Back ground and Historical Prespective:-

Damage control surgery is one of the major advances in surgical technique in the past 20 years. The principles of damage control have been slow to be accepted by surgeons around the world, as they contravene most standard surgical teaching practices-that the best operation for a patient is one, definitive procedure. However it is now well recognized that multiple trauma patients are more likely to die from their intra-operative metabolic failure than that from a failure to complete operative repairs. Patients with major exsanguinating injuries will not survive complex procedures such as formal hepatic resection or pancreaticoduodenectomy., ***(Rotondo et al,1993).***

The challenge of damage control surgery is that patients die from a triad of coagulopathy, hypothermia, and metabolic acidosis. Once this metabolic failure has become established, it is extremely difficult to control hemorrhage and correct the derangements. If the patient is to survive, the operation must be foreshortened so that they can be transferred to a critical care facility where they can be warmed and the hypothermia and acidosis is corrected. Once this is achieved the definitive surgical procedure can be carried out as necessary the "staged procedure", (***Moore, 1996).***

The principles of the first 'damage control' procedure are control of hemorrhage, prevention of contamination and protection from further injury. Damage control surgery is the most critically demanding and challenging surgery, a trauma surgeon can perform.

There is no margin for error and no place for careless surgery,***(Hirshberg et al,1995),*** The technique of abdominal packing with planned reoperation was first described in the early 1900s as perihepatic packing, At that time, liver lacerations were frequently packed with absorbable or nonabsorbable materials sutured in place. Removal of the packing often resulted in uncontrollable hemorrhage**,** ***(Schroeder,1906).*** However, perihepatic packing fell into disfavor in this era because of reports of hemorrhage, sepsis, and necrosis,(***Moore,1996).***

In 1968, hepatic packing was explored again in a case study in which a patient with bilobar hepatic injuries survived after his injury, he was treated with gauze packing that was removed 6 days later. In 1981 Feliciano et al. reported the survival in nine of ten patients who underwent temporary laparotomy pad tamponade, for hepatic injuries.The staged laparotomy approach was extended to nonhepatic abdominal injuries in 1982, when abdominal packing followed by rapid abdominal closure was used for treatment of coagulopathy,(***Stone et al,1983***).

One benefit of rapid abdominal closure is the prevention of hypothermia and further heat loss, rather than the tamponade effect caused by early closure***,(Moore,1996).***

In the 1970s and 1980s, surgeons generally made every attempt to continue aggressive surgical intervention at first operative encounter until complex surgical problems were completely resolved. Trauma centers used extreme measures such as extensive resections and reconstruction, and aggressive fluid

and blood product replacement. Surgeons were able to continue operating until serious metabolic derangements developed, most often characterized as the trauma triad of death: hypothermia, coagulopathy, and acidosis***,(Mattox,1997***).

Often, the patient survived the initial operation but died in the SICU from continued hemorrhage or multiorgan failure. During the 1980s, civilian trauma from multivisceral, high-energy mechanisms increased. This extensive multivisceral trauma frequently required lengthy and complex repairs with bleeding, often difficult to control. Traditional operative sequence was not achieving the desired level of success, and other methods to decrease morbidity and mortality were explored, (***Mattox,1997***).

Staging complex surgery during a period of physiologic instability is the primary advantage of damage control surgery. The modern concept of "damage control" with abbreviated laparotomy and planned operation allows for interruption of the vicious cycle of the trauma triad of death,***(Mattox, 1997).(Fig.1)***

Physiologic Triad of Death by Exsangunation:-

Despite of the development of trauma centers and application of standard methods of resuscitation and operative intervention the mortality rate in patients with devastating torso truma still high, due to physiologic instability. Best efforts at resuscitation and operation often lead to a lethal cascade of events including metabolic acidosis, hypothermia and coagulopathy “Trauma triad of death” ***,(Rotondo, 1995).***

