TIMING OF FRACTURE FIXATION IN POLYTRAUMA PATIENTS - ANAESTHESIOLOGIST'S PERSPECTIVES.

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The timing of definitive fixation of major extremity fractures in the polytrauma patient has been the subject of debate for the past 4 decades. Advances in orthopaedic trauma surgery, along with pivotal improvement in anesthesia and critical care medicine have increasingly enabled orthopaedic surgeons to perform definitive operation on initial presentation in polytrauma patients. The whole burden of taking care of a polytrauma patient, then falls on the shoulder of the Anesthesiologist. Apart from the problems of prolonged surgery and difficulty in managing the blood volume deficit, the Anesthesiologist is faced with a unique problem of increased incidence of respiratory failure induced by fat embolisation from the fractured bones especially long bones and pelvis. There is clinical evidence that, the polytrauma patient, who is already exposed to fat embolisation because of primary injury, may get worsened because of intra medullary reaming done during definitive fixation .So the question arises whether to submit them for immediate definitive fixation of bones or submit them for temporary bone splinting followed by definitive surgery later.

PATHOPHYSIOLOGY

When a high velocity force hits a person, the long bones get fractured and the fat droplets in the medullary cavity enter into the blood circulation. This entry is due to increase in the intra medullary pressure in the long bones just before fracture. The amount of embolism is much variable depending on trauma energy, on the kind of fracture (open fractures cause less fat embolism) and also on orthopaedic procedure used (like reaming, screws, prosthesis). At that moment, bone narrow vessels disrupt, as well as their adipose cells, from which a great amount of fat droplets result and may get embolised through medullary venules and sinusoids. Sinusoids seem to be more likely to receive embolism because, as they have walls attached to bone trabeculae and they remain open and does not collapse as venules do. If fat embolism is massive enough to occlude about 80% of the pulmonary capillary meshwork, then acute cor pulmonale results and most often the patient succumbs.

Although long bone and hip fractures are the main sources of fat embolism, recent studies demonstrated that surgical manipulation of the medullary canal can also cause fat embolism (FE)..The manipulation may be in the form of medullary reaming or applying prosthesis with or without cement. The simple reaming of the medullary canal may cause great rises in intra medullary pressure, which can reach a value up to 650mm of Hg. (The normal intra medullary pressure is 30 to 50 mm of Hg.). As a result in almost all the patients submitted to medullary channel reaming, FE occurs.

During prosthesis cementation, intra medullary pressure can reach values of 650-1500mm of Hg and embolization may last for more than 20 minutes. Fortunately the vast majority of these patients do not really develop acute cor pulmonale. But they may present with

haemodynamic and respiratory problems during this period, such as: severe arterial hypotension , cardiac arrythmias, increased pulmonary vascular resistance, increase in shunt fraction and consequently a reduction in paO2.

BIO CHEMICAL PHASE OF FAT EMBOLISATION:

As if fat embolism is an expected physiological phenomenon, lung alveolar cells are provided with the ability to produce lipase. Thus, as soon as fat droplets arrive and obstruct pulmonary capillaries, they begin to get hydrolysed by pulmonary lipase,that in general eliminates fat emboli within about 3 days. The hydrolysis of the fat, embolised in the lungs, however releases fatty acids (palmitic, oleic, and stearic), which are usually neutralized and carried away by albumin. This neutralization by albumin prevents the development of fulminant fat embolism syndrome despite showers of fat emboli. In some patients these fatty acids fail to be neutralized by albumin, subsequently promoting severe injuries in pulmonary alveoli and capillaries.

It is postulated that fatty acids played a direct traumatising role on alveolar endothelial cells. As a great concentration of calcium ions exists on intercellular joints, and as fatty acids have great affinity to this element, these acids would bind to calcium ions, because of which intercellular joints rupture. This would result in the establishment of diffuse areas of haemorrhage and oedema in pulmonary interstitium and alveolar spaces. Neutrophils also play a role in the genesis of lung injury. In the presence of free fatty acids, an enhanced adherence occurred between neutrophils and pulmonary capillary endothelium because of the integrins CD11b and CD18.

The injured pneumocytes stop producing surfactant, favouring lung collapse. The end result of this complex cell and physiochemical changes is the establishment of exhaustive lung areas in which alveoli are perfused but not ventilated(shunt) and of other areas where the opposite occurs(dead space effect).

The fat embolisation seems to be an exclusive feature of femur reaming, rather than tibial reaming. The reason being, the bone marrow cavity of tibia is smaller, the configuration of tibia allows more back streaming of the content and the venous drainage system in the distal tibia is much less extensive than in the supra condylar area.

Secondary injury- “hit”

When the fat embolism incidence following long bone fractures is quoted between 30-90%, the question of when to fix the fracture arises. As the definitive surgery for long bone fracture fixation involves medullary reaming, there is every possibility of second showering of fat embolism into the pulmonary circulation which can act as a Second Hit.

