# SURGERY

GENERAL SURGERY


1

2.50

# GENERAL SURGERY

## . Index .

# PREOPERATIVE EVALUATION

Source: Washington Dossier. (1)

## • GENERAL EVALUATION

. **Hx & P/E**: Ask PMHx, PSHx (& its complications), Social Hx (Alcohol / Smoking)
Drug hx, FHx (bleeding disorders / hemophilia / Schwinecephea)
& hx of bleeding tendency

. **Routine diagnostic testing** — See table

. **Preop. Meds** — All meds should be continued in the immediate Preop. Period **EXCEPT**:

- . Anticoagulants
- . Antiplatelets
- . Diabetic meds.

* ACEI & Statins (individualized)

| Invx. | Indication for test |
|---|---|
| CBC | . As baseline (due to possibility of blood loss)<br>. or if estimated blood loss >500 ml<br>. Pts w Chronic illness or sx of anemia. |
| U/A | . Urologic Sx<br>. Instrumentation of UT<br>. Possible surgical placement of Prosthesis |
| Serum electrolytes, Cr, BUN (KFT) | . Age >50<br>. Chronic Diarrhea<br>. Major Procedure.<br>. Renal d. / Liver d. / Cardiac d. (ex. CHF) / HTN/DM<br>. Diuretic use / Digoxin use / ACEI use |
| Coagulation Study (PT, PTT, INR, & bleeding time) | . Anticoagulation use<br>. FHx of bleeding disorder<br>. Hx of abnormal bleeding<br>. Liver d. / Malnutrition / Alcoholic |
| β-hCG | . In any ♀ Pt (in childbearing age) |
| LFT (Including Albumin) | . Major Procedure<br>. Hx of liver / Billary d.<br>* **Albumin** is a strong Predictor of Preop. morbidity & mortality |

2

| | |
|---|---|
| Type & Cross match | . If Estimated blood loss >500 ml<br>Otherwise, no need to do it. |
| CXR | . Acute cardiac / Pulmonary Sx<br>. Smokers |
| ECG | . All Pts >50 yrs (do it within 6 months Prior to surgery)<br>. Pts w Known heart d. (do it within 3 months of surgery)<br>. Diabetic pts (silent MI) |

## SPECIFIC CONSIDERATIONS

### • CARDIOVASCULAR Risk

— A leading cause of death esp. in pts w EF <35%

NL EF 55%

**Risk Factors**

- Classifications for Cardiac risk
  1. Modified cardiac Risk Index
  2. Goldman cardiac Risk Index

. Age >70 yrs
. DM
. Unstable Angina — Elective surgery is CI ⊘ & Should be Postponed until further invx.
. Recent MI (within 6 months)
. CHF (manifested as ↑JVP)
  — WORST finding Predicting high cardiac risk.
  — ## Should Precede surgery.
. Valvular HD — esp. Aortic Stenosis (AS) — ↑risk x14
. Arrhythmias & conduction defects.
. Peripheral vascular d.
. Poor Renal impairment.

Periop. **β-Blockers** — ↓ Periop. cardiac events (↓ Risk of MI in high-risk pts)

* So titrate the dose of β-Blockers to maintain HR btw. 60 – 80 b/min in the ABSENCE of hypotension.

## • PULMONARY DISEASE

### Risk Factors

m.c.c of ↑ Pulmonary Risk

- **COPD** — ↑risk by x3-x4
- **SMOKING** — Risk is ↓ after **8** wks of smoking cessation, however, there are physiologic benefit to stop smoking at least **48** hrs before surgery.

*Note
Postpone elective surgery for pts who are actively wheezing.

- Advanced Age
- Obesity (BMI > 30)
- Acute respiratory infxn — elective surgery should be postponed.
- Poor Fxnal status

** ABGs should be done preop. in pts w/ hx of lung d. or smoking as baseline — for comparison w/ postop. studies.

~~** So in Smokers~~

** ECG should be done in any pt >30 yrs w/ SOB to exclude myocardial ischemia.

## • RENAL RISK

### Risk Factors

- Coexisting illnesses (ex. DM, HTN, CAD)
- Metabolic & Physiologic derangement of CKD (Chronic Kidney Disease)

m.c Abnormalities: — Hyperkalemia
— IV volume overload
— Infectious complications

- Type of Procedure — usually major procedures are ass. w/ ↑ morbidity/mortality

~~Hypothyroidism~~

## Evaluation

**Hx** – Ask about the specific **etiology** of CKD
(Bcz Pts ω CKD due to HTN or DM are at ↑ risk of perioP. morbidity & mortality)

– Ask about **dialysis** — 1st time of dialysis
Amount of fluid removed
PreoP. wt

This gives imP. info. about Pt's expected volume status.

* Also urine output should be documented.

**P/E** Assess vol. status — ↑ JVP
crackles on lung exam. ] Indicate vol. overload

**Invx**

– CBC, Electrolytes, Cr & BUN should be done
– U/A, & culture (as indicated)

**Mgt**

– Dialysis should be done within 24 hrs of surgery — if indicated

– Intravascular volume status monitory

* **BOTH** Hypovolemia & volume overload are BAD!! ☹

**Indix for dialysis**
- Intravascular vol. overload
- Hyperkalemia
- Severe metabolic acidosis
- Complications of uremia (Encephalopathy/ Pericarditis)

**NL platelet number can mask platelet dysfunction in Pts ω Chronic uremia**

* Chronic uremia causes Plt dysfxn.

** CAD is the m.c.c of DEATH in Pts ω Chronic renal insufficiency.

* Risk of Acute Renal Failure (ARF) in Pts **ω/out** Pre-existing CKD is 1.5 – 2.5% for cardiac surgical Procedures while it's >10% for Pts undergoing AAA repair!

**Risk factors of ARF**
- ↑BUN / ↑Cr
- CHF / Intraop. Hypotension
- Advanced age
- Sepsis
- Aortic cross clamping
- Administration of nephrotoxic meds or radiocontrast agents

Prevention ⇒ **HYDRATION!**

## • CEREBROVASCULAR RISK

— Uncommon  < 1% in general population
2-5% in cardiac surgical pts

* Majority of events are postop — mostly due to hypotension or cardiogenic emboli during AFib.

* Pts w RECENT CVA, surgery should be delayed at least 2 wks (Ideally, 6 wks)

**Asymptomatic Carotid Bruit**

- Relatively common in pts >55 yrs
- NO ↑ in risk of stroke (if asympt.)

## • INFECTIOUS DISEASE

(inf.)

* Assessment of Risk Preop.:-

**• Pt-specific RF:**

- Age
- DM
- Obesity
- Immunosuppression
- Malnutrition
- Pre-existing infxn
- & other Chronic illnesses.

**• Procedure-specific RF** — see table.

"Washington"

| Wound Class | Definition | Example | Wound infxn rate | Microorganism |
|---|---|---|---|---|
| CLEAN | • Non-traumatic<br>• NO entry of GI / biliary / tracheobronchial / Resp. / or GUT. | - Wide local excision of breast mass<br>- Hernia repair<br>- Thyroid surgeries | <2% | Staph. |
| CLEAN-CONTAMINATED | • Resp./GU/GI entered BUT minimal contamination<br>i.e. controlled entrance to cavities | - Gastrectomy<br>- Hysterectomy<br>- Cholecystectomy | <10% | Related to viscus entered. |
| CONTAMINATED | • Open, fresh, traumatic wounds.<br>• Uncontrolled spillage from UNPREPARED hollow viscus.<br>• minor break in sterile technique. | - Ruptured appendix<br>- Resection of unprepared bowel. | 20% | Related to viscus entered. |
| DIRTY | • Open, traumatic, dirty wounds<br>• Traumatic perforated viscus<br>(PUS) in the operative field | - Intestinal fistula resection<br>~~- Sinus tract Abscess~~<br>- Hartmann's for diverticular perforation. | 30-70% | Related to viscus entered |

## Prophylaxis

→ Strict Sterile techniques
maintain NL body temp. & NL blood glu levels & hyperoxygenation

→ Antibiotic prophylaxis ( ↓ superficial wound infxn risk)
within (0-60) min of incision.

→ Preop. Skin antisepsis by Chlorhexidine-alcohol (better) or Povidone-iodine Scrub.

**ANTIBIOTIC PROPHYLAXIS**

| NATURE OF OPERATION | Likely PATHOGENS | RECOMMENDED ANTIBIOTICS |
|---|---|---|
| **CARDIAC:** Prosthetic valve & other Procedures | • Staphylococci<br>• Corynebacteria<br>• Enteric G-ve bacilli | - Vancomycin & Cefazolin (1st g.)<br>- Vancomycin & Aztreonam (If penicillin/ ceph. allergy) |
| **THORACIC** | • Staphylococci | • Cefazolin (1st g.)<br>• Vancomycin (If allergy) |
| **VASCULAR:** Peripheral bypass or aortic surgery w/ Prosthetic graft | • Staph.<br>• Streptococci<br>• Enteric G-ve bacilli<br>• Clostridia | • Cefazolin (1st g.)<br>- Vancomycin & Aztreonam (If allergy) |
| **ORTHOPEDIC** Total joint replacement or internal fixation of #s | Staph. | • Cefazolin (1st g.)<br>• Vancomycin (If allergy) |
| **GI** | | |
| • Upper GI & hepatobiliary | Enteric G-ve bacilli<br>Enterococci<br>Clostridia | • Cefazolin (1st g.)<br>• Cefotetan (2nd g.)<br>• Cefoxitin (2nd g.)<br>• Clindamycin + Gentamycin (If allergy)<br>• Ciprofloxacin & Metronidazole (If allergy) |
| • Colorectal or appendectomy (w/out perforation) | Enteric G-ve bacilli<br>Anaerobes<br>Enterococci | • Cefoxitin<br>• Cefotetan<br>• Ciprofloxacin & metro (If allergy) |

| | | |
|---|---|---|
| OBS/GYNE | - Enteric G-ve bacilli<br>- Anaerobes<br>- Group B Strep.<br>- Enterococci | . Cefotetan (2nd g.)<br>. Cefoxitin (2nd g.)<br>. Cefazolin (1st g.)<br>. Clindamycin & Gentamycin (IF allergy) |

## • DM

- Pts w DM have 50% ↑ risk of mortality & morbidity vs. nondiabetics.
- They have more infectious complications & impaired wound healing.

* VASCULAR DISEASE & silent CAD must always be considered!
* When undergoing an elective surgery blood glu level should be controlled btw. (100-250) mg/dl

### Preop. evaluation

#### ① Pts w diet-controlled DM

— maintained safely w/out food or glu infusion before surgery.

#### ② Pts on oral hypoglycemic meds

— Discontinue the evening before scheduled surgery.

BUT Pts who take long-acting oral hypoglyc. → discontinue 3-4 days before surgery.

#### ③ Pts who take insulin

— These require insulin AND glucose Preop to Prevent Ketosis & catabolism

So Pts undergoing major surgery should recieve ½ insulin dose & 5% dextrose IV at (100-125) ml/hrs

MI (often w/ atypical presentation) is the leading cause of DEATH Periop. among diabetic Pts.

## ANTICOAGULANTS

Preop.

— Considered safe to perform surgery → when INR is < 1.5

— If INR 2-3 → discontinue meds for 4 days preop.
>3 → discontinue meds for longer periods.

∴ So measure INR preop. (the day before)

For emergent procedures → you can give Factor VII — immediate effects
Vit. K — win 8hrs

M.C indications for warfarin tt
① A. Fib
② VTE
③ mechanical heart valve.

## STEROID DEPENDANCE

— All pts who need steroids should take hydrocortisone in the IV form, even if they used to take them orally bcz their adrenals depend on external steroids.

* Stress dose is 100 IV :→ one in the evening before surgery.
→ another at the beginning of the operation.

Yash Ghaith
The end.

# POSTOP CARE

## • IV Fluids

* Intravascular volume of surgical Pts is ↓ by BOTH — Insensible fluid losses
  \ Redistribution into 3rd space.

∴ So as a general RULE:-
Pts should be maintained on IV fluids until they're tolerating oral intake.

NOTES
- Extensive abdominal procedures require AGGRESSIVE fluid resuscitation.
- Insensible fluid losses ass. w an open abdomen can reach 500–1000 ml/hr !!

## • DVT Prophylaxis

— Should be started Pre-op in Pts undergoing major procedures (bcz venous stasis & relative hypercoag. occur during operation).

| Levels of VTE Risk & Recommended Prophylaxis | | | |
|---|---|---|---|
| LEVEL OF RISK | | Risk without Prophylaxis | Recommended Prophylaxis |
| LOW | - Major surgery in mobile Pts.<br>- Pts who are fully mobile | <10% | Early & Aggressive Ambulation |
| MODERATE | - Open gyne/uro surgical Pt<br>- Pts who are on bed rest "Sick"<br>- Mod. VTE risk + high bleeding risk | 10-40% | • LMWH, UFH or Fondaparinux<br>• Mechanical thromboproph. (Pneumatic compression devices) |
| HIGH | - Total hip & Knee Replacement<br>- Major trauma.<br>- Spinal cord injury<br>- High VTE risk + high bleeding risk | 40-80% | • LMWH, ~~UFH~~ Fondaparinux, Warfarin (INR 2-3)<br>• Mechanical thromboproph. |

## • PULMONARY TOILET

* Pain & immobalization ↓ clearance of secretions & ↓ recruitment of alveoli.

- Early mobilization
- Incentive Spirometry
- Cough
- & Deep breathing exercises

} Should be done Postop.

**Incentive Spirometry**

*The Pt can document Tidal volume & will have an "incentive" to increase it.

⇒ Pt breathes in from the device as slowly & as deeply as possible, then holds breath for 2-6 sec.

⇒ Pts w inadequate Pulmonary toilet can develop fever, hypoxemia, Pneumonia, & ATELACTASIS.

## • MEDICATIONS

### ➤ Antiemetics

Postop. nausea is common in { Pts after GA; Pts receiving narcotics.

### ➤ Ulcer Prophylaxis

Indx: . For Pts w hx of PUD
. For Pts w coagulopathy or Prolonged ventilator-dependent.

** Give acid-reducing agents or cytoprotective agents (ex. Sucralfate)

### ➤ Pain Control ex. Morphine (2-10)mg IV shot

It's imp. to control Pain, because if inadequatly controlled { Slow recovery; ↑ Post-op. complications; Pts less likely to ambulate & take deep breath; more likely to be tachycardic.

### ➤ Antibiotics

For specific cases.

- **LABS**

**CBC**

In any procedure w/ significant blood loss.

**KFT**

Important in ① NPO pts, ② renal insufficiency, ③ pts recieving large volume of IV fluids & ④ TPN or transfusion.

**Coagulation Studies**

Indx. Pts who have had insults to the liver
- large transfusion required.

**Daily ECG & series of 3 troponin I levels** (8hrs apart)

To monitor MI in pts w/ significant cardiac risk factors.

**CXR**

Indx:
- If thoracic cavity is entered
- Central venous access is attempted.
- Pts w/ significant Pulmonary d. or CVD.

Yash Ghaith
The End.

# POSTOP COMPLICATIONS

Source: Washington Surgical Recall 11

## ① FEVER

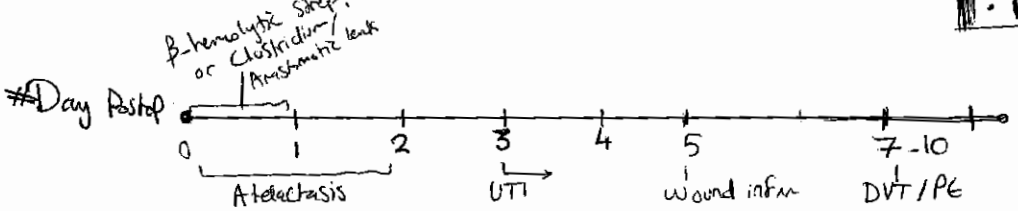
IF Intraop. fever THINK →
- Malignant Hyperthermia
- Transfusion Reaction
- Pre-existing infxn.

IF Postop. fever >38.5° THINK of → 5 W's

- **W**ind — Atelectasis
- **W**ater — UTI
- **W**ound — Wound infxn
- **W**alking — DVT / Thrombophlebitis
- **W**onder drugs — drug fever.

(imp.)

Complete fever work up (Septic workup)
- P/E (temp. / look at wound / ...)
- CBC
- U/A
- CXR
- Blood culture




* Notes

* IV site infxn / central line } fever can occur ANYTIME
* Drug fever

* UTI — occurs anytime AFTER day 3 Postop.

⇒ Pneumonia will happen in about 3 days if atelectasis is not resolved!

⇒ Clostridia infxn appears as painful bronze-brown weeping wound.

**Malignant Hyperthermia** — AKA Scholine apnea

- Develops shortly after the onset of anesthesia (Halothane / Succinylcholine)
- Temp. >40°
- Usually +ve FHx
- also malignant hypercalcemia & metabolic acidosis

(IV) Dantrolene (Antidote)
100% oxygen
Correct acidosis & cooling blankets
** Watch for developing myoglobinuria

# ② CARDIOVASCULAR COMPLICATIONS — Postop. MI / CHF

## • Postop MI

2/3 of Periop. MI occur Postop day #2 to 5

Usually Silent or w/ atypical Sx. Why? c/z most Pts Postop. are taking Painkillers, so MI passes unnoticed. (like Pts w/ DM)

### ** Risk Factors

- Hx of Angina / MI (esp. if recent)
- Advanced age
- CHF
- Extensive surgical Procedure.
- Q's on ECG / ECG Changes
- $S_3$ / Aortic stenosis.

### ** C/P

- Often w/out Chest Pain (or atypical)
- New-onset CHF
- New-onset cardiac dysrrhythmia
- Abnormal V/S: Hypotension / tachypnea / tachycardia or bradycardia.
- Neck / Arm Pain.

### ** ECG Findings

- Flipped T-waves
- STE or ST depression.
- Dysrrhythmia (New-onset Afib. / PVC/VT)

### ** Labs

Troponin I (3 samples, 8 hrs apart)
Cardiac isozymes (↑CK-MB)

### ** "BEMOAN"

**BE**ta-blockers.
**M**orphine — for Pain control
**O**$_2$
**A**spirin
**N**itrate (after r/o hypotension)

## • Postop. CHF

** Cause . THINK Fluid overload (due to excessive fluid administration)
. MI

** P/E
look for SxS of fluid overload. (ex. tachypnea / edema / ↑JVP crackles)

** Labs
- Troponin I / BNP
- CBC / ABG
- KFT

PA catheterization is an INVASIVE measure that can be used to assess vol. status.

** Mgt
- $O_2$
- Diuretics
- Morphine
- Arterial vasodilators (↓afterload)
- Inotropic agents — vasopressors

## ③ RESPIRATORY COMPLICATIONS

- Atelactasis & Pneumonia
- Postop. resp. failure.
- PE
- Aspiration Pneumonia
- Pneumothorax
- COPD/Asthma exacerbation.

### • Atelactasis — the m.c.c of postop fever day 1-2
└ It's collapse of the alveoli

** Risk Factors
- COPD / Smoking
- Abd. / thoracic surgery
- Poor Pain control
- Oversedation

Mechanism:
Pt can NOT breathe deeply 2ry to Pain on inspiration.

** CAUSES
- Inadequate alveolar expansion:
  (Poor ventilation of the lungs during surgery / or inability to fully inspire 2ry to Pain)
- High levels of inspired $O_2$

** SIGNS
Fever / tachypnea / tachycardia
↓ breath sounds & rales
↑ density on CXR

** PROPHYLAXIS
= Preop. smoking cessation — Good Pain control
— Postop incentive spirometry

III
- Postop. incentive spirometry
- Deep breathing
- Coughing
- Early ambulation
- Chest physiotherapy & NT suctioning

## • Aspiration Pneumonia

Pneumonia following aspiration of vomitus.

### ** RF

- Intubation / Extubation
- Impaired Consciousness (Drug/ethanol overdose)
- Dysphagia
- Non-fxning NG tube
- Trendelberg Position
- Emergent intubation on full stomach
- Gastric dilatation.

### ** SFS

- Resp. failure / Cyanosis
- Chest Pain
- ↑ Sputum Production/cough
- Fever / tachypnea
- Infiltrate on CXR.

**Common lobes**

- Supine — RUL
- Sitting/Semirecumbinant — RLL

**CXR FINDINGS**

- Early — Fluffy infiltrate or NL CXR
- Late — Pneumonia or ARDS

### ** Common Pathogens

- Community aquired → G+ve / mixed
- Hospital / ICU → G-ve rods

### ** Invx

- CXR
- Gram stain / Sputum culture
- BAL

### ** ttt

Abx — If Pneumonia
Intubation — If respiratory failure.
Ventilation ± PEEP — If ARDS develops.

**Mandelson's Synd.**

Chemical Pneumonitis 2ry to aspiration of stomach content (gastric acid).

- NO antibiotic Prophylaxis is required in aspiration Pneumonia.

# RENAL COMPLICATIONS
- Urinary retention
- Acute Renal Failure (ARF)

## • Urinary Retention — common!
- It's enlarged urinary bladder resulting from **medications** or **spinal anesthesia**

Remember!
Classic sx of urinary retention in Elderly is
↓
CONFUSION!

