VASCULAR INJURIES IN A TERTIARY CARE CENTER CRITICAL ANALYSIS FOR IMPROVING LIMB SALVAGE

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CERTIFICATE

This is to certify that this dissertation on **"VASCULAR REPAIR IN TRAUMA – CRITICAL ANALYSIS FOR IMPROVING LIMB SALVAGE"** is an bonafide work done by **Dr.P.S. BALA KUMAR** under my guidance during the time period 2003-2006 This has been forwarded for the award of M.Ch., Degree in Vascular Surgery by the Tamil Nadu Dr. M.G.R. Medical University.

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CONTENTS

MANAGEMENT OF VASCULAR INJURIES

HISTORY & EPIDEMIOLOGY

With continued urbanization over last few decades there has been an alarming and unabated increase in the incidence of traumatic vascular injuries. Between 1-5% of trauma cases involved injury to a major vascular structure which confronts us with the twin problem of haemorrhage on one hand and ischemia on the other hand. The lives and limbs jeoparadised in these critical vascular injuries often belong to young people and their salvage may mean many years of satisfactory function.

Though most advances in treatment of vascular injuries has evolved from war surgery it is quite surprising that it was not until **1950s** that effective vascular repair was attempted. In an analysed series of 2471 acute arterial wounds from **world war-2** end to end anastomosis was accomplished in only three instances. In fact ligation of a artery was the main stay of treatment then and resulted in an amputation rate of about 40%. Reconstruction of the artery was first employed routinely during the **Korean war** and the amputation rate dramatically dropped to 13%. Further improvement was achieved in **Vietnam war** largely as a result of further reduction in time lag and availability of expert vascular surgeon these lessons of war time surgery have been applied in peace time injury and restortation of normal blood flow and not merely saving life is prime objective now.

ERA	MAINSTAY OF TREATMENT	TREATING PHYSICIAN	AMPUTATION RATE
World War – 2	Ligation	Gen. Surgeon	> 40%
Korean War	Re-construction	Gen. Surgeon	13%
Vietnam War	Re-construction	Vascular Surgeon	5%
Current Civilian Series	Re-construction	Vascular Surgeon	<5%

Currently in large civilian series a 95% limb salvage rate has become the standard.

DEMOGRAPHIC AND ETIOLOGIC CHARACTERISTICS OF VASCULAR INJURIES IN RURAL AND URBAN SETTINGS

CHARACTERISTIC	RURAL	URBAN
Age (mean, yr)	51	31
Gender (% male)	79	84
Race (% nonwhite)	38	60
Mechanism Blunt trauma (%)	43	27

EPIDEMIOLOGY

The actual incidence of vascular trauma in most European countries is unknown. In some countries, national vascular registries have been established to gain nationwide information on vascular procedures and outcome, especially because these injuries are not always treated by vascular surgeons.

Based on the information from the Finnish Vascular Registry (Finnvasc), covering the years 1991 into 1999, the annual incidence of vascular injuries was 1.3 per 100,000 inhabitants, varying from 0.9 to 2.0 (J.P. Salenius, evaluation of the incidence of vascular trauma in the past 30 years showed that the total incidence had increased from 1.1 to 2.3 per 100,000 inhabitants, and this increase was almost totally ascribable to the increase in iatrogenic vascular injuries.

European Experience

Country	Years	No. of Patients	Trauma Mechanism (%)		
			Penetrating	Blunt	Iatrogenic
Spain	1980-1995	451	20	40	40
Sweden	1987-2000	1000	31	23	46
Finland	1991-1999	503	39	19	42
Turkey	1993-1997	190	74	25	1
Austria	1993-2001	131	32	33	35

LOWER EXTREMITY ARTERIAL INJURIES

Femoral vessels are among the most commonly injured vessels, comprising approximately 70% of all arterial injuries. More than 90% of these injuries are the result of penetrating trauma, with most resulting Gun shot wound as noted in recent study in years.

Injury to the popliteal artery accounted for 12% of all arterial injuries among survivors in World War Ist, 20% of those in World War II, 26% of those in the Korean War, and 21.7% of 1000 arterial injuries in US troops in the Vietnam War. Over the past 25 years, the civilian sector has provided the bulk of experience with these injuries, in which setting blunt mechanisms account for 20% to 75% of all cases. Popliteal artery injuries account for 19% of all extremity arterial injuries and have an incidence of 5.6 percent 1000 cases of penetrating trauma and 1.6 per 1000 cases of blunt trauma.

Incidence

The true incidence of shank arterial injuries is difficult to determine. In the absence of physical findings suggesting arterial injuries, arteriography is usually not performed for injuries below the

knee. As a result, many injuries may be missed. Contributing to this is the absence of large series of shank injuries described in the literature.

Articles that do address these injuries usually report very few cases. Therefore, the true incidence of shank arterial injuries is not known; however, infrapopliteal injuries are thought to comprise approximately 5% of civilian arterial injuries. In all, the incidence of shank arterial injury may be low, but a high index of suspicion in patients with no obvious signs of vascular injury is needed to adequately identify all injuries.

Femoral vessels are most commonly involved vessel comprising approx. 70% of all arterial injuries. More than 90% is due to penetrating trauma. Absent pulses, shock, and neurological deficits are observed in more than 80% of the patients with vascular injuriy. ABI of less than 0.9 is highly suggestive of vascular injury and requires angiogram.

Injury to popliteal artery accounts for 12% of all arterial injuries and 19% of extremity arterial injury and most commonly due to penetrating trauma than blunt trauma (5.6/1000 vs 1.6/1000 cases.

But Blunt Trauma resulted in higher amputation rates (27.5% to 71%) than penetrating trauma (10.5 to 27%) presence of any one or more hard signs mandate immediate surgery.

Popliteal vein injuries also occur most commonly with popliteal arterial injury and only 5-10% occurs in isolation, but skeletal injuries are most commonly associated injury. Popliteal vein should be repaired and ligated unless life threatening problems exist and more complex repair is required.

Combined popliteal vascular injuries with skeletal injuries poses higher risk to limb loss and amputation rates as high as 70% because of delay in diagnosis, disruption of collaterals and extreme soft tissue Ischemia with compartment syndrome.

Unstable fractures which requires stabilization before vascular repair may benefit from shunting and on other hand initial revascularisation is done if stable fracture.

Shank Vessel Injury (Infrapopliteal vessel)

These injuries forms 5% of arterial injury.

Amputation are required in 15% of patients with injury involving Tibioperoneal Trunk, anterior or posterior tibial and peroneal artery. Ligation of one tibial vessel alone results in 14% amputation, while ligation of both the tibial arteries end up in 65% amputation.

In Shank Vessel injuries adjacent nerve injury is between 8 to 58% and long term deficit seen in 20%. Associated skeletal injuries is pesnet 35% cases.

Arterial Injuries with Fractures and Dislocations

*	Fracture mid third of femur in adductor canale	SFA
*	Fracture supracondylar area of femur	Pop.Artery
*	Posterior dislocation of knee	Pop.Artery
*	Fracture proximal tibia or fibula	Pop. Artery
		Tibioperoneal Trunk
		Anterior tibial artery

UPPER EXTREMITY VASCULAR INJURY

The incidence of upper extremity vascular trauma from all modern US wars and civilian trauma has been estimated at 30%. Penetrating injuries are much more common than are blunt injuries. Of these injuries, brachial artery injuries occur in approximately 50%, with

radial and ulnar artery injuries accounting for approximately 25% each. Modern series of these types of injuries continue to report low mortality rates, with cause of death usually related to concomitant injuries. Amputation rates are very low (0.8%).

Level of Ligation	Percentage of Amputation
Combined popliteal artery and vein	70%
Anterior tibial or tibioperoneal trunk	14%
Anterior tibial and tibioperoneal	65%
Axillary Artery	8.8 to 43.2%
Brachial Artery above elbow	57.5%
Brachial Artery below elbow	25.8%
Radial Artery	5%
Ulnar Artery	1.5%
Both radial artery and ulnar	39%

Site of Injury	Associated Nerve Injury
Shaft of humerus	Radial nerve
Elbow	Median nerve
Pelvic fracture	Sciatic / Sacral Plexus
Tibial condyle and upper fibula	Common peroneal nerve and anterior tibial nerve

Site of Injury	Associated Venous Injury
Fracture clavical & First rib	Subclavian vein
Fracture femoral (Distal)	Popliteal vein
Fracture upper 1/3 of tibia involving soleal line	Tibial vein

AIM OF THE STUDY

This prospective study of vascular injuries in extremities was conducted in Government General Hospital, Chennai during the period from September 2003 to September 2005. The objectives of the study are:-

1) To study the various etiopathological factors involved and modes of clinical presentation.

2) To assess diagnostic difficulties

3) To analyse the factors influencing ultimate result.

4) To compare and contrast injury pattern and results achieved with world literature.

5) Methods to improve limb salvage.

CLASSIFICATION OF VASCULAR INJURIES

Current classification of Vascular Injuries

a) Arterial Injuries

b) Venous Injuries

Acute	**Late**
Complete transection	False Aneurysm
Lateral Tear	AV Fistula
Intimal Tear with thrombosis (mostly in arterial injuries)	
Vasospasm	

Based on Injuries

1) **Vascular Injuries without musculo skeletal injuries**
   E.g. Assaults, sharp objects, glass pieces etc..

2) **Vascular Injuries with musculo skeletal injuries**

   E.g. Fracture of adjacent bone and nerve injury.

3) **Iatrogenic Injuries**

PATHOLOGY OF VASCULAR INJURIES

In civilian life automobile accidents, industrial mishap, aggressive acts of violence all provide sources of vascular injury. Open injuries of artery may be caused by stab, bullet, glasspiece, metal projection, fracture –bone etc. In a equal number of patients blunt injury is at fault. But even in seemingly blunt trauma artery could sustain open injury due to a fractured bone. In addition to this medical mis-adventure also add up to the insult. All this injuries can be resolved into three basic pathologic patterns which help in clinical characteristics.

Transected Artery

It is commonly encountered in penetrating wounds as the cut ends constrict profoundly and retract into adjacent tissue and as a firm clot plug develops, bleeding usually arrest spontaneously and is minimal. But distally ischemia is greater.

Lateral Tear

Here the disruption of only a portion of arterial wall occurs. Since only partial constriction and partial retraction are possible, the arterial wound gaps further.

Also it can't enjoy the 'tourniquet effect' of the surrounding tissue and the blood clot that covers the arterial defect with more of a patch than a plug. Consequently rapid exsanguinations and delayed / recurrent bleeding are the lateral tear in artery. The distal pulses are usually felt since the lumen is seldom narrowed and pulse wave transmitted through intact portions of the arterial wall. Distal ischemia is thus minimal which makes partially severed artery difficult to diagnose by its peripheral effects. Moreover it is this type that is the forerunner of both false aneurysms and arterio-venous fistulas.

Intimal Tear with Thrombosis

A direct blunt injury excessive stretch of the artery or a near miss by a high velocity missile all results in injured but non severed artery. Distal pulses initially appear healthy, but gradual obliteration of arterial lumen either because of intimal flap, intra luminal thrombus or

intramural thrombus. The intima is relatively inelastic and tears first when artery is excessively stretched. The proximal intimal flap curls up while distal flap is dissected of, occluding the lumen. Thus this injury emphasizes the need for repeated evaluation of arterial circulation. Also the finding at site of injury are minimal. Infact even when exposed operation the artery may be just found narrowed and pulselesss, hence spasm should never be ascribed to it.

PATHOPHYSIOLOGY

Systemic	Local
Shock	Warm ischemic time
Metabolic syndrome secondary to ischemia	Compartment syndrome

Metabolic syndrome secondary to ischemia

When revascularisation is attempted after many hrs a metabolic syndrome similar to crush syndrome can develop. Ischemic muscle undergoes rhabdomyolysis and these products are washed away into general circulation dangerous acidosis, hyperkalemia and cardiac arrest or myoglobulinuria and renal shut down may ensure this possibility should be kept in mind when revascularisation is attempted beyond 6-24hrs.

COMPARTMENT SYNDROME

Compartment syndrome is a clinical condition that results in compromised perfusion to neuromuscular structures within confined myofascial space of the upper or lower extremities. The underlying pathology resulting in impaired tissue perfusion is compartmental hypertension. Acute increases in myofascial compartment pressure may result from blunt or penetrating injuries, associated long-bone fractures or vascular trauma, severe soft-tissue crush injury, delayed treatment of sudden arterial occlusion.

Diagnosis of this condition can usually be made on the basis of progressive motor sensory limb dysfunction associated with provocative trauma, and the onset of common clinical findings such as tense swelling and edema of compartment muscles, disproportionate muscle pain aggravated by passive extension, decreased peripheral pulses, and slow capillary refill.

The diagnosis of ACS should be suspected clinically when blunt or penetrating trauma is followed by limb swelling, motor-sensory dysfunction, and vasomotor instability. Surgery should be performed when these conditions exist. Clinical diagnosis of ACS may be more difficult to confirm in a comatose or confused patient and may require compartment pressure measurements for confirmation. Compartment pressures can be measured with a Wick catheter, the Whitesides needle method or by use of a handheld computerized needle transducer (Stryker, Kalamazoo, Mich). There is no absolute pressure associated with clinical development of compartment syndrome. Normal resting pressure in the lower leg compartment is less than 15mm Hg. Circulatory impairment begins to occur when compartment pressure increases to within 30mm Hg of normal diastolic pressure. When this happens, normal venous drainage is impaired, aggravating further edema and soft-tissue swelling. When clinical symptoms of ACS coexist with resting compartment pressures that exceed 30mm Hg, open fasciotomy should be performed. Patients in shock may have swelling at lower pressures. Transcutaneous Doppler venous flow has been used in conjunction with serial measurement of compartment pressures to more accurately diagnose impending acute compartment syndromes. Loss of phasic flow is the first Doppler change the occurs, and these changes correlate with pressures that exceed 25mm Hg. Loss

of venous flow augmentation correlates with pressures exceeding 30mmHg. Although there is no predictable clinical relationship between the development of compartment symptoms and pressure

measurements between 30 and 60mm Hg, ischemic symptoms uniformly develop when pressures exceed 60mm Hg.

Laboratory Test

Acute compartment syndrome raises CPK levels usually to 1000 to 5000 U within 4 to 6 hours, although the authors have seen elevations of up to 100,000 U when large muscle groups are involved.

