

The Orthopaedic Foundation Doctor Guide

June 2018 4th Edition

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Preface to 2016 3rd Edition

This handbook is a guide for Orthopaedic FY doctors. It outlines the basic points, which must be kept in mind when managing orthopaedic patients.

I would like to thank Ms A Hawkins, Clinical Lead, Orthopaedic Consultant and Mr C Cree, Orthopaedic Consultant, for their valuable review and editing of this handbook.

I would also like to thank Dr J Robson, Director of Medical Education, and Prof C Isles, Consultant Physician, for their support in the development of this web-based hospital handbook.

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I would like to thank Dr M Vella Baldacchino for her contributions in updating the 3rd Edition: 'Clerking in' section and her audit: A Closed loop audit: Post operative CRP monitoring following elective total knee and hip replacements.

4th Edition revised by Miss Amanda Hawkins June 2018

INDEX

Title	Page
Abbreviations	4
Documentation	5
Orthopaedic continuation sheet	8
Discharging a patient	8
Writing a referral letter	9
In-patients	10
Requesting ultrasound scan	11
Referring a patient to another department	11
Post-operative assessment checklist	12
Post-operative routine investigations	13
Post-operative delirium	14
Musculoskeletal emergencies	15
Neurological assessment	21
Spinal protocol checklist	26
Radiographs of common fractures admitted	27

Abbreviations

PC - Presenting Complaint	GCS – Glasgow Coma Score
HPC – History of Presenting Complaint	THR – Total Hip Replacement
RHD – Right Handed	TKR – Total Knee Replacement
C/O – complaints of	NOF – Neck of femur
MVA – Motor vehicle accident	CS – Compartment syndrome
RTA – Road traffic accident	C & S – Culture & Sensitivity
LOC – Loss of consciousness	FES – Fat embolism syndrome
BP – Blood pressure	RM – Rhabdomyolysis
SpO ₂ – Peripheral capillary O ₂ saturation	MG – Myoglobin
ROM – Range of motion	ARF – Acute renal failure
CRT – Capillary refill time	CP – Creatine phosphate
FFM – Fast from midnight	PR – Per Rectum
ROS – Removal of stitches	TED – Thromboembolism deterrent
HPC – History of presenting complaint	LMW – Low molecular weight
FBC – Full blood count	UMNL – Upper motor neurone lesion
U & Es – Urea & Electrolytes	DHS – Dynamic hip screw
G & S – Group & save	ORIF – Open reduction & internal fixation
CXR – Chest X-ray	MUA – Manipulation under anaesthesia
NBM – Nil by mouth	
ERP – Enhanced recovery protocol	
USS – Ultrasound scan	
eCn – e-Casenotes	
AVPU - Alert, voice, pain, unresponsive	
AMT - Abbreviated Mental Test score	

DOCUMENTATION: CLERKING IN ORTHOPAEDIC PATIENTS

First page of your clerk in sheet

SURGICAL ADMISSION CLERK-IN

Patient Name:	Attach patient demographic label	Date: ✓	Time: ✓
Hospital/CHI No:		Emergency/Elective: ✓	Ensure all ticks are filled in
Age:		GP Name:	
Address:		Address:	
Tel No:		Tel No:	E-Mail:

Presenting Complaint and Further Questioning:

Age, gender
 Presenting complaint: Ankle pain

History of presenting complaint: Short story about injury - eg if MVA: Alleged MVA (Motor Bike vs Car)mph.....@.....pm today
 Pt was pillion rider, helmet buckled, was hit by car from left side
 No LOC, no nausea or vomiting, no ENT bleeding, no open wound, no head injuries

Document if left/right dominant with respect to arm/forearm/hand injuries

Page two of your clerk in sheet:

Section 1: Past medical history

Past Medical history
 Use SCI Store to list all previous conditions/surgeries, and confirm with patient

Past Medical history:
 Past surgical history:

Section 2: Systemic enquiry

Systemic Enquiry:
Ask about any symptoms systematically:

Cardio: Chest Pain/Palpitations/Syncope

Resp: Copd/Asthma

GI: Abdo pain, weight loss, loss of appetite, PR bleeding, change in bowel habit

Neuro: Seizures/headaches/CVA/TIA

LL: DVT/PE

GU: Prostate symptoms/ UTI symptoms

Page three of your clerk in sheet:

Social History:
Smoker and alcohol: How much and for how many years
Lives with...
Works as....

Family History:
Father/ Mother: History and age of previous MI/CVA/Malignancy

Clinical Examination

Observations: BP, Pulse Rate/rhythm, Temp, SpO2

General:

Alert, conscious, oriented

CVS: Heart sounds

Chest: Auscultate lungs

Abdomen: Examine abdomen

Lower limb:

Orthopaedic:

LOOK: e.g. Deformity, swelling, hematoma, abrasions, wound...etc

FEEL: e.g. temperature, tenderness

MOVE: ROM e.g. limited due to pain, ROM of fingers full Neurological

**assessment: sensation, motor power, reflexes Distal circulation: CRT
(Capillary refill Time)<2sec, distal pulses**

Always ensure:

1. A VTE assessment form is completed and appropriate VTE prophylaxis prescribed on HEPMA.
2. Ensure all blood results are printed or documented in your clerk in sheet

Orthopaedic Continuation Sheet**Trauma inpatients:**

After the ward round, the Consultant on-call will dictate notes that by midday will be added to the patients' notes.

FY doctor needs to document new events e.g.: input from Medics, results of investigations, new symptoms, etc....

Elective inpatients:

Need postop review (e.g.: new blood tests, radiographs, blood transfusion, management of postop confusion, etc....)

If unsure about what to do with a patient contact the team they are under, if they are not available contact the on call team or any member of the Orthopaedic Team.