** Dx
- P/E — Palpable bladder
- Bladder residual vol. upon placement of cath.

** tt
Foley's catheter

inf. — Note
- With **massive** bladder distention, YOU **DON'T** drain all urine immediately! (Why?)
  To AVOID vasovagal reaction.
  Myb clamp after 1 L drainage & then drain the rest Slowly

## • Postop Renal Failure
↑ Serum Cr & ↓ Cr Clearance
— usually ass. w ↓ UOP

DDx
- **Prerenal** ⇒ Inadequate Perfusion
  (Inadequate Fluids // Hypotension // CHF)
- **Renal** ⇒ Kidney Parenchemal dysFxn.
  (ATN // Nephrotoxic contrast or drugs)
- **Postrenal** ⇒ Obstruction to outflow of urine
  (Foley's cath. obst. / Stone // BPH // Bladder dysfxn — meds / spinal anesthesia)

UOP
- Anuria < 50 cc /day
- Oliguria > 50 cc but < 400 cc /day

## * Work up for ARF

- KFT
- U/A
- FENa (Fractional Excretion of $Na^+$)
- Renal U/S — to r/o obst. uropathy
  - to assess chronicity
  - to evaluate renal vasculature (by doppler U/S)

**Formula of FENa**

Remember it as "You Need Pee"

$$FENa = \frac{U_{Na^+} \times P_{Cr}}{U_{Cr} \times P_{Na^+}} \times 100$$

These measurements should be obtained before administration of diuretics.

**Interpretation**

- BUN/Cr ration
  - > 20:1 → Prerenal
  - < 20:1 → Renal
- Specific gravity
  - > 1.02 → Prerenal (as the body tries to hold on to fluids)
  - < 1.02 → Renal (Kidney has ↓ ability to concentrate urine)
- U Na
  - < 20 → Prerenal
  - > 40 → Renal
  - $FE_{Na^+}$ < 1 prerenal, > 1 Renal
- Urine osmolality
  - > 500 → Prerenal
  - < 350 → Renal

*** Indx of Dialysis**

- Fluid overload
- Refractory hyperkalemia
- Severe metabolic acidosis
- BUN > 130
- Uremic compl. (encephalopathy/ Pericarditis)

## NEUROLOGIC COMPLICATIONS — Delerium Tremens, Postop CVA

- **Delerium Tremens (DT)** — 3rd/4th day Post-op
  - very common in alcoholic whose drinking is suddenly interrupted by surgery.

Minor alcohol withdrawal occurs 6-8 hrs after cessation of alcohol intake → resolves in 24-48 hrs.

** C/P
- Confusion ⓦ hallucination & pt become combative.
- HTN, tachycardia, fever & extensive diaphoresis.

** Tt
- IV benzodiazepines — Standard Tt
- IV alcohol (5% in 5% dextrose) — not usually done.

## • Post-op CVA

* S&S
  - Aphasia
  - motor/sensory deficits

* work up
  - Head CT (must r/o hmg if anticoagulation is going to be used)
  - Carotid doppler U/S — to evaluate for Carotid occlusion.

* ~~tx~~
  - Aspirin ± heparin (if feasible Postop.)
  * Thrombolytics are NOT usually a postop option.

* Prevention
  - AVOID Hypotension
  - Continue Aspirin Preop. in high-risk pts
  - Preop. Carotid doppler Study.

## GI COMPLICATIONS

- Constipation
- Paralytic ileus
- mechanical bowel obst.
- Postop Pancreatitis
- Short bowel synd.
- Blind loop synd
- Dumping synd.
- Jaundice

**NG tube complications**

- Aspiration pneumonia/ Atelectasis (esp. if NGT is clogged)
- Sinusitis
- Minor UGI bleeding:
  - epistaxis
  - Pharyngeal/gastric irritation.

## • Paralytic ileus — occurs in the 1st few days Postop.

↳ Postop. small bowel obstruction (NOT mechanical) but functional

**CAUSES**
- Laparotomy
- Hypokalemia
- Narcotics
- Intraperitoneal infxn.

**S&S**
- Mild distention
- NO Passage of flatus
- ABSENT bowel sounds

* Ileus resolves spontaneously

**Signs of resolving small bowel obst.**

↳ Passage of flatus / stool

***The order of recovery of bowel fxn Post-op.**

1st — Small Intestine
2nd — Stomach
3rd — Colon

- **Mechanical** small bowel obstruction

  CAUSES
  - ADHESIONS (m.c.c)
  - Incarcerated hernia

  AXR Finding
  - Dilated loops
  - Multiple air-fluid levels.

  CT
  ↳ for confirmation

  Mgt
  Surgical intervention.

- **Constipation**

  CAUSES:
  - Narcotics
  - Immobility

- **Short bowel Synd.**

  ↳ Malabsorption & diarrhea resulting from extensive bowel resection (<120 cm of small intestine remaining)

  Initial tt: TPN followed by many small meals chronically.

- **Blind Loop Synd.**

  It's bacterial overgrowth in the small intestine.

  CAUSES: Anything that disrupts the NL flora of intestinal contents — causing stasis.

- **Dumping Synd.**

  ↳ Delivery of HYPERosmotic chyme to the small intestine ⇒ causing massive fluid shifts into the bowel.
  (Normally, the STOMACH will ↓ osmolarity of the chyme prior to its emptying)

  Ass. Condition

  Any procedure that bypasses the Pylorus or compromises its Fxn (gastrectomy / pyloroplasty) thus "dumping" the chyme into small intestine.

**Surgical causes of B12 def.**
- Gastrectomy (↓Sx of intrinsic factor)
- Excision of terminal ileum (site of B12 abs)

## S4S

~~Postprandial~~ Post Prandial diaphoresis / tachycardia / emesis / dizziness / weakness / ↑flatus / abd. pain / distention

**Dx**

Hx

**Tx**

- Small, multiple low-fat meals. that are high in Protein content.
- Avoidance of liquids w meals (to slow gastric emptying)
- Surgery — ONLY last resort!
  (Conversion to Roux-en-Y)

## • Jaundice

CAUSES PostOP:-

**PREHEPATIC:**
- Hemolysis (Prosthetic v.)
- Resolving hematoma
- Transfusion reaction
- Post-Cardiopulmonary bypass

(Labs)
- ↓ Hb / ↓ Hct
- ↑ LDH / ↑ Retic.
- fragmented RBC on Peripheral smear.

**HEPATIC:** DRUGS
Hypotension / hypoxia
Sepsis
Pre-existing cirrhosis
Rt-sided HF
Gilbert / Criglar-Najjar Synd.

**POST-HEPATIC:**
- Choledocholithiasis.
- Stricture
- Cholangitis.

## INFECTIOUS COMPLICATIONS

- Catheter-related infxn
- Prosthetic-device related
- Facial / muscle infxn
- Intrabdominal abscess / Peritonitis
- Resp. (GI)/GU infxn → ex. Pseudomemb. colitis
- Wound infxn

- Pts requiring mechanical ventilation for longer than 48 hrs are at risk of developing ventilator-ass. Pneumonia.

# WOUND COMPLICATIONS

- **Wound infxn**
  - * S&S: Erythema/Swelling/Pain/heat
  - * ttt: - open wound, leave open w/ wet to dry dressing changes.
    - Antibiotics (IF cellulitis present)

- **Wound hematoma**
  - Collection of blood (blood clot) in operative wound.
  - ttt: Acute → remove w/ hemostasis
    Subacute → Observe (heat helps resorption)

- **Wound Seroma**
  - Postop. collection of lymph & serum in the operative wound.
  - ttt: Needle aspiration. (Repeat if necessary)
    Prevent this w/ closed drain.

# ENDOCRINE COMPLICATIONS

- **DKA**
  - Hx deficiency of body insulin → resulting in hyperglycemia ↑↑ ketoacids osmotic diuresis & metabolic acidosis.

  - * Signs & Symptoms
    - Tachypnea / dehydration
    - Polyuria
    - Confusion
    - Abd. Pain

  - * Labs
    - ↑Glu
    - hyperkalemia (bcz insulin will ↓ K+ entry to the cells) → Transcellular K+
    - high anion gap met. acidosis
    - ± Urine Ketones

  - * ttt
    - Insulin drip
    - IVF rehydration
    - K+ supplement
    - ± bicarbonate

  - Indx of bicarbonate: ONLY IF PH < 7.1

  - K+ Replacement
    - CI: - hyperkalemia w/ ECG Changes
      - K+ > 6
    - Doses

| [K+] | |
|---|---|
| < 3 | 30 - 40 mEq/hr |
| 3 - 4 | 20 - 30 |
| > 4 - 5 | 10 - 20 |
| | STOP! |

## • Addisonian Crisis

Acute adrenal insufficiency due to stressor (surgery/trauma/infxn)

**CAUSE**
- Postop.
- Inadequate cortisol release.

Presentation in infancy
Tachycardia & hypotension REFRACTORY to IVF & pressors!

**SFS**
- Tachycardia, **Hypotension** (& eventual hypovolemic shock) ± fever.
- Progressive lethargy
- N, V, abd. Pain & diarrhea.

Mnemonic
**ADD**ison ≡ **AD**renal **D**own
THINK:
Addisonian crisis
= adre**Na⁺L** insuff.
= **Na⁺** insuff.

**Labs**

↓ $Na^+$ ↑ $K^+$ ] 2ry to ↓ Aldesterone

**Tx**
- IVF (D5NS)
- Hydrocortisone IV
- Fludrocortisone PO
  (for mineralocorticoid replacement — aldesterone)

## • SIADH (Syndrome of Inappropriate ADH SXN)

Mnemonic
**SIADH**
≡ **S**odium **I**s **A**lways **D**own **H**ere

**Causes**

**Mainly Lung /CNS** causes
- CNS trauma / Stroke
- oat cell lung CA
- Postop.

**Labs**

↓ $Na^+$, $Cl^-$
↓ Serum osmolarity
↑ urine osmolarity

**Tx**
- Treat the cause
- Restrict fluid intake.

## • Diabetes Insipidus (DI)

Labs HYPERnatremia

↑ Serum osmolarity
↓ urine osmolarity

# OTHER COMPLICATIONS

## DIC (Disseminated Intravascular Coagulation)

- It's activation of the coagulation cascade leading to **thrombosis** & **consumption** of clotting factors & Platelets → & activation of fibrinolytic system (fibrinolysis) resulting in **BLEEDING!**

**DIC work up**
- PT, PTT
- D-dimer
- Fibrinogen
- Platelets.

- **Sxs**
  - Acrocyanosis (& other signs of thrombosis)
  - Then, diffuse bleeding from incision sites/ venipuncture site / cath. site / mucus membranes.
- **Causes**
  - **Massive tissue injury**: Trauma, burns, extensive surgery
  - Infxns / Sepsis
  - CA
  - Obstetric causes
  - Miscellaneous: **Shock** / Liver disease.
- **ttt**
  - treat the cause
  - Supportive ttt ← IVF, O2, Platelets / FFP / Cryoprecipitate.

## Pseudomembranous Colitis

Antibiotic-associated diarrhea

**Sxs**
- **Diarrhea**
- Fever
- hypotension / tachycardia

- **PATHOGEN is** **Clostridium Difficile**

**Classic abx** → **Clindamycin!** (BUT almost all abx can cause it) (that causes C.difficile)

**Dx**
- C.difficile toxin in Stool
- Fecal WBC
- Flex. sigmoidoscopy (you see mucus "pseudomembrane" in lumen of colon)

**ttt**
- Flagyl (Metronidazol) Po/IV
- Po Vancomycin — If refractory to metronidazol.

# SUMMARY

A ♯ Postop. pt came ῶ :

- FEVER ⟶ THINK of 5 W's
- Chest Pain ⟶ THINK of Perιop. MI / PE
- SOB (ῶ resp. Sx) ⟶ THINK
  - Atelectasis / Pneumonia
  - PE
  - MI / CHF
  - Asthma / COPD exacerbation
  - Pneumothorax.
- Disorientation / Coma ⟶ THINK
  - Hypoxia, ARDS ] usually due to sepsis
  - Delirium tremens
  - Hypo/Hyper natremia.
  - Ammonium intoxication ] ↓ [Common source of coma in the cirrhotic pt ῶ bleeding esoph. varices who undergoes a Portocaval shunt]
- Urinary retention (V. common!)
  - Zero UOP → usually mechanical obst
  - ↓ UOP
    - Fluid deficit
    - ARF
- Abdominal distension ⟶ THINK
  - Paralytic ileus
  - Early mechanical bowel obst. (adhesions)

** DDx for Postop. Pleural effusion:

- Fluid overload
- Pneumonia
- CHF

The End
Gabe Gfrerer

## Enteral nutrition.

In general, the enteral route is preferred over the parenteral . Enteral feeding is simple, physiologic and relatively inexpensive.

Enteral feeding maintains the GI tract cytoarchitecture and mucosal integrity (through trophic effects), absorptive function, and normal microbial flora. This results in less bacterial translocation and exotoxin release from the intestinal lumen to the bloodstream.

Choice of an appropriate feeding site, administration technique, formula, and equipment may circumvent many of these problems.

**INDICATIONS :**

for patients who have a functional GI tract but are unable to sustain an adequate oral diet.

**Contraindications:**

1.intestinal obstruction / ileus
2. GI bleeding,
3. severe diarrhea/ vomiting
4. Enterocolitis
5. high-output enterocutaneous fistula

1.**Feeding tubes.**

. Nasogastric/ nasojejunal/ gastrostomy/ and jejunal tubes
.Gastrostomy tubes can be placed using minimally invasive techniques, such as edndoscopic or laparoscopic insertion.
.Jejunal tubes are preferred for long-term access and require a continuous infusion rather than bolus administration.

**2. Enteral feeding products**.

- A variety of commercially available enteral feeding formulas are available
-Standard solutions provide 1 kcal/mL;
-calorically concentrated solutions (>1 kcal/mL) are available for patients who require volume restriction.

-Currently available dietary formulations for enteral feedings can be divided into *polymeric* (blenderized and nutritionally complete commercial formulas), *chemically defined formulas* (elemental diets), and modular formulas.

**NOTES : Enteral formulas**

a.Blenderized tube feedings can be composed of any food that can be blenderized. Caloric distribution of these formulas should parallel that of a normal diet.

b.Nutritionally complete commercial formulas (standard enteral diets) vary in protein, carbohydrate, and fat composition They are recommended for patients experiencing minimal metabolic stress who have normal gut function.

c.Chemically defined formulas are commonly called elemental diets. The nutrients are provided in predigested and readily absorbed form. However, they are more expensive than nutritionally complete commercial formulas and are hyperosmolar, which may cause cramping and diarrhea. .

d.Modular formulations include special formulas that are used for specific clinical situations (e.g., pulmonary, renal, or hepatic failure or immune dysfunction).

## 3.Enteral feeding protocols.

It is recommended to start with full-strength formula begun at a slow rate and steadily advanced. This reduces the risk of microbial contamination and achieves full nutrient intake earlier.

Conservative initiation and advancement rates are recommended for patients who are critically ill, those who have not been fed for some time, and those who are receiving high-osmolality or calorie-dense formula.

### *a.Bolus feeding.*

-Reserved for patients with nasogastric or gastrostomy feeding tubes.
-Feedings are administered by gravity and begin at 50–100 mL every 4 hours and are increased in 50-mL increments until the intake goal is reached (usually 240–360 mL every 4 hours).

****Tracheobronchial aspiration is a potentially serious complication. To prevent this, the patient's head should be elevated to 30–45 degrees during feeding and for 1–2 hours after each feeding.

-The residual gastric volume should be measured every 4 hours and before administration of the feeding bolus. If the gastric residual volume is greater than 50% of the previous bolus, the next feeding should be withheld.

The feeding tube should be flushed with approximately 30 mL water after each use.

*b.Continuous infusion*

administered by a pump is generally required for nasojejunal, gastrojejunal, or jejunal feeding tubes.

Feedings are initiated at 20 mL per hour and increased in 10- to 20-mL-per-hour increments, every 4–6 hours, until the desired goal is reached.

The feeding tube should be flushed with approximately 30 mL water every 4 hours

For some patients, the entire day's feeding volume can be infused over an 8- to 12-hour period at night to allow the patient to be disconnected from the infusion pump during the day.

**4. Conversion to oral feeding**.

When indicated, an oral diet is resumed gradually. In an effort to stimulate appetite, enteral feeding can be modified by the following measures:

1.Providing fewer feedings

2.Holding daytime feedings

3.Decreasing the volume of feedings. When oral intake provides approximately 75% of the required calories, tube feedings can be discontinued.

**5.Complications :**

a. Metabolic derangement :

Abnormalities in serum electrolytes, calcium, magnesium, and phosphorus can be minimized through vigilant monitoring
Hypernatremia may lead to the development of mental lethargy or obtundation.
Hyperglycemia may occur in any patient but is particularly common in individuals with

preexisting diabetes or sepsis. The serum glucose level should be determined frequently, and regular insulin should be administered accordingly.

b. Clogging

- can usually be prevented by careful attention to routine flushing of the feeding tube. -
-Wire stylets should not be used to unclog a feeding tube because of the risk of tube perforation and injury to the esophagus or stomach. -
Instillation of carbonated soda, cranberry juice, or meat tenderizer teaspoon papain in 30 mL water) is sometimes useful for unclogging feeding tubes.

c. Tracheobronchial aspiration

of tube-feeding solutions may occur with patients who are fed into the stomach or proximal small intestine and may lead to the development of pneumonia.
Patients at particular risk are those with central nervous system abnormalities and those who are sedated.

d.High gastric residuals

of tube feedings as a result of outlet obstruction, dysmotility, intestinal ileus, or bowel obstruction may limit the usefulness of nasogastric or gastrostomy feeding tubes. Treatment of this problem should be directed at correcting the underlying cause. If gastric retention prevents the administration of sufficient calories and intestinal ileus or obstruction can be excluded, a nasojejunal or jejunostomy feeding tube may be necessary.

e.Diarrhea

-Is a potential consequence of enteral feeding, occurring in 10–20% of patients; however, other causes of diarrhea (e.g.Clostridium difficile or other infectious colitis) should be considered. ---------
-Diarrhea may result from numerous causes: too rapid an increase in the volume of hyperosmolar tube feedings// some medications (e.g., metoclopramide)// a diet that is high in fat content// or the presence of components not tolerated by the patient (e.g., lactose).
-If other causes of diarrhea can be excluded, the volume or strength of tube feedings should be diminished. If no improvement occurs, a different formula should be used. Antidiarrheal agents (e.g., loperamide) should be reserved for patients with severe diarrhea. -

## Disease-specific nutrition

**1.Thermal injury**

has a tremendous impact on metabolism because of prolonged, intense neuroendocrine stimulation. The increase in metabolic demands following thermal injury is proportionate to the extent of ungrafted body surface. Decreasing the intensity of neuroendocrine stimulation by providing analgesia and thermoneutral environments lowers the accelerated metabolic rate in many of these patients and helps to decrease catabolic protein loss until the burned surface can be grafted

2.**Diabetes** often complicates nutritional management. Complications that are associated with TPN administration (e.g., catheter-related sepsis) are more common with prolonged hyperglycemia. Unopposed glycosuria may cause osmotic diuresis, loss of electrolytes in urine, and nonketotic coma. The goal in glucose-intolerant patients is to maintain the serum glucose level at 100–200 mg/dL. Hypoglycemia can result in shock, seizures, or vascular instability. This can be prevented by adjusting the insulin dosing, with the understanding that insulin requirements will decrease as the patient recovers from the initial stress that is associated with the illness.

3.**Renal failure** may be associated with glucose intolerance, negative nitrogen balance (resulting from increased losses through dialysis), loss of protein with decreased protein ynthesis, and diminished excretion of phosphorus. Dialysis should be adjusted accordingly, and these patient s should be nutritionally replenished according to their calculated needs. Patients who receive peritoneal dialysis absorb approximately 80% of the dextrose in the dialysate fluid (assuming a normal serum glucose level). These factors must be considered when designing a nutritional support strategy.

4.**Hepatic failure** may result in wasting of lean body mass, fluid retention, vitamin and trace metal deficiencies, anemia, and encephalopathy. More than 70–80 g per day of amino acids is required to maintain nitrogen balance in these patients. It may be difficult or impossible to limit the amount of nitrogen that a patient receives each day yet still provide adequate nutritional support. Branched-chain amino acids are metabolized by skeletal l muscle and serve as an energy source during periods of stress. These amino acids are available enterally or parenterally to decrease the levels of aromatic amino acids and, therefore, the severity of encephalopathy; however, their efficacy has not been proved

5.**Cachexia** and cancer are associated with lean muscle wasting. More than two-thirds of patients with cancer experience significant weight loss during their illness, and malnutrition is a contributing cause of mortality in 20–40% of these individuals.

Reasons for this development include decreased nutrient intake and impaired nutrient use. Antineoplastic therapies, such as chemotherapy, radiation therapy, or operative extirpation, can worsen preexisting malnutrition.Although the addition of TPN to these modalities in clinical studies has shown improvement in weight, nitrogen balance, and biochemical markers, there islittle evidence to suggest better response rates or survival. Use of specialized formulas supplemented with various substrates (arginine, glutamine, nucleic acids, and omega-3 fatty acids) may reduce morbidity and length of hospital stay, but ongoing studies need to be done before these formulas are routinely recommended.