RATIONALE FOR DELAYED FIXATION- THE 1960s

In the 1960s, immediate stabilization of long bone fracture in the patient with multiple traumatic injuries was associated with an unacceptably high mortality rate. The development of fat embolism syndrome and associated pulmonary dysfunction was feared. Perioperative cardiovascular and pulmonary support was not well established, leading to mortality rates up to 50%. As a result, long bone fractures were initially treated with splints, casts or traction until the systemic effects of fat embolism syndrome resolved. Definitive surgical stabilization was often delayed for 10-14 days until the pulmonary, cardiovascular and neurologic symptoms and the coagulation profile had stabilized.

In 1967, Kuntscher provided three recommendations for intramedullary stabilization of major fractures.

1. Do not nail as long as symptoms of fat embolization are present.
2. Take special precautions for patients with multiple fractures and extensive injury of soft tissues.
3. Do not nail immediately, but wait a few days.

NEGATIVE EFFECTS OF DELAYED FIXATION:

Delayed fixation of major fractures is fraught with local and systemic implications. Without adequate fixation, the patient cannot be mobilized and is often forced into supine recumbency for prolonged periods. This can result in dysfunction of multiple organ systems leading to a variety of disorders, including pneumonia, decubitus ulcers, vascular abnormalities, psychological disturbance and gastro-intestinal stasis. Electrolyte abnormalities frequently appear especially hyponatremia.

Frequently musculoskeletal outcomes are compromised when fracture surgery is delayed. Prolonged immobilization prevents inititation of comprehensive physiotherapy which leads to profound stiffness and disuse muscle atrophy.

RATIONALE FOR EARLY FIXATION: THE 1980's

In 1980s a radical shift occurred in the treatment paradigm of polytrauma patients. Better outcomes were achieved when intra medullary nailing of femur fracture was performed within the first few days after admission. Along with the better understanding of patho physiology after trauma, major improvements were made in the general physiologic support of severely injured patients. Ventilation strategies improved and allowed orthopaedic surgery to be performed earlier than previously.

The principle of early fixation surgery sometimes was interpreted too literally, however, resulting in an overly aggressive treatment protocol in the polytrauma patient. Surgeries were performed within 24hrs of admission, a practice that appeared to be associated with an increased complication rate. The beneficial effects of fracture fixation were often negated by the harm inflicted to the overall physiology of the patient as a result of lengthy operation associated with substantial blood loss. It led to further consideration of the timing in fracture fixation in the polytrauma patient.

DAMAGE CONTROL ORTHOPAEDICS

Damage control in general trauma surgery includes packing the major sources of haemorrhage rather than performing immediate, lengthy, definitive procedures of the visceral organs.
As part of the damage control philosophy, immediate life saving interventions, directed at stopping bleeding are applied, after which resuscitation and further stabilization are performed in the ICU. Only after the overall physiology has improved is definitive interventions performed. This change in trauma practice resulted in improved survival rates. Initial surgery was done with the goal of achieving rapid skeletal stabilization of major orthopaedic injuries to stop the cycle of ongoing musculoskeletal injury and to control haemorrhage. This approach was termed "Damage Control Orthopaedics"

CURRENT RECOMMENDATION:

Patient Assessment:

To decide upon the orthopaedic management, patients are clinically assessed about their physical status and classified as:

Stable - Grade I
Borderline - Grade II
Unstable - Grade III
Extremis - Grade IV

TABLE

Criteria used to determine the clinical condition of polytrauma patients

		PATIENT STATUS			
CRETERION	PARAMETER	STABLE	BORDERLINE	UNSATBLE	IN EXTREMIS
SHOCK	B.P in mm of Hg	≥100	80 – 100	<90	≤70
	BLOOD UNITS GIVEN IN A 2 HR PERIOD	0 – 2	2 – 8	5 – 15	>15
	LACTATE LEVELS (mg/dL)	NORMAL RANGE	2.5	>2.5	severe acidosis
	BASE DEFICIT LEVEL (mmol/ L)	DO	---	-----	>6 – 8
	ATLS CLASSIFICATION	I	II – III	III – IV	IV
COAGULATION	PLATELET COUNT (/mm^3)	>1.1 L	90,000 – 1.1 L	70,000 – 90,000	<70,000
	FACTOR II & V	90 – 100 %	70 – 80 %	50 – 70%	<50%
	FIBRINOGEN(g/L)	>1	1	<1	DIC
	D – DIMER(µg/ml)	NORMAL RANGE	ABNORMAL	ABNORMAL	DIC
TEMPERATURE	^{0}C	<33	33 – 35	30 -32	≤30
SOFT TISSUE INJURIES	LUNG FUNCTION (PaO2/FIO2)	350 – 400	300 – 400	200 – 300	<200
	CHEST TRAUMA SCORES(AIS)	1 OR 2	≥2	≥3	≤30
	ABDOMINAL TRAUMA	NONE	SLIGHT	MODERATE	SEVERE
	PELVIC TRAUMA	NONE	SLIGHT	MODERATE	SEVERE
	EXTERNAL	AIS I –II	AIS II – III	AIS III –IV	CRUSH INJURY

AIS – ABBREVIATED INJURY SCORE ATLS – ADVANCED TRAUMA LIFE SUPPORT

Surgical priorities:

The first surgical priority is to save the patient's life and when feasible, the limb, as well as to limit the time in the operating room to less than 2 hours. Within this surgical window, open fractures should be debrided and stabilized with an external fixator. A splint may be sufficient for upper extremity injuries.