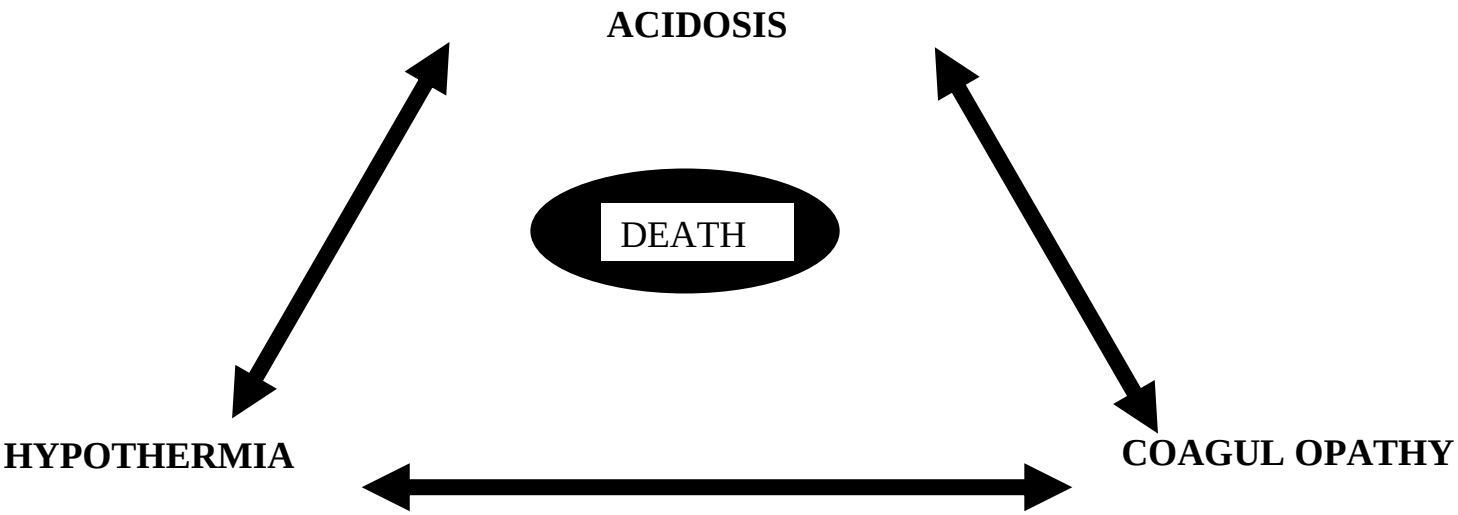


Fig1: Trauma Triade of death (Rotondo and Zonies,1997)

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 or discovered, the patient is already at high 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 types occurs, and malodour smell covers the operative field. The organs in the operative field appear dusky and puffy. Venous engorgement and venous polling appear in visible organs. The tissues are cool to touch. It is logical that the surgeon at this point wishes to terminate the operation if possible***,(Moore,1996).***

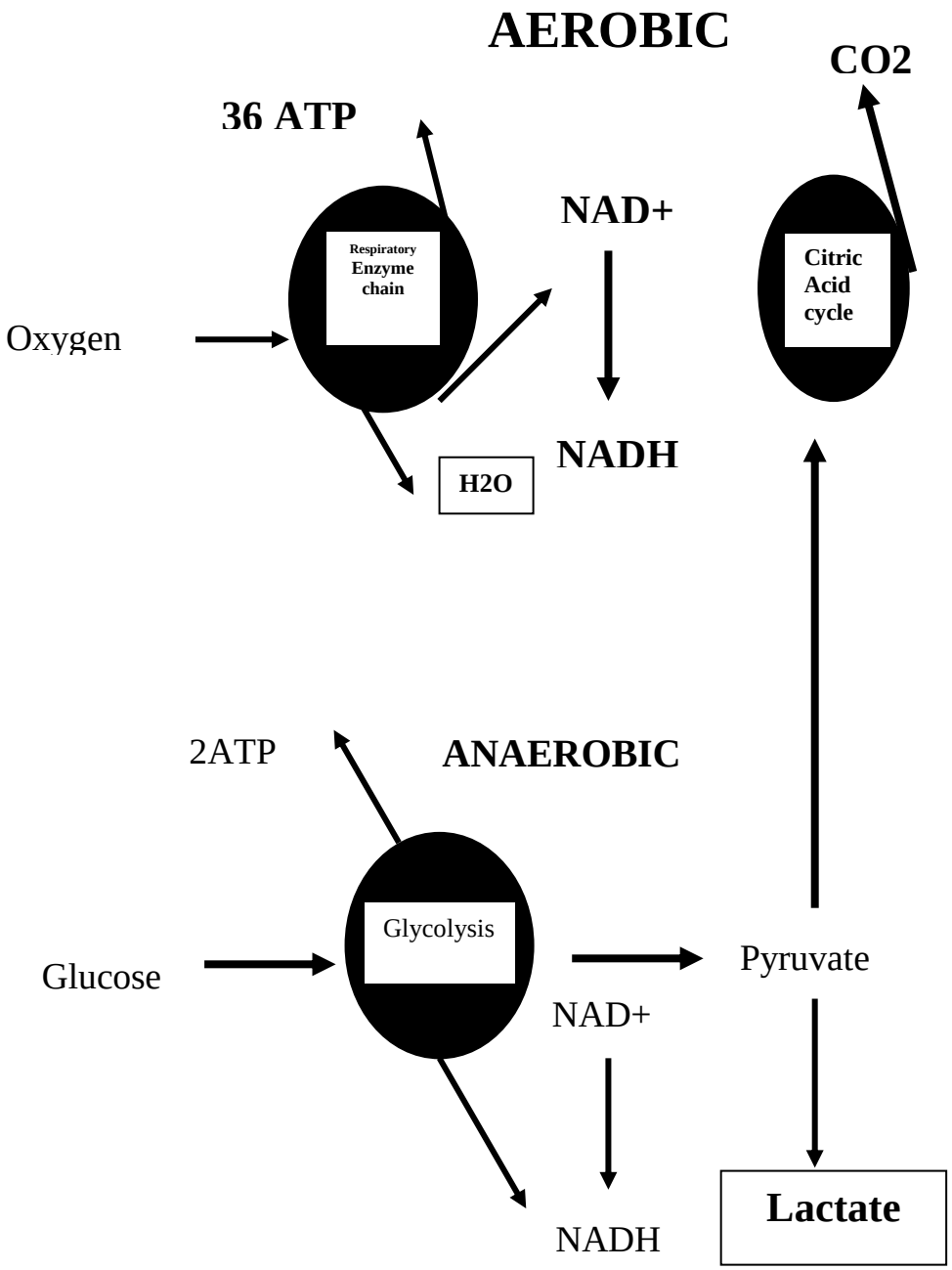
The decision to apply damage control techniques should be made very soon after the operation is begun, or even before the operation is begun, the later damage control is applied, the less successful is the out come***,(Hirshberg et al,1995***).