**6. Short-bowel syndrome**

commonly occurs in patients with less than 200 cm of functional jejunum. It may result from mesenteric ischemia, Crohn's disease, or necrotizing enterocolitis. It is characterized by nutrient malabsorption, electrolyte imbalance, diarrhea, and dehydration. Most of these patients require intravenous nutrition for life, at costs of more than $100,000 per year, with frequent hospitalizations for conditions such as

catheter sepsis, progressive organ dysfunction, and osteoporosis. The estimated length of small bowel that is required for adult patients to become independent of TPN is greater than 120 cm without colon or greater than 60 cm with some colonic continuity. Salvage of the ileocecal valve improves outcome. Intestinal adaptation may occur in some patients, thereby allowing for the transition from intravenous to enteral feeding. Uniquely formulated diets (supplemented with glutamine and growth hormone) show promise for accelerating this process (

7.**Patients with AIDS** develop PCM and lose weight. Malnourished AIDS patients require 35–40 kcal and 2.0–2.5 g protein/kg per day. In addition to the required electrolytes, vitamins, and minerals, they should receive glutamine, arginine, nucleotides, omega-3 polyunsaturated fats, branched-chain amino acids, and trace metal supplements. Those with normal gut function should be given a high-protein, high-calorie, low-fat, lactose-free oral diet. Patients with compromised gut function require an enteral (amino acid, polypeptide, or immuno enriched) diet or TPN.

# Nutritional Assessment

- Nutrition plays an imp. Role in the recovery of patients from surgery

- while most healthy patients can tolerate 7 days of starvation , subjects to major trauma /surgery/sepsis or other critical illnesses require nutritional intervention earlier.

-Poor nutrition has deleterious effects on wound healing and immune function , which increase postop. Morbidity and mortality.

## I. Types of malnutrition:

A. *Overnutrition* : obesity is defined as BMI >30
B. *Undernutrition* :
   1. **Caloric** :
      a. Marasmus

Characterized by inadequate protein + caloric intakeis
Typically caused by illness-induced anorexia.
It is a chronic nutritional deficiency marked by losses in weight, body fat, and skeletal muscle mass (as identified by anthropometric measurements). Visceral protein stores remain normal as do most lab indices .

(i.e Patients with marasmus may lose substantial body weight but are able to resist infection and respond appropriately to minor or moderate stress. )

**2. Non caloric**

a. Kwashiorkor

Charact. By catabolic protein loss ,resulting in HYPOALBUMINEMIA and generalized edema.
This malnutrition develops when the period of starvation is prolonged or if the stress is severe.
Even in a well-nourished patient, a severe stress (e.g., major burn or prolonged sepsis) may rapidly lead to the depletion of visceral protein stores and impairment in immune function.

b. Vitamins and trace elements

vitamins are involved with wound healing and healthy immune function while many trace elements are important as cofactor and enzymatic catalysts.
These substances cannot be synthesized de novo and must be part of dietary intake.

## II. Clinical Assessment :

**A .History**

Hx of weight fluctuation with intention to the timing as intent.
Recent weight loss (5% in the last month or 10% over 6 months) or a current body weight of 80–85% (or less) of ideal body weight suggests significant malnutrition.

Anorexia, nausea, vomiting, dysphagia, odynophagia, gastroesophageal reflux, or a history of generalized muscle weakness should prompt further evaluation.

A complete history of current medications is essential to alert caretakers to potential underlying deficiencies as well as drug-nutrient interactions.

**B.Physical examination**

May identify1. muscle wasting (especially thenar and temporal muscles), 2.loose or flabby skin( LOSS OF SUBCUT. FAT) ,3. and peripheral edema and/or ascites (as a result of hypoproteinemia).

More subtle findings of nutritional deficiency include skin rash, pallor, glossitis, gingival lesions, hair changes, hepatomegaly, neuropathy, and dementia

***Adjuncts to P/E :

--Anthropometric measurements : , such as triceps skinfold thickness and midarm muscle circumference, are a reflection of body-fat stores and skeletal muscle mass, respectively. These values are standardized for gender and height, and they should be reported as a percentage of the predicted value. Typically, anthropometric measurements include assessment of body weight, height, and body mass index, and these values allow the clinician to assess the patient's visceral and somatic protein mass and fat reserve.

-- Creatinine height index (CHI) : used to determine degree of malnutrition. A 24-hour urine creatinine excretion ration is measured and compared to normal standards. CHI is calculated using following equation :

where greater than 80% = zero to mild depletion, 60–80% = moderate depletion, and less than 60% = severe depletion.

**C. Laboratory tests**

Tests associated with nutrition are nonspecific indicators of the degree of illness rather than strict markers.
**Albumin , Prealbumin and transferrin** vary with hepatic metabolism ( dec. synthesis ) and capillary leak response to inflammation as well as the nutritional status.

Levels associated with illness are as follows:

1. Serum albumin of less than 3.5 g/dL in a stable, hydrated patient; half-life of 14–20 days.
2. Serum prealbumin a more useful indicator of nutritional status . . 10–17 mg/dL = mild depletion, 5–10 mg/dL = moderate depletion , less than 5 mg/dl =severe depletion. half-life of 2–3 days .
3. Serum transferring of less than 200 mg/dL; half-life of 8–10 days..

## III Estimation of Energy Needs :

1. Basal energy expenditure (BEE) : can be predicted using the Harris-Benedict equation ( in Kcal/day ):

| |
|---|
| BEE for men = 66.4 + (13.7 × weight in kg) + (5.0 × height in cm) – (6.8 × age in years) |
| BEE for women = 65.5 + (9.6 × weight in kg) + (1.8 × height in cm) – (4.7 × age in years) |

These equations provide a reliable estimate of the energy requirements in approximately 80% of hospitalized patients. The actual caloric needs is obtained by multiplying BEE by specific stress factor ( disease specific factor) .Most stressed patients require 25–35 kcal/kg per day.

2.Estimates of protein requirements:

The appropriate calorie-nitrogen ratio is approximately 150:1( calorie :protein ratio 24:1). In the absence of severe renal or hepatic dysfunction, **approximately 1.5 g protein per kg body weight** should be provided daily

**Twenty-four–hour nitrogen balance** is calculated by subtracting nitrogen excretion from nitrogen intake. Nitrogen intake is the sum of nitrogen delivered from enteral and parenteral feedings. Nitrogen output is the sum of nitrogen excreted in urine, fistula drainage, diarrhea, and so forth. The usual approach is to measure the urine urea nitrogen concentration of a 24-hour urine collection and then multiply by urine volume

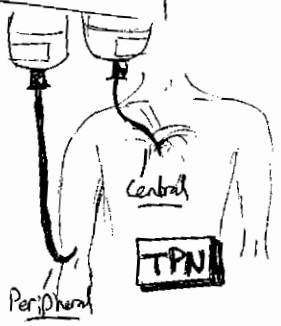
to estimate 24-hour urinary loss. Nitrogen loss equals 1.2 x [ 24-hour urine urea nitrogen (in gm) + 2 gm /day as a correction factor to account for nitrogen losses in stool and skin)

# TOTAL PARENTERAL NUTRITION (TPN)

Source: - Washington - Recall

≡ IV nutrition

→ It Provides complete nutritional support




## INDICATIONS

- NPO > 7 days
- Short bowel Syndrome.
- Enterocutaneous fistula (High-output fistula)
- Prolonged ileus.

## ACCESS

- **PERIPHERAL** : only for short-term feeding (≤ 2 wks)
- **CENTRAL** : through central venous catheter (Single / multi-lumen catheter)

* Catheter should be replaced if for unexplained fever / Bacteremia.

## SOLUTIONS

* Administered as **3-in-1** admixure:
  - **PROTEIN** as → aminoacids [10%] – 4 KCal/g
  - **FAT** as → lipid emulsion of soyabean [20%] – 9 KCal/g
  - **CARBOHYDRATES** as → Dextrose [70%] – 4 KCal/g

- Additives

**Electrolytes** ($Na^+$ / $Cl^-$ / $K^+$ / $Ca^{+2}$ / Acetate / Mg / $PO_4^{-3}$)

- Should be administered daily
- Note: $Ca^{+2}$ : $PO_4^{-3}$ ratio must be maintained to Prevent salt precipitation.

**Meds** ($H_2$-Blockers / Heparin / Iron / Dextran / Insulin / Metoclopramide)

Note: Regular insulin should initially be administered subcut. then administered via TPN (2/3 of daily subcut. dose)

**Vitamins & Trace elements** (Copper / Zinc)

Note: Vit. K is NOT included in most multivit. mixtures & must be added separately if needed.

## ADMINISTRATION

— Continuous vs. cyclic —

* It's usually given as continuous infusion (esp. if short-term)
* Cyclic TPN: feeding for (8-16) hrs during the night & fasts during the day → this gives the long-term TPN Pt freedom from the machinery to lead a less restricted life during the day

Indx for CYCLIC administration of TPN

(1) Pts who will be discharged from hospital & subsequently will receive home TPN (outpt)

(2) Pts w limited IV access who require TPN line for meds at certain time of the day

## DISCONTINUATION

TIMING When the Pt can satisfy 75% of caloric & Protein needs w oral intake or enteral feeding.

INFUSION RATE Halved for 1 hr
Halved again next hr
Then discontinue!

(Notes)
* Tapering PREVENTS rebound hypoglycemia from hyperinsulinemia
* No need for tapering if there's glycemic stability.

# COMPLICATIONS

- Central line complications — infxn / Pneumothorax
- Electrolyte disturbances
- Glucose Problems
- Loss of gut barrier (PUD)
- Acalculus cholecystitis / Gallstones
- Fatty infiltration of the liver
- Refeeding Syndrome.
-

NOTES

- **Refeeding Syndrome:**
  - It's severe fluid & electrolyte shifts in **malnourished** Pts undergoing refeeding.
  - Can occur in both TPN or enteral nutrition (BUT more common in TPN)
  - Labs: ↓ $K^+$ ↓ $Mg^{+2}$ ↓ $PO_4^{-2}$
  - This will lead to — altered myocardial fxn & arrhythmias
    - Deteriorating respiratory fxn.
    - Liver dysfxn
    - Seizures / confusion / coma / ↓ temp
    - Death!

Yada Glenda
The End.

# STOMA

Source: Surgical Recall Dossier

## DEFINITION

* It's a surgically made opening of the bowel into the anterior abdominal wall

**Notes**

The Stoma should be through the rectus abdominus muscle NOT through the apponeurosis.

If it's possible, the more distal the stoma the better (To be closer to NL anatomy)

## TYPES "Named according to the site"

- Esophagostomy
- Gastrostomy (G-tube)
- Jejunostomy (J-tube)
- Cecostomy
- Ileostomy
- Colostomy

## • TEMPORARY vs. PERMANENT STOMA

- There is distal bowel segment remaining after resection
- Done to divert the fecal stream.

- When NO distal bowel segment remaining after resection
- Done when for some reason, the bowel can not be rejoined.

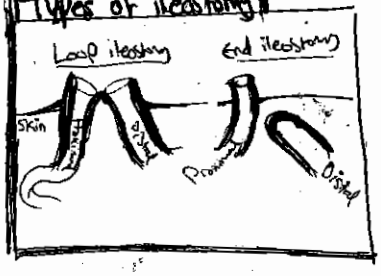
**Stoma Appliance**

A removable Plastic bag attached by adhesive to the abdominal skin.

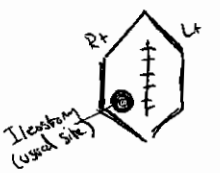
## • INDICATIONS

- Feeding or administration of drugs.
- Diversion
- Decompression

**Types of Ileostomy**




## ▶ Ileostomy — Types: End ileostomy, Loop ileostomy


* **Site**: usually at the Rt iliac fossa
* **Shape**: Fashioned as a "spout" of bowel protruding around 3 cm above the skin. (Why? to protect the surrounding skin from the highly irritant contents)
  ↳ It's called "Brooke ileostomy"
* **Bag contents**: WATERY stool (Bile / gastric juice / Chyme)
* **Smell** OFFENSIVE smell!
* **Surrounding Skin**: is usually inflamed (irritated from acid) if there is leakage of sxns.
** Median or Paramedian scar is usually seen for colectomy or Procto colectomy

## ◎ Indx for the temporary type

* Defunctioning Stoma — to Protect a more distal anastomosis that is at a particular risk of leakage or breakdown.

## ◎ Indx for the Permanent type

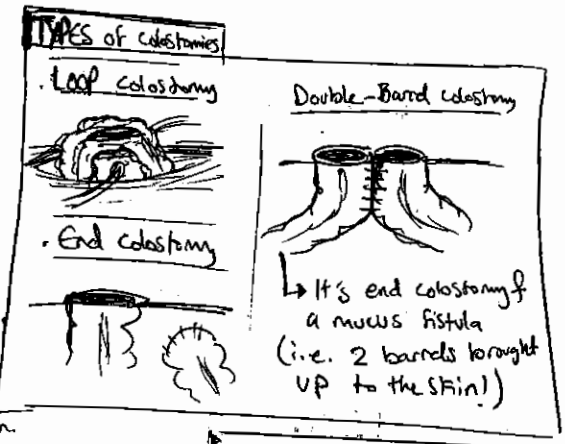
* Following Pan-Proctocolectomy — usually done in: Familial Adenomatous Polyposis (FAP) / IBD
(Removal of rectum + colon + ANUS)

### ✱✱ Notes about ileostomy

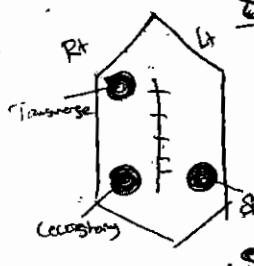
- It discharges small quantities of liquid material **CONTINUOUSLY**.
- It does NOT require irrigation.
- An appliance should be worn at ALL times

## ➢ Colostomy

**Types** — Loop colostomy
- End colostomy
- Double barrel colostomy




**Sites**




- Lt iliac fossa: Sigmoid colon (mc colostomy type)
- Rt upper quadrant: Transverse colon
- Rt iliac fossa: Cecostomy

- **Shape**: The bowel mucosa is in direct contact w the skin (unlike ileostomy) — at the skin level. (cuz the stool is not irritant to the skin)
- **Bag contents**: Formed stool
  * No skin changes around it.

**Notes about Colostomy**
- Requires irrigation daily / or every other day.
- An appliance is NOT required!
- If sigmoid colostomy expels stool once/day
- Transverse colostomy should NOT be constructed as a permanent stoma cuz it's bulky / foul-smelling / wet discharge & appliance should be worn everyday & prone to leak & prolapse is common!

## ◎ Indx for the temporary type

- To protect a more distal anastomosis (as ileostomy)
- To rest a more distal segment of bowel involved in inflammatory process.
- Emergency measure to relieve complete distal large bowel obstn causing proximal dilatation.

## ◎ Indx for the Permanent type

- Abdominoperineal resection of a low rectal / anal tumor.

## COMPLICATIONS (of BOTH ileostomy & colostomy)

### EARLY

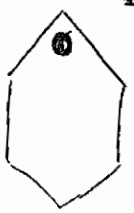
- **Obstruction** of Stoma due to edema or fecal impaction
- Mucosal **Necrosis** / Sloughing of terminal bowel due to ischemia
- Persistent **leakage** btw. Stoma & appliance → Skin **erosion**
  (usually due to inappropriate Placement of stoma ex. over skin crease)

### LATE

- **Stenosis** - of stomal orifice
- **Prolapse** of Bowel (usually the distal part)
  or **Retraction** of spout ileostomy.
- **Perforation** after colonic irrigation
- Parastomal hernia (due to abd. weakness)
- Parastomal fistula.

* Rate of complications in { ileostomy — 40%; colostomy — 20% }

## Gastrostomy



**Site**: At the epigastric area

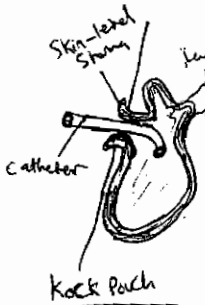
**Indx**: – Feeding
– Decompression (ex. Intestinal obstruction.)

* G-tube is Put through the abd. wall.

Usually, any fistula ABOVE ileum is used for feeding.

## NOTES

* Proximal loop: end stoma, Passes stool into the stoma appliance.
* Distal loop: Produces little mucus, called mucus fistula.
* **Kock Pouch** (AKA: continent ileostomy)
  - It's formed by the terminal ileum after ileectomy
  - It has the vol. of 500ml – 1L so that feces is stored temporarily & pt need not carry a stoma bag! ↑ improves quality of life.




## Summary

### TYPES OF STOMA

- **Loop Stoma**
  - Usually temporary
  - BOTH Prox. & distal segments drain on the skin surface through a single skin aperture.
  - Distal loop has No Fxn (defxning loop)
- **Split Stoma**
  - Defxning Stoma
  - Not used nowadays (replaced by loop)
  - Brought separately on SKIN.
- **Single-end Stoma** = most used as permanent stoma

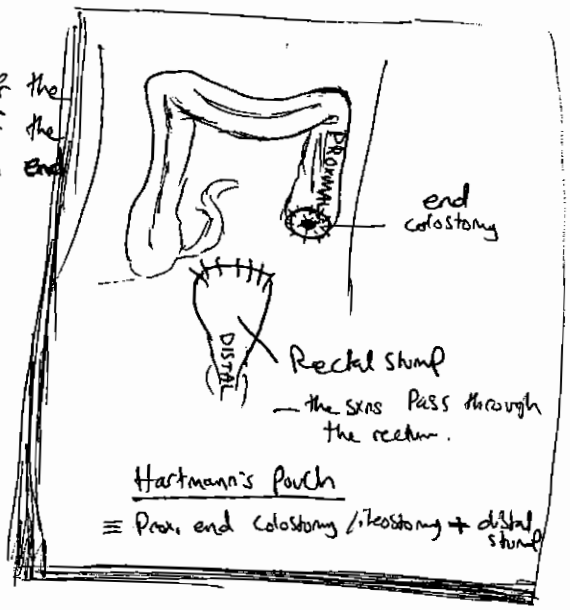
(2)

## Hartmann's Procedure

↳ It's the surgical resection of the rectosigmoid colon w closure of the rectal stump & formation of an end colostomy.

### Indxs-

- Used after emergency resection of rectosigmoid lesions where primary anastomosis is inadvisable because of obstruction, inflammation or fecal contamination.




** Why doesn't an ileostomy or colostomy close?
due to Epithelization.

** Why doesn't a gastrostomy close?
Due to Foreign body.

Gabi Goldsmith
The End.

# STOMA — Examination OSCE

- **INSPECTION**
  - Site
  - Contents : consistency & smell
  - Surrounding skin
  - Prolapse or retraction
  - Parastomal hernia (Ask the Pt to cough)
  - Parastomal fistula
  - Stenosis
- **PALPATION**
  - Lumen (One or double barreled) // Stenosis
  - Contents.
  - Palpate the ANUS If < closed → it's Permanent / Patent → it's temporary

Yalla Ghatta
The End.

# TUBES & DRAINS



Source : Surgical Recall Dossier

## TUBES

Used to drain or instill fluid from or into body cavity.

- **Nasogastric tube (NGT)**

**Indx**

① To decompress the stomach or small bowel
② To initiate an enteral feeding or medication
③ To perform gastric lavage.

**CI**

- facial bone fracture (to avoid the entry of the tube through the cribriform plate to the brain)
- Nasopharyngeal obstruction.

**Technique**

- **Placement:**

If the Pt can talk w/out difficulty & succus returns, the tube should be in the stomach.

- The pt should sit upright in his bed or lying supine w/ the head flexed 45°
- The tube should be lubricated.
- Use topical local anesthetic (as Lidocaine)

The length of the tube to reach the stomach is around 40-50 cm in adults.

* If you suspect abnormal placement → Do X-Ray (The tube has a radio opaque stripe)
* How to identify the tip location of the tube ? (3 methods)
  ① Aspiration of the fluid & looking at its nature.
  ② Infusing air & auscultation a rumbling voice in the stomach area.
  ③ Radiology through the opaque stripe.

BEFORE feeding via any tube, you should do high abd. X-Ray to confirm placement into GIT & NOT the lung!

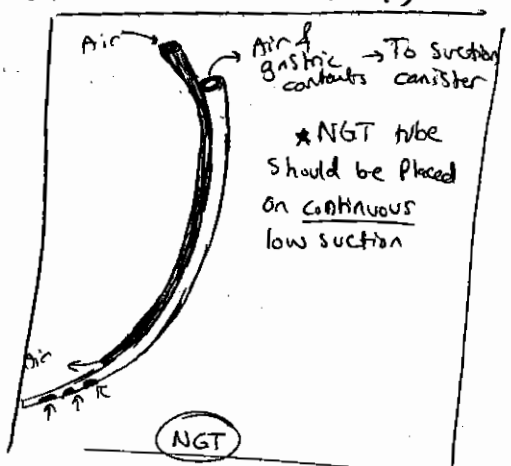
- **Removal:**
  - Give pt a tissue
  - Discontinue suction
  - Remove quickly & tell Pt to blow nose.

## Types

- **Single lumen tubes** — Best for feeding & administration of meds.
- **Dual lumen tubes** — Best for decompression fxns (since one port of the tube is always patent for air thus the tube can't be collapsed!)

## Complications

- **Obstruction** (Clogged Tube)
  - Mgt of clogged NGT
    - *Saline → flush the clear port, reconnect the suction & flush air down the blue sump port.
  - GERD (if the caliber of the tube is large)
  - tube complications:
    - esophagitis
    - stricture
    - recurrent aspiration Pneumonia
- Necrosis of the nasal skin due to pressure of the tube.
- Impairment of the nasal sinus drainage → Sinusitis!
- Passage through the cribriform Plate to the brain (The most SERIOUS)
- Perforation of esophagus or stomach leading to → mediastinitis or Peritonitis. (RARE)




*NGT tube should be Placed on continuous low suction

NGT

If NGT is clogged it will NOT decompress the stomach & will keep the lower esph. sph. (LES) open → ↑ risk of aspiration!

