

# Update in Nonpulmonary Critical Care

## Trauma Critical Care

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### "DAMAGE CONTROL" APPROACH TO TRAUMA SURGERY

Operative goals for trauma patients with severe multisystem injuries have changed in recent years. This has presented a new challenge for critical care physicians, who must now address problems that previously burdened anesthesiologists during lengthy operations on unstable patients. The historical objective of laparotomy was to provide definitive hemostasis, repair of *all injuries*, and control of enteric contamination by resection of bowel injuries, followed by anastomoses or ostomy formation. Prolonged operative times were common in patients with multiple injuries such as massive liver trauma, abdominal vascular trauma, retroperitoneal injuries, multiple intestinal disruptions, chest trauma, and open fractures. If the patient returned to the operating room for postoperative bleeding the surgeon was considered to have performed an inadequate operation.

The burden was on the anesthesiologist to provide sufficient intraoperative critical care so that the surgeon had sufficient time to definitively repair all injuries. In many of these patients progressive intraoperative deterioration in physiology occurred, typically manifested as *hypothermia*, *coagulopathy*, and *acidosis*. This triad presented as a vicious cycle that often could not be interrupted, and patients frequently died in the operating room due to "irreparable injuries."

Staged laparotomy, also known as abbreviated laparotomy, or "damage control," is a new, important, rapidly expanding surgical approach to treating unstable patients. It can be defined as a series of operations performed during several trips to the operating room at intervals of several hours to days, in order to accomplish definitive repair in a staged manner in accordance with the patient's physiologic tolerance. A minimal operation is initially performed solely to reduce or control surgical bleeding and enteric spillage. Techniques for abbreviating operations include ligation of intestinal injuries without anastomoses, leaving retained clamps on injured vascular structures or placement of temporary intraluminal shunts, packing diffusely bleeding surfaces with multiple laparotomy pads, and using towel clips to quickly close the skin, leaving the underlying fascia open. The patient is then rapidly transported to the intensive care unit for resuscitation in order to restore sufficient physiologic reserve to allow return to the operating room for definitive repair and reconstruction (1-3).

Although retrospective in nature, studies have documented a nearly 50% decrease in operative times for the most severely injured patients treated by this approach, and salvage rates of

20-60% in patients who formerly died in the operating room (2, 4). However, this shifts the primary burden of addressing the triad of hypothermia, coagulopathy, and acidosis from the anesthesiologist to the critical care physician. Prompt correction of these abnormalities is vital in order to minimize blood loss, an independent risk factor for postinjury multiple organ failure, and to allow early definitive repair of injuries.

While often lifesaving, the damage control approach is also associated with its own unique set of potentially lethal complications, most notably intraabdominal hypertension, or the "abdominal compartment syndrome" that results from large resuscitation volumes, massive bowel edema, retained laparotomy pads used for packing raw bleeding surfaces, retroperitoneal hematoma, and postoperative bleeding. The purpose of this article is to review the current physiology and treatment of the components of the "deadly" triad, and to review the pathophysiology of abdominal compartment syndrome and its effects on intensive care unit management.

### HYPOTHERMIA

Hypothermia in the trauma patient should be considered as a distinct entity from hypothermia induced by other causes. Mortality from moderate hypothermia (28-32° C) due to exposure is less than 25%, with virtually all deaths attributable to underlying diseases, rather than to hypothermia. In contrast, in trauma patients a core temperature less than 32° C is associated with 100% mortality, and any decrease in temperature below 35° C is a poor prognostic sign (5). Hypothermia is more common and more profound in more seriously injured patients, making it difficult to determine whether mortality is related to the hypothermia, or to the severity of the underlying injuries. However, studies that compared slow versus rapid rewarming methods, including a randomized prospective trial, demonstrated a significant 7-fold increase in mortality during resuscitation of patients who were deliberately rewarmed less aggressively (6, 7).

