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FLUID AND BLOOD THERAPY IN TRAUMA

MORE QUESTIONS THAN ANSWERS

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Tissue injury results in SIR, release of mediators leading to increase in vascular permeability and tissue edema. On top of this, a concurrent haemorrhage causes further reduction in intravenous volume. The initial fluid redistribution that occurs following trauma is related more to the degree of tissue trauma and ischemia than to blood loss per se. With mild hypovolemia, blood in the venous capacitance vessels is mobilised to ensure adequate venous return. When this is depleted, fluid from the interstitial space is shifted to the intravascular space (autotransfusion) and the gradient between the oncotic pressure and hydrostatic pressure decreases. If there is further blood loss, haemorrhagic shock results. A decrease in cardiac output and arterial O2 content leads to decreased O2 delivery. Cellular mechanisms fail, sodium potassium adenosine pumps fail, causing water to shift into the intracellular space further depleting intravascular fluid, cellular swelling occurs and ultimately cell death if the process is not reversed.

Fluid resuscitation can increase the interstitial edema, which is caused by the 'reperfusion injury' to the capillary interstitial membrane. This causes a rise in osmotic pressure in the extracellular spaces and glucose is primarily responsible for this. Tissue edema is rarely life-threatening but can decrease oxygenation, delay healing and can lead to subsequent sepsis. The whole goal of fluid therapy is to ensure adequate oxygen supply. In an editorial by Bickell, 'Are victims of injury victimised by attempts of fluid resuscitation,' he tries to explain how many hypotensive trauma patients are operating within the limits of physiologic compensation and the questions are not only what fluids, how much but also to whom and when to administer.

The American College of Surgeons protocol for ATLS recommends replacing each ml of blood loss with 3 ml of crystalloid fluid. This is known as the 3 for 1 rule. The patient's response to this initial resuscitation determines subsequent therapy, 3 response patterns are described:

1. Rapid: Responds rapidly and remains hemodynamically stable.

2. Transient - Responds initially then deteriorates as fluids are decreased to maintenance levels.

3. Non-responsive: Failure to respond either to crystalloids or blood.

Controversy persists as to the choice of the fluids for resuscitation-crystalloids or colloids? The basis of the 3 for 1 rule comes from the volume of distribution of electrolyte solutions. Again the proportion of crystalloid to colloid needed to ensure adequate volume expansion depends on the degree of permeability injury. Crystalloids expand the extracellular fluid space but larger volumes are required. Colloids by virtue of their oncotic pressure produce effective volume expansion with small volumes of infusate.

Crystalloids:

These can be isotonic or hypertonic. Isotonic fluids equilibrate throughout the intravascular and interstitial compartments but do not cause intracellular shifts. These can effectively replace interstitial fluid shifts. Hypertonic fluids can cause redistribution of intracellular fluid into extracellular compartments but its mainly from the interstitial space. Authors have reported no difference in outcome whether crystalloids or colloids are used but a combination of both may be efficacious.

Hypertonic Fluids:

The theoretical advantage of using hypertonic fluids is mainly the small volume requirements for resuscitation. The osmotic effects, the inotropic effects and the direct vasodilating effects of hypertonic saline leads to increase in MAP, CO and an increase in renal, mesenteric, splanchnic and coronary blood flow with the peripheral vasodilatation. But to be effective, studies have shown these solutions must pass though the lung, thus stimulating osmolar receptors. But remember, these can also predispose to increased haemorrhage from the open blood vessels. The vasodilatory effect can counteract the early compensatory vasoconstrictor response induced by hypovolemia. It can also cause hypernatremia and hyperchloremia with a resultant metabolic acidosis. The serum levels are relatively normal with small infusions.

Combined Crystalloid - Colloid resuscitation:

When using combinations, the hypertonic crystalloids can draw water out of the intraceullar spaces and the colloid component prolongs the beneficial effects of these solutions. Hypertonic saline dextran 40 (HSD) expands plasma 3-4 times the volume infused. Crystalloids expand plasma volume less then 30%. The combined beneficial effects are attractive but studies have demonstrated more bleeding with this group. Delaying or slowing of HSD solutions reduced the morbidity and mortality rates.

