Damage control surgery

Three approaches to a complex problem

A surgeon facing a complex surgical challenge has three philosophical concepts available**:**
(1) Avoidance,
(2) Aggressive attempts at complete reversal or control of the condition, and
(3) Temporizing manoeuvres.

Avoidance:-

Avoiding a complex problem is achieved by both individuals and corporations. Denying that a complex surgical problem exists is a basic form of avoidance. Unfavourable outcomes are often outgrowths of avoidance.
Another avoidance tactic is "automatic" early consultations or aggressive transfer to another facility when the sending facility does not possess the necessary equipment or resources to adequately address a surgical problem. Such transfer is appropriate, when necessary damage control tactics applied to ensure that the patient makes the transfer with minimal deterioration in condition. At times, an approach to transfer is accomplished without any temporizing measures, and the transfer is a deliberate attempt to get rid of a patient with a problem that either frightens or is beyond the scope of the physician and /or the hospital.

Attempts at complete correction of an acute complex surgical problem:-

The application of extracorporeal support, extensive resections, aggressive fluid (including blood) replacement, surgery, and primary extensive reconstructions was reported by many developing trauma centres.
Such aggressive management may lead to multiorgan failure, hypothermia, coagulopathy, or "failure to thrive."
These problems related to the advance in technology and management. These conditions, encountered in the resuscitation area of the trauma centre (emergency centre), the operating room, and the intensive care unit.

- **Hypothermia ,Coagulation and Acidosis**

All surgeons have intraoperatively encountered a patient who exhibits the triad of **HYPOTHERMIA, COAGULOPATHY, AND ACIDOSIS**. These closely linked findings are not an initial cause of a catastrophe but a relatively late manifestation of significant alteration in a patient's molecular, cellular, and hemodynamic equilibrium .Once the condition is apparent and/or discovered, the patient already is at significant risk for both death and complications.
This triad secondarily and rapidly leads to secondary complications.

- Every capillary in the surgical field seems to manifest exsanguinating haemorrhage.
- Cardiac arrhythmia of various descriptions occurs.
- A malodorous smell appears over the operative field.
- The organs in the operative field appear dusky and puffy.
- Venous engorgement and venous pooling appear in visible organs.

- The tissues are cool to the touch.
- The surgeon's pulse increases with a feeling of impending death.
- It is logical that the surgeon at this point wishes to terminate the operation, if possible.

Considerable thought is given to how this encounter could be prevented or avoided .Thus, the damage control concepts emerged in many of the nation's cities almost simultaneously

- By the time the triad of hypothermia, acidosis, and coagulopathy is manifested, the systemic responses that led to this triad already have been present for many minutes and are almost irreversible if the usual course of the operation is continued.
- The trauma patient often arrives in the operating room with a core temperature of less than 34°C. Trauma patients undergoing prehospital crystalloid fluid resuscitation have manifest abnormal partial thromboplastin time, prothrombin time, and platelet counts on the initial blood count.
- Patients in "shock" and even those with mild hypotension often have measurable arterial pH determinations in the "acidotic" range, and the same blood gases often report base excesses greater than 8 in the "shocked" trauma patients. This fact is often either unknown or ignored
- **Standardized damage control** approach provides a protocol for haemorrhage control, impedance of continuing enteric spillage, and planned secondary (and tertiary) operations.

Metabolic Acidosis

The predominant physiologic defect resulting from persistent or repetitive bouts of hypo-perfusion is metabolic acidosis. From the moment of energy transfer, normal cell physiology is altered and a shift from aerobic to anaerobic metabolism takes place, resulting in lactic acidosis.

Hypothermia

Hypothermia is an inevitable pathophysiological consequence of severe injury and subsequent resuscitation. Heat loss in the field, resuscitation manoeuvres, injury severity, age, exposure of body cavities during surgery, impaired thermogenesis, and degree of transfusion have all been implicated as important factors.

Adverse effects such as cardiac arrhythmias, reduction in cardiac output, increase in systemic vascular resistance, and a left shift in the oxygen-haemoglobin saturation curve have been described

Coagulopathy

It is clear that virtually every aspect of normal physiologic clotting is affected in the cold, acidotic exsanguinating trauma patient. It is also clear that a predominant factor governing coagulation is body temperature. The clotting cascade, governed by a series of temperature sensitive serine-dependent esterase reactions, becomes relatively inhibited during hypothermia

Hypothermia has both a qualitative and a quantitative effect on the clotting cascade, resulting in a decrease in the rate of cascade reaction and a decrease in the production of factors.
Platelet function is also affected by hypothermia **w**hich is shown to decrease plasma thromboxane levels and increased bleeding times.
Activation of the fibrinolytic system has been demonstrated following missive' transfusion& shock.
The dilutional effects of massive transfusion, defined as replacement of greater than 100% of a patient's blood volume. In this setting, thrombocytopenia and reduction in factors V and VIII have been well documented.
As expected, hypothermia alone increased both PT and PTT and the effect was even more dramatic when combined with haemodilution
Metabolic acidosis, hypothermia, and dilution all contribute in varying amounts to the development of coagulopathy and the progression to death.

THE DAMAGE CONTROL APPROACH
The term *damage control* describes a systematic three-phase approach designed to disrupt the lethal cascade of events leading to the death by exsanguinations.

Damage control part I, consists of immediate exploratory laparotomy for control of haemorrhage and contamination using the simplest techniques. Definitive reconstruction is delayed, and the application of intra-abdominal packing to all dissected surfaces and injured organs is followed by a rapid, simple closure.
Damage control part II consists of a secondary resuscitation in the intensive care unit that is characterized by maximization of hemodynamic, core re-warming, correction of coagulopathy, complete ventilatory support, and continued injury identification.
Damage control part III: When normal physiology has been restored, damage control part III, reoperation for removal of intra-abdominal packing with definitive repair of abdominal injury and closure, can take place.
If necessary, extra-abdominal injuries may be repaired at this time.

Patient Selection

Early identification of patients who require damage control promotes optimal results.

In an effort to identify these patients, *conditions, complications* and *critical factors* must be considered.

KEY FACTORS IN PATIENT SELECTION FOR DAMAGE CONTROL

1. **Conditions**
2. **Complexes**
3. **Critical Factors**

Conditions

- High-energy blunt torso trauma
- Multiple torso penetrations
- Hemodynamic instability
- Presenting coagulopathy and/or hypothermia

Complexes

- Major abdominal vascular injury with multiple visceral injuries
- Multifocal or multicavitary exsanguination with concomitant visceral injuries
- Multiregional injury with competing priorities

Critical Factors

- Severe metabolic acidosis (pH < 7.30)
- Hypothermia (temperature < 35°C)
- Resuscitation and operative time > 90 minutes
- Coagulopathy as evidenced by development of non-mechanical bleeding
- Massive transfusion *(*> 10 units packed red blood cells)

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