Hypothermia in the trauma patient is referred to as secondary accidental hypothermia, and a great deal of confusion has arisen as a result of considering it as similar to primary accidental hypothermia. Primary hypothermia occurs when heat production is normal, but body temperature decreases as a result of severe heat loss due to environmental conditions. Secondary hypothermia is a result of diminished heat production. Because body heat is generated as a result of oxygen consumption, less body heat is generated during shock, when oxygen consumption is pathologically reduced, resulting in a decrease in core temperature without severe environmental cold stress.

Uncontrollable hemorrhage is the most frequent cause of early death in trauma patients, and perhaps the most severe effect of hypothermia is its inhibitory effect on the enzymatic reaction rates of the coagulation cascade. The extent to which hypothermia causes coagulation problems is underestimated clinically because fibrometers, which measure the prothrombin time (PT), partial thromboplastin time (PTT), and throm-

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bin time (TT), are designed to detect clotting factor deficiencies. The plasma is warmed to 37° C before testing, thus correcting for any effect of hypothermia.

Clinically relevant levels of hypothermia (< 35° C) can prolong clotting times to the same extent as severe clotting factor deficiencies. Using normal human plasma and a fibrometer modified to allow measurement of clotting times at temperatures other than 37° C, one study measured the PTT at 35, 33, and 31° C. Clotting times were equivalent to that of blood with factor IX levels of 39, 16, and 2.5% of normal, respectively (8). Factor IX deficiency, the most common congenital bleeding disorder, leads to abnormal bleeding when levels drop below 25–30% of normal. In trauma patients clotting factor depletion frequently accompanies hypothermia, which compounds this effect (9). Hypothermia also affects platelet function. In a study in which hypothermia to 32° C was induced in baboons, but one forearm was kept warm with heating lamps and a warm blanket, simultaneous measurements of bleeding time in the warm and cold arm were 2.4 and 5.8 min, respectively (10). Temperature management is therefore a key issue in the management of these patients.

The body has relatively high specific heat (heat gain/loss per degree Celsius change in temperature), 0.83 kcal/kg/°C. A 70-kg patient must gain 58.1 kcal to increase average body temperature by 1° C. Basal heat production is approximately 1 kcal/kg/h if oxygen consumption is normal, which will produce a re-warming rate of roughly 1.2° C/h if all heat loss is prevented. However, most of these patients are in shock, have reduced heat production, and are incapable of spontaneous rewarming.

External rewarming methods help to prevent heat loss, but transfer little heat to the patient. Skin temperature may be 10–15° C cooler than core temperature in hypothermic patients. Heat flows from an area of higher temperature to one of lower temperature as a function of the second law of thermodynamics. External rewarming techniques therefore cannot transfer heat to the core until the temperature of the skin is raised to at least the level of the core. During this lag time core temperature may actually continue to decrease (afterdrop). Fluid-circulating heating blankets are particularly inefficient because the blanket is only in contact with a small percentage of the body surface (< 25%). Also, during hypothermia skin perfusion decreases from roughly 200 ml/min/m<sup>2</sup> to as little as 4 ml/min/m<sup>2</sup>, which reduces its thermal conductivity to a level roughly equivalent to cork (11).

Convective air rewarmers also transmit very little heat because the density of air is so low that it contains very little heat even at a high temperature. A hand can be inserted into a 212° F oven, but not into 212° F water. These devices blow 21,000 L of air per hour over the patient. The density of air is 0.0012 kg/L; therefore, 25 kg of air passes over the body per hour. The specific heat of air is 0.24 kcal/kg/°C. If the air temperature decreases by 3° C as it rapidly passes over the body only 19 kcal/h is transferred to the patient. However, as a function of the second law, if the temperature of the surrounding environment is warmer than skin temperature, heat loss cannot occur, except by sweating. Since most heat loss occurs through the skin, wrapping the patient in a convective warmer can effectively prevent heat loss (12).

