knee replacement population ranged from 10% to 40%. Minimizing perioperative blood loss, and therefore decreasing the transfusion levels, is very desirable during hip and knee replacement. There are several reasons that decreasing the number of transfusions is beneficial. First, there is the cost associated with a transfusion. A unit of autologous blood transfused to a patient costs approximately $800 at our institution. A study found that the cost of blood transfusions is commonly miscalculated and the true costs range from $522 to $1183 [2]. Not only are there direct costs of the transfusion, but there is also an associated increased cost of the hospital and duration of stay [3]. Secondly, there is a psychological impact on patients who receive a transfusion. A blood transfusion can make patients feel as though they went through a bigger operation and that they have a giant task of recovery ahead. Finally, there are inherent risks with all blood transfusions. These include issues such as blood type incompatibility, immune suppression, lung injury, hemolysis, and disease transmission. Additionally, long-term morbidity and mortality have been seen to increase with the use of blood transfusion [4]. Although these statistics would appear to implicate transfusion as the cause for poorer outcomes-associated total hip and knee replacement, there are obviously times when a blood transfusion is necessary and should be given.

Blood transfusion, as previously discussed, can have a dramatic impact on the cost and length of stay following total hip and knee surgery [3]. This has led to the question of "what is the most effective strategy to decrease blood transfusion in 2014?" A basic understanding of the principles of fluid management is a great place to begin. Traditionally, many of the postoperative clinical symptoms including orthostatic hypotension, elevated heart rate, and low urine output have commonly been misdiagnosed as being anemia

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related. However, these symptoms are mainly related to decreased intravascular volume and therefore will almost always be corrected by replacing this volume with IV crystalloid solutions (normal saline or lactated ringers). The anesthesia literature has shown that volume replacement in the setting of low hemoglobin is safe even in high-risk patients [5]. Additionally, many of the intensive care units have adjusted the threshold values for which they transfuse in the highest risk patients [6].

Many surgeons rely on the anecdotal evidence that they notice substantial improvements in the vigor of patients after providing a transfusion. Patients may "perk up" and feel much better after receiving two units of blood. However, the main reason for symptomatic improvement is likely related to replenishing the intravascular volume with roughly 700 mL of fluid from these transfusions. The combination of the volume from the transfusion paired with the mobilization of extravascular fluid into the vascular system may well improve the patient's symptoms. However, replacing the intravascular volume with crystalloid instead of a blood transfusion is the ideal first step from a cost and safety analysis.

In addition to more judicious use of fluids in the setting of orthostatic hypotension, elevated heart rate, and low urine output, we have also changed our threshold transfusion level. We have seen a transition away from the practice of transfusing all patients with hemoglobin levels less than 10 g/dL. There has been evidence to suggest that it is safe to lower the transfusion threshold to 8 g/dL even in some higher risk patient populations [7]. The American Association of Blood Banks has reflected this sentiment toward a stricter transfusion guideline with their 2012 transfusion guidelines. They now recommend the following: (1) adhere to a restrictive transfusion strategy (7–8 g/dL) in hospitalized, stable patients; (2) adhere to a restrictive strategy in hospitalized patients with preexisting cardiovascular disease, and consider transfusion for patients with symptoms or a hemoglobin level of 8 g/dL or less; and (3) consider symptoms as well as hemoglobin concentration during transfusion decisions [8].

Attempts to decrease perioperative blood loss have led many surgeons to try methods that either decrease intraoperative blood loss or utilize the patient's own blood to transfuse after the surgery. Intraoperative devices include thrombin sprays, lavage with epinephrine, or lavage with norepinephrine [9]. Each method has some inherent risks that have likely decreased widespread use. Thrombin sprays are commonly a bovine derivative, which can secondarily cause a hypersensitivity reaction. Epinephrine and norepinephrine constrict blood vessels, which may decrease intraoperative blood loss; however, postoperatively those blood vessels will relax and begin to bleed again.

Postoperative transfusions with autologous blood require patients to donate blood approximately 2–3 months prior to surgery. This allows adequate time for the patient's hemoglobin to return to baseline before the surgery. As was discussed previously, a unit of autologous blood costs approximately $800. Studies have shown that the function of red cells degrades overtime and may actually be associated with worse outcomes even after being stored for only 14 days [10]. Traditionally, blood banks are only allowed to store red blood cells for 42 days, secondary to issues with red cell degradation. The working practice of most blood banks is to use that blood that is closest to its expiration date; in effect, routinely and systematically transfusing the worst blood available. With autologous transfusion, patients must weigh the risks of having the surgery earlier to increase red cell viability versus waiting later for their hemoglobin to return to baseline. For this reason, as well as the cost, autologous transfusion has largely fallen out of favor at our institution.

In an attempt to decrease our transfusion rates, the literature was reviewed to determine what other surgical specialties were using to decrease their transfusion rates. Other surgical disciplines including cardiac surgery and trauma surgery have utilized a group of antifibrinolytic medications for many years. Aminocaproic acid, aprotinin, and tranexamic acid (TXA) have been the three most commonly used antifibrinolytics. TXA is the most extensively studied of these types of agents. In recent years, TXA has been more extensively used in orthopedic surgery, and there are now more than 90 peer-reviewed publications detailing the efficacy and, to a lesser degree, the safety in hip and knee arthroplasty patients. There is a comprehensive Cochrane database review that has further studied the risks and the benefits of TXA in the orthopedic literature.

TXA is the antifibrinolytic that we most commonly use at our institution. During the normal clotting cascade, plasminogen is converted to plasmin by tissue plasminogen activator (tPA). The purpose of plasmin is to breakdown fibrin, which typically results in further bleeding. However, TXA prevents the conversion of plasminogen to plasmin by inhibiting tPA [11]. Plasmin itself may also be inhibited by TXA, further decreasing fibrinolysis (Image). The mechanism of action for TXA is important to understand. TXA is not a

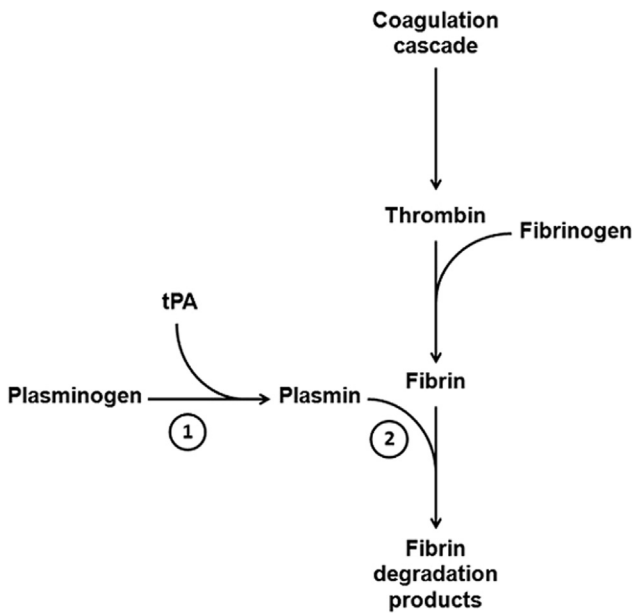

Image – The pathways leading to fibrinolysis. Tranexamic acid primarily inhibits plasminogen activation (1) and, to a lesser extent, plasmin (2). The ultimate effect is reduced fibrinolysis of existing thrombus [12]. tPA: tissue plasminogen activator.