Trauma Triad of Death:

1-Metabolic acidosis :-

The predominant physiologic defect resulting from persistent or repetitive bouts of hypoperfusion is metabolic acidosis, as a result of trauma, normal cell physiology is altered and a shift from aerobic to anaerobic metabolism takes place resulting in lactic acidosis,(***Moore,1996) .Fig.2***

Lactate clearance has been closely correlated with the degree of oxygen delivery and consumption as endpoints of resuscitation .Abramson demonstrated that the rate of lactate clearance predicts survival rate in severely injured trauma patient, the survival rate is 100% in patient with lactate clearance at the first 24 hours while 14% only survive with lactate clearance at 48 hours, ***(Abramson et al, 1993).***



(Fig.2) Cellular metabolism in the shock state
(Rotondo and Zonies, 1997)

2-Hypothermia:-

It is defined as core temperature below 35^{o}C, when O2 consumption is limited by shock in the presence of low temperature, heat production can not offset and hypothermia results***,(Gentilello et al,1991).***

Hypothermia is inevitable pathophyiologic consequence of severe injury and subsequent resuscitation.

Causes: ***1-Conductive heat loss.***

2-Evaporative heat loss.

3-Convective heat loss.

1-Conductive heat loss is due to (***Bernabei et al , 1992).***

- Impaired thermogenesis
- Transfusion of unwarmed fluid and Blood Products
- Exposure of body cavity during surgery.

2-Evaporative heat loss;

The heat evaporates from visceral and serosal surfaces and from blood saturated drapes

3-Convective heat loss;

The heat radiates directly from body surface to environment, However it is trivial.

The relationship between hypothermia and death is identified by Soltman who found 40% mortality in 100 postoperative patients

admitted to ICU With Hypothermia for more than 4hs, ***(Soltman et al, 1985).***

Effects of Hypothermia:

- Cardiac arrhythmia.
- Reduction in cardiac out put.
- Increase in systemic vascular resistance.
- A left shift in the oxygen Hemoglobin saturation curve ***(Just et al, 1992).***

The correction of the hypothermia is essential for survival, as neihter coagulopthy nor acidosis can be corrected untill the patient's core temperature reaches normal level.

3-Coagulopathy:-

It is clear that normal physiologic clotting is affected in the cold acidotic trauma patient. The clotting cascade becomes relatively inhibited during hypothermia, (***Patt et al,1988).***

The standard assays used to assess clotting function are partial thromboplastin time (PTT), prothrombin time (PT), and bleeding time (BT), which are altered with decreasing body temperature.

Rohrer and Natale showed a dramatic increase in mean PT and PTT with decreasing temperature in pooled plasma from normal volunteers and found that hypothermia has both qualitative and quantitative effect on the clotting cascade resulting in decrease in the rate of cascade reaction and decrease in the production of clotting factors, (***Rorher and Natale, 1992***).

Platelet function is also affected by hypothermia .In 1987 Valeri declared that hypothermia in an animal model decrease plasma thromboxan levels and increases the bleeding time, this implies that cyclo-oxygenase pathway and hence, the balance between thromboxan and prostacyclin are affected by changes in the temperature, (***Valeri et al, 1987) .***

Fibrinolytic system is also activated in massive transfusion, shock, and hypothermia.In severely injured patients especially with head injury there is elevation of D-dimer levels (Fibrinogen degradation products),prolongation of PT, and reduction of both antithrombin and fibrinogen levels,(***Kearney et al,1992***).

Finally,metabolic acidosis,hypothermia,and hemodilution lead to coagulopathy and progression to death,so each of these factors must be controlled to prevent or stop lethal cascade of events in trauma patient, Fig.1(***Moore,1996***).