DDx of excessive NGT drainage:
- Tip of NGT is inadvertently placed in duodenum & drains Pancreatic fluid & bile
- Ddx: X-Ray

## ● Gastrostomy (G-Tube) or Jejunostomy (J-tube)

### Indx

1. Initiation enteral feeding & medication
2. Decompression of the stomach & small bowel.

### Technique

**Placement**
- Inserted through ant. abdominal wall surgically / endoscopically / or radiologically.

*In order to replace them we have to wait for a mature tract to form & this needs 2-6 wks (depending on the type of the tube used) — Its better to replace them under fluoroscopic guidance inorder not to lose the access

**Removal** — at bed side (mostly closes spontaneously with a delay of 2)

**Types**

- Single lumen tube — Mainly for feeding
- Double lumen tube — Mainly for decompression.

* Enteral feeding through G-tube occurs in bolus fashion BUT in J-tube it must be in a continuous fashion to avoid diarrhea. (i.e. feeding like the NL Physiology!)

**Complications** — Rare! (mainly due to incorrect placement)

- Htng
- Peritonitis. & local cellulitis.

## T-Tubes

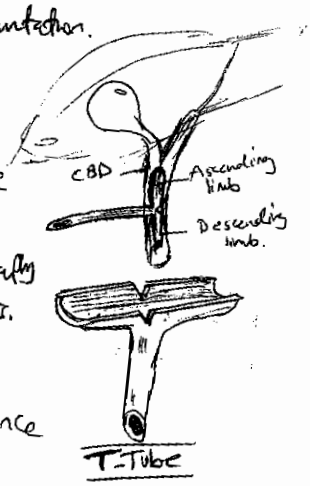
T-shaped tubes placed into the bile duct often through or adjacent to the cystic duct.

**Indx**

1. Cholecystectomy w CBD exploration
2. Biliary anastomosis after liver transplantation.
3. Many forms of biliary surgery.

**Advantages**

- They're connected to gravity drainage & thus can drain the bile easily
- Can be used to Perform T-tube cholangiography & & can give access to interventional instruments.




**Removal**

Can be removed by gentle traction once the tract has matured.

**Complications**

- Cholangitis (—Tube malfxn in the setting of bile duct obst.)
- Leakage at the site of insertion.
- Biloma or abscess formation.

## • Cholecystostomy Tube

↳ Placed surgically or Percut. w/ U/S guidance to drain the gall bladder.

## • Foley's Catheter

– Balloon-tipped catheters that are placed in the bladder through urethra & left for gravity drainage.

**Indx**
- Relief of urinary retention
- Measuring UOP accurately.
- Instill irrigant to the bladder.

**CI** — urethral injury

**Techniques**
- Lidocaine gel injected into the urethra (to make the process less uncomfortable)

If a Foley cath. can NOT be inserted, what are the next steps?
① Anesthesize urethra w/ lidocaine jelly
② Try a LARGER Foley

If urethral injury DON'T insert Foley
Alternative suprapubic catheter.

**Complications**

**UTI** (The most imp.) Mgt → Remove catheter + Abx

## • Central lines

Catheters placed into the major veins (central veins) via subclavian, internal jugular, or femoral vein approaches.

**Major Complications**
- Pneumothorax (ALWAYS obtain Postplacement CXR)
- Bleeding
- Infxn
- Malposition
- Dysrhythmias.

What is a "cordis"?
Large central line cath.
– used for massive fluid resuscitation
– or for placing a a Swan-Ganz cath.

## DRAINS

**Indx:** ① Withdrawal of fluids
② Apposition of tissues to remove a potential space by suction.

The End.

# CHEST TUBES

Source: Surgical Recall

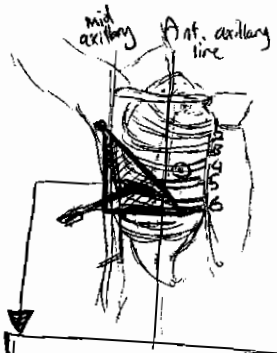
AKA: Thoracostomy tube

## Indx

- To oppose the Parietal & visceral pleura → to seal any visceral holes.
- To drain Pus/ fluid / Chyle/blood/air.

## Technique of administration

- Administer local anesthetic
- Incise skin in the 4th/5th intercostal space btwn. the mid & ant. axillary lines.
- Perform blunt Kelly-clamp. dissection over the rib into the Pleural space.
- Perform finger exploration to confirm intrapleural Placement.
- Place tube **Posteriorly & superiorly**




**Safe ▲ For insertion**

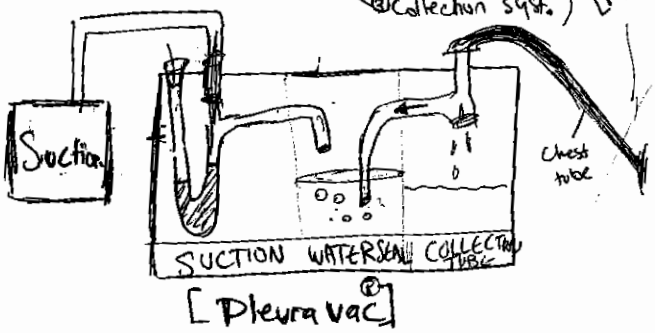
- Ant. border of lat. dorsi
- Lat. border of Pectoralis major
- A line sup. to horizontal level of nipple.
- Apex below axilla

## NOTE

- Chest tube: is Placed **OVER** the rib to avoid the vessels & nerves.
- In most cases, it should be positioned Posteriorly into the apex.

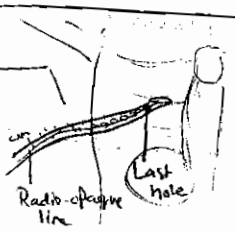
## MECHANISM

It's connected to ① Suction ② Water seal ③ Collection syst. } Three-chambered Box [Pleuravac ®]




[Pleura vac ®]

Centimeters on chest tube are measured from the last hole.




### Notes

- How can you tell on if the last hole on the chest tube is in the Pleural cavity?

— Last hole is cut through the radiopq. line in the chest tube & is seen on CXR as a break in the line—which should be in the Pleural cavity. (1)

- Mechanism of each chamber:-

## COLLECTION CHAMBER

— Collects fluid / Pus / blood / or Chyle
& measures amount

* Connects to the water seal bottle & the chest tube.

## WATER-SEAL CHAMBER

— **One-way valve**.
Allows air to be removed from the pleural space
BUT does NOT allow air to enter pleural cavity.

* Connects to the suction control bottle & to the Collection Chamber.

## SUCTION-CONTROL CHAMBER

— Controls the amount of suction by the height of the water column;
Sucking in room air releases excessive suction

* Connects to wall suction & to the water seal bottle.

### Notes

- NEVER clamp off the chest tube! (EXCEPT to "run the system" momentarily)
- Run the syst. momentarily:
To see if the air leak is from a leak in the pleural cavity (hole in lung) or from a leak in the tubing
⇒ so momentarily occlude the chest tube & if the air leak is still present, it's from the tubing or tubing connection, NOT from the chest!

**Placement of chest tube on water-seal**

By removing the suction, a tension Pneumothorax can NOT form. why? bcz of the one-way valve (water-seal) that allows release of air buildup.

- **HOW can you check for a leak?**

LOOK at the **water seal chamber** on suction:

- If bubbles pass through the water-seal fluid, a large air leak is present (air leaking into chest tube)
- * If NO air leak is evident on suction → Remove suction & ask the Pt to cough, so that
- If air bubbles through the water seal, a small leak is present.

---

- The usual course for removing a chest tube placed for Pneumothorax:

1. Suction until the Pneumothorax resolves & air leak is gone.
2. Water seal for 24-hrs
3. Remove the chest tube if NO Pneumothorax or air leak is present after 24-hr of water seal.

How fast is a small, stable Pneumothorax absorbed?

~1% daily

So a 10% Pneumothorax by Vol. will absorb in ~10 days!

## REMOVAL OF CHEST TUBE

- Cut the stitch
- Ask the Pt to exhale & inhale maximally.
- Rapidly remove the tube (split second) & at same time, Place Petroleum jelly gauze covered by 4x4's & then tape.
- Obtain a CXR

Gabe Ghantous
The End.

# Tracheostomy

S3

Definition: opening (stoma) in ant neck to create a surgical airway.

Tx (1) Relieve upper airway obstruction

causes: FB
- trauma
- Bloody / mechanical edema
  Eg. in anaphylaxis
- croup (acute laryngitis)
- Bilateral vocal cord paralysis
- congenital web/atresia

(2) Improve Respiratory Function

In cases of:
- acute severe pneumonia / chronic bronchitis ⇒ LOTS of secretions
- flail chest
- unconscious pt (eg. following severe head/chest injury)
- bulbar poliomyelitis (paralysis!)

(3) Substitute intubation

indp ↳ Any pt who needs intubation >2 w → substitute with tracheostomy

CI

Absolute:
1. if we can do translaryngeal intubation easily & quickly
2. Fractured larynx/damaged cricoid
3. Transection of trachea with distal end retracted into mediastinum

Relative:
1. ~~Infants~~ Infants & toddlers (risk of subglottic stenosis)
2. Acute laryngeal disease
3. Massive neck edema
4. Bleeding tendency

5/4

## Technique

1 Position: neck hyperextended
2 under local anesthesia
3 Skin incision: Long: or transverse 2-3 cm above suprasternal notch
4 Expose trachea at midline be — retracting strap muscles laterally
↑ thyroid isthmus superiorly (↑ thyroid vessels)
5 Palpate thyroid & cricoid cartilages
allocate 2nd & 3rd tracheal rings
Dont go beyond 4th ring → risk of ↑ tracheoinominal fistula (1st ring → stenosis)
6 Do either long, transverse, or H-shaped incision, remove small piece of trachea or do a flap (better!) (m.c)
7 use a dilator, then insert tube

note: size should be 3/4 of tracheal lumen

## Complications:

↳ Immediate (During procedure)
- Bleeding from thyroida., v., carotid
- injury to neighbouring structures → esoph, Recurrent Laryng n, pleura → tension pneumothorax, vocal cord, Larynx (# or cartilage disruption)
- obstruction
- technique related → malposition of inasion, incorrect placement, unsuccesful / Prolonged time: Do not exceed 3 mins!
- cardiac arrhythmia

↳ Intermediate:
- tracheal erosion (from movement)
- tube obstruction / displacement
- subcut. emphysema
- aspiration
- Fistula (TEF / tracheoinominate)
- Infection.

↳ Late:
- Late Bleeding
- Infection
- granuloma
- voice changes / vocal cord adhesion & incontinence
- tracheal stenosis
- tracheomalacia

* How to prevent complications?
1. Sterile technique
2. change tube daily (or every hour)
3. Proper care: - atraumatic suctioning
   - humidified oxygen
   - use mucolytics if secretion are thick
   - physiotherapy
4. CXR after placement
5. Deflate cuff q 5 mins to prevent tracheal ischemia

⊕ Advantages over translaryngeal intubation:
- easier nursing care
- facilitate oral feeding
- pt can speak
- easier for pt to move
- less work of breathing
- facilitate transfer from ICU towards

---

When to remove tracheostomy tube:-
1. ABG: $PO_2 > 60$
   $PCO_2 < 45$
2. Patient requires less suction
3. Return of cough reflex
4. Good general status

NOTE

* Cricothyrotomy VS. Tracheostomy ⊕
Cricothyrotomy is quicker & easier — Performed in emergent cases (through the cricothyroid membrane)
While tracheostomy is Placed in an OR or at bedside in ICU
— Placed btw. tracheal rings. (btw 2nd & 3rd or 3rd & 4th)

THE END!
(3)

# SHOCK

Source: Washington Surgical recall

## DEFINITION

- Inadequate tissue Perfusion resulting in generalized cellular hypoxia & dysfxn
≡ Recognized by evidence of **end-organ dysfxn**

* Compensatory Changes in response to hypotension
⇒ Release of catecholamines, aldosterone, renin & cortisol
⇒ Leading to ↑ HR, Preload & Afterload.

## TYPES OF SHOCK

| TYPE OF SHOCK | SKin | JVP | COP | PCWP | SVR | mixed venous $O_2$ content |
|---|---|---|---|---|---|---|
| HYPOVOLEMIC | Cool / Pale | ↓ | ↓ | ↓ | ↑ | ↓ |
| CARDIOGENIC | Cool / Pale | ↑ | ↓ | ↑ | ↑ | ↓ |
| SEPTIC • Early | Warm / Pink | ↑↓ | ↑ | ↓ | ↓ | ↑ |
| SEPTIC └ Late | Cool / Pale | ↓ | ↓ | ↓ | ↑ | ↑↓ |
| NEUROGENIC | Warm/Pink | ↓ | ↓ | ↓ | ↓ | ↓ |

## SIGNS

- Pale, diaphoretic, cool skin
- V/S: Hypotension, Tachycardia, Tachypnea
- ↓ mental status
- Poor capillary refill & Poor Urine OutPut (UOP)

**BEST** indicator of tissue Perfusion is **UOP** & also mental status.

## LABS to assess tissue Perfusion

Lactate(↑), Base deficit, pH (↓)

** Interventions in all types of shock < $SaO_2$ (Aim: >92%) / Hb / CaP (Aim: at least 7-9 g/dL)

# HYPOVOLEMIC SHOCK

↓ intravascular vol. > 20%

- **CAUSES**
  - Hmg
  - Burns
  - Bowel obst.
  - Crush injuries
  - Pancreatitis

  } usually differentiated by hx

- **SIGNS**
  - * Early: orthostatic hypotension, mild tachycardia/anxiety/diaphoresis, Vasoconstriction (↓PP w/ ↑diastolic P.)
  - * Late: Δ Mental Status, marked tachycardia.

| Blood Loss (%) | < 15% | 15 - 30% | 30 - 40% | > 40% |
|---|---|---|---|---|
| Blood Loss (ml) | < 750 | 750 - 1,500 | 1,500 - 2,000 | > 2,000 |
| V/S: HR | NL | > 100 | > 120 | > 140 |
| V/S: BP | NL | Syst. BP NL, Diast. BP ↑ (due to vasoconstriction) | Syst. BP ↓, Diast. BP ↓ | Syst. BP ↓↓, Diast. BP ↓↓ |
| V/S: RR | NL | ↑ | ↑↑ | ↑↑↑ |
| UOP | NL | ↓ | Oliguria | Anuria |
| Mental Status | Minimal Anxiety | Mild Anxiety | Confusion | Lethargy |

**Notes**

* The m.c V/S change ass. w/ **EARLY** hypovolemic shock is TACHYCARDIA.

* ↓ Pulse Pressure appears w/ EARLY hypovolemic shock. Why?
  Pulse Pressure (= Systolic − Diastolic) ↓ because of vasoconstriction resulting in ↑ diastolic BP

**tt**

- **STOP** the bleeding
- Volume expansion: I.V.F (isotonic LR), Blood Products if needed.

** What type of pts does NOT mount a NL tachycardic response to hypoval. shock? ① Pts on β-blocker ② Spinal shock ③ Endurance athletics.

# CARDIOGENIC SHOCK

- Cardiac insufficiency : LV failure (usually) resulting in inadequate perfusion.

**CAUSES:**
- MI
- Papillary m. dysfxn
- Cardiac tamponade
- Tension Pneumothorax.
- Cardiac valve failure.

**Sfs**
- SOB / crackles
- Loud $S_2$ ($P_2$) — on CXR → Pulmonary edema.
- Gallop rhythm.
- Pulsus alternans

**~~tt~~**

Based on dx / mechanism

If CHF → diuretics & afterload reduction (ACEI)

If LV failure (MI) → Pressors & afterload reduction

Last resort support mechanism : IABP ( Intraaortic Balloon Pump)
VAD (Ventricular Assisted Device)

# SEPTIC SHOCK

Documented infxn & hypotension

CAUSES: G-ve septicemia (m.c)
G+ve septicemia / fungus (less common)

Complications: multiple organ failure (MOF)
DIC
**Death!**

Sfs: (Initial) Vasodilation / warm skin / full Pulses / NL UOP
(Late) Vasoconstriction / Poor UOP
Mental Status Changes
Hypotension

Associated findings : Fever / Hyperventilation / Tachycardia.

**Factors ↑susceptibility of septic shock**

Anything that ↑ susceptibility to infxn:
- Steroids
- DM
- Immunosuppression
- Trauma
- Hematological d.

150

**Labs**

Early: Hyperglycemia / Glycosuria
Resp. Alkalosis
Leukopenia
Hemoconcentration

Late: Acidosis / ↑ Lactate
Leukocytosis

> Only 50% of blood cx are +ve in pts w/ bacterial septic shock!

**tt**

IV Fluids
Antibiotics (Empiric then by culture)
Drainage of infxn
Pressors - prn
Zygris® - prn

> **Zygris**
> Is activated Protein C
> ⇒ Shown to ↓ mortality in septic shock & Multiorgan failure

## NEUROGENIC SHOCK

Inadequate tissue perfusion from loss of sympathetic vasoconstrictive tone.

**CAUSES**

Spinal cord injury:
- Complete transection of SC
- Partial cord injury w/ spinal shock
- Spinal anesthesia

**S&S**

Hypotension & bradycardia
Neurologic deficit

> **RULE:-**
> ALWAYS rule out first hypovolemic shock! in pt w/ suspected spinal shock.

**tt**

- IV fluids
- Vasopressors reserved for hypotension refractory to fluid resuscitation.

** **Spinal Shock**: Complete flaccid Paralysis immediately following SC injury, may/may not be ass. w/ circulatory shock.

The End
Sarah Gluth

# SEPSIS, SIRS & MOF

Source: Washington Slides 61

## DEFINITIONS

- **Infxn**: Is presence of organisms in ~~a respiratory~~ closed space or location where not normally found.
- **Sepsis**: Known or suspected infxn • **Severe Sepsis**: Sepsis w/ acute organ dysfxn.
- **SIRS** (Systemic Inflammatory Response Syndrome): Is clinical response arising from non-specific insult manifested by 2 of the following: (Criteria)

V/S
- Body Temp. (>38° or <36°)
- HR >90 b/min
- RR >20 /min or $PCO_2$ <32
- WBC (>12 or <4 or >10% bands)

## RISK FACTORS

- Pre-existing disease (CVS / RS / Renal / HIV)
- Age (Extremes of Age)
- Gender (♂)
- Genetics (TNF Polymorphisms — TNF promoter high secretor genotype)

## RESPONSE

- Physiology — HR, Respiration, Fever, BP / COP / WBC, hyperglycemia
- Markers of inflammation — TNF / IL-1, IL-6 / Procalcitonin / PAF

## ORGAN DYSFXN

- Lung → ARDS
- Kidneys → ATN
- CVS → Shock
- CNS → metabolic encephalopathy
- PNS → Critical illness polyneuropathy
- Coagulation → DIC
- GI → Gastroparesis / Ileus / Cholestasis
- Endocrine → Adrenal insufficiency
- Skeletal m. → Rhabdomyolysis

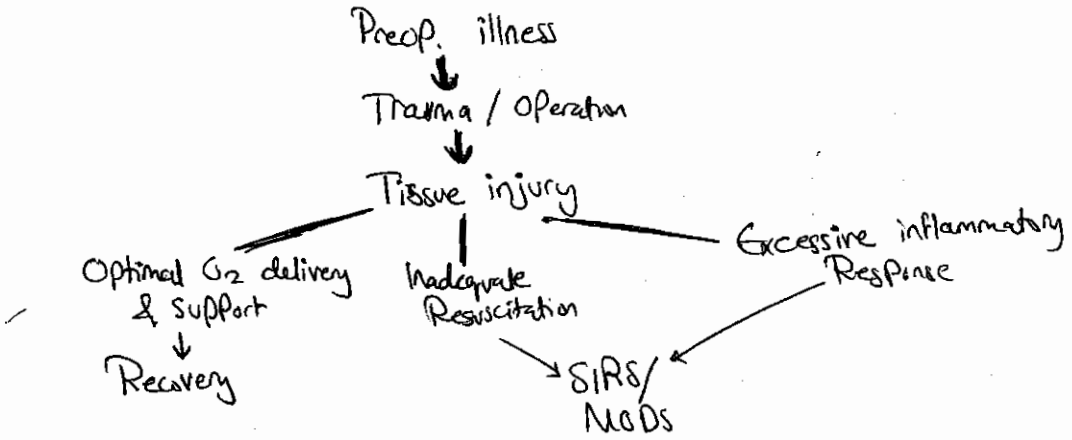
Infxn/ Trauma ⟹ SIRS (Criteria) ⟹ Sepsis ⟹ Severe sepsis

Sepsis: ↓ SIRS ῶ a Presumed or Confirmed infxn

Severe sepsis: ↓ Sepsis ῶ ≥1 sign of organ failure

Mortality 35%

## ** Pathogenesis of SIRS /MOD **




# MEDIATORS OF SEPTIC RESPONSE

### Pro-inflammatory mediators

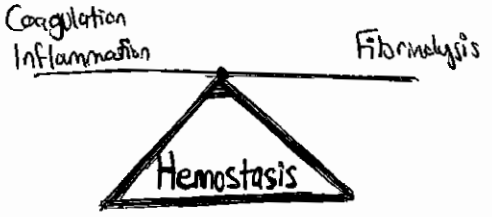
- Bacterial endotoxins
- TNF-α
- IL-1, IL-6, IL-8
- PAF
- INF-ɣ
- Prostaglandins
- Leukotrienes
- NO

### Anti-inflammatory mediators

IL-4, IL-10, IL-6, IL-12

Protein C

$PGE_2$




• Sepsis is an imbalance of inflammation, coagulation & fibrinolysis.