Discharging a patient

1. Record discharge advice: Diagnosis, Treatment, Postoperative plan (weight-bearing status, ROS, etc....), follow-up plan (hip fractures are not routinely followed-up unless they have had a Gamma Nail).
2. Prescribe Medications and always think about Thromboprophylaxis. We use as standard for most patients Low Molecular Weight Heparin (LMWH). Patients <50kg need a lower dose. If uncertain about which patients need this contact the on-call team, but any patient who is non-weight bearing or in a long leg cast needs prophylaxis, any patient who has warfarin stopped for surgery will need cover with LMWH until this is restarted and the INR is therapeutic. Patients on such drugs as Clopidogrel will need a plan from the team about when this should restart as each surgeon will have their own opinion on this based on patient factors.
 - Routinely after Knee replacement : 2/52 LMWH
 - Routinely after Hip replacement 4/52 LMWH
 - Routinely after Hip fracture 4/52 LMWH
 - Routinely after Tendoachilles rupture 3/52 LMWH for the time they are in plaster NWB
 - Spinal injury: as per protocol page 24

If a patient is starting Warfarin refer to the yellow chart for dosage as per protocol.

3. Discharge summary:

Please include the name of the treating consultant and the postoperative plan that are recorded in the Operative Notes section and Ward Round Dictations.

Review analgesia (e.g.: Co-codamol 30/500 PO 2 tabs qds, Oramorph PO 10 mg 4 hourly (PRN))

Review the need for laxatives

Ensure all changes to medications are included on the discharge letter (with reasons when possible)

Writing a referral letter

To: Doctor	Designation: Registrar	Referred to:Consultant
From: Dr	Designation: FY Dr	From: Orthopaedic Consultant
<p>Dear Doctor, <i>Thank you for seeing this patient....</i></p> <p>COPY HPC (history of presenting complaint)</p>		
<p>Diagnosis: COPY from ORTHO SHEET or OPERATIVE NOTES</p>		
<p>Laboratory investigations: <i>FBC, CRP, INR....</i></p>		
<p>Medication: <i>COPY from CARDEX</i></p>		
<p>Surgical procedures: COPY FROM OPERATIVE NOTES</p>		
<p>Management: COPY FROM WARD ROUND DICTATIONS OR OPERATIVE NOTES</p>		
<p>Purpose of ref: <i>E.g.: For Rehab, for 2nd opinion, for further management, etc....</i></p>		

In-patients

Elective patients attend a pre-operative assessment clinic. On admission, FY doctor needs to ensure all appropriate investigations have been requested and completed. Blood tests and ECGs less than 3 months old do not routinely need repeating as long as there have been no changes in symptoms or medications and no new acute events during that time.

You are responsible for following up and recording every test you order.

1. All patients over 60 years old require a minimum of: FBC, U& Es and ECG (pre-operative routine investigations)
2. CXR should be requested for patients with:
 - Significant cardio-pulmonary disease and unstable symptoms,
 - Recent onset of significant respiratory signs or symptoms,
 - Recent exposure to tuberculosis.
 - As part of the surgical work up for many cancers to exclude metastases.
 - Patients scheduled for critical care.
 - Patients with well-controlled cardiopulmonary disease do not routinely need a CXR.
 - Age alone is not an indication for CXR.
3. Pre-operatively anticoagulant (e.g.: Warfarin) and antiplatelet (e.g.: Clopidogrel) therapy in patients undergoing joint replacement is stopped. If the patient is on aspirin this is not stopped. On admission, FY doctor should check if they have been stopped. Clopidogrel / Warfarin are usually stopped 7 days pre-operatively and alternative thromboprophylaxis is prescribed accordingly.

*N.B. In emergency admissions: if a patient is on Warfarin (e.g. for AF) and needs surgery (e.g. hip hemiarthroplasty), the registrar / consultant will ask for **Reversal of Warfarin** (Vit K 5mg slow IV, or Beriplex 50 units/Kg IV then repeat clotting screen after 20 min)*

4. NBM >6 hours, prescribe fluids PRN and sliding scale in DM type 1

5. Prescriptions:

Enhanced Recovery Protocol (ERP) - Preoperative medication (before THR and TKR):

- Gabapentin 300mg PO 2 hrs Pre-op (omit if renal impairment)
- Paracetamol 1g PO 2 hrs Pre-op
- Oxycontin 10mg PO 2 hrs Pre-op

Postoperative ERP analgesia:

- Paracetamol 1g PO/IV qds (500 mg PO/IV qds if body weight < 50 Kg)
- Oxycontin PO (*or Longtec*): **3 doses**
 - After THR 10 mg/12 hrs (5md bd in > 80 yrs or renal impairment eGFR< 30ml/min)
 - After TKR 15mg/12 hrs (5md bd in > 80 yrs or renal impairment eGFR< 30ml/min)
- OxyNorm (or Oramorph) 10 mg 1 hourly (PRN)
- If any concerns: discuss with Pain Management Team

Laxatives: Lactulose 10ml bd **and** Senna 15mg at bedtime ON (once daily)

Antiemetic PRN: Cyclizine 50mg, PO/IV, 8 hourly (Max/24 hrs: 75-150)

Requesting ultrasound scan (USS)**You are responsible for following up every test you order**

Make sure you know your patient well (history, clinical presentation, lab results) and reason for requesting the investigation.

Fill a request form (use X-ray Request Cards)

Contact the on-call radiologist (to discuss the case)

Send request form.