Myoglobinuria is also another marker of muscle cell lysis. A positive urine benzidine test for occult blood in the absence of red blood cells is key to the diagnosis. Myoglobin is toxic to the glomeruli of the kidney and leads to renal failure if the compartment syndrome remains untreated.

PATHOPHYSIOLOGY OF I/R INJURY

Ischemia – Reperfusion (I/R) Injury

I/R Injury is encountered in variety of settings from conditions such as stroke, Haemorrhage, Trauma, and shock which manifests as

SIRS and MODS. Thus during Ischemic time several changes occurs at Endothelium, arterioles, capillaries and venules.

Changes occurring at Endothelium are:-

Membrane depolarization, due to ATP depletion during Ischemia that causes impairment of K^+ channels and release of reactive O_2 species that mediates deleterious effects of I/R Injury like cell swelling, cytoskeletal derangements, recruitment and activation of inflammatory cells.

Changes occurring at Arterioles

Primary manifestation at arteriolar level is impaired endothelium dependent vasodilatation. This is due to depletion in endothelial NO synthasel and depletion of Arginine due to increased activity of

arginase and depletion of BH_4. This results in Increased Arterial Resistance.

Changes occurring at Capillaries

Occlusion and Narrowing of capillaries occur following Ischemia due to platelet adhesion and leucocytes recruitment causing congestive occlusion. Further increasing fluid filtration causes intersitial edema and leads to compression of capillaries. This causes a phenomenon called "NO REFLOW PHENOMENON".

Changes occurring at Post Capillary Venules

Changes at this level is mainly due to leucocyte localisation and adhesion which disrupt's intercellular junctions during emigration across endothelial barrier and release of ROS. These effects causes diffuse Tissue edema and hemodynamic instability as a consequence of increased endothelial permeability and depletion of Intravascular volume.

Also endothelial injury due to hypoxemia causes neutrophil localisation due to release of adhesion molecule P SELECTIN. Further leucocytes enhances platelet adhesion and Thrombus formation.

Release of cytokines like IL-1 and TNF α causes remote Ischemic injury.

XO (Xanthine Oxidase) is released in to Blood stream following Ischemia which is taken up by LIVER and activates PMN & Kupffer cells which causes MODS. Thus Allopurinol is useful in inhibiting Xo

TRIAGE IN VASCULAR INJURY

Primary Survey

Initial assessment on receiving the patient in trauma ward is made to assess the severity and site of injury in order to determine priority of resusitation

Resusitation

The best method for temporary control of arterial bleeding is proper application of firm local pressure over the site of bleeding. The use of proximal tourniquets is to be condemned. Similarly attempts to find the artery or control bleeding with clamps or ligature usually dislodge clots and also risk damage to the arterial wall or adjacent neurovascular structures.

Haemodynamic stabilization should be obtained at the earliest as hypotension decreases limb blood flow and aggravates ischemia immediate blood transfusion and oxygen therapy are helpful.

Secondary Survey

The presence and extent of all injuries are established and their priority of repair determined. A secondary survey is made after initial resusititation to assess the patient for any missed out injuries.

Indewelling Arterial Shunt

If a delay in reconstruction is required for attention to other major injury, an indwelling arterial shunt (Teflon / Polyurethane) is preferable to temporarily restore flow.

CLINICAL FEATURES

The diagnosis of vascular injury needs to be prompt, correct complete and exact. The trauma surgeon is often caught between delay in patient presentation referral or transportation and an urgent need to proceed with the operation. The diagnosis of vascular injury is difficult in trauma setting.

The six P's of acute arterial insufficiency namely pain pallor, pulselessness, paraesthesia, paralysis and poikilothermia may not be rewarding in a trauma patient. Pain may be because of other injury and pallor may be due to associated hypovolemia shock. Similarly sensory and motor weakness could well be due to associated nerve injury. But glove and stocking paraesthesia of ischemia should be differentiated from segmental / dermatomal distribution of nerve lesion. Systemic vasoconstriction and swelling and edema following trauma may preclude reliable palpation of pulses. Moreover distal pulses may be palpable despite severe arterial injury. It could happen in following setting.

1) Partially severed artery
2) Injury to a branch Profunda femoris art

3) Short segmental occlusion

4) Transmission of pulses through soft clot.

In Parkland Hospital (Dallas), it has been reported that 25% of the patients with serious vascular injury had palpable pulses. It useful to divide sign of arterial injury into definite or hard signs and equivocal or soft signs. Hard signs are pathognomic of underlying arterial injury and they mandate operative exploration. Soft signs suggest presence of arterial and arteriography or exploration may be necessary to exclude or confirm it.

HARD Signs	**Equivocal or Soft Signs**
Distal circulatory deficit	Small or moderate size stable haematoma
Distal Ischaemia (6 P's)	Adjacent nerve injury
Pulses diminished or absent	Hypotension (Transient / Prolonged)
Bruit Or thrill	Proximity injury
Expanding or pulsatile hematoma	
Active Arterial bleeding.	

INVESTIGATION

While clinical evaluation alone is inadequate, noninvasive testing and arteriography have their own limitations. Plain radiograph merely helps by demonstrating a fracture or locating a foreign body.

Unfortunately in many instances they offer little assistance and negative X-ray of the bone and soft tissue should not discourage consideration of arterial injury.

Role of non invasive testing

The potential value of non-invasive testing is much more limited in acute trauma setting because of absence of well defined diagnostic criteria. Also haemorrhage around vessels may prevent normal transmission of doppler velocity signals. Yet the readily available portable doppler velocity meter gives immediate information at the bed side to augment clinical evaluation. A triphasic doppler signal rules out significant proximal occlusion. While low pitch monophasic signal indicates inflow obstruction. Absence of any signal indicates total obstruction with no collateral flow and prompt arterial repair is

indicated. A systolic ankle pressure measurement using hand held doppler of about 60 mm of mercury always indicate adequate collateral flow to maintain tissue viability. However an ABI of 0.9 and less indicates definite vascular injury.

Thus doppler is useful in following clinical settings: when pulse examination is in doubt, it determines arterial patency. When major arterial injury is obvious it assesses the severity of ischemia and therefore of urgency of repair. Serial monitoring after orthopaedic manipulation or post op period.

Duplex Scan

An extremity / brachial pressure index in the affected limb of less than 0.90 has been used as a threshold to prompt duplex evaluation. Patients undergoing duplex evaluation are by definition hemodynamically stable. Evaluation should include veins as well as arteries to assess for traumatic arteriovenousfistulae, as well as isolated venous injuries.

Sites of disturbed or absent color flow, or extravascular flow should prompt closer inspection.

Arterial injuries from penetrating trauma detectable by duplex scanning include arterial stenosis or occlusion (from intramural hematoma or transmural laceration), dissection or intimal flap formation, pseudoaneurysm, and arteriovenous fistula. Venous injuries include thrombosis, extrinsic compression (from hematoma or soft tissue swelling), and fistula. In diagnosis of compartment syndrome duplex scan demonstrates changes characteristic of venous congestion and increased arterial impedance.

Arteriography

It is single most useful procedure of detecting arterial injury. Exclusion arteriography: this is done in patients with soft signs of arterial injury either to confirm or exclude the diagnosis. Characterisation arteriography: it is indicated in patients with hard signs of arterial injury. But in whom there are multiple penetrating trauma, extensive blunt trauma or presence of bruit which needs more precise information.

Arteriographic findings in arterial injury includes:

- sudden 'cut off' of contrast
- Angiographic gap when efficient collateral allow reformation of distal main artery.
- Extravasation of contrast.
- Asymmetrical narrowing or filling defect.
- Visualisation of false aneurysm
- Visualisation of fistulous tract and of adjacent venous system in AVF.

Arteriography by necessity delays the surgical treatment for the sake of more precise diagnosis. It may change the operative exposure or may make exploration unnecessary.

Spiral / Helical CT Angiography

This recent advancement in investigatory modality is very safe and has 90-94.5% sensitivity diagnosis vascular injuries.

Intravascular Ultrasound

It is been currently used in some tertiary care centers and has been proved highly useful in detecting intimal tear and dissection.

MR Angiography

In the past 5 years, MR angiography has become another alternative for the diagnosis of vascular trauma. Even though it is not widely used, it has the advantages, compared with other modalities, of being noninvasive, obviating intravascular iodine contrast material, and imaging several vascular structures simultaneously.

TREATMENT OPTIONS

Primary correction of shock, control of bleeding, establishing hemodynamic stability, assessment of injury to other systems like Neurological, Cardiothoracic Injuries are assessed before cases are taken up for vascular re-vascularisation.

Non Operative Management

The management of minimal, nonoclusive, clinically symptomatic arterial injuries detected by arteriography remains controversial . Some surgeons have proposed above management if following clinical and radiological criteria are present.

1) Low-velocity injury
2) Minimal arterial wall disruption (<5mm) for intimal defects and pseudoaneurysms.
3) Adherent or downstream protrusion of intimal flaps
4) No active hemorrhage

Surgical treatment

'All major arterial injuries should be repaired promptly once diagnosed'. When a main artery is injured the collateral can never be relied upon 'every effort must be made to complete arterial repair within six hours of injury'. Every hour of delay increases effects of ischemia, diminishes of chance for successful repair.

Vascular Anaesthesia

Regardless of anaesthesia used an experienced anaesthetist is required as intra operative blood loss may be substantial. Smooth induction is vital to prevent dislodgement of tamponading clot. Drugs causing hypotension should be avoided.

A wide operating field including a entire limb on proximal torso should be prepared to ensure access to proximal vessel for control and distal vessel for assessing distal pulses. An uninvolved lower extremity is also prepared for ready access to saphenous vein.

Intraoperative assessment of extent of vascular injury

Completely severed and partially severed artery are easily recognized but the true extent of intimal injury has to be properly assess in these. The injury in a non severed artery may appear in as innocous superficial spasm. This spasm may be ruled out by distending that segment of artery with heparnised saline injected with needle. Applying gauze pledgets soaked in 2.5% papaverine is less effective. If doubt exists arteriotomy will reveal whether it is contusion, intimal tear or thrombosis.

Adequate debridement of injured vessel is important as leaving a transmural injury may result in thrombosis or future false aneurysm while leaving a damaged intima promotes the risk of occlusion by clot or intimal dissection thus resection upto 1cm beyond the visible limits of arterial injuries required in blunt injury till the normal pearly white intima is seen.

Intraluminal propagated thrombosis is often present and proximal and distal balloor catheter. **Thrombectomy** of the injured and artery is accomplished, if collateral is inadequate with poor back bleed. The distal vessels is irrigated with copious amounts cold

heparinized saline and further clot formation is discouraged by instilling 100-200ml of heparinized saline into the distal arterial tree and 5-10ml proximal to proximal clamp.

The type of vessel injured and the extent of debridement required will determine the best method of repair. Minor laceration and punctured wounds are treated by lateral repair. In cases where **lateral repair** will cause narrowing **patch angioplasty** is required, but segmental arterial resection are needed in most of the cases. If after mobilization of vessel there is no tension in an un flexed limb **End to End Anastomosis** is possible. A loss of more than 1-2cm of arterial wall may require interposition graft. In patients with vascular trauma 20-50% require **interposition graft** and **reversed saphenous vein graft**. Interposition is ideal for more extremity vascular injury LSV has mean diameter of 4-6mm and is the thickest autogenous vein in the body and tolerates high arterial pressure.

Its other advantages is rapid availability applicability near joints and long term functions. But when it gets infected transmural necrosis, disruption and fatal haemorrhage can ensue. In the absence of suitable saphenous vein, **Cephalic vein** is also the choice. **Prosthetic graft**

material is avoided if possible except for larger arteries They increase risk of infection and late graft failure is common, if the injured vessel is minor ligation can be done.

Smooth intimal coaptation is ensured by continuous, simple over and over everting sutures, using 5/0, 6/0 or 7/0 prolene, PTFE or polyester material. They are placed approximately 2mm from cut ends with bites 2mm apart if larger vessel like Aota and iliac artery and 1mm bites for medium and smaller vessel. **In children an interrupted suture repair** is preferred to ensure circumferential growth.

Once the repair is completed distal clamp is removed first on this causes blood to expand the suture line and tighten the seal. Pulsation should be restored immediately if not **operative angiogram** to assess the anastomosis and the status of distal arterial tree is indicated. In combined arterial and venous injury normally injured vein should be repaired first. This minimizes distal ischemia. This provides the better out flow and enhances the success of arterial repair.

Adjunctive pharmacological measures

No. of pharmacological agents have been designed to scavenge O_2 free radicles, reverse harmful cytokine production and decreased I/R injuries.

a) Mannitol : Decreases edema and produces osmotic and decreases myoglobinuria

b) Anti oxidants : Deferoxamine, Xoinhibitor, prostacyclin and calcium regulators. Efficacy of all these agents are not proven.

Vasodilation

Intra arterial vasodilator injection increases the flow and lessens stasis and thrombosis. It has been successfully used in traumatized ischemic extremities.

Adjunctive surgical measures

Thorough debridement of non viable tissue is important to minimize the subsequent infection other injuries should be identified and repaired as necessary.

Soft tissue coverage

Prompt covering of the exposed vascular repair is vital in order to minimize the chance of infection and to protect the repair old desiccation and subsequent trauma. Contaminated and questionable wound should be left open managed by delayed primary closure. Skin graft does not provide adequate protection, but vascularised muscle provideds good coverage therefore this could be accomplished by local muscle flap, myocutaneous flap or free tissue transfer.

Fasciotomy

It's indication and values are discussed in chapter on compartmental hypertension.

Closure of Fasciotomy

Fasciotomial should be closed as early as possible because they are major metabolic brain to patient wound can be closed primarily or by SSG. There is no ideal timing and decision for closure should be made individually.

Postoperative Care

Patients must be observed closely in post-operative period as early complication like thrombotic occlusion and bleeding require immediate exploration. Hourly pulse monitoring and doppler examination are mandatory. Thrombotic occlusion for trauma occurs approximately in 10% of the cases if the pulses that are initially present disappears the diagnosis is thrombosis and immediate re-exploration is indicated.

Systemic anticoaguloation is not required in post-operative period. If the repair is technically imprecise no amount of heparin will sustain patency. But still it has a role in presence of repaired venous injury. Heparin is indicated for DVT prophylaxis and full dose heparin for venous injury patients and proven DVT patients.