• In sepsis, coagulation, inflam. > Fibrinolysis & continued process of proinflammatory overwhelms the anti-inflam. cytokines.

* Coagulation & inflammation are closely linked
⇒ The cytokines from inflammation ⊕ coagulation pathways this results in the forming of the enzyme thrombin which produces CLOTTING in the body → microclotting → impaired Blood flow.

* Fibrinolysis (i.e. Breakdown of clots)
It's the body's response to the ↑ clotting & inflammation.

• In sepsis, fibrinolysis is inhibited or slowed bcz of mediators — PAI-1 (Plasminogen Activator Inhibitor-1)
\ TAFI (Thrombin Activatable Fibrinolysis Inhib.)

**NOTES**

• Sepsis is the leading cause of death in the **non-coronary ICU**.
• ⅓ of pts who develop severe sepsis will die within 1 month!!
• Severe sepsis is common! & is increasing in incidence.
• CAUSES of sepsis — **Bacterial** infxn (m.c)
\ Fungal / Parasitic / Viral

# ttt

## ① Addressing the infxn (Empiric then according to culture)

- Broad-spectrum IV abx
- Source control: Drainage/Debridement/ or removal of infxn source.

## ② Circulating Support

In the 1st 6 hrs:

Goals
- CVP 8-12 mmHg
- MAP ≥65 mmHg
- UOP ≥0.5 mg/kg/hr
- Mixed $SvO_2$ ≥ 70%

* Vasoactive meds (Dopamine, NE)

Phenylephrine is NOT beneficial in the setting of sepsis.

## ③ Adjunctive ttt

ex. Activated Protein C — has been demonstrated to ↓ mortality

↳ **Disadv.:**

* Short $t_{1/2}$
* ↑ risk of serious bleeding — so it must be used w/ caution.

↑ Pt's risk for infxn w/ resistant organisms:

- Prior ttt w/ abx during hospitalization.
- Prolonged hospitalization
- Presence of invasive devices.

Mike Smith
The End.

# SURGICAL INFXN



Source : Surgical Recall

*Classic S/S of Inflammatn/Infxn : Swelling / heat / Pain / Erythema.

## • Definitions

**Bacteremia** : Bacteria in the blood

**SIRS** : Syst. Inflammatory Response Synd. (Fever / Tachycardia / Tachypnea / Leukocytosis)

**Sepsis** : Documented infxn & SIRS

**Septic shock** : Sepsis + Hypotension

**Cellulitis** : Blanching erythema from superficial dermal/epidermal infxn (usually strep. > staph.)

**Abscess** : Collection of Pus w/in a cavity.

**Superinfxn** : New infxn arising while a pt is recieving abx for the original infxn at a diff. site.

**Nosocomial Infxn** : Infxn originating in the hospital

*The m.c nosocomial infxn is → UTI

* The m.c noscomial infxn causing DEATH → is RTI (Pneumonia)

## UTI

Invx
- U/A
- Culture
- urine microscopy for WBC

*What constitutes +ve U/A:
- +ve Nitrite (from Bact.)
- +ve leukocyte esterase (from WBC)
- >10 WBC / HPF
- Presence of bact. - Supportive

What * of colony-forming ~~bact~~ unit (CFU) confirms the dx of UTI?
- on urine culture, $10^5$ CFU

Tt of Bladder Candidiasis
- Remove/Change foley catheter.
- Administer syst. fluconazole or amphotericin bladder washings

microorganisms
- E. coli
- Klebsiella
- Proteus.

Tt
Abx w G-ve spectrum
ex. SMX/TMP (Bactrim)
Gentamicin / Ciprofloxacin / Aztreonam
*Check culture & sensitivity

# CENTRAL LINE INFXNS

Angiocatheters (Peripheral IV catheters) should be changed every 3-4 days.

- **S&S**
  - Unexplained Hyperglycemia.
  - Fever
  - Mental Status change
  - Hypotension
  - Tachycardia → **Shock**
  - Pus & erythema at central line site.

- **m.c.c of "catheter-related blood stream infxns" are:**
  - **Coagulase-ve** Staph. (33%) — m.c
  - Enterococcus
  - Staph. aureus.
  - G-ve rods.

**TPN** ↑ risk of central line infxns.

- Timing of changing the central line:
  When they're infected → there is NO advantage to changing them every 7 days in Non-burn pts.

**ttt**
- Remove central line (send for culture) ± IV abx
- Place a **NEW** central line in a diff. site.

# WOUND INFXNS

→ Discussed later on

# SUPPURATIVE HYDRADINITIS

Infxn/abscess formation in **APOCRINE** sweat glands.

Sites:
- Perineum / Buttocks
- Inguinal area
- Axilla

} sites of apocrine glands.

Causative organism
- Staph. Aureus.

ttt
- Abx
- Incision & drainage (Excision of skin & glands for Chronic infxns)

## Peritoneal Abscess :

- It's an abscess in the peritoneal cavity
- CAUSES :
  - Postop status after a laparotomy.
  - Ruptured appendix
  - Peritonitis
  - Any inflammatory intraperitoneal process.
  - Anastomotic leak.
- SITES :
  - Pelvis
  - Subphrenic
  - Lesser sac
  - Paracolic gutters
  - Morison's Pouch. (hepatorenal recess)
- S&S
  - Fever (classically spiking)
  - Abd. Pain
  - Mass
- Dx
  - Abd. CT (or U/S) — done AFTER day# 7 Postop.
    (Otherwise, it won't be organized & will look like a NL Postop. fluid collection)
    - FINDINGS on CT :
      - Fluid collection w fibrous rind
      - Gas in fluid collection.
- ~~Tt~~
  - Percut. CT-guided drainage.
  - Other options : Transrectal drainage (or transvaginal)

* ALL Abscesses should be drained EXCEPT Amebiasis !

# PSEUDOMEMBRANOUS COLITIS

It's abx-induced colonic overgrowth of C. difficile, 2ry to loss of competitive nonPathogenic bact. that comprise the NL colonic flora.

* Note: It can be caused by ANY abx but esp. Penicillins / Cephalosporins / & Clindamycin

## Sts

Diarrhea (Bloody in 10% of pts)
± Fever
± ↑WBCs
± Abd. cramps
± Abd. distention.

## Microorganism

Exotoxin released by C. Difficile

## Dx

Assay stool for exotoxin titer
± Fecal leukocytes

on Colonoscopy — FINDING: Exudate that looks like a membrane (hence, "Pseudomembo.!)

## ttt

- STOP the causative agent (abx)
- PO metronidazole (Flagyl) or PO Vancomycin

NEVER give antiPeristaltics!

Yale Griffith
The End.

# SURGICAL SITE INFXN (SSI)

* Infxn due to Surgery *

2/3 involved in Surgical incision (superf. / deep)
1/3 Deep structures accessed by incision

The m.c nosocomial infxn is UTI

- 3rd most reported nosocomial infxn.
- Most common Surgical nosocomial infxn.
- Deaths in pt w nosocomial infxns — 77% are related to infxns!!

EARLY SSI occur usually w/in 24 hrs Post op & are usually due to Strep. or Clostridium

LATE SSI: 1-5 days post op

## LEVELS OF SSI

### • SUPERFICIAL INCISIONAL SSI [Skin & Subcut. tissue]

Definition + Criteria

Infxn w/in 30 days Post op.
& ONLY involves Skin & Subcutaneous tissue.
& at least one of the following:

- Purulent drainage from SUPERFICAL incision
- +ve culture from closed surgical site obtained aseptically
- One of S&S of infxn: (Pain / Tenderness / localized swelling / Redness / Heat.)

### • DEEP INCISIONAL SSI

Definition + Criteria

Infxn w/in 30 days Post op. if no implant is left in place or w/in 1 year if implant is left in place.
& involves Deep Soft tissues (muscles / fascia)
& at least one of the following:

- Purulent drainage from DEEP incision BUT NOT from organ / space compartment
- Deep incision dehiscence or opened by Surgeon when Pt has at least one of: Fever (>38°C) / localized Pain / tenderness (unless culture is -ve)
- Abscess or other evidence of infxn of deep incision (on PE / reoperation / histopathology / Imaging)

& infxn appears to be related to the operation.

# ORGAN/SPACE SSI

**Definition + Criteria**

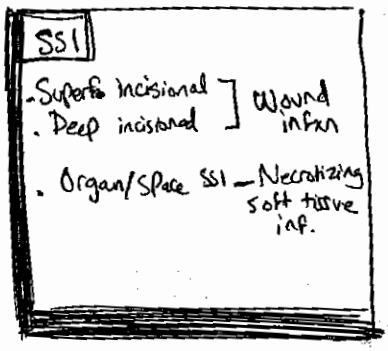
Infxn w/in **30** days Postop. if **no** implant is left in place
or w/in **1 year** if implant is left in place.

& infxn appears related to the operation.
& involves any part of the anatomy (organs & spaces)
& at least one of the following:

- Purulent drainage from a drain that is placed into the organ/space.
- +ve culture from aseptically obtained fluid or space/organ.
- Abscess or other evidence of infxn of organ/space (on P/E, reoperation, histopath., imaging)

## MICROORGANISMS

**Staph. Aureus**
Coagulase –ve staph
Enterococcus spp.
E. coli
Pseudomonas. Aeruginosa.




## RISK FACTORS

- Long operation (>2 hrs)
- Length of preop. Hospitalization
- **Procedure-specific** RF: Clean / Clean-contaminated / Contaminated / dirty
- **Pt-specific** RF:
  - **LOCAL**: – ↑Bacterial load
    - Wound hematoma/seroma
    - Necrotic tissue
    - Foreign Body
    - Obesity (fat)
    - Dead space (that prevents delivery of phagocytic cells to bact. foci)
    - Poor approximation of tissue
    - ↑ tension (tight sutures)
  - **SYSTEMIC**: – Age (>70)
    - DM
    - Chronic d. (Renal / Liver d.)
    - Malnutrition
    - Alcoholism / Smoking
    - Immunosuppression
    - Drugs: Steroids / CTX
    - Pre-existing infxn.
    - Shock / ↓ Blood flow will result in Poor delivery of PMNs & antibiotics
    - PAD

# PREVENTION

- Strict sterile techniques
- Maintain NL temp., NL blood glu (<110 mg/dL), ↓ Hyperoxygenation.
- Antibiotic ProPhylaxis —— within 1 hr of incision.
- Preop. skin antisepsis (The use of Chlorhexidin-alcohol is better than Providare-iodine scrub).

The End

# WOUND INFXNS

## Definition

Infxn of an operative wound

## TIMING

Classically, Postop day ** 5 - 7

## S&S

- PAIN at incision site
- Erythema
- Drainage
- Induration
- Warm skin
- Fever

## ~~**~~ Mgt

- Remove skin sutures/Staples
- RULE OUT fascial dehiscence.
- Pack wound open
- Send wound culture
- Give abx

The m.c bacteria that cause fever & wound infx in the 1st 24 hrs Postop. (EARLY infxn) are:

- **Streptococcus**
- **Clostridium** (Bronze-brown weeping tender wound)

## Micro-organisms

- Staph. Aureus. ~20%
- E-coli ~10%
- Enterococcus.
- Other causes: Staph. epidermis/Pseudomonas / Anaerobes / Strep.

## CLASSIFICATION**

- CLEAN
- CLEAN-CONTAMINATED
- CONTAMINATED
- DIRTY

** Discussed in details in the "Preop. evaluation" summary. **

## COMPLICATIONS

- Fistula / Sinus tracts
- Abscess
- Sepsis
- ↓ wound healing
- Superinfxn
- Hernia

## RISK FACTORS

- **Procedure-related:**
  - Clean / clean-contaminated / contaminated / Dirty
  - Long operation (>2 hrs)
  - Hypothermia in OR
  - Length of preop. hospitalization
- **Pt-Specific:**
  - LOCAL: - ↑ Bacterial Load
    - Wound hematoma, Necrotic tissue, FB, serum
    - Obesity (fat)
    - Poor approximation of tissue / Tension (tight sutures)
    - Dead space (that ⊖ delivery of phagocytic cells to bacterial foci)
  - SYSTEMIC:
    - Age (>70)
    - DM
    - Chronic diseases (Renal / Liver d.)
    - Malnutrition
    - Alcoholism / Smoking
    - Immunosuppression
    - Drugs: Steroids / CTX
    - Pre-existing infxn & uremia
    - ↓ Blood flow (ex. Shock) — Poor delivery of PMNs & antibiotics

## INVESTIGATIONS

- **CBC**: leukocytosis or leukopenic
- Blood **CULTURE**
- **CT** or other imaging studies (to localize an abscess for example)

## ~~Tt~~

Incision & drainage — if abscess

Abx - for deep abscesses.

- Some sup. abscesses (see box)

> • **Fluctuation** is a sign of superficial abscess.

> **Indx of abx after drainage of a subcut. abscess:**
>
> - DM
> - Surrounding cellulitis
> - Prosthetic heart valve
> - Immunosuppression.

The End.

# NECROTIZING SOFT TISSUE INFXNS

Sources: Washington Dossier Recall

## DEFINITION

- It's a rare, BUT very severe, **PROGRESSIVE** bacterial of the **skin, soft tissue, fascia,** or **muscles.**

## RISK FACTORS

- Old age
- Immunosuppression
- DM
- Chronic d. (co-morbidities) – 90%
- Alcoholism
- Blood supply compromise (ex. PAD)

> * It's ass. w **HIGH** morbidity & mortality (40%)
> So the earlier the tt, the better!

## MICROORGANISMS

Usually POLYMICROBIAL (Type I) or due to a single orgnism as **Group A Streptococcus Pyogens** (type II)

> Polymicrobial infxns work as synergism!

## S&S – Characteristics

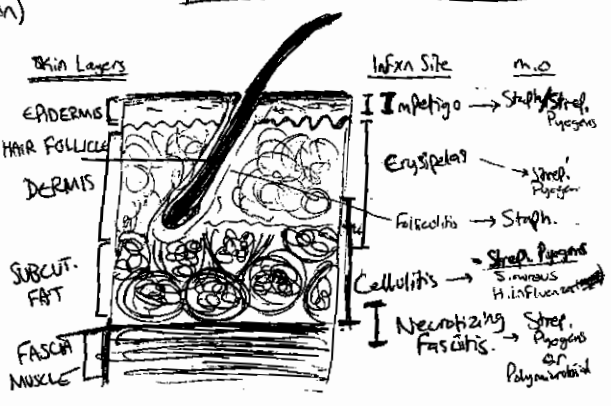
- Pain OUT OF PROPORTION TO examination
- Bullae
- Systemic signs of toxicity
- WBC > 15,400
- Tenderness beyond the area of erythema.
- Crepitus
- Cutaneous anesthesia
- Cellulitis refractory to abx tt.

> Flesh-eating bact. is **Strep. Pyogens**

> Early dx & intervention are the most imp. factor in determining the outcome.

## FORMS (Anatomical Classification)

- Necrotizing **FASCIITIS**
- Necrotizing **MYOSITIS**
- Necrotizing **CELLULITIS**




## General tt for all types

- Resuscitation as necessary.
- Empiric broad-spectrum abx.
- **DEBRIDEMENT!**
- Nutrition (x1.5 – x2 basal requirements)

FORMS

## ① * CELLULITIS

- Acute bacterial infxn
- Involves the Skin & Subcut. tissue (including the superficial fascia only)
- **SITE** m.c in the Lower Limb

  other sites: – Periorbital regions
  – Incisions
  – Puncture wounds (Skin Popping / illicit drugs)
  – Bites
  – Areas of Pre-existing Skin condition (venous stasis / Ischemia / decubitus ulcers)

- **Microorganism**

  **Streptococcus Pyogens** (m.c)
  Staph. Aureus (uncommon)
  H. Influenza (rare)

## ② * FACIITIS — Flesh-eating disease

- Involves the **subcut.** tissue & deep fascia
- It's an **EMERGENCY**! Pt must be taken to the OR immediately
- C/P may be masked! as changes in the overlying skin may only be observed later in the disease Process.
- **Microorganism**
  - Classically, Group A strep. Pyogens
  - BUT most often it's Polymicrobial.
- **CAUSE**

  m.c.c is **TRAUMA** ~80%

  However, **20%** of cases occur in healthy Pts w/ NO known injury

- ~~III~~
  - IV Fluids
  - IV Abx
  - Aggressive early extensive surgical debridement
  - Culture
  - Tetanus Prophylaxis

**Fornier's Gangrene**

It's a type of necrotizing infxn or gangrene — usually affecting the **Perineum / Scrotum**

* Usually seen in Pts w/ DM

(III) Triple Abx & Wide debridement – a surgical EMERGENCY!

## ③ MYOSITIS (AKA: Gas Gangrene)

It's clostridial muscle infxn.

**m.c micro-organism**

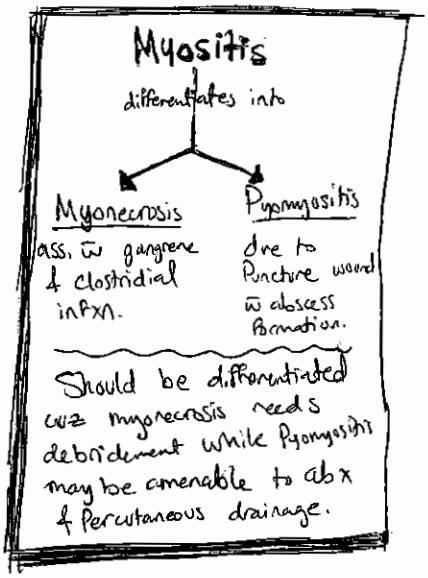
Clostridium Perfringens — m.c.c.

**SPS**

- Pain, Fever, Shock
- Crepitus
- Foul-smelling brown fluid
- Subcut. air on X-Ray.

**Tx**

- IV abx
- Aggressive surgical debridement of involved muscle.
- Tetanus Prophylaxis.




Jade Galbraith
The End

# CRITICAL CARE
## [ICU Monitoring]

Source: Washington Recall

### ICU Checklist (Mnemonic: FAST HUG)

Pneumonic it!

**F**eeding
**A**nalgesia
**S**edation
**T**hromboembolic Prophylaxis

**H**ead-of-bed elevation (Pneumonia Prevention)
**U**lcer Prevention
**G**lu control

> The m.c bacteria in ICU Pts is ↓ **G-ve rods**

## MONITORING

**① Temp.** : Q 4 hrs
— obtain **core** temp. (BEST is rectal)

> Core temp. : is the temp. of internal organs, # It's better than skin temp.

**② ECG** : Continuous
— To detect HR, Rhythm, & dysrrhythmia.

**③ Arterial Pressure monitoring**

- **Types**
  - **Indirect** — by sphygmomanometer, at least Q1hr
  - **Direct** — by intra-arterial catheter.
- **Indx**
  - Need for continuous monitoring (beat-beat) — as in unstable Pts
  - If frequent ABG measurements are required.
- **Site of insertion**
  - m.c is **Radial artery**
    Why? due to its accessibility & good collateral blood flow.
  - others: Femoral art., & less commonly; dorsalis Pedis or axillary art.
- **Complications**
  - Occlusion → limb ischemia
  - Infxn (cellulitis / Bacteremia) due to cath. colonization or contamination.

**INTRA-ARTERIAL LINE NOTES!**

* Intra-arterial line should be AVOIDED in **infants** bcz occlusion may cause limb ischemia → & subsequent deformity!
* Always assess extremity distal to catheter before & after insertion.
* If evidence of ischemia → immediate removal!

## ④ Central Venous Pressure monitoring

Indx: to measure CVP
- to measure $CvO_2$
- to administer vasoactive drugs
- TPN

## ⑤ Pulmonary Art. Cath. (AKA: SwanGanz cath.)

- To determine cardiac filling pressure
  - COP / SVR / Mixed venous $O_2$ sat. ($SvO_2$)
  - PA Pressure
- Used in unstable pts to assess responses to tt w/ fluid & cardioactive agents.

**Requirements & considerations**

ECG must be checked BEFORE placement to r/o LBBB. Why? bcz PA cath. causes transient RBBB.

∴ So if Pt has LBBB → before placement, you should place a transcutaneous Pacemaker.

**Complications**

- **Balloon Rupture** → Risk of air embolism
  - → SUSPECT when air inflated doesn't return.
  - → CONFIRM by aspiration of blood from balloon Port.
  - Mgt: Remove it!
- **PA Perforation** — c/f: Hemoptysis.
- **Dysrrhythmia** — usually self-limited.

**NOTE**
The use of PA cath has NOT been demonstrated to change mortality.

Alternative to Swan-Ganz is Esophageal Doppler
- less invasive
- Measures desc. aortic flow velocity over time & SV — to titrate fluid administration.

