Both Injury And Surgery Result In Trauma To Blood Vessels And May Jeopardize Tissue Vascularity: How can the orthopaedic surgeon minimize these effects?

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Abstract

Injury can cause direct and indirect trauma to blood vessels. Vascular trauma can result in bleeding, ischaemia and reperfusion injury, all of which can decrease tissue vascularity. Surgery directly traumatizes tissues and blood vessels but can also produce a devastating "second-hit" of inflammation which potentiates the ill effects of the initial injury. The orthopaedic surgeon can reduce these effects in several ways including good surgical planning and technique. Understanding the trauma burden of the patient and the physiology of trauma are essential. Concepts such as the lethal triad of acidosis, hypothermia and coagulopathy to identify a patient with high morbidity are important. Newer and less invasive surgical methods and implants are also vital to this aim.

This article will discuss the ways in which injury and surgery can cause trauma to blood vessels and as a result damage tissue vascularity. It will then answer the question of how the surgeon can minimise the effects.

Injury can cause direct and indirect damage to vessels. Direct damage includes lacerations to or complete transection of a vessel. Blood vessels can also be contused. They can form true aneurysms involving all three layers of the vessel wall or false aneurysms which result from a split between the tunica intima and media. This allows blood to track between these layers and form an out-pouching of the blood vessel. Intimal flaps can be elevated and the resultant damaged vessel wall can lead to thrombosis. Haematoma formation can occur beneath the intima of a contused vessel. If a vein is damaged adjacent to an artery an arteriovenous fistula can form. Indirect damage can occur when a vessel is compressed externally. The systemic response to injury can also have an indirect deleterious effect on blood vessels.

The major consequences of vascular trauma are bleeding, ischaemia and reperfusion injury. Bleeding can be immediately life-threatening. It causes hypovolaemia and will eventually result in circulatory failure with inadequate tissue perfusion causing cellular hypoxia. In a patient who is actively bleeding there is a catecholamine drive to help preserve blood supply to vital organs. This drive stimulates

gut and skin alpha-receptors to cause vasoconstriction. The vasoconstriction causes some areas not to be perfused which can lead to further tissue ischaemia.₄

Hypoxia affects red blood cells. With decreased oxygen tension red blood cells become rigid and are unable to pass through the microcirculation. They can become lodged in the microvasculature and can contribute to the formation of microcoagulation. This in turn can lead to disseminated intravascular coagulation (DIC). These effects can all jeopardize tissue vascularity. Lack of oxygen in tissues results in an intracellular energy deficit as there is not enough oxygen for oxidative phosphorylation in mitochondria. The cell relies on anaerobic metabolism and lactic acid builds up resulting in metabolic acidosis. The cell membrane becomes more permeable and the energy-dependant ATPase pump fails resulting in potassium leak and sodium and water influx. The cell will eventually swell and die.4

As toxic metabolites in ischaemic tissue build up there is damage to membrane permeability and oedema occurs. Oedema can indirectly worsen tissue perfusion by compressing blood vessels and reducing oxygen delivery. It also puts pressure on other tissues and will eventually result in metabolic dysfunction and necrosis of tissue.

Reperfusion injury is an important consequence of blood

vessel injury. If an ischaemic area is reperfused the accumulated toxins are released into the circulation and can worsen acidosis. Reperfusion with oxygenated blood can lead to the formation of highly reactive oxygen free-radicals which can further damage the tissues. 4,6 After reperfusion there is increased capillary permeability which leads to plasma protein extravasation and interstitial oedema. 4

Surgery can compound all of these effects. There will be more direct damage to tissues and blood vessels. Extensive approaches will damage skin and other soft tissue along with their blood vessels. Tissues can become bruised by rough handling and in that way tissue vascularity can be jeopardized further. In the fixation of fractures periosteal stripping will compromise the blood supply to the bone. Poor surgical technique such as excess tension on wounds during closure will damage the skin and reduce the blood flow to the healing edges. All of these can cause necrosis and increase the risk of infection which in turn can reduce tissue vascularity.

Indirectly surgery can cause more damage to vessels and tissue vascularity by further blood loss. It can also produce a devastating "second-hit" inflammatory response. Moore et al. performed a prospective study of four-hundred and fifty-seven trauma patients who developed multiple organ dysfunction syndrome (MODS). They investigated the time at which these patients developed MODS post-injury. The hypothesis was that the injury produced a systemic inflammatory response "first-hit" that could, if large enough, precipitate MODS. If the patient was then exposed to an early "second-hit" such as surgery they stood more chance of developing MODS.₇ In the presence of impaired microvasculature and hypoxia there is more chance of damage occurring to tissue vasculature.