Airway rewarming depends on the principle that the amount of water capable of being held as vapor depends on the temperature of the air. When a hypothermic patient inspires saturated warm air, condensation occurs on contact with the cold airway. Fully saturated 41° C air holds 0.05 ml of H<sub>2</sub>O per liter of air, whereas 30° C air suspends only 0.03 ml of H<sub>2</sub>O per liter. When a hypothermic patient inspires 1 L of saturated 41° C air, 0.02 ml of H<sub>2</sub>O condenses within the airway. With a minute ventilation

of 10 L/min (600 L of air per hour), 12 ml of H<sub>2</sub>O per hour condenses within the airway. The amount of heat liberated when water condenses (latent heat of vaporization) is 0.58 kcal/ml H<sub>2</sub>O. Thus, the amount of heat transfer is only 6.9 kcal/h (0.58 kcal/ml H<sub>2</sub>O × 12 ml H<sub>2</sub>O/h). This technique reduces the small amount of heat lost by breathing room temperature air, but has a negligible effect on body temperature (13).

Pleural or peritoneal lavage results in significant heat transfer. Because the specific heat of water is 1 kcal/L° C, the amount of heat transfer depends on the difference between the inlet and outlet water temperature. If 1 L of 40° C water exits the body cavity at 35° C (exit temperature depends on dwell time), 5 kcal of heat is left in the body. Under these conditions, nearly 12 L of irrigation is required to raise body temperature by 1° C.

The administration of warm intravenous fluids is a critical step in preventing and treating hypothermia. A patient will have to generate an extra 16 kcal to warm 1 L of room temperature (21° C) crystalloid to 37° C. If the patient cannot generate this additional heat because of fixed oxygen consumption this results in a loss of 16 kcal, which will decrease body temperature by 0.28° C/L. Warming blood products is particularly important, because they are stored at 4.0° C.

Modern countercurrent fluid warmers pass fluids through a 42° C water bath contained in a length of thin aluminum tubing that has a 1,000 times greater thermal conductivity than standard plastic intravenous tubing. This enables the infusion of up to 800 ml of saline, or 1 unit of blood, per minute at a temperature of 37–40° C, which in a 32° C patient is equivalent to transfusing nearly 5–8 kcal of heat per liter. Patients requiring massive resuscitation may benefit significantly from warmed intravenous infusions. Cardiopulmonary bypass, the most rapid rewarming method available, relies on the principle of warm intravenous fluid administration, but circumvents any limitations imposed by the patient's fluid requirements by recirculating the same fluid.

Continuous arteriovenous rewarming (CAVR) is a relatively new method of performing extracorporeal circulatory rewarming that does not require a mechanical pump or heparin, and can be performed by intensive care unit personnel with minimal training (14). The technique is analogous to continuous arteriovenous hemofiltration. Percutaneously placed 8.5F femoral arterial and venous catheters are connected to a standard countercurrent fluid warmer, and the patient's own blood pressure creates a circulatory fistula through the heating mechanism. The tubing circuit is heparin bonded. The typical fistula flow is between 250 and 350 ml/min when systolic blood pressure is above 80 mm Hg. If the patient's temperature is 32° C and blood is reinfused at a temperature of 40° C, nearly 8 kcal of heat will be transfused every 3–4 min. In a prospective, randomized study involving 57 critically injured hypothermia (< 34.5° C) trauma patients that compared standard rewarming methods with CAVR, rapid rewarming significantly increased the likelihood of successful resuscitation.

## COAGULOPATHY

In addition to the effects of hypothermia, the coagulopathy that occurs in severely injured patients is complex, and extremely difficult to manage. *In vivo* coagulation is considerably different from the conventional view of a mutually exclusive intrinsic and extrinsic cascade, a paradigm that was originally designed to detect congenital clotting factor deficiencies *in vitro*. *In vivo* clotting is affected by a variety of events, and in many cases fibrinolysis, rather than thrombin formation, is predominant. This may especially be true at the site of injury. The generation of clot results in production of

clotting inhibitors that limit the formation of thrombus to the site of injury. If there is continued bleeding there may be extensive local fibrinolytic activity, producing a "local" coagulopathy. Similar to the difficulties in detecting local tissue anaerobic metabolism by measuring lactate from a blood sample obtained from the entire circulatory pool, these local changes may not be apparent by standard clinical testing.