The rheologic agent, low molecular weight, dextran by its antithrombotic and microcirculatory enhancement property increase flow in microcirculation. The use of hypertonic manitol has been advocated to decrease the post ischemic swelling and aggressive management of the infection before it endangers the life of the patient or anastomosis is mandatory.

Treatment of Pseudoaneurysms

This requires a dedicated sonographers to apply between 10 and 120 min of compression to the "neck" of the pseudonaeurysm. The technique has the obvioius labor-intensive drawback of occupying a significant amount of time for the sonographer. The procedure has a reported initial success rate of 60% to 90%, obviating the need for surgical repair in these cases. However, recurrence rates have been reported to be a high as 25% to 30% .

More recently, the use of ultrasound has been combined with thrombin injection for much improved results. An overall success rate of 96% has been reported in the largest series.

INTERVENTION TECHNIQUES IN VASCULAR TRAUMA

Angioembolisation

Management of significant bleeding following blunt pelvic fracture, penetrating pelvic injury can be done by angiographic transcatheter embolisation by GELFOAM which remains the vascular space for several weeks and then absorbed. This method is used in controlling Lumbar Arterial Bleed, Peripancreatic, Perinephric bleeding and also for post operative liver bleed if in right lobe of liver.

Use in Extremity Injuries

Some indications like bleeding from axillary arterial branch vessels like Throaco-acromial and circumflex humeral branch. In lower limb deep muscular branch which is inaccessible can be embolised more so in profunda femoral artery. Coil embolisation of more distal PFA, AV fistula and laceration can be done.

Solid	Liquid
1) Coils 2) Gelfoam 3) Ivalon Sponge 4) Steel Coils 5) GUG LIELMI detachable coils	Isobutylcyano acrylate

Endovascular Treatment of Vascular Injuries

Several options exists for endovascular treatment of traumatic vascular injuries.

1) Intravascular stents
2) Stentgrafts
3) Occulsive devices like Balloons, Coils and other Thrombogenic Materials.

INSTITUTIONAL PROTOCOL FOR VASCULAR REPAIR IN TRAUMA

Selection Protocol

1) All vascular injury patients with atleast distal venous flow in extremities by hand held doppler were taken up for revascularisation.

2) Adequate resuscitated patients with stable vital parameters.

3) Neurosurgical clearance in suspected head injury patients.

4) Vascular repair after arrest of intracavitary bleed elsewhere (intra abdominal, intra thoracic)

Diagnostic Protocol for Vascular Intervention

1) Clinical examination – absent or feeble pulses
2) ABI of less than 0.6
3) Duplex scan in late presentation to localise site of injury, false aneurysm and AV fistula's.
4) Peripheral Angiogram for multi level injuries, false aneurysm and AV fistula's.
5) CT with contrast in case of intra abdominal and pelvic injuries

TREATMENT PROTOCOL

1) End to end anastomosis in sharp penetrating wound without segmental loss of artery.
2) RSV graft in infra inguinal vascular injuries with end to side anastomosis proximally and distally.
3) Harvesting of LSV from normal limb
4) Synthetic graft in case of Aorta / Iliac
5) Adequate surgical debridement in case of open wounds.
6) Muscle cover for graft & anastomosis where skin loss is present
7) Prevention of reperfusion injury

    - Adequate hydration
    - IV mannitol
    - Allopurinol 200mg preop & 100mg bd
    - Inj NaHco3 IV immediately after release of clamps and SOS based on ABG reports

Post Operative Monitoring

1) Monitoring of vital signs / hrly urine output.
2) ABG – 2hrly
3) Pulse oximetery
4) Monitoring of peripheral pulse
5) Postoperative full dose heparin if DVT / venous repair
6) Prophylactic dose of injection heparin in arterial repair

POST OPERATIVE COMPLICATIONS

Three most complications are:-

1) Thrombosis
2) Infection
3) Stenosis
4) Miscellaneous complications

Local	Systemic
1) Chronic pain	Fever
2) Decreased function	Toxemia
3) Ischemic changes	Atlectasis
4) Systemic complications	Pulmonary embolisem
5) AVF & false anuerysim	MODS
6) Arteriosclerotic changes	
Anuerysmal graft changes	

FACTORS INFLUENCING LIMB SALVAGE

Haemorrhage

As already mentioned completely severed artery causes minimal bleeding. Infact even completely severed leg or forearm heamostasis is effective enough to prevent bleeding to death but a partially severed artery can give rise to rapid exsanguinations or a delayed / recurrent bleeding. With the modern resuscitative measures the dangers of haemorrhage have been superseded by problems of ischemia.

Ischemia

The degree of ischemia depends upon interaction of several local and general factors.

Local factors

Damage to certain is especially liable to have serious consequences (popliteal artery and brachial art) the adequacy of collateral circulation plays an important part. Extensive blood injury destroys many collaterals and cause more ischemia than clean penetrating wounds. The increased incidence of amputation on blunt injuries is attributed to this. Use of tourniquets and development of compartmental hypertension adds up to the ischemia.

General factors

Old age, atherosclerosis, diabetes – mellitus anemia influence degree of ischemia. The degree and duration of shcok may determine the onset of gangrene after vascular damage. In shock both the low B.P. and compensatory peripheral vasoconstriction compromise the peripheral perfusion. The survival or loss of the limb may depend upon whether the B.P improves within first two or three hours after injury. Urgent blood replacement and adequate oxygenation go along way in forestalling irreversible tissue damage.

The ischemic damage is also dependant upon the tolerance of various tissues of limbs to anoxia. The peripheral nerves are most sensitive within 30-60 mins. They temporarily loose the power of conduction resulting paralysis and paraesthesia. The next most vulnerable tissue is striated muscles and its period of tolerance is in the range of 6-8hrs. The muscle fibres begin to die between 6-12 hrs after vascular occlusion. The skin tendon, bone have greater tolerance and skin can survive for 24 hrs.

The fate of tissues therefore is dependent on the speed with which circulation is restored by surgery. If free and vigorous the circulation is restored within 6 hrs the recovery can be complete. Restoration of circulation beyond 6 hrs may still allow partial recovery though the end result may be ischemic contractures or amputation at lower level. If severe and unrelieved ischemia persist for 12-24 hrs chance of limb salvage is removed.

COMPARISON WITH WORLD LITERATURE

This study is based on analysis of 65 patients with Arterial Injury of extremities treated at Govt. General Hospital, Chennai for the period of 2 years from September 2003 to September 2005. The patients were admitted in Trauma Ward, managed initially by General Surgeon and subsequently treated by vascular surgical team of the hospital. Those patients with severely damaged extremities with resultant primary amputation were excluded from the study. 60 Patients presented acutely soon after the Injury while 5 present with post traumatic false aneurysm. (Table 1). Their age ranged from 10 years to 60 years of which 96.1% were in 3rd decade and only 6 patients were female.

Distribution and type of Injury

The 65 injured patients had Arterial Injury with few sustaining more than a single vessel injury. The lower limb was most commonly involved and superficial femoral artery is most frequently injured vessel closely followed by popliteal artery and in upper limb brachial artery was most common vessel involved.

Table – I

No. of Patients	Total (65)	%
Acute Presentation	60	92.3%
False Aneurysm	5	7.7%

Table – II

Sex Incidence	No. of Patients	%
Male	59	90.8%
Female	6	9.2%

Table – III

Upper Limb

Artery	No.	Our Series	Gill 1976	Burnet USA
	Total = 65			
Axillary	1	1.5%	2.4	4.7
Brachial	17	26%	27.6	18.8
Ulnar	2	3.1%	3.4	9.4
Radial	2	3.1%		

Abdomen & Lower Limb

Artery	No.	Our Series	Gill 1976	Burnet USA
	Total = 65			
Abdominal Aorta	1	1.5%		
Iliac	6	9.2%		
CFA	1	1.5%	8.6	9.4
SFA	23	35.3%	22.4	45.3
Popliteal	10	15.4%	25.9	12.5
Anterior Tibial	1	1.5%	8.6	7.8
Posterior Tibial	1	1.5%	-	-

Table – IV

Etiology of Injury	Our Series (65)		Gill	Bole
RTA	53	81.5	58.6	16.6
Assault	3	4.6	18.9	33.3
Iatrogenic	3	4.6	6.9	-
Gun shot	Nil		15.6	50.0
Others	5	7.7	-	-

Table – V

Type of Arterial Injury

Pathology	Our Series (65)		Gill	Bole
Transection	24	36.9	44.8	51.2
Lateral Tear	Nil	-	10.3	37.9
Intimal Tear with Thrombosis	29	44.61	24.1	10.6
Spasm	8	12.3	5.28	-
False Aneurysm	5	7.7	6.9	0.8
AV Fistula	Nil	-	5.2	3.5
Subadventital Rupture	-	-	-	-

Table – VI

Associated Injury

Structures	Our Series (65)		Gill	Moris USA
Vein	33	50.76	19	13.3
Nerve	12	18.46	18	21.3
Ortho	56	86.15	55	35.5

Table – VII

Associated Venous Injury

Total = 33

Site	
IVC	1
Femoral Vein	10
Popliteal	18

Pathology	
Contusion	29
Lateral Tear	3
Transection	-

Management	
Ligation	28
EEA	2
Interposition Graft	Nil
Lateral Venography	3

Table – VIII

Associated Nerve Injury

Total = 12	
Site	
Median Nerve	6
Ulnar Nerve	3
Common Peroneal	3

Table – IX

Associated Orthopaedic Injury

Total = 56	
Fracture Site	
Clavicle	7
Humerus	13
Femur	24
Both Bone Leg	6
Pelvis	6

Table – X

Shock

Total = 30	
Injured Artery	
Brachial	9
Axillary	2
Radial and Ulnar	1
Femoral	13
Popliteal	4
Post. Tibial	1

Table – XI

Clinical Indicators of Vascular Injury

Acute Presentation

Total = 65		**%**
Pulse Deficit	54	82.5
Distal Ischemia	29	44.4
Profuse Arterial Bleed	11	17.4
Pulsatile Haematoma	14	22.2
Expansile Haematoma	8	12.6
Proximity Injury	56	86.1

Late Presentation		
Pseudoanuerysym	5	7.5%

Table – XII

Type of Arterial Repair

Type	Our Series 65		Gill(51)	Burnet (49)
	No.	%		
Interposition Graft with SV	48	73.8	47	31
EEA	5	7.6	41	45
Synthetic Graft	2	3.07	2	-
Patch Angioplasty	6	9.2	-	-
Thrombectomy	2	3.07	-	-
Ligation	2	3.07	4.0	10

Table – XIII

Fasciotomy

Total = 40	
Upper Limb	22
Lower Limb	28

Table – XIV

No of Cases Improved following fasciotomy

Upper Limb	20
Lower Limb	20

Automobile accidents were responsible for most of 53% vascular Trauma and acts of violence caused 16.7% of injury (Table IV) Traffic accidents commonly affected the lower limb while stab injury were equally common in both limbs. Of the three latrogenic injury all were following transl femoral angiogram in common femoral artery. Two latrogenic injuries resulted in false aneurysm.

Among the acute injuries Intimal Tear with Thrombosis was the most frequent finding 44.61%, as seen in Table V in our series. Transection was next most common injury (36.9%) in our series and vasospasm in about 12.3% of cases. Post traumatic false aneurysm was the presentation in 5 patients. All of them resulted from penetrating injury and had lateral tears in Artery. The Arteries involved in false aneurysm include common Femoral Artery (3), Brachial Artery (2). The cases that came with obvious gangrene were due to RTA with pelvic fracture associated with injury to left external Iliac artery and common femoral artery, with gangrene of anterior and adductor compartment of thigh. Another case of myonecrosis with popliteal artery injury due to RTA had to undergo above knee amputation.

Associated injuries (Table VI)

50.76% was found to have adjacent venous injury transection was commonest type of venous injury. 6.5% of upper limb arterial injury had associated venous injury, while it was 43.1% in lower limb. The common veins involved were popliteal vein and femoral vein respectively.

18.46% of cases had associated nerve injury. Of which 75% were in upper limb. Median nerve was the most common involved and transection was the commonest pathology encountered.

Similarly 86.15% patients had associated orthopaedic injury (Table IX). 90% was following RTA. Nearly 2/3 were in lower limb and fracture of lower end of femur was commonest.

Clinical Features and diagnosis

Of the 65 patients with Acute presentation 30 cases presented with hypovolemic shock 3 arterial injury were clamped and ligated to control haemorrhage at referral hospital. 80% of cases with shock were lower limb injury which were femoral artery and popliteal artery injury. The arterial injury was transaction in 24 cases and lateral tear in

7 cases. Among the femoral arterial injury with shock all but one had fracture femur.

Almost all cases had injury or fracture in close proximity to the arteries. Indicating 'proximity' to be an important clue to arterial injury (Table IX). Notable distal ischemia was observed in 44 of the cases and profuse arterial bleeding occurred in 17.4%. Late recurrent bleeding was present in one case of lateral tear of Brachial artery. Bleeding was arrested in these cases with local pressure dressings. Blind clamping and tourniquets were avoided.

Once the shock was overcome and ongoing bleeding was arrested patients were rapidly assessed for other injuries and priorities established. Ultrasonic hand held doppler flow assessment was used in all cases. It was especially useful in minor vessel injury in cases where distal pulses palpation were precluded by edema. Patients with equivocal signs were further evaluated by duplex examination by which distal arterial pressure can be measured. An arterial pressure index of greater than 0.9 rules out an arterial injury in non diabetics.

Arteriography was required in 7 patients. It was done either to exclude / confirm arterial injury or to localise the injury and to

diagnose multiple level injuries. The frequent indications were extensive blunt injury, multiple penetrating trauma, infrapopliteal injury or absence of distal pulses. In all 7 cases it revealed extravasation in 2, cut off with distal reformation in 3 and intraluminal thrombosis with cut off in 2. five cases presented several days to months after the injury with pulsatile false aneurysm. Distal pulses were present in all cases.

Management

The time lag between injury and definitive treatment varied enormously from 4 to 30 hrs. The average pre admission delay was 16.30 hrs with post admission delay of 4.56 hrs. Thus an average of 20.30 hrs lapsed between injury and surgery in our series.