Colloids:

The concept was introduced by Starling. A plant derived colloid, gumacacia was used in World War I. Blood and components were used in World War II. Albumin then came but the high cost led to development of synthetic colloids like the dextrans, gelatins and hetastarches.

Blood substitutes:

Blood substitutes were developed in the search for a non- antigenic disease-free, oxygen-carrying fluid. Three haemoglobin based products are available:

1. Stroma free haemoglobin

2. Modified stroma free heamoglobin

3. liposome encapsulated haemoglobin.

Side-effects like renal failure, leukopenia, platelet dysfunction and vasoconstriction are unacceptable. The Hb based O2 carriers (HbOC), other blood substitute products from outdated human blood, bovine Hb, recombinant Hb are being investigated. With plasma half life of several days . HbOCs can serve as a bridge to transfusion, that can reduce the banked blood requirements of acute trauma patients. The way these products interact with the shock state has not been studied. Hypertension often seen with these is probably due to the vasoconstriction from haemorrhage. The NO scavenging either improves performance or worsens control of bleeding.

Blood Transfusion

The response to the initial fluid bolus provides information on the type and amount of additional fluid needed. Patients who become hemodynamically stable and have no ongoing blood loss can continue with crystalloid infusion. If not, they would require erythrocyte and blood products and this world require consideration of complications. A hemoglobin level alone cannot be used as a transfusion trigger. Patient's O2 delivery and O2 consumption should be considered. The effects of hypovolemia must be separated from those of anemia. A loss of upto 30% of the blood volume can be treated with crystalloid. If more, then blood may have to be used as a replacement fluid. The 4 major reasons for transfusing blood and blood products in trauma are 1. Improvement of oxygen transport 2. restoration of red cell mass 3. correction of bleeding caused by platelet dysfunction 4. correction of bleeding caused by factor deficiencies.

Massive transfusion:

Transfusion of at least one blood volume or 10 units of blood in a 24 hr period is massive transfusion. In 1982, Millers study on trauma patients reported that in those receiving more than 40æ with in the first 24 hrs had a survival rate of less than 15%, those from 30-39 units had a 40% survival rate and those receiving between 20-29 units had a survival rate of 69%. Survival has been reported even with 100u of blood and the current survival rate following massive transfusion is 50%. Partial cross-matching and uncross matched blood are essential considerations according to the type of trauma, About 1 in 800 have unexpected serum antibody during cross match and only 1 in 2500 have antibodies capable of causing hemolysis. If for some reason, more than 4 units of type 'O' Rh negative packed red blood cells (PRBcs) have been given, its best to switch over to type specific blood when it becomes available because the high anti -A, anti -B titres could cause hemolysis of type specific donor blood. O-ve whole blood should not be given as the high donor anti-A and anti-B titres could cause hemolysis of recipient RBCs.

Autotransfusion:

Salvage of shed blood from wounds, body cavities, drains finds use in trauma patients. The blood can be directly anticoagulated and reinfused into the patients using a macroaggregate filter. Another method is the use of a cell-saver and provision of washed RBCs. Several complications can occur and is usually seen with autotransfusion of more than 1500ml of shed blood.

Blood component transfusion:

Massively transfused patients require transfusion of specific hemostatic components, platelets, frozen plasma, cryoprecipitate. The American Society Of Anesthesiology Task Force on blood component therapy gives recommendations for specific therapy. The complications of massive transfusion are given in any book.