So, how can the surgeon minimize these effects? Of vital importance is obtaining a full history. Discovering how, when and where the injury occurred and the degree of force that caused the damage can give important clues as to the nature of the injury. These factors help to understand the trauma burden of the patient. The time since injury, blood loss and details of other injuries and what needs to be done to these injuries is necessary. The polytrauma patient will already have had a large first inflammatory reaction and the second-hit of surgery must be avoided. This knowledge can help to plan surgical tactics in repairing injured vessels, stabilising fractures and treating soft tissue damage. Stabilising fractures are treating soft tissue damage.

to resuscitate and stabilise the patient as much as possible prior to any operative intervention.

ATLS protocols should be adhered to. The basic principles of identifying and treating life-threatening injuries first should not be forgotten. Although ischaemia is a consequence of vascular injury it is not treated in the primary survey whereas bleeding is. It should be remembered that the window of opportunity to reperfuse an ischaemic limb can be a couple of hours, whereas bleeding can be immediately life-threatening.₂

Recognising which patients to operate on immediately and which would benefit from a damage control approach is another way in which the surgeon can help to reduce the damaging effects on tissue vasculature. Patients who are haemodynamically unstable should be taken to the operating theatre straight away in order to locate and stop the source of bleeding. In blood vessel trauma these patients should not have pre-operative angiograms.

It has been recognised that there is a lethal triad of hypothermia, acidosis and coagulopathy which should be avoided in the injured patient. Each component can contribute to reduced tissue vascularity. Acidosis has been discussed. Hypothermia causes clotting derangement and can worsen coagulopathy. Coagulopathy can occur with leaching of clotting factors or it can result in DIC. Patients who are found to be hypothermic, coagulopathic and acidotic are best treated using a damage control approach to stop any bleeding, stabilise fractures and then plan definitive vessel, fracture or soft tissue repair at a later date.

Another way in which the surgeon can identify the patients that will have a poor outcome with extended surgery is by monitoring lactate levels. Blow et al. showed that a lactate greater than 2.5mmol in a patient with normal haemodynamic parameters was highly suggestive of occult hypoperfusion. They advocated aggressive resuscitation to correct this prior to surgery in order to reduce the incidence of Adult Respiratory Distress Syndrome (ARDS) and MODS.₉ The surgeon can therefore recognise which patients need further resuscitative measures before surgery and resuscitate them adequately in order to improve tissue vascularity.

There are specific vascular techniques that the surgeon can employ to reduce damage to blood vessels and vasculature. It is also important to use simple measures such as the use of a finger or balloon tamponade to control bleeding.₂ A solid knowledge of anatomy is essential for the surgeon to try to minimise any ill effects on blood vessels of tissue vasculature. Bleeding can sometimes be controlled by identifying a vessel proximally to an anatomical barrier. In a groin injury for example the femoral artery can be controlled proximally to the inguinal ligament. This avoids blindly approaching the bleeding injured vessel and results in swifter control with less blood loss and less damage to the vessel and surrounding issues.₂

With blood vessel injuries after proximal and distal control has been achieved it is advisable to optimise the incision and clear around the injured vessel to define the extent of the damage. The vessel should be dissected along the periadventitial plane directly onto the arterial wall to prevent cutting the vessel or stripping any branches.2 The surgeon has to decide whether to repair the vessel, to ligate it or to create a temporary shunt. If the patient is stable and not coagulopathic then the injured vessel can be repaired. When repairing the vessel care should be taken in handling so not to cause further damage, for example vascular clamps should not be applied so tightly that they crush the vessel. If a simple repair is carried out then the sutures should be short with the needle held perpendicular to the vessel wall. Complex repairs include the use of vascular patches, anastamosis or interposition grafts., In an unstable patient or a patient who is bleeding from a second location or when there is not enough equipment or expertise to perform repair then the vessel should be ligated or a temporary shunt applied. Temporary shunts can be formed from a variety of implements such as nasogastric tubing and Foley catheters.,

Pre-empting problems comes with experience but is another way in which the surgeon can help to reduce damaging effects on tissue vascularity. An example of this is the use of pre-emptive fasciotomy prior to a popliteal artery repair. Compartment syndrome can occur in a previously ischaemic limb once the vessel has been repaired, especially following prolonged surgery. This will damage muscle and jeopardise the blood flow to the limb. Performing the fasciotomy first rather than waiting for it to happen prevents this damage.