Complication: Esophageal perforation (not common)

## ⑥ Respiratory monitoring

### ** Pulse oximetry

- Used in ALL critically ill Pts
- It Provides quantitative, continuous assessment of arterial $O_2$ sat. ($SaO_2$)

▶ **CAUSES of Poor Signal detection/unreliable measurement**

- Probe malposition/motion
- Hypothermia / vasoconstriction
- Hypotension

▶ **CAUSES of falsely ↓ $SaO_2$**

- Nail Polish
- Dark Skin
- ↑ Serum lipids

▶ **CAUSES of falsely ↑ $SaO_2$**

- Carboxyhemoglobin
- Methemoglobin

### ** Capnography

- Provides quantitative, continuous assessment of $CO_2$ conc. & gradient btw $PCO_2$ & end-tidal $CO_2$ ($ETCO_2$)

▶ **CAUSES of ↑ $ETCO_2$:**

- ↓ Alveolar ventilation
- ↑ $CO_2$ Production.

**CAUSES of ↑ $CO_2$ Production:**

- Sepsis / fever
- Over feeding
- Exercise
- Acute ↑ in CO

▶ **CAUSES of ↓ $ETCO_2$:**

- ↑ Alveolar ventilation
- ↑ Dead space (w/ out Δ in $P_aCO_2$)
  - ↳ due to PE / Air embolism / ET intubation

# SEDATION & ANALGESIA

* Sedation allows critically ill pts to tolerate invasive supportive interventions (intubation & mechanical ventilation)

Delirium is ass. w ↑ in-hospital & ICU stay.

## CONTROL OF AGITATION

- m.c drug used → Benzodiazepines (BDZ)
  - ↳ Potent for sedation, anxiolysis & Amnesia
  - ↳ mediated through GABA receptors. (⊖ γ-Aminobutyric Acid receptor)

BDZ is ass. w higher rates of delirium!

**Modified Ramsey Sedation Scale**
** For titration of sedation **

| | |
|---|---|
| 1 | Anxious / Agitated |
| 2 | Cooperative / Oriented |
| 3 | Responds to commands only |
| 4 | Asleep, BUT responds to stimuli |
| 5 | Asleep, BUT responds sluggishly to stimuli |
| 6 | NO RESPONSE |

** Pts tolerant to BDZ (require ↑ doses)
- Prev. intake of similar agents
- Alcoholic / Smokers

** Pts Susceptible to BDZ (require less doses)
- Elderly — >50 yrs
- Pts w Pre-existing cardiopulm. / hepatic / or renal dysfan.

Medications

**Midazolam** (Class: BDZ)
- Onset (1-3) min
- Short t1/2
- ✓ Active metabolites.

**Lorazepam** (Class: BDZ)
- Onset (10-20) min
- Longer t1/2
- NO active metabolites

If Prolonged use, metabolites accumulate ↓ takes longer time to be fully awaken!

S/E of Propofol
- HYPOTENSION
- Hypertriglyceremia
- Met. acidosis
- Risk of Pancreatitis

**Propofol**
- It does NOT accumulate like BDZ → so earlier ambulation
- more expensive
- Short t1/2

S/E
- Hypotension
- ↓ HR

**Dexamedetomide** (— Selective α2-adrenergic agonist)
- Used in short term sedation, & more easily arousable!
- max. use for 24 hrs

## ••• CONTROL OF DELERIUM

By antiPsychotics — Haloperidole

* used to treat delerium emergently.

S/E :

- Hypotension
- Arrhythmia
- Prolonged QT interval
- Extrapyramidal SX

] ∴ so always do ECG in Pts on long-term Haloperidole!

## ••• CONTROL OF PAIN

Meds

### . Morphine

- PRN (administration as needed)
- Beware of active metabolites accumulation in pts w renal impairement
- S/E : Histamine Release → Hypotension

### . Fentanyl

- m.c drug used as opiate for cont' drips
- t½ (30-60) mn. — Short! due to rapid redistribution.
- Unlike morphine, it does NOT cause histamine release → LESS hypotension.

### Hydromorphine

- Used in Pts allergic to morphine/fentanyl
- NO active metabolites → so used in Pts w Renal failure

### Meperidine

- least freq. used due to many S/E
- Risk of accumulation of metabolites (normeperidine)
- S/E Seizures.

### Methadone

- Narcotic w long t½
- Facilitates withdrawal from narcotics due to long t½

### Thoracic / Lumber epidural cath.

- well tolerated. S/E : hypotension

8:9

**NOTE**

For Pts on long-term sedation & analgesia; daily **interruption** of sedation to wakefulness Produces ↓ time on mechanical ventilation → SHORTER ICU stays.

BUT this is **NOT** applied on surgical ICU Pts (only typical medical ICU Pts) due to higher analgesia requirements than medical ICU Pts.

# RESPIRATORY FAILURE

Inadequate exchange of $O_2$ &/or $CO_2$

**Dx**

- Hx & P/E
  sfs of resp. impairement ← ↑RR, SOB, ↓LOC
- Pulsoximetry & ABGs
  $SaO_2$ <90%
  $PO_2$ < 60 mmHg
- CXR
  to know the cause.

**NOTES**

- **Ventilation**: Air through lungs
  - MONITOR by → $PCO_2$
  - HOW to ↑vent. (↓$PCO_2$)? ↑RR, ↑TV — min vent.
- **Oxygenation**: $O_2$ delivery to alveoli
  - MONITOR by → $PO_2$, $SaO_2$
  - HOW to ↑oxyg. (↑$PO_2$)? ↑$FiO_2$, ↑PEEP

Adequate oxygenation does NOT guarantee adequate ventilation.

**tt**

- $O_2$
- Airway management
- Ventilation

**Extremes of V/Q mismatch**

1. **Dead space vent.**
   V/Q = ∞
   ex.. PE, COPD
2. **Shunt**
   V/Q = zero
   ex. ARDS / Pulmy edema / Pneumonia

**SHOCK**
**SEPSIS** ] Discussed later

## UGI hemorrhage PROPHYLAXIS

ICU pts are at ↑ risk for stress-induced mucosal ulceration → resulting in GI Hmg.

RF
- Head injury (Cushing Ulcers)
- Burns (Curling Ulcers)
- Requirement of mechanical vent.
- Prev. hx of PUD
- Use of NSAIDs or steroids
- Presence of Shock / RF / Portal HTN / or coagulopathy

PROPHYLAXIS
- $H_2$-Blockers (↓ the incidence significantly!)
- PPI (if pt bleeds despite being on appropriate $H_2$-Blocker ~~tt~~)

## RENAL DYSFUNCTION

- Presents as Progressive oliguria (in the setting of ↑ BUN & Cr)

↳ This can Progress to Renal Failure & Anuria (~5% of all ICU Pts)

NOTE
Renal insuff. may present as Polyuria!
(AKA: High-output RF)

- **PRERENAL**: — dve to hypovol.
  - m.c.c of RF in ICU pts
  - LABS: BUN/Cr >20
    Urine osm. > 500 mosm.
    FENa <1 (NL)

**Nephrotoxins**
- Aminoglycosides
- Amphoterzin
- IV radiocontrast
- CTX

*PREVENTION by Good hydration!

- **RENAL**: — dve to ischemia (Prolonged Prerenal state) or toxins.
  - Susceptible Pts: DM or Pre-existing renal d.
  - LABS urine osm. <350 mosm.
    FENa >1
- **POSTRENAL** — dve to BILATERAL renal flow obst.
  - do U/S
  - Urinary cath. malfxn must always be r/o.

(7)

8s

## ANEMIA

* Indx of transfusion (usually Packed RBC)
  - Hb < 7 mg/dL
  - Hb 7–10 mg/dL in pts w/ — Acute coronary Synd., Severe hypoxemia, Active bleeding

Goal ⇒ Hb > 10 mg/dL

⊘ DON'T transfuse if Hb > 10 mg/dL.

Why? cuz it's ass. w/:

1. TRALI (Transfusion-Related Acute Lung Injury)
2. ↑ Hospital & ICU stay
3. Death!

## BLOOD GLU CONTROL

* Tight glycemic control is ass. w/ ↓ mortality.

Goal ⇒ < 140 mg/dL — safe & beneficial!

* Hypoglycemia is still a major risk of tight glycemic control.

Yale Gluth
The End

# ARDS

## Acute Respiratory Distress Synd.

### DEFINITION

Acute lung injury, lung damage & release of inflammatory cells leading to ↑ Permeability & Pulmonary edema, often associated w multiorgan failure.

### CAUSES

- **DIRECT**
  - Pneumonia
  - Aspiration
- **INDIRECT**
  - **SEPSIS** (m.c.c)
  - Severe trauma
  - Acute Pancreatitis
  - Drug overdose (Aspirin / Heroin)
  - Fat embolism
  - Near drowning

ARDS is **type I** respiratory failure — Hypoxemic RF

### CLINICAL FEATURES

- Cyanosis
- Tachypnea / Tachycardia
- Peripheral vasodilation
- **Bilateral** Fine inspiratory crackles

$PO_2/FiO_2$ NL is >300

- Acute Lung injury 200-300
- ARDS → <200

In ARDS, A-a gradient is ↑

### INVESTIGATIONS

- Blood tests: CBC / electrolytes / clotting (PT, PTT) / Amylase / CRP / & Blood culture
- ABG
- CXR: BILATERAL Pulmonary infiltrates.
- Pulmonary art. catheterization: to measure PCWP

8.3

## DIAGNOSTIC CRITERIA (Mnemonic: Remember it as ARDS!)

**A**cute onset
**R**efractory hypoxia ($PO_2/FiO_2$ < 200)
**D**iffuse bilateral infiltrates.
**S**hunt hypoxemia mechanism (**NO** Lt-sided HF) – PCWP < 18 mmHg

## PHASES OF ARDS

### ① EXUDATIVE PHASE — Cellular Phase

- ALOT of cells
- Alveolar spaces damage
- Capillary injury

CXR findings

- BILATERAL infiltrations
- Can't be differentiated from cardiogenic Pulm. edema if heart size is NL.

### ② PROLIFERATIVE PHASE — Fibrosing Alveolitis.

few pts develop this phase

- Improper healing Process → "Fibrosis"!!
- Significant Pulm. HTN ↑ fibroblasts.

CXR Findings

- NOT as Prominent as exudative Phase.

** Here, You can do NOTHING! 🤷

### ③ RECOVERY PHASE 🙂

- Gradual resolution of hypoxemia
- CXR is NL
- Functionally, lung is back to NL.
- Alveolar septa is back to NL.

## MANAGEMENT

- ADMIT the pt to ICU
- Supportive therapy :
  - ✱ Mechanical ventilation
    - ↳ Low $V_T$ (< 6 ml/kg) & Pressure-limited approach (< 30 cm $H_2O$))
      - ✱ This improves outcome! ☺
  - ✱ IV Fluids
    - ↳ Give the least fluids as possible
      - — Monitor by swan-ganz catheter.
- Treat the underlying cause ex. sepsis by abx.

## PROGNOSIS

The mortality decreased from 60% → 30%!

✱ Poor Px is ass. w

- Chronic liver d.
- Sepsis (esp. in the end-stage)
- Old age
- Non-pulmonary organ dysfxn (esp. if ≥ 3 organs)

✱✱ Still, mortality rate is very HIGH!

Good Night
The End.

৭৬

# FLUIDS & ELECTROLYTES

Source: Washington Surgical Recall Dossier

Total Body Water (TBW)
(60% of body wt)

- **INTRA**cellular — 40% of body wt (2/3)
- **EXTRA**cellular — 20% of body wt (1/3)
  - Intravascular — 5% (1/4)
  - Interstitial (3rd space) — 15% (3/4)

Mnemonic
"TIE"
Think 60, 40, 20
Total body fl. 60%
Intrac. 40%
Extrac. 20%

* The Principle **EXTRA**cellular — Cation → $Na^+$; Anions → $Cl^-$ & $HCO_3^-$

* The Principle **INTRA**cellular — Cations → $K^+$ & $Mg^{+2}$; Anions → $PO_4^{-2}$ & negatively-charged proteins

## Notes

- Lean tissues (muscles & solid organs) have higher water content than fat & bone.
- ♀ TBW = 50%
  ♂ TBW = 60%
  Newborns TBW = 80%

** The concentration gradient btw. compartments is maintained by ATP-dependant Na-K pump, & movement of water across a cell membrane depends primarily upon osmosis.

**Serum Osmolarity** $= 2\,Na^+ + Glucose/18 + BUN/2.8$

$= (290 - 310)$ mosm  **N 300** mosm.

a2

## Definitions

- **Osmolality**: no. of osmoles / Kg of water
  Comprised of effective AND ineffective components

- **Tonicity**: The physiologic parameter that the body attempts to regulate.
  — Equals only effective osmolarity

* **Effective osmoles**: Can NOT freely permeate through cell membrane.

e.g. Extracellular ▸▸ $Na^+$ / glu / mannitol / glycine
Intracellular ▸▸ $K^+$ / aminoacids / & organic acids.

∴ So any asymmetric accumulation of effective osmoles will cause transcompartment. movement of water (hence the name!) "effective"

* **Ineffective osmoles**: Can freely cross cell membrane & UNABLE to affect water movement (Ineffective!)

e.g. Urea / ethanol / methanol

## NOTES:-

- Avg. intake of water / day = 2L < 75% from oral intake; 25% extracted from solid food.

- Daily water losses:
  - Urine → (1 - 1.5) L
  - Feces → (250) ml
  - Insensible loss from < Skin (sweat) → (200 - 400) ml; Respiratory loss → (500 - 700) ml

Levels of $Na^+$ & $Cl^-$ in sweat ~40 meq/L

- **PHYSIOLOGIC RESPONSES TO HYPOVOLEMIA:**
  1. $Na^+$/water retention → via renin - aldosterone
  2. water retention → via ADH
  3. Vasoconstriction via < Angiotensin II; Sympathetics
  4. ↓UOP

Role of ADH (Vasopressin)

Δin plasma osmolality
↓
sensed by osmoreceptors in the hypothalamus
↓
Sxn of ADH (sensed by Δin EC volume)

(2)

# ELECTROLYTE DISTURBANCES

- The common cause is LAB ERROR!

**Remember!**
- Major extracellular cation → $Na^+$
- Major intracellular cation → $K^+$

## SODIUM ($Na^+$)

NL range: (135 - 145) mmol/L

- Sodium balance is maintained mainly by the KIDNEYS.
- All (but 2-5%) of $Na^+$ in the body is located in the extracellular fluid & difference in distribution is maintained by $Na^+$-$K^+$ Pump.
- $Na^+$ salts accounts for >90% of total osmolality of ECF
- Plasma $Na^+$ concentration reflects the RELATIVE Proportions of $Na^+$ & water NOT the absolute amount of $Na^+$ in the body.

## HYPONATREMIA

< 135 mmol/L

CAUSES

Serum osmolality

### HYPOtonic hyponatremia

- **HYPOVOLEMIC**
  - . GI losses
  - . Skin losses
  - . lung losses
  - . 3rd spacing
  - . Renal losses
- **HYPERVOLEMIC**
  - CHF
  - Cirrhosis
  - Iatrogenic fluid overload
- **ISOVOLEMIC**
  - Water intoxication / SIADH
  - $K^+$ losses → (for GI losses / diuretics due to cellular exchange of cations)
  - Drugs [Sulfonylurea / Carbamazepines / Phenothiazines / Antidepressants]

### HYPERTONIC hyponatremia

- Hyperglycemia (Pseudohyponatremia)
- Hypertonic infusions:
  - Glu
  - mannitol
  - Glycine

### ISOtonic hyponatremia

- Pseudohyponatremia:
  - Hyperlipidemia
  - Hyperproteinemia
- Isotonic infusions:
  - Glu
  - mannitol
  - Glycine

Work up:
urine osmolarity
urine $Na^+$ → >10 → renal cause
→ <10 → extrarenal cause

## * Sxs of hyponatremia

** Predominantly NEUROLOGIC (due to brain cells' swelling – cerebral edema)

- Lethargy / confusion
- N, V
- Seizures / coma

Sx are rare when Na+ > 125 mmol/L

– Chronic hyponatremia is usually asympt. until serum Na+ is < 110 mEq/L

## * tt

- Isotonic / Hypertonic hyponatremia → treat the cause
- Hypotonic hyponatremia
  - Hypovolemic → Give N.S & correct ~~hydration~~ ongoing losses.
  - Hypervolemic → water restriction ± diuretics (loop) ± BNP (in acute HF)
  - Euvolemic
    - SIADH → lasix + fluid restriction
    - water intoxication → fluid restriction

* In ① Symptomatic hyponatremia or ② Extreme hyponatremia (<110); HYPERtonic saline is indicated (3% NaCl)
  → Goal: >120 mmol/L (when it's reached, discontinue the hypertonic saline)

Hypertonic saline Should NOT be administered w/out concomitant diuretic tt.

## CENTRAL PONTINE DEMYLINATION

- It's a complication of RAPID correction of hyponatremia
- Signs:
  - Confusion
  - Spastic quadriplegia
  - Horizontal gaze Paralysis
- **To AVOID** this, the rate of correction Should be ≤ 12 meq/L/day

Measurement of Na+ Deficit

= 0.6 X Wt * X (120 – measured Na+)

# POTASSIUM ($K^+$)

NL range (3.5 - 5.3) mmol/L
Requirements (50-100) mmol/day

- Major INTRAcellular cation (only 2% is extracellular!)
- It's influenced by — acid-base balance
  — ↑ hormones
- Renal excretion

Mnemonic
alKalosis
≡ $K^+$ low / (Hypokalemia)

## HYPOKALEMIA

### CAUSES

GI: ↓intake (rare!)
GI losses — vomiting / Diarrhea / villous adenoma / fistula

Renal: metabolic alkalosis
Diuretics / steroids
RTA / 2ry aldosteronism.
↓$Mg^{+2}$

Shift into cells: Insulin effect
Alkalosis

*Hypomagnesemia is frequently ass. w hypokalemia & must be corrected so always give $Mg^{+2}$ before $K^+$ replacement
↓ It ⊖ reabsorption of $K^+$ from renal tubules.

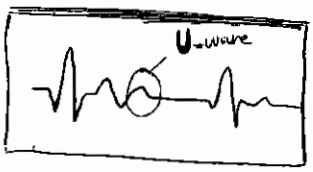
### C/P

mainly CARIOVASCULAR & also Neuromuscular

→ If mild (>3) → Asympt.

→ If severe (<3): weakness / tetanus / Paresthesia
N, V, Ileus

Hypokalemia exacerbates Digitalis Toxicity




ECG Changes — T-wave depression
ST depression
Prominent U wave
ectopy (PAC / PVC / A-fib.)

### ~~tt~~

- Oral replacement (KCl) — If mild
- IV replacement (KCl) — Indx: • Severe <3
  • Significant Sx
  • Oral intolerance.

### ⊗ CAUTION

- Maximum amount of $K^+$ infusion is 40 mmol/L
- Maximum infusion RATE of $K^+$ via — Peripheral line: 10 mmol/hr
  — Central line: 20 mmol/hr

why? Rapid infusion may cause cardiac arrest!
also thrombophlebitis may result.

(6)

# HYPERNATREMIA ≡ always hypertonic hypernatremia >145

CAUSE
Typically due to loss of water in excess of solutes.

* Surgical causes

. Inadequate hydration
. Renal losses < DI / Diuresis
. GI losses < vomiting / Diarrhea
. Sweating / tachypnea / Iatrogenic TPN

**SIS**

* Mainly NEUROLOGIC (due to dehydration of the brain cells)
. Lethargy / weakness / irritability / fasciculation / seizures / coma
~~Respiratory paralysis~~

**Tx** Give oral water or IV G/W 5% (hypotonic) or NS 0.45% / 0.9 — slowly over 12-24 hrs
. Treat water deficit ass. w/ hypernatremia

$$\text{Water deficit (L)} = 0.6 \times \text{Wt(kg)} \times \left(\frac{\text{current } Na^+}{140} - 1\right)$$

. Treat the underlying cause.

* What is the MAJOR COMPLICATION of ↓ $Na^+$ level too fast?
Seizures (NOT central Pontine demyelinolysis)

## Renal $Na^+$ Excretion/Reabsorption

① **Effective blood volume**: ↓ vol. ⊕ RAAS system
↓ filtered $Na^+$
↑ Renal reabsorption

② **Natriuretic hormones**:
Secreted by atria
Fxn: ↑↑ excretion of $Na^+$ by ↑ filtration & ⊖ reabsorption

**Proximal** (Changes in Starling forces + symp. n.)
**Distal** (Aldosterone)

# HYPERKALEMIA > 5.3 mmol/L

## CAUSES

Always think first of simple things!

- **Pseudohyperkalemia** (Spurious hyperkalemia) due to:
  - Poor venipuncture technique
  - Leukocytosis / Thrombocytosis
  - In vitro hemolysis
- Excessive **intake**
- Impaired **excretion**:
  - Renal failure / Tubular d.
  - Adrenal insuff. / Addison's
  - Diuretics: $K^+$ sparing (Spironolactone)
- **Shifts**:
  - Acidosis
  - Insulin def.
  - Tissue damage (hemolysis / internal bleeding / muscle damage)
  - Drugs: digitalis Poisoning / Arginine.
- Blood tx

## C/P

CARDIOARRHYTHMIAS & muscle weakness.
also ↓ DTR/areflexia

ECG abnormalities:
- **Peaked T-wave**
- Prolongation of PR interval
- Prolonged QRS complex
- Complete heart block
- Cardiac arrest!