Patients with massive injuries may also develop systemic clotting factor depletion and diffuse coagulopathy due to the body's continuous attempts to form clots at multiple injury sites. Fibrinolysis is activated by clotting, and teleologically serves to clear thrombi from the microvasculature and to limit the formation of thrombus. Massive clotting factor activation due to multiple injuries may result in uncontrolled fibrinolysis, and a cycle of clotting factor activation with further production of antithrombins.

A number of platelet inhibitors also found in the circulation of severely injured patients, such as prostaglandin I<sub>2</sub> (PGI<sub>2</sub>) and antithrombin III, prevent stabilization of the platelet plug by blocking fibrin formation, resulting in a negative feedback mechanism that prevents platelet activation. Patients with major blood loss alone generally do not exhibit dilutional thrombocytopenia because of compensatory mobilization of platelets from the bone marrow, spleen, and other sites. However, an equivalent amount of blood loss accompanied by shock is associated with a decrease in platelet count, indicating that shock, not dilution, is the primary mechanism responsible for thrombocytopenia. This was confirmed by a study in which platelets were routinely administered to seriously injured trauma patients, regardless of platelet count, who ultimately exhibited the same platelet count as patients not receiving routine platelet administration (15).

Transfusing clotting factors and platelets until the consumptive process resolves is an essential first step in treatment. However, therapy should be primarily directed at the underlying shock. Rewarming is also vital, as empiric clotting factor repletion without rewarming is unlikely to staunch hemorrhage because clotting enzyme kinetic activity is significantly reduced in the cold patient.

Clinically, treatment of coagulopathic bleeding is critically compromised by current coagulation monitoring technologies. These tests are time consuming; 40–60 min elapses before results are obtained. If clotting factors are needed an additional 30–40 min is required for thawing and transport. The entire blood volume of the bleeding trauma patient may have been exchanged during that time interval, making the results of the laboratory tests obsolete. Empiric clotting factor and platelet administration is rarely indicated, but in the massively bleeding trauma patient can be justified because of inadequacies of current testing methodologies.

One technology that has shown considerable promise in predicting hemorrhage and that can be performed at bedside is thromboelastography (TEG), which measures the viscoelastic properties of blood (16). Clot formation consists of the interaction of platelets with fibrin; thus, platelet and clotting factor interactions are not independent. TEG uses a warm cup containing a fixed piston that does not touch the sidewalls. Approximately 0.4 ml of blood is placed in the cup, which rotates around the piston until a bridging clot has formed. A paper tracing provides the amount of power required to maintain the rotational movement of the cup. TEG examines whole blood coagulation, and provides information on how fast the clot forms, the speed of clot growth, clot strength, and whether clot strength is maintained or breaks down early. These are the key elements in determining the likelihood of platelet and clotting factor deficiencies, as well as increased fibrinolysis

(17). This device has influenced both blood utilization and reoperation rates in cardiac surgery, and its successful use is being increasingly reported in the trauma literature (18, 19).

## ACIDOSIS

It is important to correct acidosis by aggressively resuscitating the patient to appropriate end points. The current understanding of the pathophysiology of hemorrhagic shock is based on a series of studies by Wiggers. Controlled hemorrhage led to a reduction in oxygen consumption ( $\dot{V}O_2$ ), and on reperfusion, to an increase in  $\dot{V}O_2$  above baseline, as if "in the nature of repayment of an oxygen debt." When the duration of shock was prolonged the subsequent hypermetabolic phase did not occur, and the animals went on to die. This suggests that shock eventually results in intracellular metabolic derangements in oxygen and substrate utilization that are no longer responsive to increases in oxygen delivery.

This phenomenon was demonstrated in a study of trauma patients who were adequately resuscitated. One group had persistently elevated lactate concentrations and reduced oxygen consumption and extraction. Compared with the group whose acidosis and extraction defect had resolved, the multiple organ failure rate was 35% compared with 5% ( $p < 0.001$ ), and mortality was 50% compared with 9% ( $p < 0.01$ ) (20).