When a bone fractures there is damage to the surrounding soft tissues and blood vessels due to an implosion-like process. A reduction in blood flow has been observed due to vasoconstriction of medullary and periosteal vessels as a response to trauma. In cortical bone this can be as much as fifty percent reduction in blood flow. As the fracture

separates a space is created which causes blood vessel rupture both inside and outside of the bone, and soft tissue damage also occurs. The blood supply to bone is damaged in many ways and it is important that the surgeon understands these in order to properly treat a fracture with minimal damage to blood vessels. The accident itself causes rupture of endosteal and periosteal vessels with stripping of the periosteum. Soft tissues are necessary for the repair process and these too are damaged. Transporting the patient to hospital without stabilizing fractures causes motion at the fracture site which can further damage the blood supply and soft tissues. Any surgical approach used can cause vessel damage.11 The implant used for fracture fixation can also cause blood supply compromise. With plates this occurs due to soft tissue stripping required to apply the plate to bone, also the contact between the bone and the implant damages the bone immediately below the plate. Necrosis takes place in this place and the bone remodels slowly by creeping substitution. The remodeling causes temporary porosis which predisposes to infection and in rare cases this can result in sequestration. Decreasing the area of the plate that is in contact with the bone by undercutting the plate can help to reduce the area affected and therefore enhance fracture healing. Locking plates are a further improvement to this. The screws lock into the plate so there can be no contact between the plate and the bone, the plate acts as a splint. It also has the advantage of allowing the use of unicortical screws; this can reduce the amount of soft tissue irritation at the far cortex.₁₀ The surgeon can therefore choose the implant which causes the least amount of disruption to blood supply.

Further improvements are the techniques of Minimally Invasive Plate Osteosynthesis (MIPO) which involves small incisions to apply a plate and then slide it along the bone. This avoids a large surgical approach and can decrease softtissue vasculature damage. Farouk et al performed a cadaveric study on ten fresh cadavers. Each underwent conventional plating via a lateral approach to one femur and MIPO via a two centimeter incision contralaterally. Silicone dye was inserted to identify perforating and nutrient vessels. They showed less disruption to the vessels with the MIPO technique. It should be remembered that there were small study numbers and the experiment was not in living patients.₁₂The Less Invasive Stabilisation System is a locking plate again designed to hover above then bone held in place by screws locked into the bone. It is also inserted through small incisions.₁₀

When using intramedullary implants reaming reduces endosteal blood flow. The return of blood flow depends on the extent of reaming and the size of intramedullary nail used. Without reaming the endosteal blood supply is affected to a lesser degree, bone healing is faster and the avascular area is reduced. Thus, the surgeon can avoid reaming in order to preserve blood supply.₅

If a fracture is to heal by callus formation then the surgeon must recognize that the majority of the blood supply to the callus is from the soft tissues. It is therefore important to disrupt the soft tissues as little as possible.

If a fracture has occurred with extensive damage to the soft tissues and disruption to the blood supply of the bone then it has a better chance of healing the more stable it is. The ideal is to achieve interfragmentary compression and absolute stability. The blood vessels can then easily cross the fracture site. This is especially important at articular surfaces. In the diaphysis if there is a multifragmentary fracture attempts at achieving absolute stability may disrupt the fragment biology and damage the blood supply to prevent healing. 5 The surgeon must have an understanding of the principles of fracture fixation using absolute and relative stability and know when to apply them.

A final consideration is the surgeons' role in advocating injury prevention. There have been many successful campaigns in recent years with this aim. The promotion of wearing seat belts whilst driving has reduced the severity of injuries sustained in car accidents. The use of speed cameras to reduce speeding and thus accidents has had another positive effect. It could be argued that as someone who sees the disastrous effects of trauma the surgeon is in a prime position to fight to reduce the incidence of it. If there was no injury to the blood vessels in the first instance then there

would be no need to try to reduce the effects on tissue vascularity.

Injury and surgery result in trauma to blood vessels and jeopardize tissue vascularity. The surgeon can not reverse these once they have occurred but can minimise them as far as possible. This can be achieved by a combination of understanding the nature of the injuries and the patient's physiological state, a thorough knowledge of anatomy, experience and good surgical technique, combined with an appreciation of the principles of haemostasis, vascular repair and bone fixation.

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