If the patient is in extremis (Grade IV) , haemorrhage control is paramount, followed by stabilization of vital parameters in the ICU. Major fractures are considered to be a secondary priority, if possible, stabilized with external fixators.

In the borderline patient who responds to resuscitation, definitive procedures (intra medullary nailing) can be performed but within an upper surgical limit of less than 2 hrs. The patient with several lower extremity fractures should be continuously reassessed, with particular attention paid to the following parameters:

- PaO_2/ FIO_2 should not drop below 250mm of Hg
- Temperature should not be less than 32°C
- Requirement of fluids (should not exceed 3L or 5 units of blood)
- Absence of significant coagulopathy

Provided that the patient maintains these levels, the surgeon may address the next major fracture. Otherwise a temporizing approach may be selected.

In the stable patient all the fracture can be definitely fixed within the first day.

STRATEGY IN PATIENTS WITH HEAD INJURY:

Following significant head injury, the brain loses the capacity for auto regulation of blood flow in zones of contusion. These patients are at greater risk for decreased CBF during the first 12-24 hours.

Intra operative hypotension is an important risk factor for secondary brain injury. For that reason, regional anaesthesia with extensive sympathetic block is to be avoided in these patients. The primary goals of management of traumatic brain injury are maintenance of adequate cerebral perfusion pressure and avoidance of secondary insults.

The priority between head injury and long bone fracture rests on individual basis, depending upon the severity of each.

STRATEGY IN PATIENTS WITH CHEST INJURY:

Chest injury in the polytrauma patients consists of either chest wall fracture or lung contusion. Both may precipitate respiratory failure because of different reasons (flail chest, restriction of breathing due to pain, ARDS)

Early diagnostic studies may not adequately reveal the event of the evolving lung injury. Even when ABG and chest x-ray are normal pulmonary contusions may occur as a result of immune response, resulting in ARDS

The timing of fracture management in these patients remains controversial. Because all authors agree that severe chest injury represents a risk factor for ARDS, ruling out severe lung contusion by early CT scan is advisable. Presence of severe chest injury with established ARDS precludes any major surgery. Other cases should be individualized and decision taken.

PREVENTION OF FAT EMBOLISM SYNDROME (FES)

Regarding the FES that may occur after orthoplastic or bone reaming, today many surgical maneuvers exist for reducing the risk. As expected all of them aim to avoid pressure rise inside medullary channel.

1. Medullary channel depletion: Medullary channel cleaning with saline solution in high pressure pulsed streams, followed by aspiration of medullary content.
2. Fluted rods: when comparing the reaming with either cylindrical rods or fluted rods, definitely cylindrical rods cause much rise in intra medullary pressure.
3. Venting: venting the medullary canal before filling with cement by making a drill or with suction catheter, maintaining a negative pressure inside thus avoiding intra medullary pressure rise.

4. Retrograde filling: filling the medullary channel with the cement, from the distal end of prosthesis.

5. Viscosity of cement: Using low viscosity cements avoid rise in IMP

6. Distal over drill: Entrance port at distal femur is enlarged to 12.mm from the usual 8mm. This is called as over drill which helps to vent out rise in IMP

7. Prosthesis without cement: in selected cases.

8. Cortico steroids: They may play an important protection role when administered before FES is completely established. But this view is not completely accepted by everybody.

SUMMARY:

The patient with polytrauma may be classified as

STABLE
BORDERLINE
UN STABLE
IN EXTREMIS

- Early definitive fixation is recommended for the stable patients and in borderline and unstable patients who respond well to initial resuscitation.
- In patients with severe haemorrhagic shock or any other life threatening condition, prolonged surgical procedures should be avoided, and staged fracture fixation to be done. The damage control approach, which uses external fixation as a primary tool, may be applied in such cases.
- For the patient who presents as borderline or in poorer condition, a multi disciplinary approach is required to determine the best timing of musculoskeletal case.

CONCLUSION:

The decision to fix the fractures in the polytrauma patients in the single sitting has to be decided, considering the overall physical status of the patient and the availability of a multi disciplinary care.

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References:

1. Journal of American Academy of Orthopaedic Surgeons- September 2009, Vol 17: No 9. Hans – Christoph Papetal et al
2. Fat embolism: A review for current orthopaedics practice- Luiz Tarciso et al – Acta ortho brasilia – 13(4)- 2005.