Of the 65 patients who underwent arterial surgery, three patients died of hypovolemia, 4 patients died due to sepsis . Two cases due to gangrene of lower limb other due to toxemia and extensive myonecrosis. Other cases were due to hypovolemia and patients was found unfit for any form of anaesthesia or repair. Regional anaesthesia was used in 2/3 of cases and rest managed with general anaesthesia after meticulous debridement the extent of arterial injury was assessed.

Arteriotomy was done in injuries in continuity to assess the type of injury sustained. Damaged arterial walls were excised carefully and fogarty balloon thrombectomy done proximally and distally and back bleeding established. Heparinized saline was injected into distal artery. End to end anastomosis without tension done in 5 cases. While in 48 cases reversed saphenous vein graft used with end to end bypass both proximally and distally. Synthetic graft (PTFE) was used in 2 patients for iliofemoral segmental injury. Later on repair was done with 5/0, 6/0 or 7/0 prolene suture.

Associated venous injury was treated by interposition graft, and ligation and EEA. Vascular repair was covered with healthy muscle by transposition of adjacent muscle required.

Associated orthopaedic injury was managed by 'K' wire fixation, External fixation, splint and POP immobilization were also done.

Fasciotomies were done in 40 cases to decompress the compartment . It was done mostly in patients with obvious distal ischemia, longer time lag and extensive blunt injury and venous injury. Successful revascularisation was followed with periodic pulse palpation and hand held doppler assessment in postoperative period.

FACTOR'S INFLUENCING AMPUTATION

LOWER LIMB AMPUTATIONS TOTAL = 8	
ASSOCIATED INJURY	
Skeletal	**7**
Venous Injury	**8**
SITE	
Distal Popliteal	**5**
SFA	**2**
AVERAGE ISCHEMIC TIME	**27.5hrs**

UPPER LIMB AMPUTATIONS TOTAL = 2	
ASSOCIATED INJURY	
Skeletal	**1**
Gross Wound Contamination	**1**
SITE	
Axillary Artery	**1**
Brachial Artery	**1**
AVERAGE ISCHEMIC TIME	**16.5 hrs**

RESULTS

- 65 patients underwent acute arterial surgery
- Restoration of pulse — immediate = 73.85%
  Delayed = 10%
  Failure = 15.35%

- Disappearance of pulse on 1st day = 9.2%
- Amputation Rate = 15.38%
- Limb Salvage Rate = LL 75%
  = UL 95%
- Death = 7 Cases (10.7%)
- Post Reconstruction Edema = 8 Cases
- Ischemic Contracture = 2 Cases
- Residual Nerve Palsy = 4 Cases
- Blow out = 13 Cases
- Average Ischemic Time in amputated = UL 16 hrs.
  Cases = LL 27.5hrs

Among the 65 patients who underwent immediate arterial surgery. In 48 patients pulse restoration was achieved immediately and delay in about 7 cases. Pulse did not return in one case of femoral arterial injury with crush injury of leg and subsequently ended in amputation. In one case restored pulse disappeared on the 2nd day following extensive arterial thrombosis and pulse restored after thromboembolectomy. The amputation rate in the series was 15.38% pointing to limb salvage of 95% in UL & 75% in LL comparable to international literature.

There was seven death's in this series, related to multiple injury resulting in hypovolemic shock. Significant post reconstruction edema was present in 8 cases and infection in 2 patients. 2 patients developed ischemic contractures and residual nerve palsy seen in 4 patients when last seen after a median follow up time of 6 months.

FOLLOW-UP

According to the Swedvase, the mortality rates among 931 patients with vascular injuries secondary to trauma were 1.7% after penerating trauma, 1.4% after blunt trauma, and 4.5% after iatrogenic vascular injuries (R. Takolander, Swedish Vascular Registry, personal communication, 2001). In Finland, the overall mortality rate among 503 patients with vascular injuries was 2.8% (J.P. Salenius, Finnish Vascular Registry, personal communication, 2001).

In a single hospital series of 4.7 patients with vascular injuries from the University of Athens, the overall mortality rate was 3.7%; the amputation rate after revascularization, 2.3%; and the wound infection rate, 14.9% (G. Androulakis, personal communication, 2001).

In follow-up of our patients 18 patients out of 65 operated patients attended our Outpatient Department for a period of six months. Out of the 18 patients 8 patients had a combined arterial and venous injury who had post reconstruction edema, 4 patients had residual nerve palsy, 2 patients had ischemic contracture.

CONCLUSION

65 Patients with arterial injury of extremities were treated during 24 month period. It is significant that 96.1% were in peak of their youth (3rd decade). A marked male preponderance of 98% was noted. Automobile accidents and penetrating injury were predominant causes. Iatrogenic injury occurred in about 4.6% of the patients. About 90% of acute arterial injury endangered the limbs superficial femoral artery, popliteal artery and brachial artery were seen most frequently. Average ischemic time was 16.75 hours with preadmission delay contributing much. 'Proximity' was a important clue in our series as almost all cases had injuries or fracture close to the arteries. But nearly ¼ had palpable distal pulses inspite of arterial injury, indicating that absent pulses alone was inadequate to be the sole indication for exploration. Portable doppler velocity meter gave valuable immediate information at the bed side augment the clinical diagnosis.

Arterial repair was accomplished with autogenous vein graft in 73.8% patients and end to end anastomosis in 7.6% patients and venous repair done in 15.1% percentage of patient. Associated orthopaedic and nerve injury were responsible for most of the

functional impairment and limb losses. A limb salvage rate of Lower Limb 75% and Upper Limb 95% was achieved. There was seven death due to arterial injuries in this series.

In our series none of the international studies had such prolonged ischemic time and hence comparative analysis could not be made.

Main cause for amputation in our series was due to (1) delayed presentation (2) due to no reflow phenomenon (3) infection

Although useful as a guide, strict reliance on 6hrs golden period indicating the need fro primary amputation was not followed in our institution. In most situations vascular reconstruction can be accomplished. Currently in the study any single scoring system was not adhered but associated major nerve disruption has a greatest impact on successful rehabilitation of injured extremity.

The false aneurysm were managed without any significant complication. Most of the cases which presented with gangrene were due to delay as well as mismanagement at the referring hospital.

The high limb salvage rate achieved was mainly as a result of ``prompt resuscitation from shock, prompt diagnosis and proper treatment. The availability of expert vascular surgeon in a short period resulted in gratifying results. Selective use of arteriography, repair of associated venous injury, liberal fasciotomy all contributed to success. Further improvement can be achieved by shortening the referral, transportation easily resusitation and identification of vascular injury.

With recent technological advancements, reduction in Transportation time due to better road and air ambulance services combined continuous monitoring right from site trauma through personal status monitor worn as wristwatch and a vehicular status monitor mounted on Ambulance vital signs can be received through GPS (Global Positioning System).

Earlier identification at primary care center at Trauma assistant can be achieved by better training with help of VIRTUAL REALITY using computer generated simulation . Better resusitation and improvement of Haemodynamic stability can be achieved by Hb-base red cells substitutes which are readily available and do not require cross matching and enhances flow and O_2 delivery. Further with surgical assistance of ROBITICS and Telemanipulation remote

controlled operation can be one of benefits of leap in technology useful to train and work with live patients.

AMBROISE PARE in 1552, reinstroducing the ligature and avoiding the brutal cautery remarked “I dressed him and god healed him” from there the frontiers of vascular surgery have now leaped to the extent of 95% limb salvage and microvascular reimplantation. No doubt application from the lessons of the past have contributed to us a great deal.

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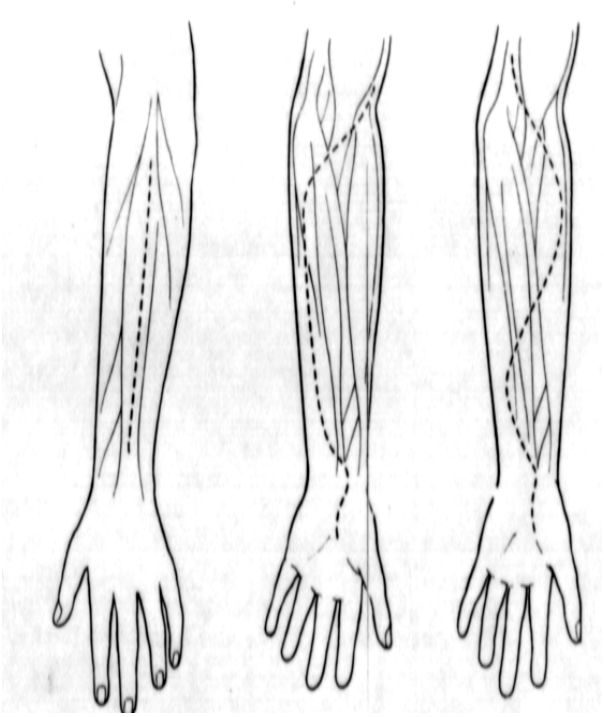
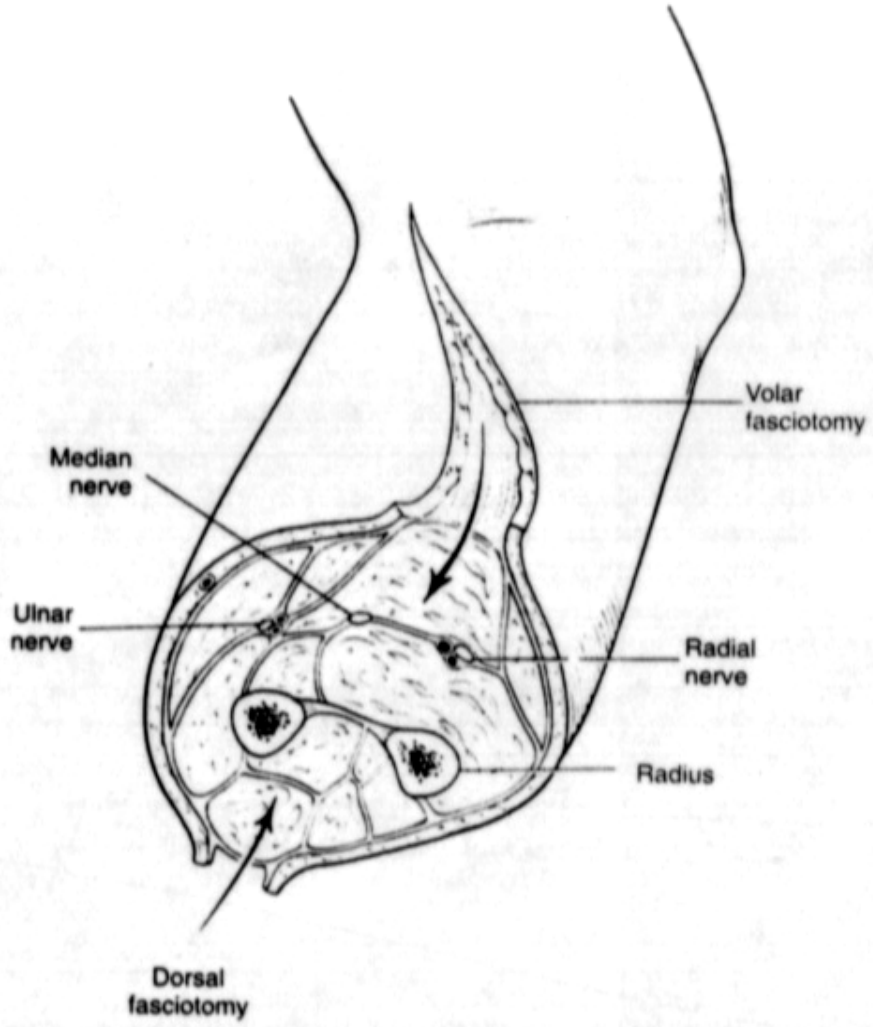
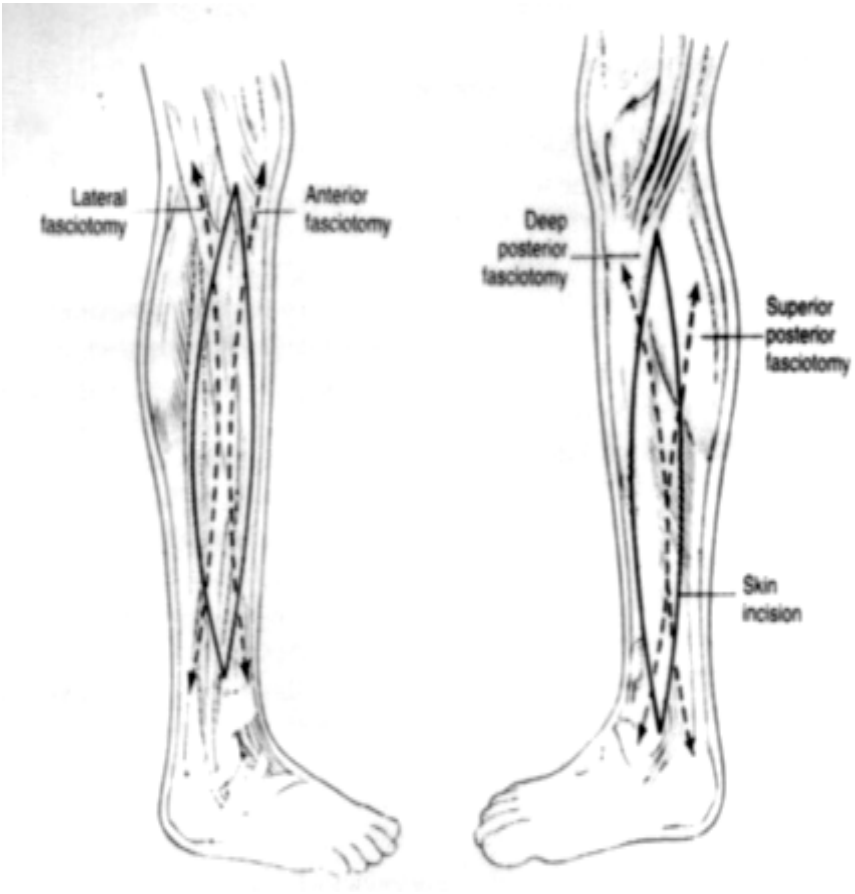