## ttt

usually if >6.5 mmol/L

The critical value is >6.5 mmol/L

**URGENT ttt** Remember "CB DIAL K"

- **C**alcium IV — cardioProtective (w ECG monitoring)
- **B**icarbonate ($NaHCO_3^-$) IV — alkalosis drives $K^+$ into the cells.
- **D**ialysis_Definitive! (Indx if severe/refractory / or life-threatening hyperKalemia)
- **I**nsulin / dextrose
- **A**lbuterol ($\beta$-agonist)
- **L**asix (Furosemide)
- **K**ayexalate ($Na^+$ Polystyrene sulfonate)

**Non-acute ttt**
- Lasix
- Kayexalate.

To sum up,

**HYPOKALEMIA**
- AlKalosis
- Insulin
- β-agonists
- Osmotic diuretics
- Sodium Bicarbonate (Alkalosis)

**HYPERKALEMIA**
- Acidosis
- ACEI
- β-Blockers
- CTX
- K+-sparing Diuretics
- Digitalis toxicity
- NSAIDs
- Succinylcholine

ABCDD

## CALCIUM

NL range (8.5-10.5) mg/dL

- 3 FORMS:
  - Ionized; 45% → The ONLY active form!
  - Protein-Bound 40%
  - Complex w/ freely diffusible compounds 15%
- METABOLISM is controlled by:
  - PTH (from bone & kidney)
  - Vit D (from intestine)

## HYPOCALCEMIA

* Dx should be based only on IONIZED form NOT total $Ca^{+2}$.

CAUSES
- Vit D def.
- Calcium sequestration:
  - Acute pancreatitis
  - Rhabdomyolysis
  - Rapid transfusion
- Surgical causes: After,
  - Total thyroidectomy — 2ry to vascular compromise to Parathyroid glands.
  - Parathyroidectomy — Serum $Ca^{+2}$ reaches its lowest level w/in 2-3 days Post-op. returning to NL in 2-3 days.

Usually it causes transient hypocalcemia

- In conjunction w/ $Mg^{+2}$ depletion (which simultaneously impairs PTH sxn & fxn)
- Acute alkalemia (from rapid administration of $HCO_3$ or hyperventilation) — due to abrupt ↓ in the ionized fraction (w/ NL total $Ca^{+2}$)
- Drugs:
  - Aminoglycosides
  - Diuretics (loop)

## C/P of hypocalcemia

- Perioral numbness & tingling
- Tetany (2 Signs) / Chvostek sign \ Trousseau's Sign
- ↑DTR
- ECG changes: QT Prolongation
  Ventricular arrhythmias

**Chvostek Sign**

Facial muscle spasm elicited by tapping over the branches of facial n. on CHeek!

(Remember)

**CH**vostek ≡ **CH**eek

## ~~tt~~

Acute tt — Calcium Gluconate 10% IV
Chronic tt — oral $Ca^{+2}$ & vit. D.

$Ca^{+2}$ infusion may Potentiate Digitalis toxicity!

* During therapy, always monitor $Mg^{+2}$, Phosphorus & $K^+$ levels
  — Replete if necessary.

* Complication of infused Ca if IV infiltrates → Tissue Necrosis!
  So Never administer peripherally unless absolutely necessary
  [Calcium gluconate is LESS toxic than CaCl.]

* **HYPO**albuminemia ↓ **total** $Ca^{+2}$ (bound fraction)
  While ionized form is unaffected.
  So
  ↓1 g/dL albumin will ↓ Serum $Ca^{+2}$ 0.8 mg/dL

* Formula to measure $Ca^{+2}$ in pts w hypoalbuminemia
  = (4 − measured albumin) × 0.8
  add this value to the measured $Ca^{+2}$ level → corrected $Ca^{+2}$

# HYPERCALCEMIA

## CAUSES Mnemonic: CHIMPANZEES

- **C**alcium Supplements IV
- **H**yperParathyroidism (1°/3°) / **H**yperthyroidism
- **I**mmobility / **I**atrogenic (thiazide diuretics)
- **M**etz / **M**ilk alkali Synd.
- **P**aget's disease (bone)
- **A**ddisons / **A**cromegaly
- **N**eoplasm (colon / lung / breast / Prostate)
- **Z**ES (as Part of MEN I)
- **E** xcessive Vit. D
- **E** xcessive Vit. A
- **S**arcoid

## C/P

- " Stones, Bones, Abdominal groans & Psychiatric overtones)
  - Nephrolithiasis
  - bone disease & Pain
  - N, V, constipation & dehydration
  - Δ mental status.
- Polyuria, Polydypsia & constipation

.ECG changes: - Short QT
- Prolonged PR

## ttt

If mild (< 12) — $Ca^{+2}$ restriction & treat underlying cause
\ Hydration.

If severe — Hydration w/ N.S + diuretics (BUT NOT thiazide)
- Steroids / Calcitonin / Bisphosphonates (Pamidronate) / mithramycin
- Dialysis (Last resort!)

## PHOSPHORUS

NL range (2.5 - 4.5) mg/dL

* Usually derangement in concentration of phosphate & $Ca^{+2}$ coexist.

### HYPOPHOSPHATEMIA

CAUSES

- ↓intestinal absorption — malabsorption (vit. D def.)
  \ use of phosphate binders:
  - Aluminium
  - Mg
  - $Ca^{+2}$
  - iron
- Renal loss — Acidosis / Alkalosis
  - Diuretic ttt (esp. acetazolamide)
  - Hyperglycemia — osmotic diuresis
  - During recovery of ATN
- Shift (from extra → intra) — Resp. alkalosis
- Born pts.

**C/P**

Usually asx, if severe (<1) mg/dL

- Resp. muscle dysfxn → Resp. failure!
- Diffuse weakness
- Flaccid Paralysis

**Risks of IV Phosph Replacement**

- Hyperphosphatemia
- Hypocalcemia
- Hypomagnesemia
- Hyperkalemia (from Potassium Phosphate)
- Hypotension & Hyperosmolarity
- Metastatic calcification
- Renal Failure

**ttt**

ttt is important esp. in critically ill pts.

IV Replacement - 7-10 days

once it's > 2 mg/dL → oral ttt (4 times / day) (Sodium Phosphate)

## HYPERPHOSPHATEMIA

**CAUSES**

- Impaired renal excretion
- Transcellular shift (intra → extra) due to: tissue trauma, tumor lysis (CTX), Insulin def., Acidosis
- Postop hypoparathyroidism.

**C/P**

In the Short term → Hypocalcemia & tetany

w chronicity → Soft tissue calcification (ectopic) 2ry hyperparathyroidism.

**ttt**

- Dietary restriction
- Hydration (to ↑ excretion) w diuresis (acetazolamide)
- Aluminium hydroxide (binds phosphorus)
- Dialysis — only in extreme / severe cases.

## MAGNESIUM

NL range (1.5 - 2.5) meq/L

- Mainly intracellular
- RENAL excretion & retention play the major physiologic role in regulating body stores.
- Mg is NOT regulated under hormonal regulation

# HYPOMAGNESEMIA → Usually ass. w hypocalcemia & hypophosphatemia.

Frequently encountered in trauma pts.

## CAUSES

SURGICAL causes:
- TPN
- hypocalcemia
- gastric suctioning
- Diarrhea, vomiting
- Aminoglycosides
- Renal failure

- GI or Renal losses
- Shift (extra → intra)
  - → Acute MI
  - → Alcohol withdrawal
  - → Recieving glu-containing solutions
- After Parathyroidectomy.

ALWAYS fix hypomagnesemia w hypokalemia

## C/P MAINLY NMS & CVS

**NMS** Δ Mental Status
Tremors / hyperreflexia (↑DTR) / tetany (like hypocalcemia)

**CVS** QT, PR & QRS Prolongation
V. arrhythmias (esp. in pts recieving digitalis)

## tt

- Acute tt → MgSO4 IV
- Chronic tt → oral Mg oxide (s/e: diarrhea)

Indx of **IV** MgSO4
- In severe cases (<1)
- In symptomatic pts
- if pt developed Torsade de Pointes

# HYPERMAGNESEMIA

## CAUSES

- IATROGENIC! (TPN, RF (Renal Failure), IV oversupplement)

(seen mostly in the setting of RF — due to impaired excretion)

## C/P

mild (5-6) — asympt.
Severe (>8) — ↓DTR
- Hypotension / sinus bradycardia
- CNS depression / Resp. failure
- Prolonged PR, QRS & QT

The **antidote** of MgSO4 is Calcium Gluconate!

## tt

- STOP exogenous Mg
- Give Ca gluconate 10% IV in life threatening cases
- Dialysis → the definitive therapy

Good Luck
The end.

# IV Fluid Therapy

Source: Washington

## SOLUTIONS

- Crystalloids < Isotonic, Hypertonic, Hypotonic
- Colloids < Albumin, Dextran, Hydroxyethyl starch

| | |
|---|---|
| N.S | 154 meq $Cl^-$ / 154 meq $Na^+$ |
| ½ N.S | 77 meq $Cl^-$ / 77 meq $Na^+$ |
| ¼ N.S | 39 meq $Cl^-$ / 39 meq $Na^+$ |
| D5W (5% dextrose) | (50g) dextrose in $H_2O$ |
| Lactate Ringer (LR) | 130 meq $Na^+$, 109 meq $Cl^-$, 28 $HCO_3^-$ (lactate), 3 meq $Ca^{+2}$, 4 meq $K^+$ |

## CRYSTALLOIDS

- Relatively inexpensive

**Indx**
- Volume Expansion
- Maintenance infusion
- Correction of electrolyte disturbances.

### ● ISOTONIC CRYSTALLOIDS

Ex. 0.9% NaCl N.S
Ringer's Lactate (RL) } usually used interchangably

* Distribute uniformly throughout the extracellular fluid compartment.
  ∴ So after 1 hr, only 25% of total vol. remains in the intravascular space.

* N.S is Preferred in < HYPERKalemia, HYPERCalcemia, HYPONatremia, HYPOChloremia, Metabolic Alkalosis

Lactate base becomes $HCO_3^-$

The lactate in RL will be converted into bicarbone

So it can NOT be used for maintenance cuz pts will become alkalotic!

* Ringer's Lactate is designed to mimic extracellular fluid (Plasma) ≡ Balanced Salt sol.

* The m.c trauma resuscitation Fluid is ⟶ Ringer's Lactate

## • HYPERTONIC SOLUTION

Ex. 7.5% NaCl
3% NaCl

Indx
- Used in Shock / burns (usually in combination w colloids like dextran)
- TPN

Effects

Studies showed that it causes significant blunting of neutrophil activation. w transient ↑ in serum Na that normalizes w/in 24 hrs.

*This effect may help in ⊖ widspread tissue damage & multiorgan dysfxn seen after traumatic injury.

S/E
- Hypernatremia (Hyperosmolarity) & Hyperchloremia
- HypoKalemia
- Central Pontine demyelination.

## • HYPOTONIC SOLUTIONS

Ex. D5W
0.45% NaCl

- Should NOT be used for volume expansion (cuz it only expands 10% of vol. infused!)

Indx
Replacement of free water deficit

## COLLOID SOLUTIONS

- It contain High-molecular wt substances that REMAIN in the intravascular space
- More expensive than crystalloids.

Indx
When crystalloids fail to sustain plasma vol. bcz of low colloid osmotic pressure in burn pts & Peritonitis (↑ Protein loss from vascular space)

S/E
- Pulmonary Edema
- Renal failure (max. dose)
- Bleeding disorders
- Reaction
- Expensive!

* Early use of colloids in the resuscitation regimen may result in more prompt resuscitation of tissue perfusion & may ↓ total vol. of fluid required.

- **ALBUMIN PREP.** 5% or 25% Albumin.

  Indx Vol. Expansion

  NOT indicated for pts w adequate colloid oncotic pressure — Albumin >2.5
  Total Protein >5 mg/dL

- **DEXTRAN** < Dextran-40 / Dextran-70

  — Synthetic glu Polymer (undergoes renal elimination)

  Indx
  - Volume Expansion (Expands the intravascular vol. by an amount **EQUAL** to the volume infused!)
  - Thromboembolism Prophylaxis

  S/E
  - Renal Failure
  - Osmotic diuresis
  - Coagulopathy
  - Laboratory abnormality < ↑ blood glu & Protein / Interferes w blood cross matching

- **HETASTARCH** (Hydroxyethyl starch)

  — Synthetic molecule (similar to glycogen)
  - Replaces SAME amount of fluid infused.
  - LESS expensive than albumin.
  - LESS S/E than dextran.

  S/E ↑ Amylase x2.

# • PRINCIPLES OF FLUID MANAGEMENT

- NL individual consumes 2-2.5 L of water daily.
- Daily water losses — 1-1.5 L in urine
  - 250 ml in stool
  - 750 ml insensible losses — Skin 250 ml, Resp. 500 ml

* The minimum amount of fluid needed to excrete catabolic end products is ~~(800 ml)~~

- NL daily electrolyte loss — $Na^+$ & $K^+$ ⇒ 100 meq
  - $Cl^-$ ⇒ 150 meq.

* **Preop. Mgt**
Any pre-existing electrolyte disturbance should be corrected BEFORE operation. ↑ w/ hypermetabolism/ hypervent. /& Fever.

* **Intraop.**
↳ Replace Preop. losses (deficit) & ongoing losses.
So Replacement includes — maintenance
- Hmg
- 3rd space losses (depends on incision)

NL urine output: At least 0.5-1 cc/kg/hr

* **Postop.**
- Monitor UOP
- Monitor GI losses from NGT/Stoma.

## 3rd Spacing

Fluid accumulation in the interstitium of tissues (as in edema) from intravascular → to interstitium.

(int) — * When does it return back into intravasc.?
**Postop day #3**
(So BEWARE of fluid overload, once the fluid begins to return back to intravascular compartment, SWITCH to hypotonic fluids & ↓ rate)

* **CLASSIC SIGNS** of 3rd spacing: - Tachycardia
- ↓ UOP

(tt) IV hydration w/ isotonic fluids.

## ∞ IV Replacement by anatomical site

Gastric (NGT) — D5 ½ N.S +20 KCl
Biliary — LR ±$NaHCO_3$
Small Bowel (Ileostomy) — LR
Colonic (Diarrhea) — LR ±$NaHCO_3$

**NOTES**

- Most common trauma resuscitation fluid → LR
- Most common Postop. IV Fluid after laparotomy: LR / D5 LR for 24–36 hrs
- After laparotomy, when should a Pt's fluid be monitored? Postop day #3
- What IVF is used to replace Pancreatic Fluid loss? → LR (Bicarbonate loss)
- * What Portion of 1L N.S will stay in intravascular space after laparotomy? After 5 hrs — only 20%!

**Daily Secretions:**

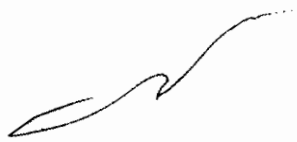
Saliva — 1.5 L / day
Bile — 1 L
Pancreatic sxn — 600 ml
Gastric — 2 L
Small bowel — 3 L

* **Almost all** sxns are reabsorbed.

"$B_1G_2S_3$" Remember it alphabetically & numerically.

Bile — 1 L
Gastric — 2 L
Small bowel — 3 L

The End.



# FLUID MANAGEMENT
(Calculations)

IV Fluid Replacement:

**MAINTENANCE + DEFICIT + ONGOING LOSSES**
(Insensible losses).

## CALCULATION of Maintenance

FLUIDS

- Roughly it's 30-50 ml/kg/d. (Mean of 40)

- **100/50/20 Rule:** (/24 hrs)

  1st 10 kg → 100 ml/kg/d.
  2nd 10 kg → 50 ml/kg/d.
  Rest → 20 ml/kg/d.

- **4/2/1 Rule:** (/1 hr)

  1st 10 kg → 4 ml/kg/1 hr
  2nd 10 kg → 2 ml/kg/1 hr
  Rest → 1 ml/kg/1 hr.

Minimal UOP:
for adults 0.5-1 cc/kg/hr
for children 1-2 cc/kg/hr

ELECTROLYTES

* Maintenance fluid → usually N.S

Sodium 2-4 meq/kg/d.
K+ 1-2 meq/kg/d.

- fluid is administered as following:
  1) ½ of total fluid over first 8 hours
  2) ½ of total fluid over next 16 hours.

The End.

# TRAUMA

Source: Recall
Kaplan

* The widly accepted Protocol is Advanced Trauma Life Support (ATLS)

## • ELEMENTS OF ATLS Protocol •

- PRIMARY SURVEY / RESUSCITATION
- SECONDARY SURVEY
- DEFINITIVE CARE.

Hx is obtained while Completing the Primary survey.

## PRIMARY SURVEY

5 STEPS: THINK ABCDE

**A**irway (& C-spine stabilization)
**B**reathing
**C**irculation
**D**isability
**E**xposure & Environment.

Principles are followed in completing the Primary Survey:

* Life-threatening Problems discovered during the Primary survey are ALWAYS addressed BEFORE Proceeding to the next step.

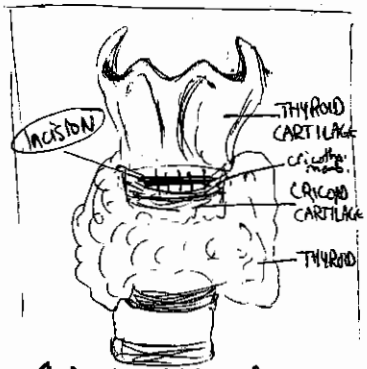
**A** ➤ **Airway** + C-spine

Goal: Securing the airway & protecting the spinal cord.

* SPINAL IMMOBILIZATION must be considered in airway assessment. HOW?
  — Use a full backbone & rigid cervical collar.

* **Maneuvers** used to establish an airway:
  Chin lift, Jaw thrust, or BOTH
  — If successful, often an oral/nasal airway can be used to temporarily maintain the airway.
  — If unsuccessful, do endotracheal Intubation (ET intubation)
  — If ET intubation is unsuccessful, do cricothyroidotomy (Surgical airway)

Ask the Pt a question, if he can speak → then the airway is intact!




Cricothyroidotomy:-
Incise the cricothyroid membrane (btw cricoid cartilage Inf. & thyroid cartilage Sup.)

* So ALWAYS consider spinal immobilization & adequate oxygenation.

B

# Breathing

Goals — Securing oxygenation & ventilation
— Treating life-threatening thoracic injuries.

Assessment: **Inspection** — For air movement / RR / Cyanosis / ↑JVP / Tracheal shift / Asymmetric chest expansion / Use of accessory muscles.

**Palpation** — For presence of subcutaneous emphysema, Flail segments

**Percussion** — Hyperresonance / dullness over either lung field.

**Auscultation** — For breath sounds.

Life-threatening conditions that MUST be diagnosed & treated during this step:
- Tension Pneumoth.
- Open Pneumothorax
- Massive Hemothorax
- Tamponade.

## PNEUMOTHORAX

It's injury to the lung, resulting in release of air into the pleural space btw. the normally apposed Parietal & visceral pleura.

**SpS** — Usually asymptomatic
- Chest pain, SOB, Anxiety
- Hyperresonance of affected side
- ↓ Breath sounds of affected side

**Dx**
Clinical
CxR (~83% sensitive) — demonstrates absence of lung markings where the lung has collapsed.

**Tt**
Tube thoracostomy (Chest tube)

### Open Pneumothorax

**Dx** usually obvious w/ air movement through a chest wall defect
↑ pneumothorax on CxR

**Tt**
- Chest tube.
- occlusive dressing.

### *Tension Pneumothorax

— Life-threatening emergency!
— It causes total ipsilateral lung collapse & mediastinal shift (away from injured lung) → Impairing venous return → ↓CO → ↓BP

**Dx**
Clinical
(No time for CxR!)

**SpS** Same as mentioned above + mediastinal shift. Shock!

**Tt**
- Immediate needle decompression → then Chest tube

## Flail Chest

2 separate fractures in 3 or more consecutive ribs.

Dx
- Flail segment of chest wall that moves **paradoxically** (sucks in w/ inspiration & pushes out w/ expiration, opposite the rest of the chest wall)

Tx
Intubation w/ PPV & PEEP prn (let ribs heal on their own)

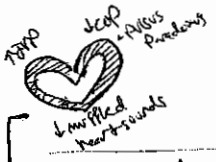
*MAJOR cause of respiratory compromise w/ flail chest is **underlying contusion!**

## Cardiac Tamponade

Bleeding into the pericardial sac, resulting in constriction of heart, ↓COP (The pericardium does NOT stretch!)

Dx U/S (echocardiogram)

Tx Pericardial window — if blood returns then median sternotomy to r/o & treat cardiac injury!




**Beck's Triad**
- Hypotension
- Muffled heart sounds
- ↑JVP

**Characteristics**
- Pulsus Paradoxus
- Kussmaul's Sign (↑JVP w/ inspiration)

## Massive Hemothorax

Dx Unilateral ↓/absent breath sound
Dullness on Percussion
CXR, CT
Chest tube output

Tx
- Volume replacement
- **Chest tube**

Index for emergent thoracotomy for hemothorax:
- Massive Hemothorax =
  - > 1.5 L of blood on initial placement of chest tube.
  - Persistent, >200 cc of bleeding via chest tube per hr x4 hrs.