Referring a patient to another department**You are responsible for following up every referral you make**

Make sure you know your patient well (history, clinical presentation, lab results) and reason for referral (if uncertain discuss with on-call team)

1. Call switch board and ask for name of SHO/Registrar on call for that dept. Ask operator to connect you.

2. Greet Dr. _____ and introduce yourself: Name, FY doctor of ward _____

3. Propose to discuss a patient: *I would like to discuss and refer a patient*

4. Give **name** and **CHI** no., and present case, and current management plan

5. Ask for recommendation: Can you please recommend a plan of management?

Or ask for a review: Can you please come and review the patient in Ward...

6. Update Continuation Sheet: *Spoken to Dr _____, for team to review in ward later.*

Post-operative assessment checklist
(Framework to use when asked to review post-operative patients)

Review case notes (or eCn) and post-operative instructions

- Past medical history
- Medications
- Allergies
- Intraoperative complications
- **Postoperative instructions (very important)**
- Recommended treatment and prophylaxis.

Complete a respiratory status assessment

- Oxygen saturation
- Effort of breathing/use of accessory muscles
- Respiratory rate
- Trachea - central or not?
- Symmetry of respiration/expansion
- Breath sounds
- Percussion note

Complete a circulatory volume status assessment

- Hands - warm or cool, pink or pale
- Capillary return - less than two seconds or not?
- Pulse rate
- Pulse rhythm
- Blood pressure
- Conjunctival pallor
- Urine colour and rate of production
- Wound soakage and drainage from drains (if any)

Complete a mental status assessment

- Patient conscious and normally responsive (AVPU Alert, voice, pain, unresponsive)
- If abnormal determine whether confusion is present (AMT Abbreviated Mental Test score)
- If abnormal determine GCS, oxygen saturation and blood glucose.

In addition to the above physical assessment, record:

- Any significant symptoms, such as chest pain or breathlessness
- Pain and adequacy of pain control
- Urine retention
- Abdominal pain, distension, constipation, bowel sounds and **rule out ileus**

Postoperative routine investigations:**You are responsible for following up every test you order:**

- All patients (elective & trauma) need FBC, U&Es checked 24 hours after surgery
- All elective joints need Check X-ray:
 - THR: Pelvis (for hips) AP
 - TKR: (left / right) Knee AP & Lat
 - Shoulder replacement: (left / right) true AP shoulder (in scapular plane) & Scapular Y view
- All NOF fractures treated by hemiarthroplasty need Check X-ray: Pelvis (for hips) AP
- All femoral nails need Check X-ray: Full length (L or R) femur AP & Lat
- All tibial nails need Check X-ray: Full length (L or R) Tib & Fib – AP & Lat

What is a CRP test?

It is an acute phase protein, which measures the acute phase response to local and systemic events that accompany inflammation.

All surgeries induce an increase in CRP secondary to surgical trauma.

- CRP will always be high up until:

- **DAY 3 for Total Knee replacements**
- **DAY 4 for Total Hip replacements**

FACT:

- In an audit organised between 02/2015 and 06/2015 we looked at the number of CRP requests each time FY1s changed shifts

A Closed loop audit: Post operative CRP monitoring following elective total knee and hip replacements.

M. Vella-Baldacchino, M. Brown, E. O'Flaherty, O Bailey, E. Crane

- CRP requests are **higher** when junior doctors **start their orthopaedic** job and the number of requests decrease with experience.

**As a junior doctor: What should YOU know?**

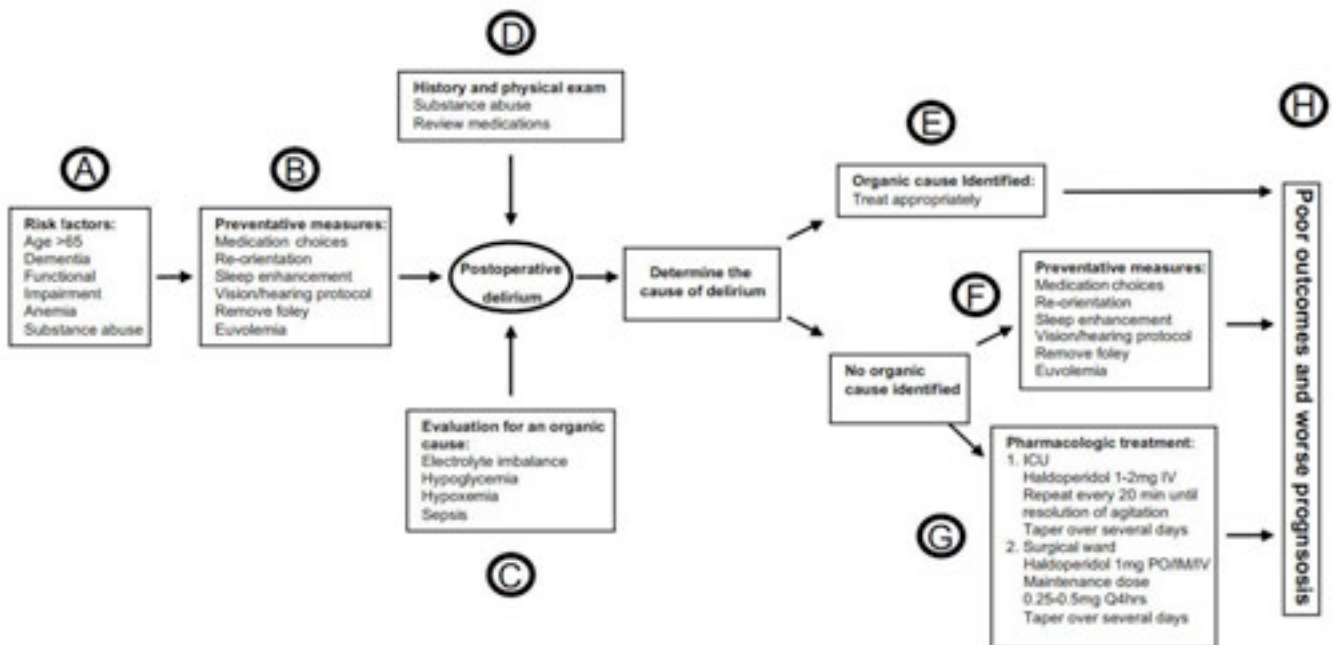
- A rise in CRP in the post-operative period is **expected**
- Therefore, there is **no clinical benefit** of requesting a CRP in the early post-operative period