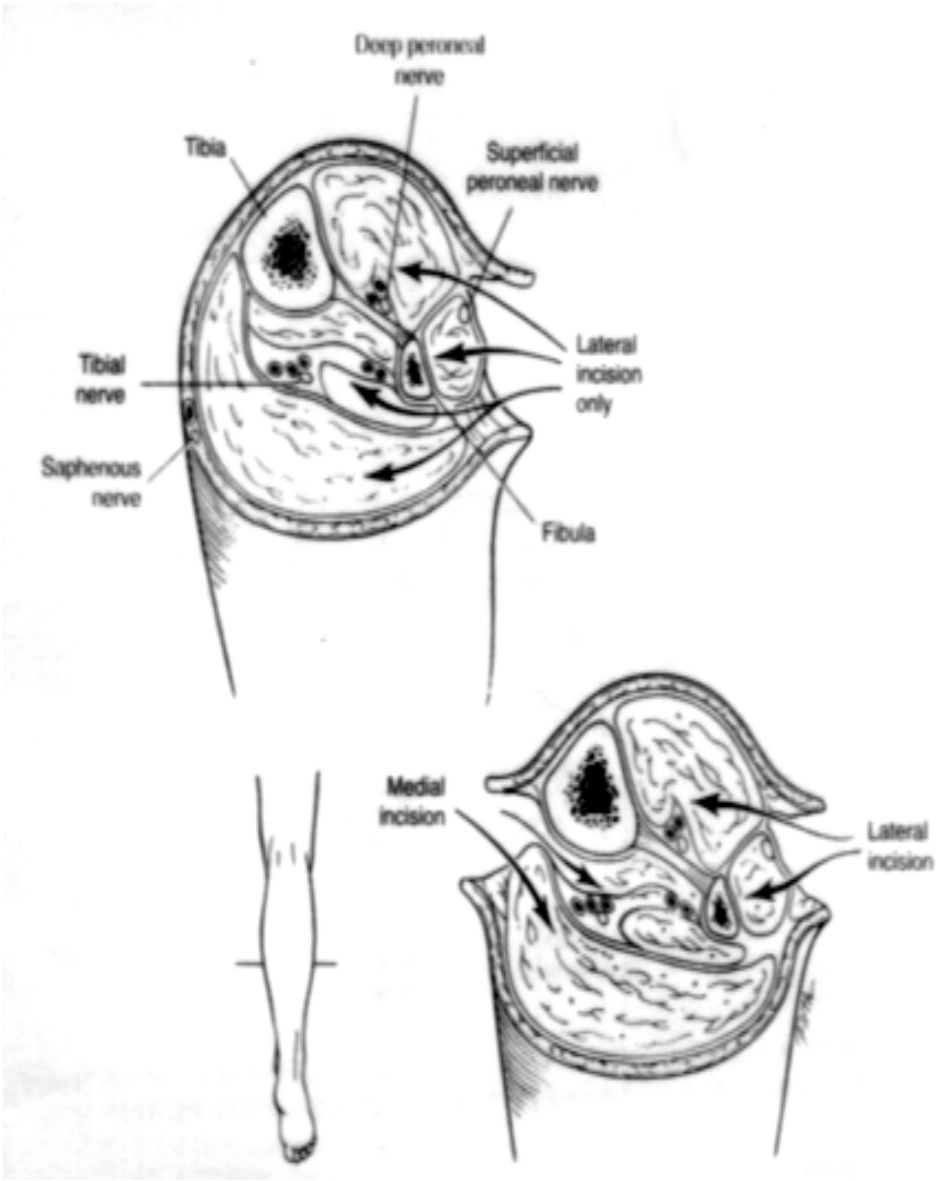
Volar (S-type or Straight) and dorsal (Straight) incisions for forearm fasciotomies



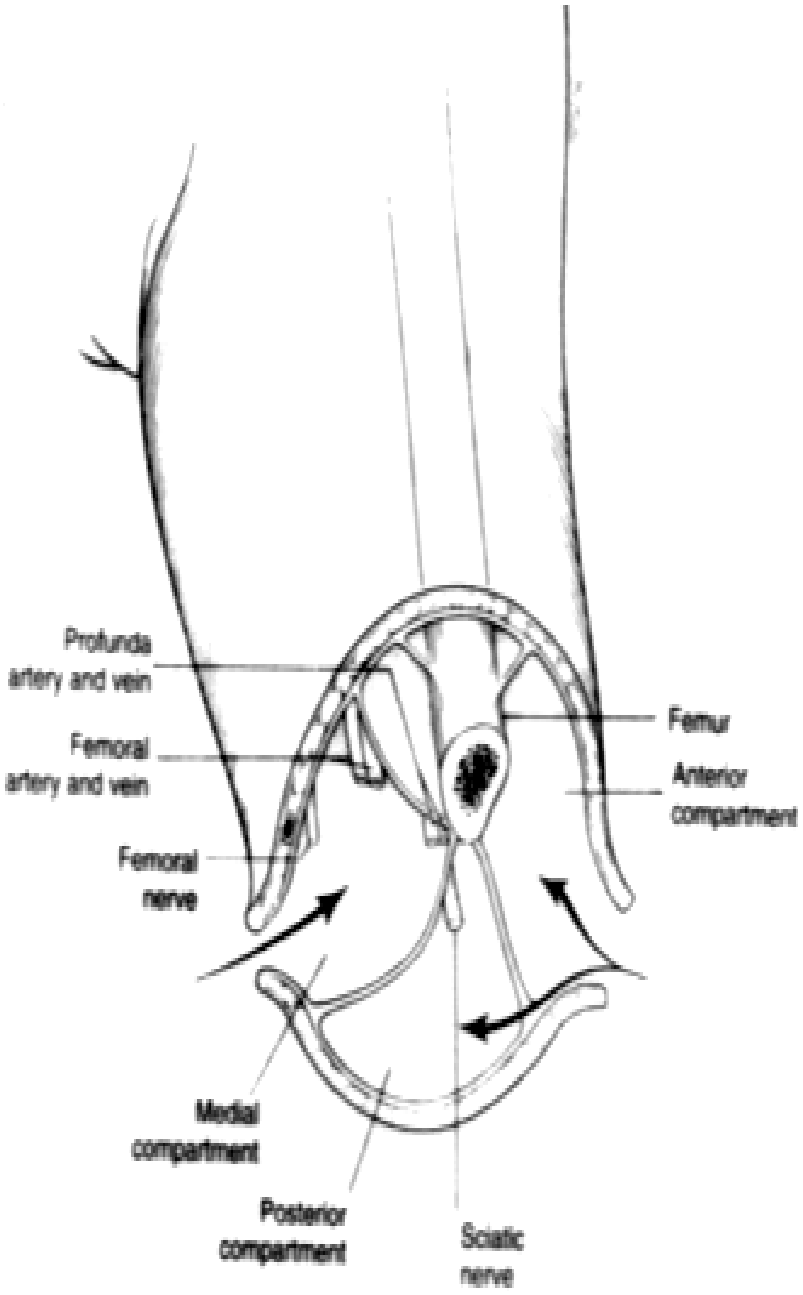
Decompression of compartments of the forearm.



Lateral and medial leg incisions for a four-compartment fasciotomy. The lateral and anterior compartments are decompressed through the lateral incision and the superificial and deep posterior compartments through the medial incision

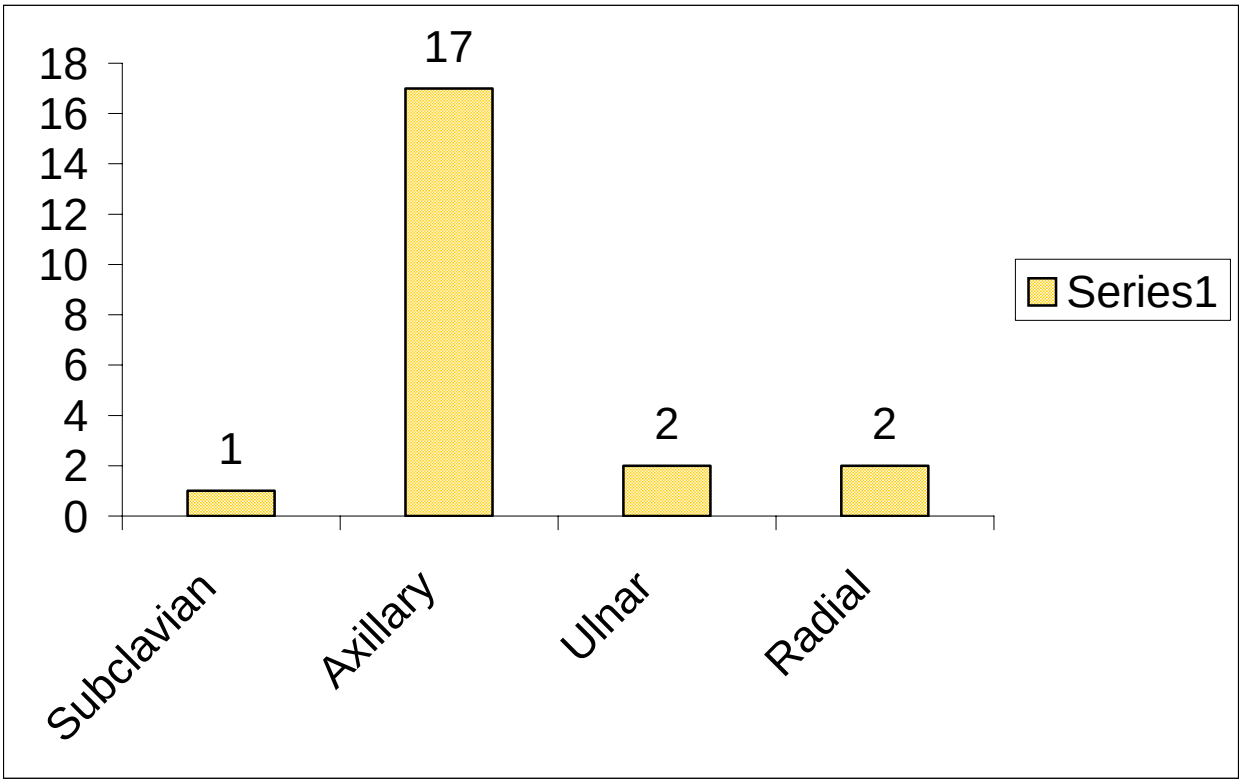


Four-compartment fasciotomy can be accomplished by two incisions (a medial and a lateral) or a single lateral incision.



Thigh fasciotomies. The anterior and posterior compartments are decompressed through a lateral incision and the medial compartment through a medial incision.