Studies demonstrate a strong correlation between the development of coagulation abnormalities and the duration of hypotension. There are also studies demonstrating that hypoperfusion is associated with consumptive coagulopathy and microvascular bleeding independent of the amount of blood loss (15). In one study shock-induced acidosis lasting greater than 150 min independently resulted in significant prolongation of the PTT and decreases in factor V activity (21).

## ABDOMINAL COMPARTMENT SYNDROME

Compartment syndrome is defined as increased pressure within a closed space sufficient to impair tissue function within that space. Abdominal compartment syndrome (ACS) often occurs in severely injured patients, especially those who undergo abbreviated laparotomy with abdominal packing. It is reported to occur in as many as 14% of patients who undergo laparotomy and who are found to have serious intestinal or hollow organ injury (22). Early recognition may prompt decompressive laparotomy, leaving an open abdomen that is covered by a temporary prosthetic material. This typically provides immediate improvement in organ function and physiologic status.

ACS may be recognized by the presence of a tensely distended abdomen, elevated peak airway pressures, inadequate ventilation, and hypoxia. However, these findings are relatively nonspecific. Intraabdominal pressure can be monitored by instilling 100 ml of saline into the catheterized bladder, which at that volume remains a passive reservoir. The intraabdominal portion of the bladder can then serve as a transducer to record abdominal pressure, without any contribution from its own musculature (23). The tubing is held parallel to the patient at the level of the pubis until urine forms a meniscus distal to the sampling port. A clamp is placed distal to the port and a needle is inserted through the port and connected to a water manometer or electronic pressure transducer.

A pressure greater than 30 mm Hg is invariably associated with oliguria due to a decreased renal blood flow associated with increased renal venous pressure and a calculated increase in renal vascular resistance (24). ACS is also associated with a 30–40% decrease in cardiac output related to decreased venous return and an increase in systemic vascular resistance (23, 25). Experimental studies demonstrate decreases in splanchnic

blood flow as great as 75%, with gut mucosal acidosis that is independent of cardiac output. In humans there is an independent correlation with ACS and subsequent multiple organ failure (26).

An intraabdominal pressure of 25 mm Hg can also cause elevation in intracranial pressure, presumably by increasing central venous pressure, resulting in significant decreases in cerebral perfusion (27). Maintenance of a cerebral perfusion pressure greater than 70 mm Hg is now widely recognized as a vital component of management of traumatic brain injury.

ACS has profound effects on pulmonary function, with progressive hypoxemia and CO<sub>2</sub> retention, or a requirement for very high peak airway pressures to maintain adequate tidal volume. The risk of barotrauma correlates most strongly with transpulmonary pressure, rather than peak or static airway pressure. When intraabdominal pressure is elevated increased pleural pressure should be assumed.

One experimental study demonstrated a linear relationship between intraabdominal and pleural pressure as intraabdominal pressure was increased. When abdominal pressure reached 25 mm Hg pleural pressure was 18 mm Hg, which corresponds to 24.5 cm H<sub>2</sub>O pressure (24). If the amount of end-expiratory pressure is below pleural pressure, the patient is experiencing negative, rather than positive, end-expiratory pressure, which may result in progressive alveolar collapse. Many of these patients develop refractory hypoxemia due to inappropriate avoidance of sufficient positive end-expiratory pressures to maintain functional residual capacity. Higher levels of positive end-expiratory pressure may be needed, as long as circulation can be supported.

## SUMMARY

The surgical approach to the most injured patients has changed in recent years. Many patients arrive in the intensive care unit with problems that in the past would have been definitively addressed in the operating room, or led to the patient's demise due to continued attempts to complete all surgical procedures, despite deteriorating physiology. As a result, the triad of hypothermia, acidosis, and coagulopathy, along with the frequent complication of abdominal compartment syndrome, are critical factors that require correction in the intensive care unit. Prompt correction is necessary not only to allow expeditious completion of required surgical procedures, but because this triad, unless interrupted, invariably leads to death during resuscitation.

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