Postoperative delirium:

Postoperative delirium is a frequent complication in elderly patients following operation for hip fracture. Current research literature notes that 10% to 50% of elderly postoperative patients experience delirium. Patients who have had femoral neck fractures can experience delirium three times more than patients undergoing non-orthopaedic surgery. Postoperative delirium is associated with increased morbidity and mortality

Several theories concerning the pathophysiology of delirium include metabolic encephalopathy, intoxication by drugs [especially anticholinergic ones] and polypharmacy, hypoglycaemia, surgical stress responses, peri-operative hypoxemia, and hypotension. It has been suggested that the type of anaesthesia, administration of opioids, sleep deprivation and non-relieved pain may play a role in the development of postoperative delirium. Despite these, the pathophysiology of delirium has not been studied much and is not well understood.

Systematical approach:



In practice, the commonest causes are drugs, infections (fever, raised white blood cell count) , fluid imbalance and metabolic disorders, cerebral hypoxia, pain, sensory deprivation, urinary retention, and faecal impaction (especially in people with pre-existing dementia).

If suspecting stroke (asymmetrical facial weakness, asymmetrical arm weakness, speech disturbance) contact on-call team to organise urgent CT brain.

Musculoskeletal Emergencies

(i) Compartment Syndrome

Introduction

Compartment syndrome is 'a condition in which the circulation and function of tissues within a closed space are compromised by an increased pressure within that space'. The muscles and nerves of the extremity are enclosed in osteofascial compartments and are therefore susceptible to this condition. It is a surgical emergency which if not recognised and treated early can lead to ischemic contractures, neurological deficit, amputation, renal failure and even death. Compartment syndrome is most commonly seen following trauma, but may occur after ischemic reperfusion injuries, burns and positioning during surgery. Fractures of the tibial shaft and the forearm account for 58% of compartment syndromes.[1]

Pathophysiology

Three theories have been proposed to explain the development of tissue ischemia:

- (1) The increased compartmental pressure may lead to arterial spasm.
- (2) When tissue pressure rises or arteriolar pressure drops this reduces the transmural arteriolar pressure difference to maintain patency and arterioles close.
- (3) If tissue pressure rises then the veins will collapse and venous pressure will rise until it exceeds tissue pressure. This reduces the arteriovenous gradient and as a result reduces tissue blood flow.[2]

When muscles become anoxic histamine-like substances are released and these increase endothelial permeability. Transudation of plasma occurs and this increases the pressure within the compartment. It is only in the late stages of compartment syndrome that arterial flow into the compartment is compromised. Neural tissues demonstrate functional abnormalities (paresthesia and hyperesthesia) within 30 min of the onset of ischemia, and irreversible functional loss after 12 h. Muscle shows functional changes after 2–4 h and irreversible changes beginning at 4–12 h.[3]

Diagnosis

- Clinical

The classical signs of impending compartment syndrome are pain, pallor, paresthesia, paralysis and pulselessness (The 5 p's). However by the time all these symptoms have developed (especially pulselessness) the limb will be non-viable. Clinical diagnosis is made on a combination of physical signs and symptoms. These include **pain out of proportion to the stimulus, pain on passive stretch of the affected muscle compartment**, altered sensation, muscle weakness and tenderness over the muscle compartment.[4]

- Intracompartmental pressures (ICPs)

Kits have been developed to measure ICPs. If on clinical examination an obvious compartment syndrome is present pressure measurement may not be necessary. However it can be a useful adjunct in the diagnosis of compartment syndrome especially in children, unconscious patients and those with equivocal clinical findings. There is inadequate perfusion when the pressure within a closed compartment rises to within 10–30mmHg of a patient's diastolic blood pressure. The diastolic pressure minus the ICP is called the delta pressure. The most commonly used delta pressure is 30mmHg or less.[5]

Treatment

A high index of suspicion is required and early decompression of all at risk compartments is the treatment of choice. Removal of all dressing down to skin, followed by **open extensive fasciotomies** with decompression of all muscle compartments in the limb is the treatment of choice.

In patients whom the diagnosis is being considered and in those in whom resuscitation is proceeding the following steps should be performed:

- (1) Ensure the patient is normotensive, (2) Remove any circumferential bandages all the way down to skin, (3) Maintain the limb at heart level (4) Give supplemental oxygen.[6]

References

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(ii) Fat Embolism Syndrome**Introduction**

Fat Embolism Syndrome (FES) is 'a condition in which fat globules are demonstrated within the lung parenchyma or peripheral microcirculation'. It manifests clinically as acute respiratory insufficiency.[1]

Causes [2]

FES is most common after skeletal injury, and is most likely to occur in patients with multiple long bone and pelvic fractures. Other causes include acute pancreatitis and burns.

Pathophysiology [2]

Two theories have been proposed:

4. **Mechanical theory:** Increased intramedullary pressure after injury forces marrow into injured venous sinusoids leading to obstruction of the pulmonary and systemic vasculature.
5. **Biochemical theory:** Hydrolysis of triglyceride emboli by pneumocyte lipase together with excessive mobilization of free fatty acids from peripheral adipose tissue by the catecholamines results in toxic pulmonary concentration of these acids. The biochemical theory helps to explain non-traumatic forms of FES.