Upper Limb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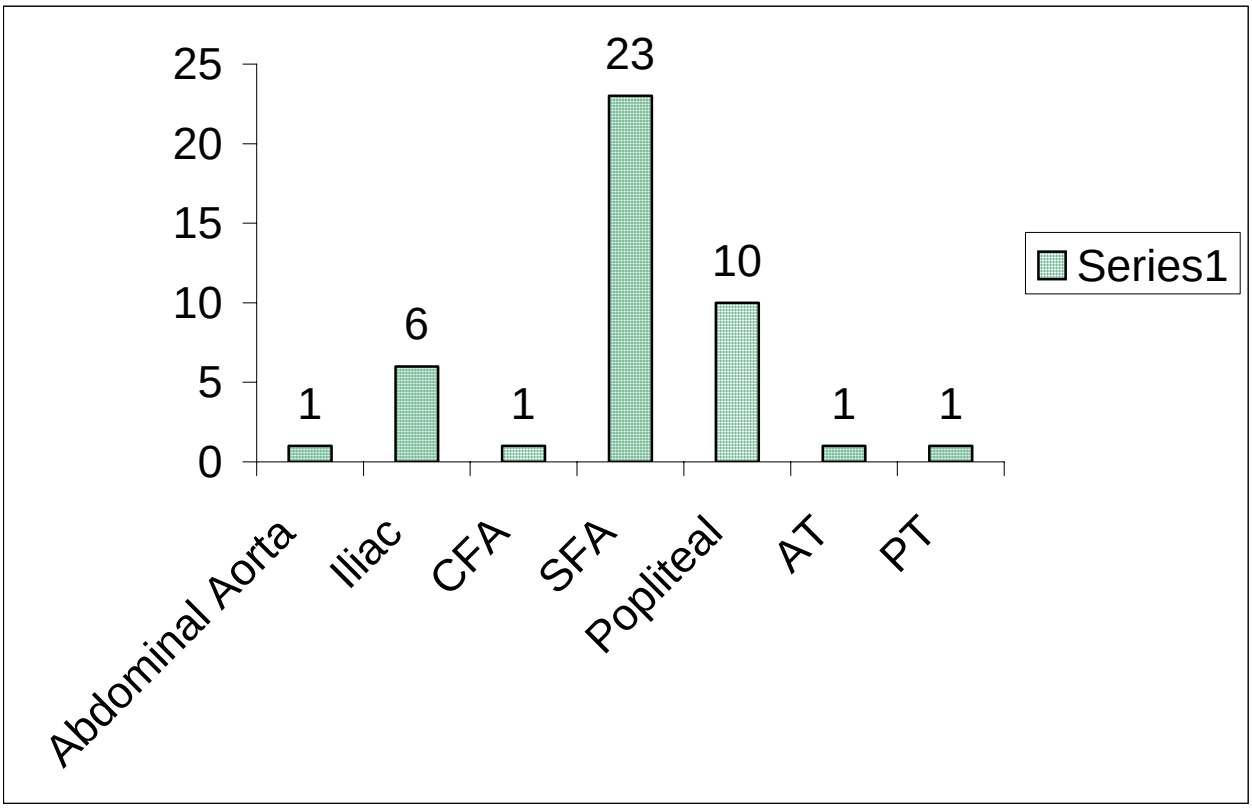
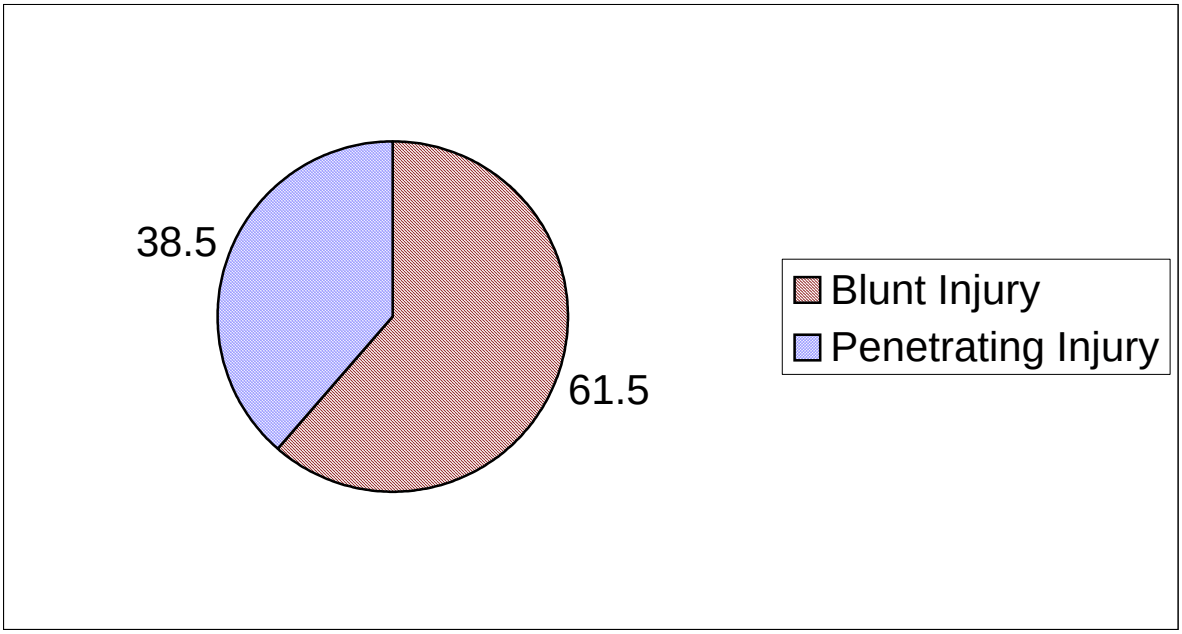
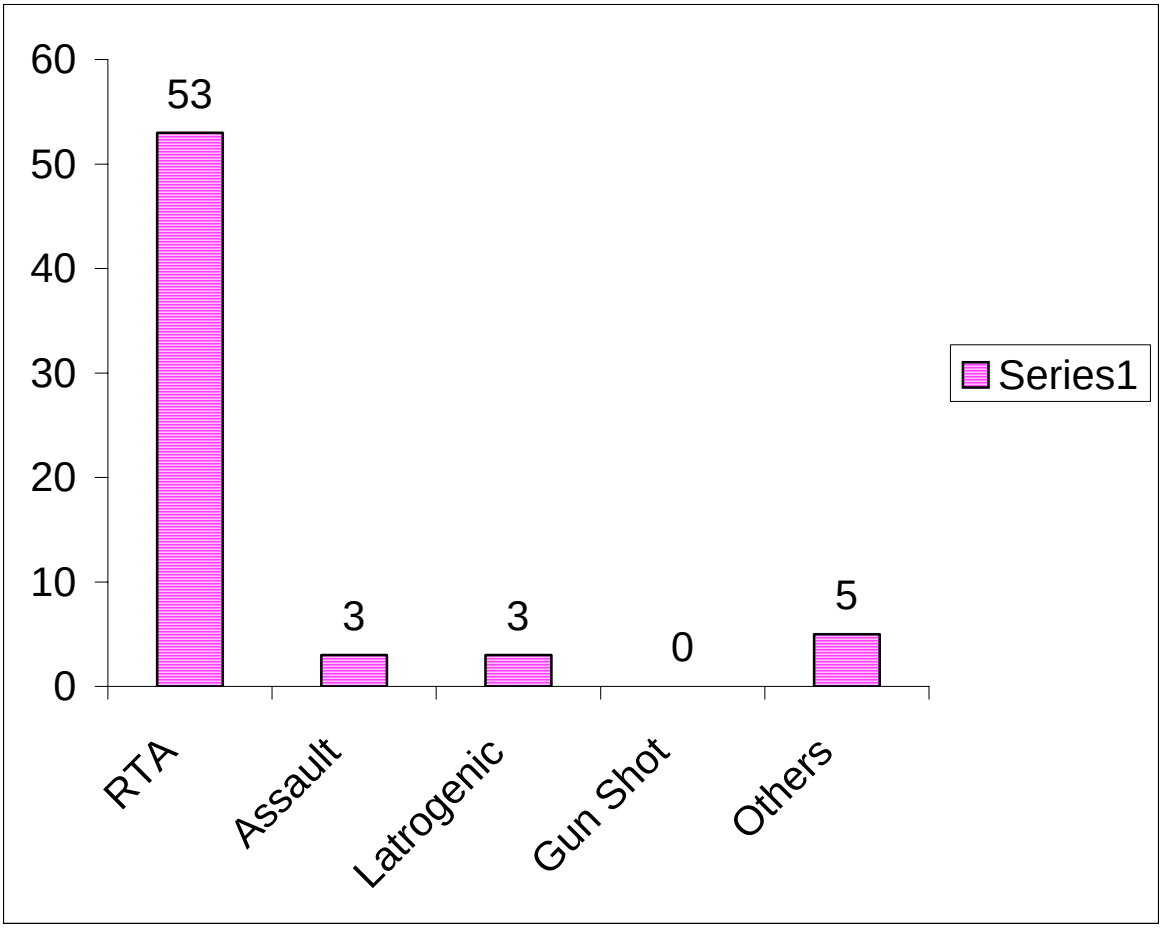
Abdominal & Lower Limb