End points of Resuscitation:

Control of bleeding, restoration of circulating blood volume and providing adequate oxygenation at the cellular level must remain cornerstones of care for trauma patients. No single end point has found to be sufficient by itself and these have to be considered concurrently with other vital signs. Blood pressure and heart rate are poor indicators of severity of shock and do not correspond to the cardiac index, though these are ATLS guidelines. It is difficult to monitor blood volume, cardiac index and DO2 before and during administration of large volumes of fluids in the emergency department or OR. How do we know that the patient has been adequately resuscitated?. BP, HR, urine output, mental status, pulse oximeter and capnogram are all used but will not reflect the situation at the cellular metabolic level. More aggressive monitors have been shown to improve mortality especially in elderly patients like the CVP, pulmonary artery occlusion pressure and ABG monitoring but studies have shown that the mean values of these are the same in the surviving and non surviving trauma patients. There are studies which show cardiac index, DO2, and O2 consumption as better end points following trauma. The time frame to achieve the survivor values seems to be more important then the absolute survivor values. This is probably because the patients are not allowed to go into the irreversible O2 debt. These end points have also been questioned. Perfusion related variables such as A-V oxygen content difference, mixed venous pH, arterial base excess can also predict survival and adequacy of resuscitation. These can give some indication to the whole body O2 debts. The mortality rate is shown to increase with the degree of acidosis at admission and the subsequent 24 hrs. Lactate levels, a measure of anaerobic metabolism correlates with survival. If the lactate level was normalised in 24 hours, there was a 100% survival rate and a 75% survival rate if it took 48 hours to clear the lactate. Gastric tonometry may provide definitive assessment of resuscitation as an indicator of restoration of splanchnic blood flow. Tissue O2 monitoring is another good indicator. Skeletal muscle blood flow decreases early in shock states and is restored late during resuscitation, making skeletal partial pressure of O2 a sensitive indicator of low flow. Subcutaneous tissue is another sensitive area where flow dependent O2 consumption may be detected.

Current issues in Resuscitation:

Administration of prehospital fluids is a balance between the physiologic benefits of intravenous volume loading against time spent establishing IV access and consequences of increasing systolic blood pressure and dilution of coagulation factors. In uncontrolled haemorrhage, optimum survival is thought to be achieved by allowing blood pressure to remain low until surgical hemostasis is achieved, a technique known as 'permissive hypovolemia or hypotensive resuscitation' and systolic BP of 70-80mm Hg has been suggested. This is not appropriate for head injury patients though. Crystalloids and colloids can be used, but the colloids should be used only when BP is below 50mmHg. Aggressive resuscitation with crystalloids may increase the pulse pressure at a time when blood viscosity is decreased greatly and the clot associated with vascular injury has little time to stabilize. Stern et al compared the effects of saline resuscitation of MAP 40mm Hg, 60mmHg and 80mmHg. Mortality was greater in the MAP 80 mmHg. group. The MAP 40mmHg had the least intraperitoneal hemorrhagic volume and lowest mortality rate but was shown to have marked metabolic acidosis and reduced DO2. MAP of 60mmHg showed markedly improved tissue perfusion. They attained higher MAP than the aggressively treated animals and these were attributed to be changes in pulse pressure. Severe hemodilution may be a factor for increased mortality as increased CO implies increased SV and myocardial O2 demand which all trauma patients may not be able to achieve. So normotension is not the ideal therapeutic end point. One trial in humans to test the concept of delayed resuscitation or controlled under-resuscitation showed improved survival if IV fluid administration was delayed until they reached the OR. The death rate was found to be higher in patients who underwent immediate fluid resuscitation. The arguments against immediate resuscitation is that it reverses vasoconstriction, dislodges early thrombus when given in huge volumes, dilutes coagulation factors and changes viscosity because of the resistance to flow. So timing may be important. Optimal timing and rate of infusion are other important factors to be considered. At higher infusion rates, the blood loss is also higher. The potential risk of inducing major hemorrhage from blood vessels before surgical control could be reduced by avoiding an infusion rate that is too fast and at a very early stage of the injury. Penetrating injuries are easy to study but blunt injuries are more difficult to reproduce. Hypertonic solutions were shown to be more useful here, probably because more solutions remained intravascularly compared to the other 2 groups. Extracorporal supports, heparin-bonded circuts, are all being tried. The extra corporal circuit maintain the body perfusion while isolating the vascular injuries intra operatively. As we enter the next century, Resuscitation Medicine remains an open field for research. Being familiar with end points of resuscitation and making interventions as and when indicated will improve outcome finally.

Further Reading:

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