Diagnosis [3]

Various criteria were proposed by different authors such as Gurd and Wilson. Table 1 [4]

□ **Clinical features**

Classic presentation - asymptomatic interval for about 12-72 hours followed by triad:

- Pulmonary changes - Earliest manifestations.
 - Dyspnoea, tachypnoea and cyanosis
 - Respiratory failure - 10% of cases
- Cerebral changes - Due to cerebral edema.
 - Acute confusion, convulsions and coma
- Dermatological changes - Petechial rash due to occlusion of dermal capillaries.
 - Appears within 36 hours and disappears within a week
 - Distributed to the upper anterior portion of the body – conjunctivae, chest, neck, axilla and upper arm. It is theorized to be due to fat particles floating in the aortic arch and embolizing through the carotids and subclavians

Other features:

- Retinal Signs: retinal haemorrhage, and presence of fat droplets in the vessels
- Renal Signs: transient oliguria, lipuria, and haematuria
- **Laboratory studies**
 - Thrombocytopenia, anemia and hypofibrinogenemia.
 - Decreased hematocrit is attributed to intra-alveolar hemorrhage.
 - Cytological examination of urine, blood, CSF and sputum may detect fat globules.
 - ECG findings may show right heart strain or ischemia.
- **Imaging Studies**
 - **Chest radiography:** Diffuse bilateral pulmonary infiltrates (snow storm appearance).
 - **Head CT:** May reveal diffuse white-matter petechial hemorrhages

Treatment [5]

No specific drug therapy for FES is currently recommended. Treatment is essentially preventive (early stabilization of long bone fractures) and supportive (cardiovascular and respiratory resuscitation). Maintenance of intravascular volume (albumin binds to fatty acids) and adequate analgesia are important.

Major criteria (one essential for diagnosis)	Petechial rash
	Respiratory insufficiency
	Cerebral involvement
Minor criteria (four essential for diagnosis)	HR >120 beat per minute
	Temp > 39.4°C
	Retinal signs - fat or petechiae
	Jaundice
	Renal signs - anuria or oliguria
Laboratory findings (one essential for diagnosis)	Thrombocytopenia
	Anaemia
	High ESR
	Fat macroglobulinemia

Table 1: Gurd and Wilson's diagnostic criteria for FES.

References

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2. Gossling HR, Pellegrini VD; Fat Embolism Syndrome: A review of the patho physiology and physiological basis of treatment. *Clin Orthop.*, 1982; **165**: 68-82.
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(iii) Rhabdomyolysis**Introduction**

Rhabdomyolysis (RM) is the “dissolution of sarcolemma of muscle and the release of potentially toxic intracellular components into the systemic circulation and the attendant consequences”. [1]

Causes

A prerequisite for the development of this disease process is muscle injury. There are various causes of RM: vascular interruption, ischemia-reperfusion, crush injury (crush syndrome), improper patient positioning, seizures, extreme exercise, electrical injury and infection. [2]

Pathophysiology [1,2]

As the ischemic time lengthens irreversible muscle damage occurs allowing the release of toxic metabolic by-products:

- Cell membranes are damaged leading to leakage of its contents (e.g. potassium, myoglobin, and hydrogen), depletion of intracellular ATP (due to oxidative phosphorylation malfunction) and vulnerability to oxygen free radicles.
- Intracellular hypocalcaemia (due to Ca^{++} -ATPase malfunction) leads to the activation of intracellular autolytic enzymes (proteases and lipases).
- Release of myoglobin (MG) leads to myoglobinemia. MG contains iron which subsequently becomes an electron donor leading to the formation of *free radicals*. MG also has the potential to release *vasoactive agents* such as platelet activating factor and endothelins that may lead to renal arteriolar vasoconstriction, thus worsening renal function. A high concentration of MG in the renal tubules leads to the formation of tubular casts and resultant tubular obstruction and myoglobinuric Acute Renal Failure (ARF). The incidence of ARF in RM is 10-30%.
- Reperfusion-induced injury: Reestablishment of blood flow after prolonged ischemia aggravates the tissue damage, either by causing additional injury (mediated by oxygen free radicles, leukocytes, leukotrienes and inflammatory mediators) or by unmasking injury sustained during the ischemic period (influx of MB, potassium and phosphorus into the circulation).

Diagnosis [3]➤ **Clinical features:**

- A high index of suspicion is necessary to allow prompt recognition and treatment to avoid the development of ARF and need for hemodialysis.
- Patients present with signs of the underlying cause, muscle pain and shock. With worsening renal function patients develop oliguria and classic “tea colored urine”.

➤ **Laboratory studies:**

- Elevation of serum CPK (its level has been seen to correlate with the development of ARF): Creatine phosphate (CP) is found in striated muscle. CPK catalyzes the regeneration of ATP from the combination of CP with ADP. In RM, muscle cells die and release this enzyme into the bloodstream.
- Urine is found to be dipstick “positive” for blood despite the absence of erythrocytes on microscopic examination due to myoglobinuria.
- Increasing blood urea nitrogen (BUN) and creatinine,
- Other findings include: hypocalcaemia, hyperkalemia (potential for cardiac toxicity), hyperuricemia, hyperphosphatemia, lactic acidosis, and disseminated intravascular coagulation (DIC) from thromboplastin release.

Treatment [4]

- The cornerstone of treatment is aggressive volume resuscitation (maintain a urinary output of >100 mL/hour) and correction of electrolyte imbalance (hyperkalemia, hypocalcaemia and acidosis).
- Bicarbonate use increases MG solubility and induces solute diuresis.
- Mannitol is an *osmotic diuretic*. It is a *volume expander*, *reduces blood viscosity*, and acts as a *renal vasodilator*. Perhaps more importantly, it has been found to be an *oxygen free radical scavenger*.
- Another key element in the treatment and prevention of renal failure is the avoidance of other iatrogenic renal insults such as the use of nephrotoxic antibiotics, IV contrast medium, ACE inhibitors, NSAIDS and so forth.