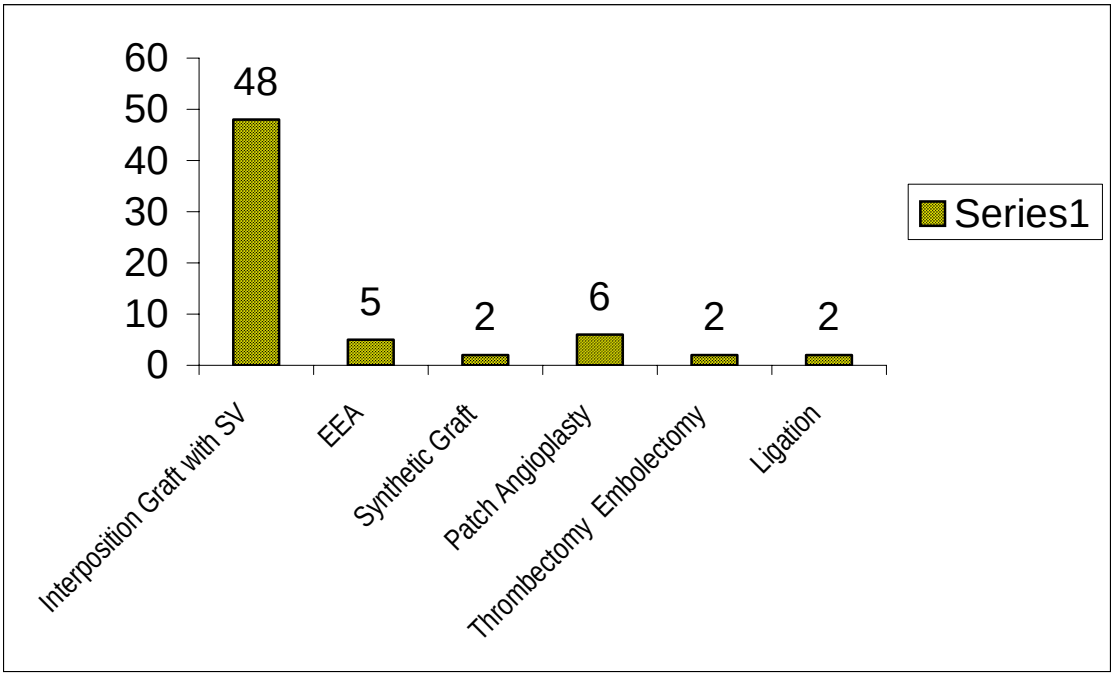
Mechanism Of Injury

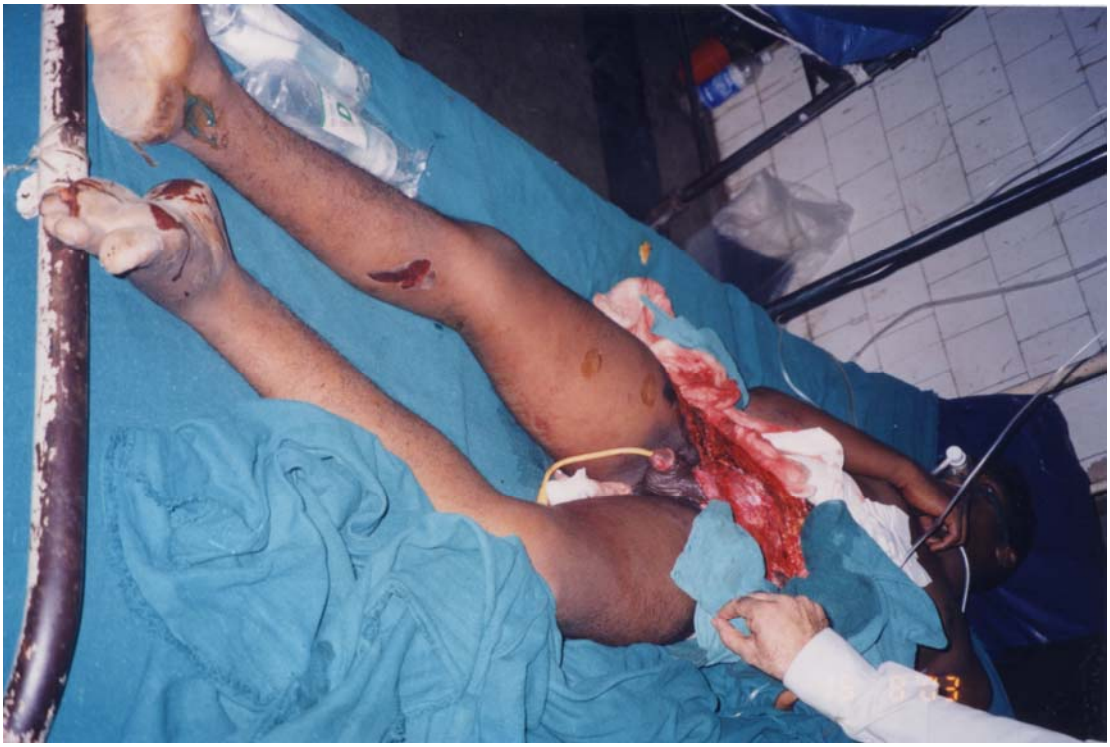


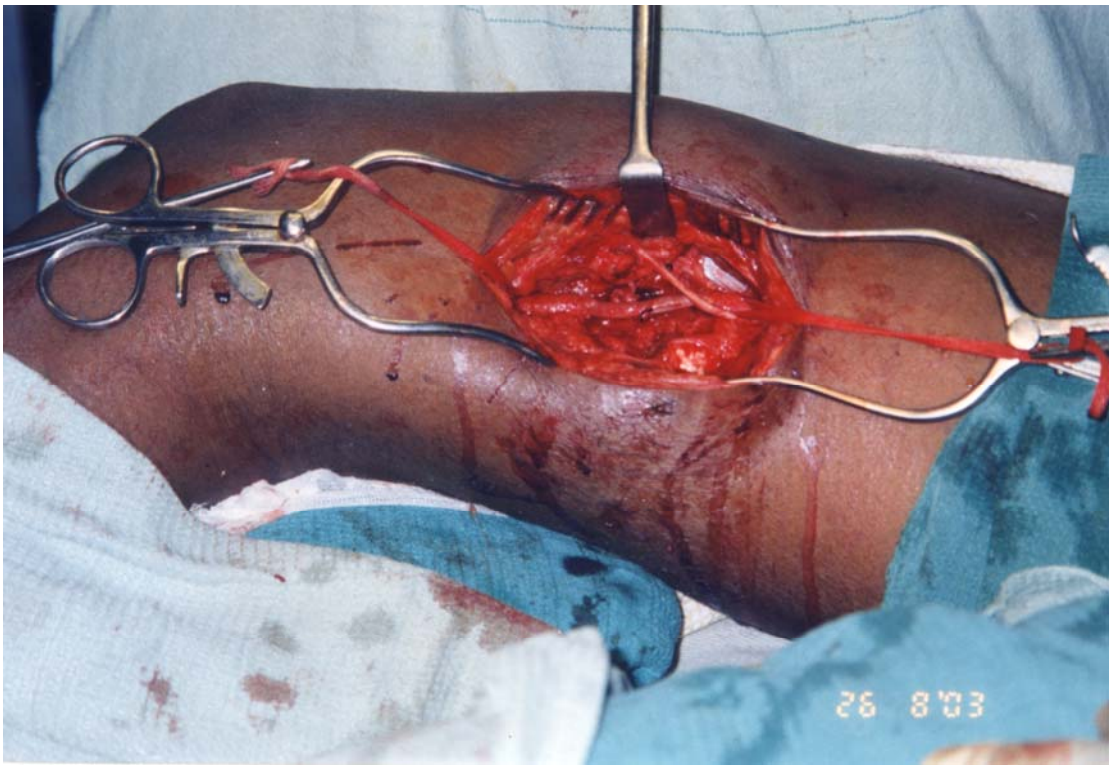
Etiology of Injury



Type of Repair



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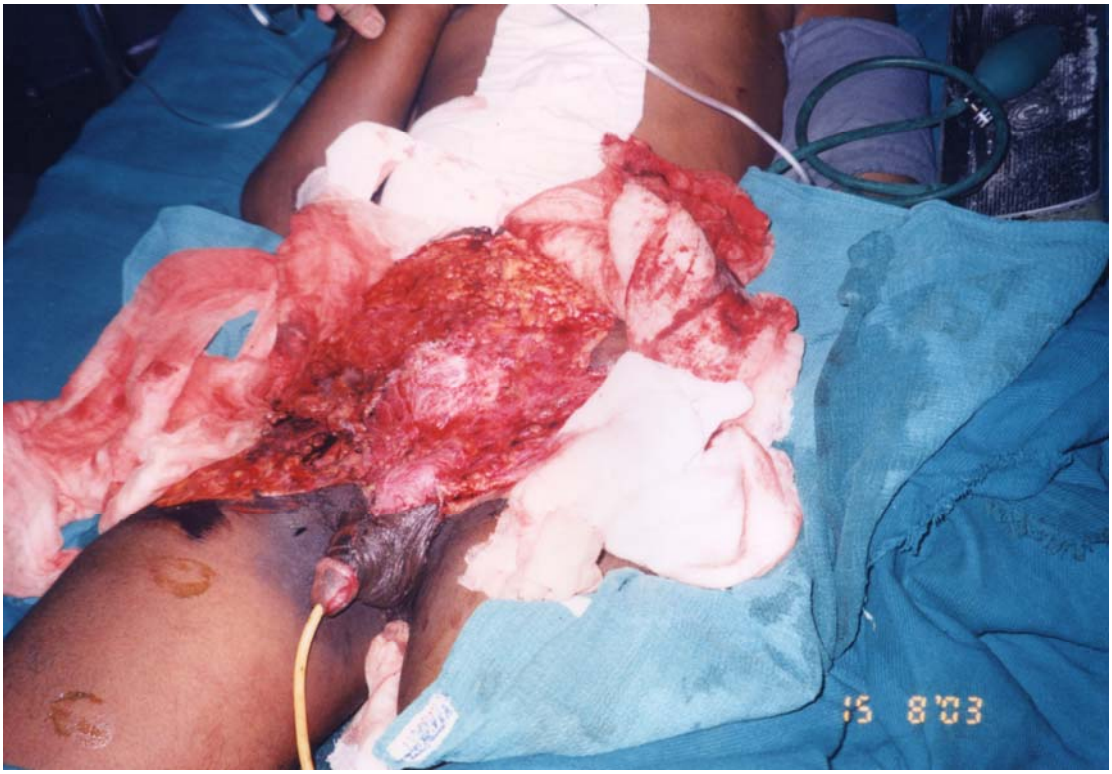
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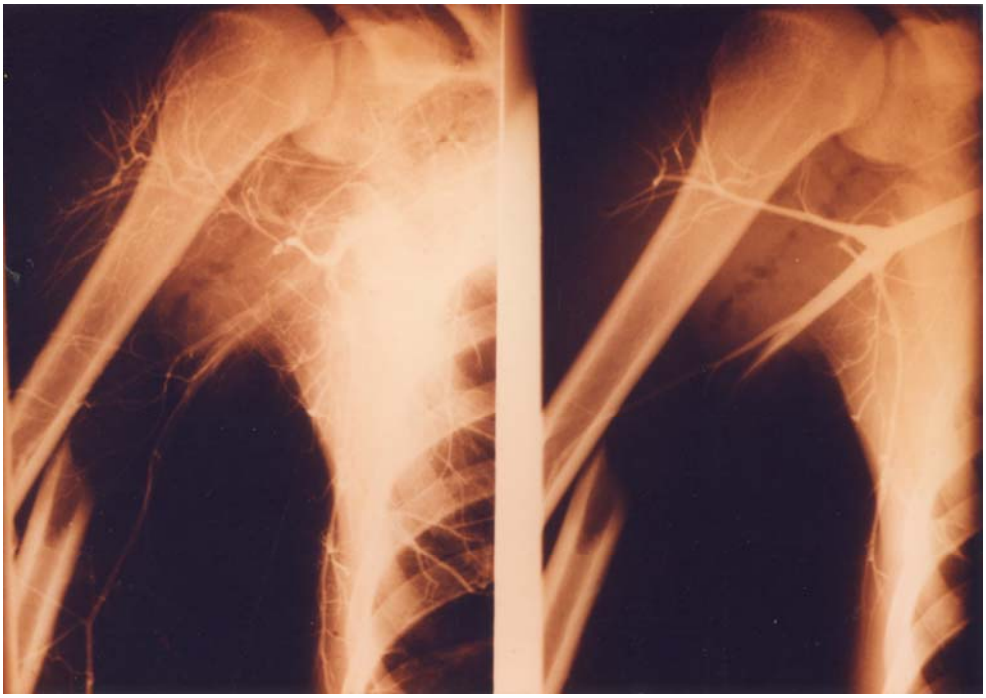