References

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2. Visweswaran P, Guntupalli J. Rhabdomyolysis. *Critical Care Clinics* 1999; **15**:415-28.
3. Homsy E, Barreiro M, Orlando JMC, Higa EM. Prophylaxis of acute renal failure in patients with rhabdomyolysis. *Renal Failure* 1997; **19**: 283-88.
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Neurological assessment**Glasgow Coma Score**

Score	Eye Opening	Speech	Motor Function
6			Obeys
5		oriented	Localizes
4	spontaneous	confused at times	Withdraws
3	to voice	inappropriate words	abnormal flexion
2	to pain	incomprehensible	abnormal extension
1	none	none	None
15 (best)			

MOTOR**Grade Strength**

- 5 Full ROM against gravity and resistance; normal muscle strength
- 4 Full ROM against gravity and a moderate amount of resistance; slight weakness
- 3 Full ROM against gravity only, moderate muscle weakness
- 2 Full range of motion when gravity is eliminated, severe weakness
- 1 A weak muscle contraction is palpated, but no movement is noted, very severe weakness
- 0 Complete paralysis

Upper Limb

Nerve Root	Key Muscles	Right	Left
C5	Elbow flexors		
C6	Wrist extensors		
C7	Elbow extensors		
C8	Finger flexors (distal phalanx of middle finger)		
T1	Finger abductors (little finger)		

Lower Limb

Nerve Root	Key Muscles	Right	Left
L2	Hip flexors		
L3	Knee extensors		
L4	Ankle dorsiflexors		
L5	Big toe extensors		
S1	Ankle plantar flexors		

Reflexes are graded using a 0 to 4+ scale:

0	Absent
1+	Hypoactive
2+	Normal
3+	Hyperactive without clonus
4+	Hyperactive with clonus

Upper Limb

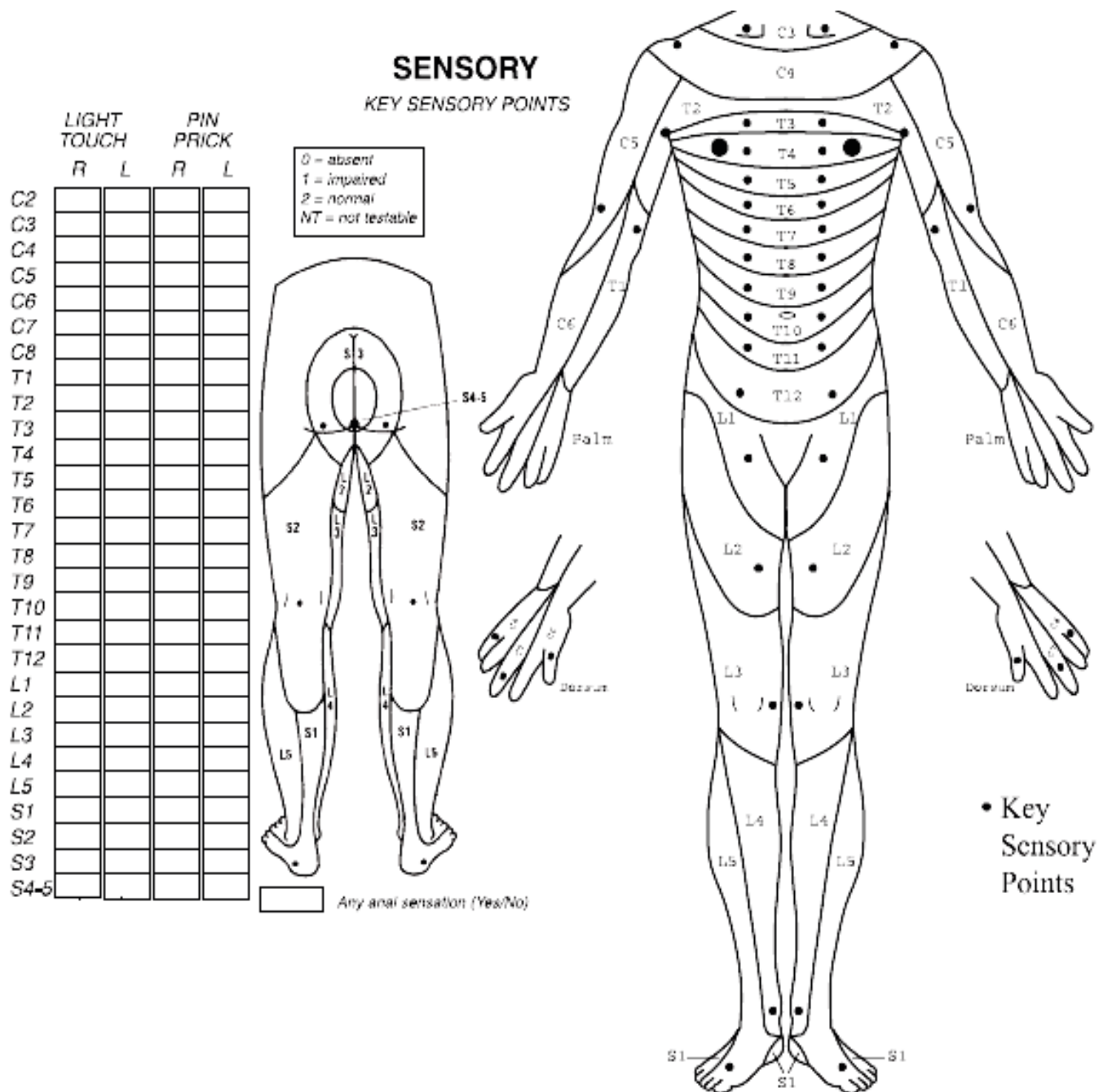
Nerve Root	Reflex	Right	Left
C5	Biceps		
C6	Brachioradialis		
C7	Triceps		

Lower Limb

Nerve Root	Key Muscles	Right	Left
L4	Knee		
S1	Ankle		

Per Rectum (PR) examination:

1. Perianal sensation (Yes / No)
2. Voluntary anal contraction (Yes / No)
3. Faecal Mass (Yes / No)



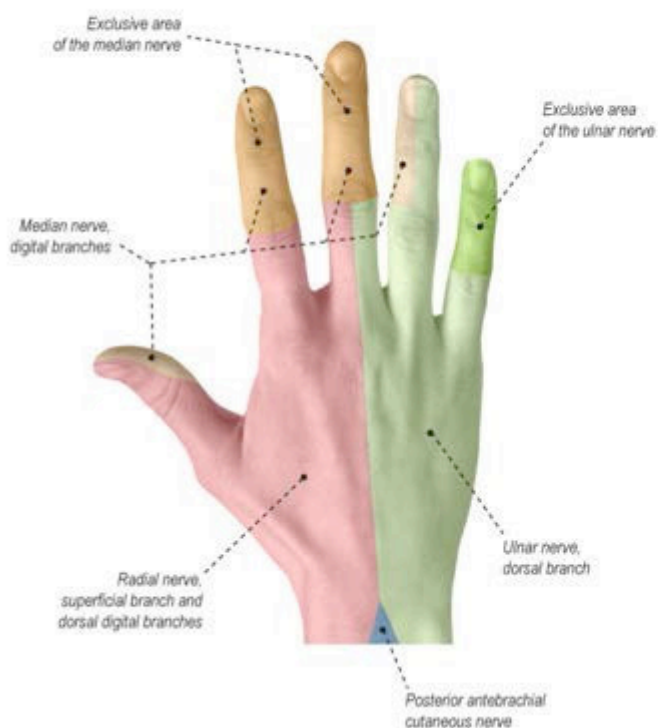
Sensory Territories and Innervations (Volar View)



Dermatomes (Volar View)

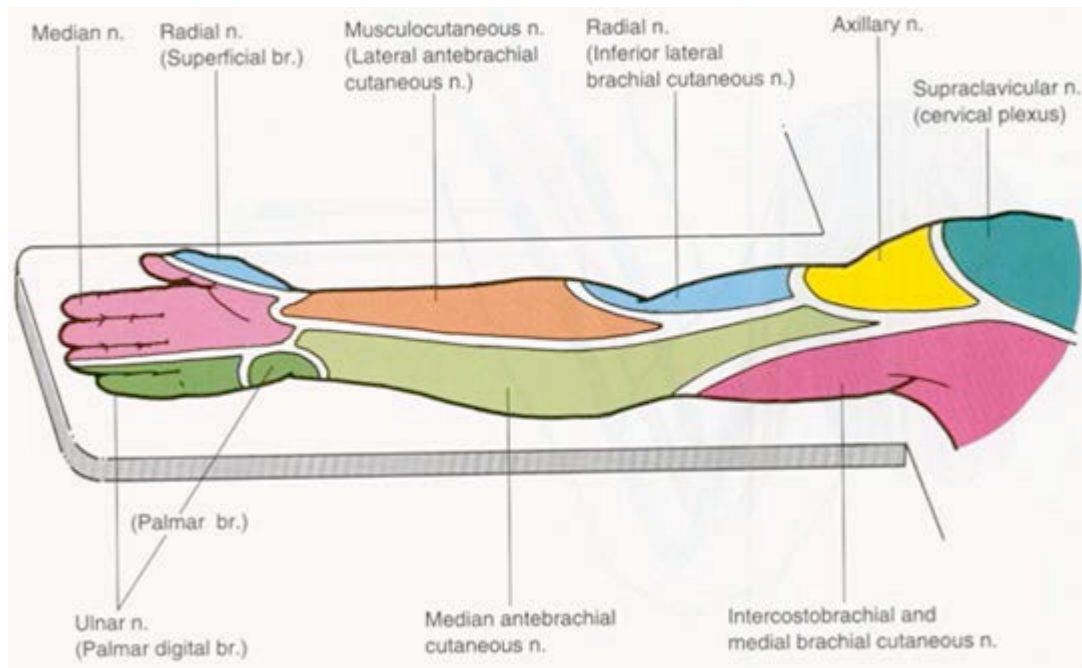


Sensory Territories and Innervations (Dorsal View)



Dermatomes (Dorsal View)





Spinal Protocol Checklist

Spinal immobilization, Log-rolling	<input type="checkbox"/>
H ₂ blocker	<input type="checkbox"/>
TED stockings	<input type="checkbox"/>
LMW Heparin	<input type="checkbox"/>
C ₂ H ₅ OH withdrawal (sedative & thiamine)	<input type="checkbox"/>
Urinary catheter	<input type="checkbox"/>
NBM & NG tube	<input type="checkbox"/>
Pressure area care (spinal bed)	<input type="checkbox"/>
MRSA status/swabs taken	<input type="checkbox"/>
Tetanus status	<input type="checkbox"/>
Respiratory care (airway, O ₂ , chest physiotherapy)	<input type="checkbox"/>

Important definitions:

Spinal shock:

Is a state of transient physiologic (rather than anatomic) reflex depression of cord function below the level of injury, with associated loss of all sensorimotor functions. An initial increase in blood pressure due to the release of catecholamines, followed by hypotension, is noted. Flaccid paralysis, including of the bowel and bladder, is observed, and sometimes sustained priapism develops. These symptoms tend to last several hours to days until the reflex arcs below the level of the injury begin to function again (e.g. bulbocavernosus reflex)