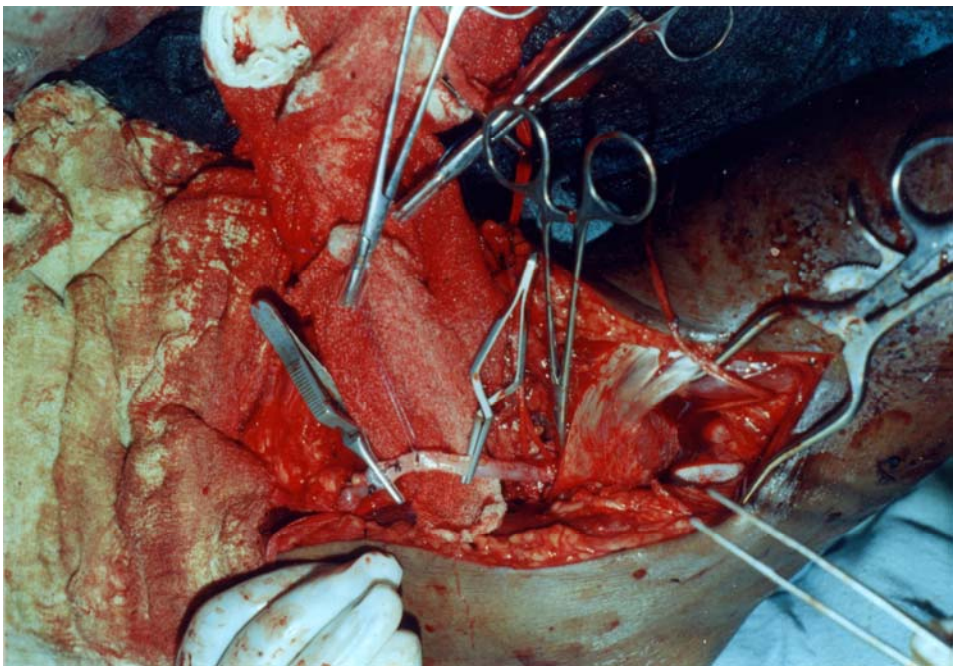

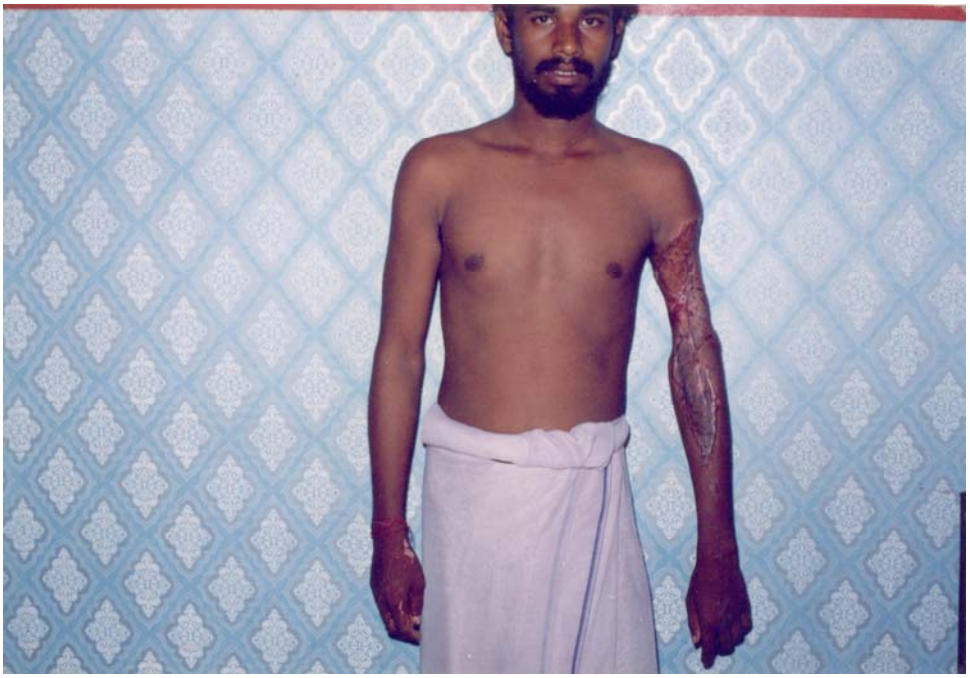

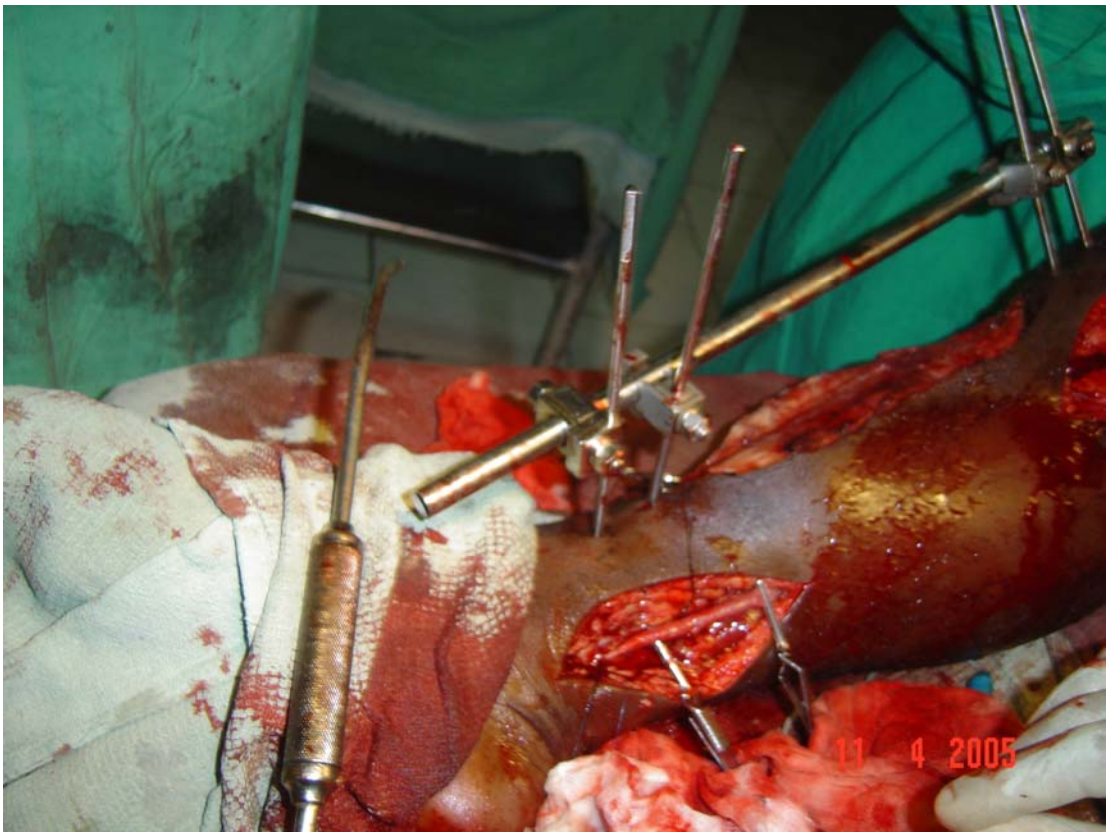
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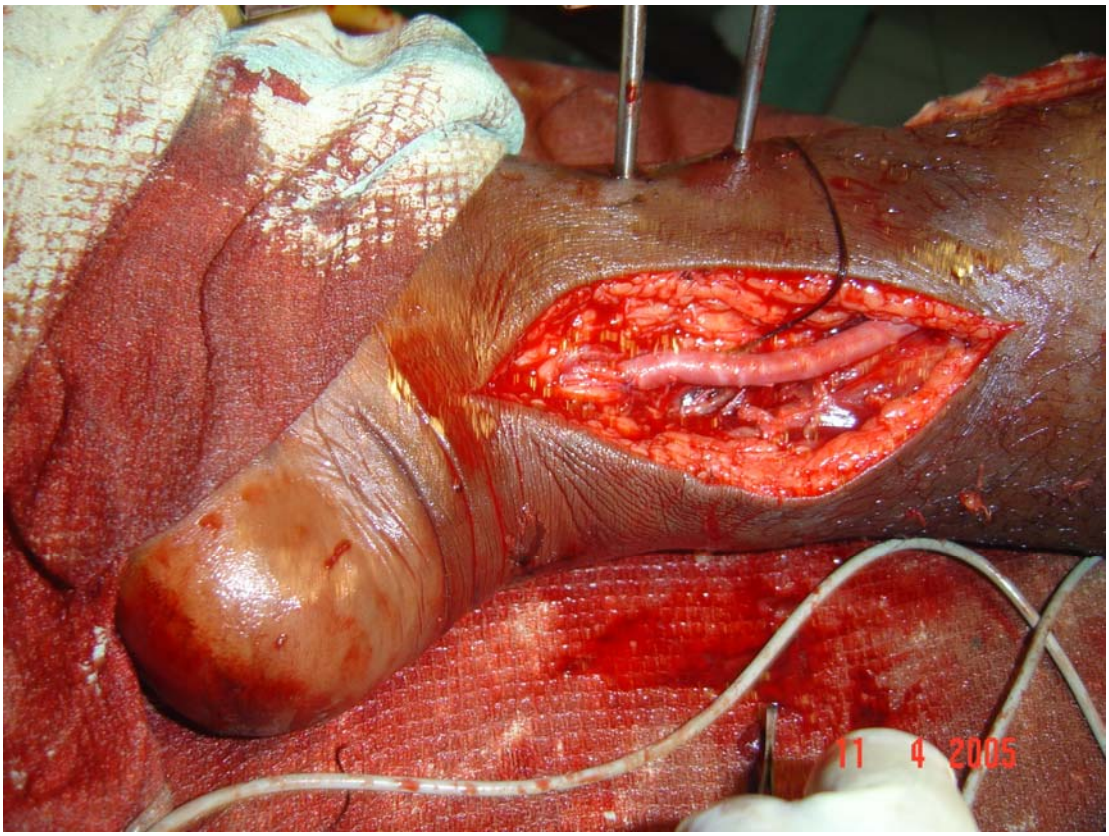
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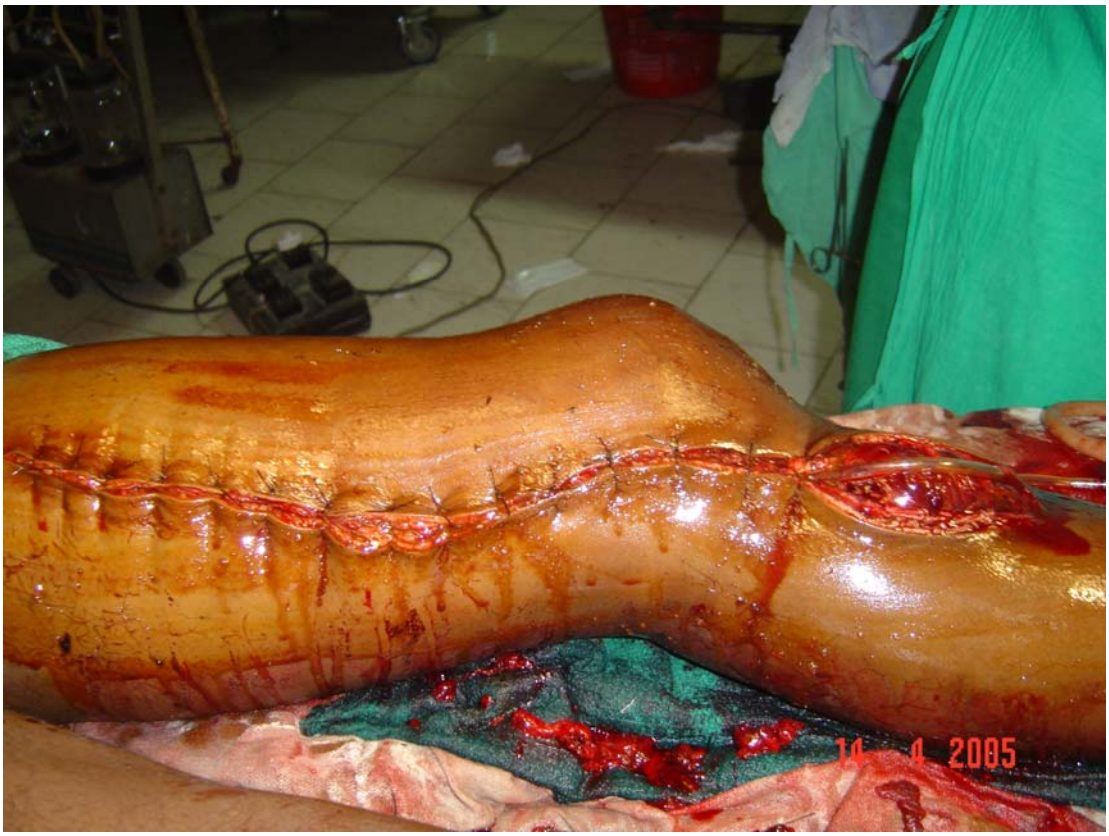
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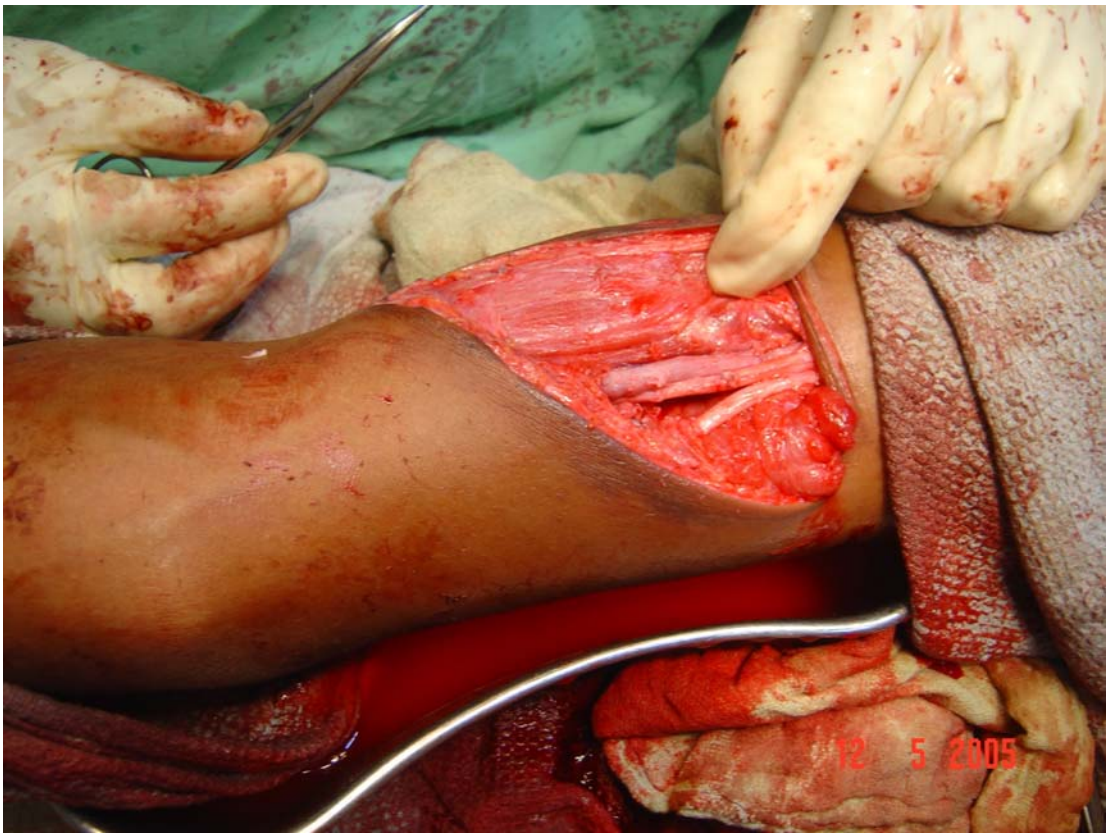
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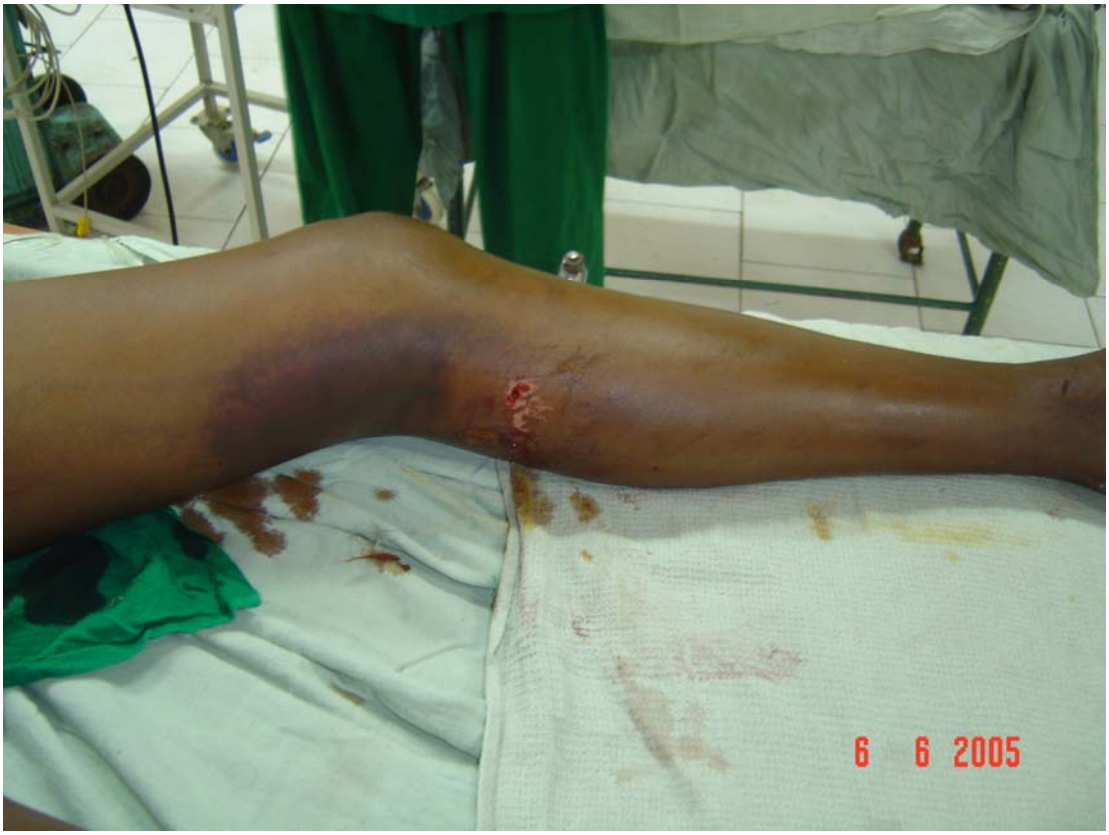
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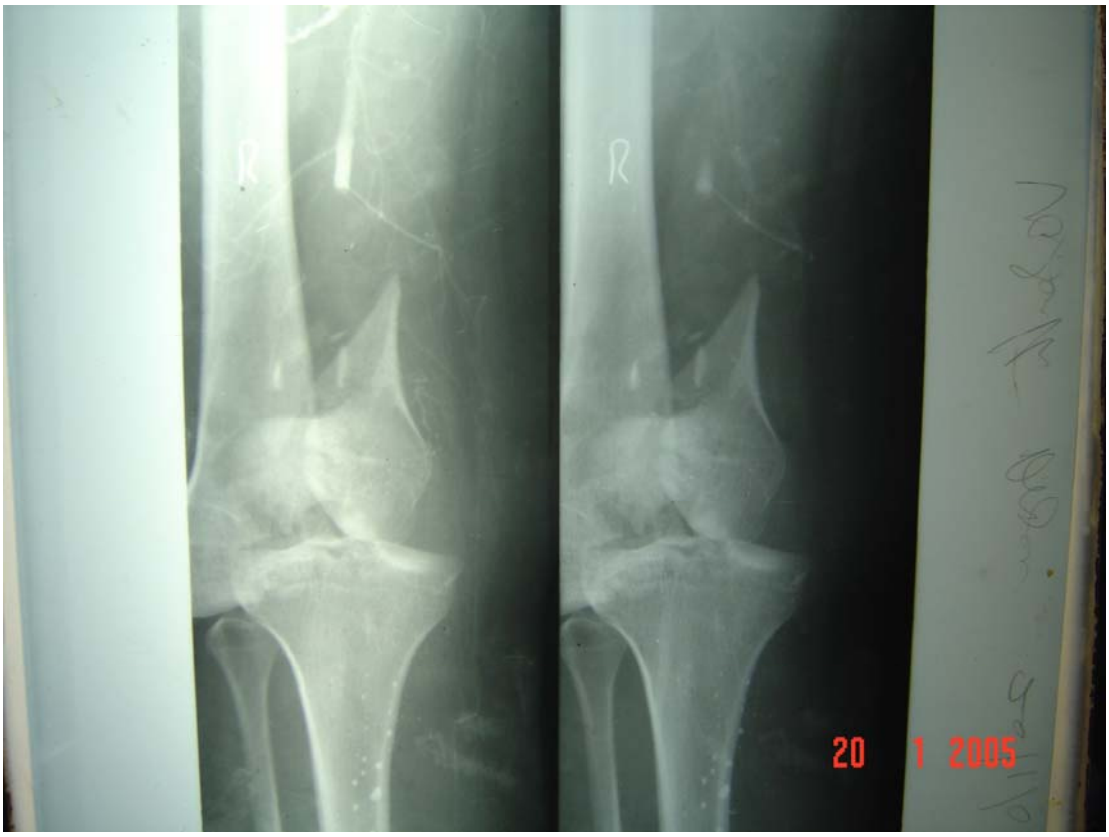
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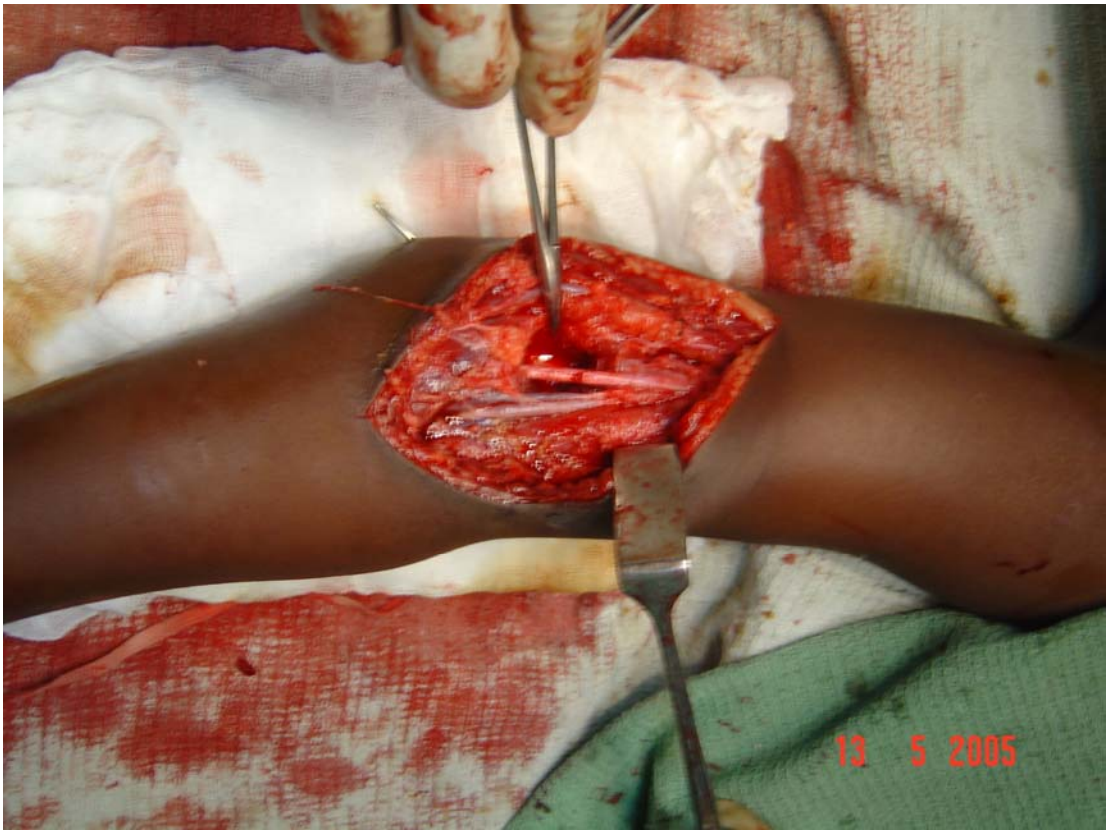
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