Neurogenic shock:

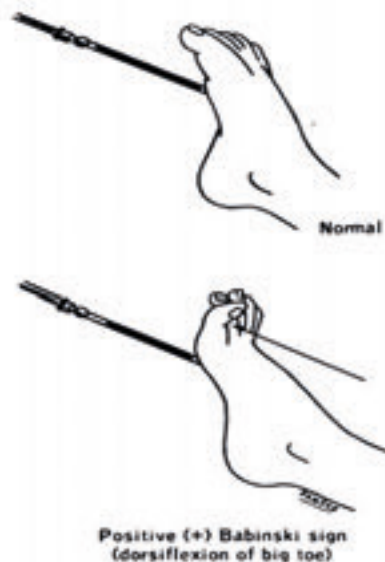
Is manifested by a triad of hypotension, bradycardia, and hypothermia. Shock tends to occur more commonly in injuries above T6, secondary to the disruption of the sympathetic outflow from T1-L2 and to unopposed vagal tone, leading to a decrease in vascular resistance, with associated vascular dilatation. Neurogenic shock needs to be differentiated from spinal and hypovolemic shock. Hypovolemic shock tends to be associated with tachycardia.

Nerve root lesion:

In the absence of spinal shock, motor weakness with intact reflexes indicates SCI, while motor weakness with absent reflexes indicates a nerve root lesion.

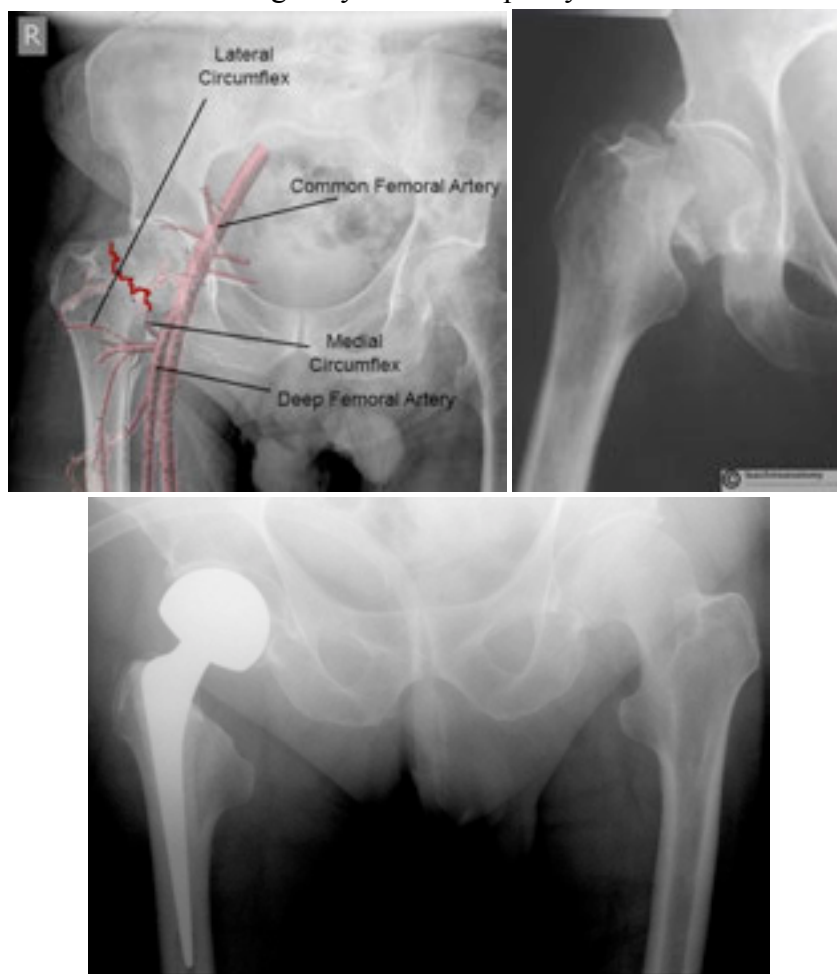
Plantar reflex:

+ve Babinski sign = Upper Motor Neurone Lesion (UMNL)

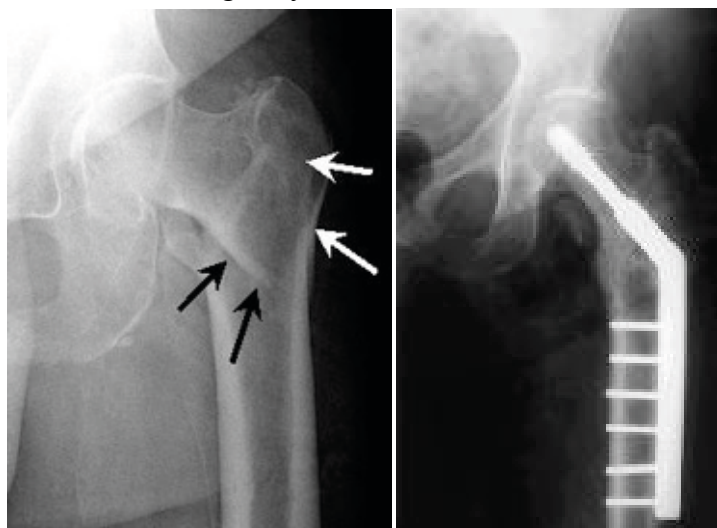


Radiographs of common fractures admitted for management and rehabilitation.

Intracapsular neck of femur fracture managed by hemiarthroplasty



Extracapsular neck of femur fracture managed by a DHS



Surgical neck of humerus fracture managed by ORIF



Distal end radius fracture managed by MUA + K-wires



Tibia fracture managed by Intramedullary nail



Ankle fracture managed by ORIF and a syndesmotomic screw