## C Circulation

Goals: Securing adequate tissue perfusion & ctrl of external bleeding

INITIAL test for adequate perfusion: Palpation of Pulses!
Roughly, if palpable radial pulse (then) Syst. pressure at least 80 mmHg
if palpable femoral/carotid (then) Syst. pressure > 60 mmHg (3)

**Assessment for circulation:** HR / BP
Peripheral perfusion / capillary refill (NL < 2 sec)
UOP
mental status
exam of skin: cold, clammy → hypovolemia.

**Sites of external bleeding:** Direct pressure ± tourniquets

**BEST IV access in trauma pts:**
- 2 large-bore IVs (14-16 gauge)
IV catheters in upper extremities (Peripheral IV access)

Pts who may not mount a NL tachycardic response to hypovolemic shock:
- pts on β-blockers
- DM (orthostatic hypotension)
- pts w SC injuries
- well-conditioned athletes.

**Alternative sites of IV Access:**
- Percutaneous & cutdown Catheters in lower leg saphenous
- Central access — femoral, jugular, subclavian.

**FLUID of CHOICE in trauma pts:** Ringers' lactate! — lactate helps buffer the hypovolemia — induced metabolic acidosis.

**TYPES of decompression in trauma pts**
- Gastric decompression w NGT
- Bladder decompression w Foley's after NL rectal exam.

**CI of Foley's**
- Signs of urethral injury
- Severe pelvic ♯ in ♂
- Blood at urethral meatus / Perineal oozing)
- "High-riding" Prostate
- Scrotal / Perineal injury / ecchymosis.

## D Disability

**Goals:** Determination of neurologic injury (THINK: Neurologic Disability)

**Assessment**
- Mental status — Glasgow Coma Scale (GCS)
- Pupils — A blown pupil suggests ipsilateral mass (blood) as herniation of the brain compresses CN III
- Motor / Sensory — Screening exam for lateralizing extremity movement / sensory deficits.

# Glasgow Coma Scale (GCS)

## Eye Opening
4 - Opens Spontaneously
3 - Opens to Voice/command
2 - Opens to Painful Stimuli
1 - Does NOT open eyes

THINK: 4 Eyes!

## Motor Response
6 - Obeys commands
5 - Localizes Pain
4 - Withdraws from Pain
3 - Decorticate Posture
2 - Decerebrate Posture
1 - NO movement

## Verbal Response
5 - Appropriate Oriented
4 - Confused
3 - Inappropriate words
2 - Incomprehensible sounds
1 - NO sounds

THINK: Verbal ≡ V ≡ 5

GCS NL 15

If intubated → the score is out of 11 not 15 (verbal response is not evaluated)

Score for a Pt in Coma ≤ 8

Score for a DEAD man. 3

## Exposure / Environment

(E)

Goals: Complete undressing to allow a thorough visual inspection & digital palpation of the Pt during the 2ry survey.

Environment ≡ Keep a warm Environment (i.e. keep the Pt warm, a hypothermic Pt can become coagulopathic!)

# SECONDARY SURVEY

A "Trauma Series" consists of radiographs of
- C-spine
- Chest
- Pelvis

## Trauma Hx

It begins once the primary survey is complete & resuscitative efforts are done.

* Whenever possible take an "AMPLE" Hx :-
  - **A**llergies
  - **M**edications / **M**echanism of injury
  - **P**MHx / **P**regnant?
  - **L**ast meal
  - **E**vents surrounding the mech. of injury

DON'T forget to examine Pts back in trauma Pts! - often forgotten.

- Complete Pt/E, including ALL orifices: Ears, Nose, Mouth, Vagina, Rectum → i.e. Head-to-toe evaluation!
- Neuro exam (GCS) / Procedures / Labs / Imaging

## ** NOTES on Plt

- On **eye** examination, look for traumatic **Hyphema** (Blood in the ant. Chamber of the eye)
- On **ear** examination, look for **Hemotympanum** (a sign of basilar skull #)
- On **nasal** examination, DON'T miss **Nasal Septal Hematoma**.
  - Hematoma must be evacuated if not, it can result in pressure necrosis of the septum.
- Best indication of a mandibular # is **Dental malocclusion**.

# TYPES OF TRAUMA

## HEAD TRAUMA

- Penetrating head trauma → As a RULE, requires Surgical intervention & repair of the damage.

**Open skull #:** i.e there's overlying wound.
**Closed skull #:** No overlying wound.

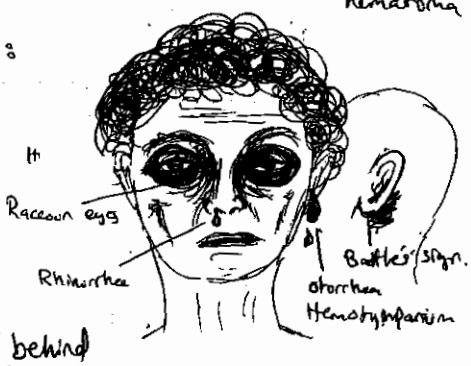
- Linear skull # → if closed → Left alone
  - if open → Requires wound closure
  - if comminuted or depressed → must be treated in the OR.

Hypovolemic shock can NOT happen from intracranial bleeding. Why? bcz there is NO enough space inside the head.

*Any pt w head trauma becomes unconscious → Do **CT** (look for intracranial hematoma)

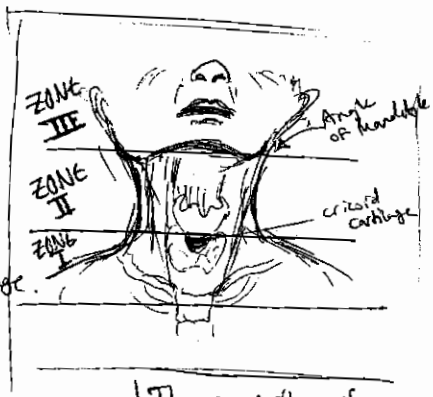
### *Signs of Basilar Skull #:

1. Raccoon eyes.
2. Clear rhinorrhea
3. Otorrhea
4. Hemotympanum
5. Battle's sign. (Ecchymosis behind the ear)



# NECK TRAUMA

Mech. of injury: Penetrating vs. Blunt




## ANATOMY OF THE NECK BY TRAUMA ZONES

- **ZONE III** : Angle of mandible & up
- **ZONE II** : Angle of mandible to the cricoid cartilage.
- **ZONE I** : Below cricoid cartilage

* These divisions help drive the diagnostic & therapeutic mgt decisions for Penetrating neck injuries.

The majority of the vital structures of the neck lie ⍵ in the **ANTERIOR** ▲

* **Penetrating injury** (Penetrating through PLATYSMA) MUST be further evaluated!.

**MgE** of Penetrating injuries (acc. to the zone)

ZONE III) Selective exploration
ZONE II) Surgical vs. selective exploration
ZONE I) Selective exploration

**SELECTIVE EXPLORATION**

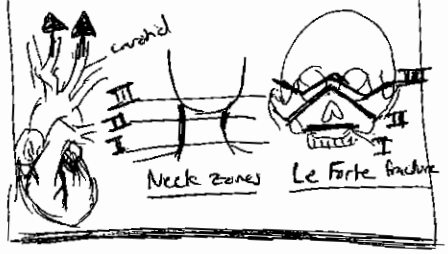
is based on diagnostic studies that include:
- A-gram
- CT A-gram
- Bronchoscopy
- Esophagoscopy

**Indic for surgical exploration**
- Expanding hematoma / Pulsatile hematoma
- Subcutaneous emphysema.
- Tracheal deviation.
- Δ in voice quality
- Air bubbling through the wound
- **SHOCK**
- Neurologic injury.

Penetrating injuries
- Vascular → ↑ common, life-threatening
- Nonvascular

**NOTES**
- Hyoid # indicates significant neck injury.
- C-spine injuries are much more common ⍵ BLUNT neck injury.

To remember the order of neck trauma zones & Le Fortes ⇒ in the direction of carotid blood flow:



# SPINAL CORD INJURIES

- Complete transection : Nth works (sensory / motor) below the lesion

- Hemisection (Brown-Sequard):
  - Typically from clean-cut injury (knife. blade)
  - Paralysis & loss of Proprioception distal to the injury side & loss of Pain Perception on the other side


- Ant. cord Synd.:
  - Typically seen in burst fractures of vertebral bodies.
  - Loss of motor fxns / pain & temp. sensation on both sides distal to the injury w/ Preservation of Vibration & Positional sense.

- Central cord Synd.
  - usually in elderly w/ forced hyperextension of neck.
  - Paralysis & burning Pain in upper extremities. w/ Preservation of motor fxn in lower extremities.

Dx

- For precise dx → Do MRI
- Corticosteroids immediately after injury may help.

# CHEST TRAUMA

- Rib Fracture

Can be deadly in elderly. why? bcz progression of Pain → hypoventilation → Atelectasis → Pneumonia!

Tt local nerve block & epidural catheter.

- Pericardial Tamponade

- Remember Beck's triad: Hypotension / Pulsus Paradoxus, ↑JVP, Muffled heart sounds.
  - It's life-threatening emergency!

Dx - Hx & PE
- Confirmed by U/S

Tt immediate decompression via needle Pericardiocentesis / Pericardial window / or thoracotomy w/ manual decompression

Clinically apparent tamponade may result from 60-100 ml blood

- **Blunt cardiac trauma**
  - Usually 2ry to motor vehicle collision / Fall from heights / Crush injury / Direct violent trauma.
  - Screening: ECG.
  - tt is focused on its complications (as arrhythmias)
- **Pneumothorax** (mentioned before)
  - Air in the pleural space.
- **Hemothorax**
  - Blood in the chest
  - >200 cc of blood must be present to be apparent on CXR
  - * massive hemothorax if >1.5 L of blood.
- **Flail chest**
- **Air embolism**
  - Suspected when sudden death occurs in a chest trauma pt.
- **Fat embolism**
  - Typical scenario- pt w multiple trauma (including several long bone #s) who developed petechial rashes in the axilla & neck, Fever, Tachycardia, ↓ Plt count.
  - PLUS full blown picture of resp. distress. w hypoxemia & bilateral patchy infiltrates on CXR.
  - (tt) Respiratory support.

| |
|---|
| 1/4 of hemothorax cases have an ass. Pneumothorax |
| 3/4 of hemothorax cases are ass. w extrathoracic injuries. |

# ABDOMINAL TRAUMA

Penetrating vs. Blunt

| PENETRATING | BLUNT | STUDIES |
|---|---|---|
| • Direct injury by gunshot / stab wound<br>• Injury from fragmentation of the bullet<br>• Indirect injury from the resultant "shock wave" | • Injury caused by direct blow<br>• Crush injury<br>• Deceleration injury.<br>• Decompression<br>• Shearing | • FAST<br>• CT scan<br>• DPL |

Gunshot wound → requires exploratory laparotomy for repair of intraabd. injury

Stab wounds → Individualized: If it's clear that Penetration has occured (not necessarily removing the bullet)
(Penetrating the viscera) or if unstable → Do exploratory laparotomy.

(9)

a Blunt trauma to abdomen → If signs of Peritoneal irritation → do exploratory laparotomy (Acute abdomen)

# TRAUMA STUDIES

## TRAUMA LABS

Hct won't be low after an acute massive hmg cuz there's no time to equilibrate

- CBC / Chemistry / Amylase / LFT / Lactic acid /
- Coagulation studies.
- **Blood typing & crossmatch** — Major: Donor RBC + Recipient Serum; Minor: Recipient RBC + Donor serum
- U/A (ABO Rh)

## IMAGING

CT is usually done ONLY if pt is STABLE

- ✱ Classic **blunt** trauma ER x-Rays → AP CXR, AP pelvis film.
- ✱ C-spine evaluation (after P/E) → X-Ray → CT (to r/o C-spine ✱)
- ✱ for eval. of C-spine LIGAMENTOUS injury → MRI & lateral flexion-extension C-spine films.
- ✱ To r/o thoracic aortic injury → Do CT of mediastinum looking for mediastinal hematoma ⓦ CTA → Thoracic arch aortogram (Gold Standard!)

**m.c Site** of thoracic aortic traumatic tear is → Just distal to the take-off of the Lt subclavian art.

**FINDINGS** on CXR that are suggestive of thoracic aortic injury:

- **WIDENED** mediastinum (m.c)
- Apical Pleural capping
- loss of aortic contour/Knob
- Depression of Lt main bronchus
- Tracheal deviation
- Pleural Fluid
- Elevation of Rt main bronchus

**FAST** (Focused Assessment ⓦ Sonography for Trauma)

- By U/S
- +ve if free fluid is demonstrated in the abdomen.

**4 Views** are utilized to search for free intraperitoneal fluid that collects & appears as hypoechoic areas on U/S:

1. RUQ (Morrison's Pouch): btw. liver & kidney.
2. LUQ (Splenorenal recess): btw. spleen & kid.
3. Pouch of Douglas: lies above rectum (Probe placed in suprapubic region)
4. Subxiphoid & Parasternal views — to look for hemopericardium.

**CT advantages**

- used for specific organ injuries.
- can be used for retroperitoneal injuries. very specific 98%

CT disadv.: misses diaphragm / GIT & Pancreas.

## Adv. of FAST

- Rapid bedside screening study.
- Noninvasive
- NOT time consuming
- 80–95% sensitive for intrabd. bleed.

## Disadv. of FAST

- Operator dependant.
- ↓ specificity for individual organ injury.

**DIAGNOSTIC TEST OF CHOICE for eval. of unstable Pt w blunt abd. trauma:**

**FAST**

## DPL (Diagnostic Peritoneal Lavage) — mostly for bowel injury detection (abdominal)

Indx
- Unstable Pt
- stable BUT U/S (FAST) not available.
- Prev. surgery / morbid obesity / coagulopathy / cirrhosis

**OPEN DPL:** Similar to open Port Placement in laparoscopic surgery (Peritoneal cavity is entered under direct vision) using the Hassan Port.

**CLOSED DPL:** A catheter is placed through the needle & advanced into the Peritoneum. (Placed below umbilicus)

If there's Pelvic #, Place the cath. ABOVE umbilicus to ~~close~~ avoid false +ve DPL

THEN, you aspirate for blood & if < 10 cc are aspirated → infuse 1 L of saline of Ringer Lactate.

THEN, drain the fluid (by gravity) & analyse.

NOTE "Grossly +ve" DPL → ≥ 10 cc blood aspirated.

### Indx of POSITIVE DPL

**Classic:**
- Blurred / cloudy Lavage fluid
- RBC ≥ 100,000 /mm³
- WBC ≥ 500 /mm³
- Lavage fluid (N.S/LR) drained from Chest tube/ Foleys / NGT

**Less common:**
- Bile Present
- Bact. Present
- Feces Present
- ↑ Amylase level.

**Celiotomy:** incision through the abdominal wall to gain access into the abdominal cavity.

NGT & Foley must be in Place before DPL!

### Adv. of DPL

- Done at bedside.
- Widely available.
- Highly sensitive for hemoperitoneum.
- Rapidly removed.

### Disadv. of DPL

- Invasive
- Risk of iatrogenic injury (<1%)
- ↓ Specificity (many false +ve)
- Does NOT evaluate retroperitoneum.

# Indx for EXPLORATORY LAPAROTOMY

- Abdominal trauma & unstable Pt.
- Evisceration
- Peritonitis.
- Diaphragmatic injury.
- Hollow viscus Perforation — free intraperitoneal air.
- Intraperitoneal bladder rupture (Dx: cystography)
- +ve DPL
- Surgically correctable injury diagnosed on CT.
- Removal of impaled weapon.
- Rectal Perforation.
- Gunshot wound injury in the abdomen.

## NOTES

- m.c solid organ injured ⓦ PENETRATING trauma → is LIVER
  m.c solid organ injured ⓦ BLUNT trauma → is SPLEEN & LIVER
  ⟹ BUT recent studies say that **LIVER** is the m.c in BOTH (Penetrating & Blunt.)
- "3-for-1" Rule : is that trauma Pt in hypovolemic shock acutely requires **3L** of crystalloid (LR) for every **1L** of blood loss.
- minimal UOP for an adult trauma Pt is **50** ml/hr
- Pancreatic injury is usually Penetrating.
- Indx of abd. CT in blunt trauma → NL V/S ⓦ abd. Pain/tenderness
- Indx of DPL / FAST in blunt trauma → Unstable Pt!
- Signs of laryngeal #:
  - Subcut. emphysema in neck
  - Altered voice
  - Palpable laryngeal #
- tt of rectal **Penetrating** injury:
  1. **Diverting Prox. colostomy**
  2. Closure of Perforation
  3. **Presacral drainage.**
- Bleeding from Pelvic # is m.c caused by **VENOUS** (85%) more than arterial.

- tt of extensive irreparable biliary / duodenal / ± Pancreatic injury is → Whipple.

- tt of Penetrating injury to the COLON — If in shock → resection & colostomy
  If stable → Ivy anastomosis / repair

- tt of small bowel injury → Ivy closure or resection & Ivy anastomosis.

- tt of minor Pancreatic injury → Drainage

- tt of massive tail of Pancreas injury → Distal Pancreatectomy (± splenectomy)

- "Lethal Triad" = "ACH" Think ACHe!
  1) Acidosis
  2) Coagulopathy
  3) Hypothermia

- What can present after BLUNT trauma w/ neurological deficits & NL brain CT?
  - Diffuse Axonal Injury (DAI)
  - Carotid art. injury.

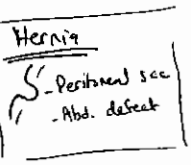
Linda Griffith
The End.

# HERNIAS

Source: Recall 127

## DEFINITION

It's the Protrusion of Peritoneal sac through a musculoperitoneal barrier (Abdominal wall); a fascial defect.




## INCIDENCE

Overall 5-10%

- Indirect hernia 50% ♂
- Direct hernia 25% ♂
- Femoral hernia 5% ♀
- Obturator hernia less ♀

## RISK FACTORS

**↑Intraabdominal Pressure:**

- Chronic constipation
- Straining on urination (Prostate enlargement)
- Chronic cough (smoker, asthmatic Pt, COPD)
- Obesity / Pregnancy / Ascites.

*Age (Elderly have weak abd. muscles)

Indx of ttt → To AVOID complications!

**Small hernia defects are more dangerous than large hernia defects.**

## COMPLICATIONS

- Incarceration / Strangulation
- Bowel Necrosis
- Small bowel obstruction
- PAIN

---

Terminology

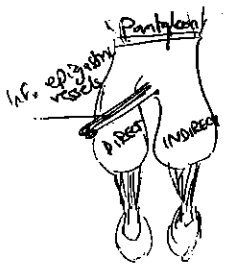
* **Reducible**: The ability to return the displaced organ or tissue/hernia contents to their usual anatomic site.
* **Incarcerated**: Swollen or fixed within the hernia sac (incarcerated = imprisoned) ⇒ May cause intestinal obstruction (i.e. an irreducible hernia)
* **Strangulated**: Resulting in ISCHEMIA → S&S of ischemia & intestinal obst. or bowel necrosis (THINK: Strangulated = choked)

**Complete** : Hernia sac & its contents / Protrude all the way through the defect.

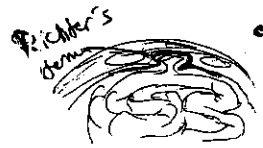
**Incomplete** : Defect Presents Without sac or contents Protruding completely through it.

# TYPES OF HERNIAS

- **Indirect Inguinal** : Inguinal hernia lateral to Hasselbach's Δ
- **Direct Inguinal** : Inguinal hernia Within Hesselbach's Δ
- **Incisional Hernia** : Hernia through an incisional site (m.c.c is wound infxn)
- **Femoral Hernia** : Hernia medial to femoral vessels (UNDER inguinal ligament)
- **Obturator Hernia** : Hernia through obturator canal (♀ > ♂)
- **Umbilical Hernia** : Hernia through the umbilical ring
  - In adults, it's ass. w/ obesity/Pregnancy/ & Ascites.
- **Hiatal Hernia** : Hernia through esophageal hiatus. (Types < Sliding, Paraesoph)
- **Internal Hernia** : Hernia into or involving intraabdominal structure.
- **Spigelian Hernia** - Hernia through the linea Semilunaris (or Spigelian fascia)
  AKA Spontaneous lat. ventral hernia
  (Spigelian ≡ Semilunaris)
- **Sliding Hernia** : Hernia sac partially formed by the wall of a viscus (bladder / cecum)




- **Pantaloon Hernia** : Hernia sac exists as BOTH a direct & indirect hernias straddling the inf. epigastric vessels & Protruding through the floor of the canal as well as the internal ring (2 sacs separated by inf. epigastric vessels)
- **Parastomal Hernia** : Hernia adjacent to an ostomy (e.g. Colostomy)
- **Ventral Hernia** : Incisional hernia in the ventral abd. wall.




- **Richter's Hernia** : Incarcerated / Strangulated hernia involving only one sidewall of the bowel, which can spontaneously reduce → gangrenous bowel & perforation w/in the abdomen Without sign of obstruction!
  - Gangrene Without obstruction!
- **Diaphragmatic Hernia** → (Types) : **Bochdalek's Hernia** : Hernia through Post. diaphragm
  **Morgagni's Hernia** : Ant. Parasternal hernia
- **Amyand's Hernia** : Hernia Sac containing a ruptured Appendix
  (Amyands ≡ Appendix) (2)

# LAYERS OF ABDOMINAL WALL

**S**kin
**S**ubcutaneous fat
**S** Camper's fascia
} 3 S's

External oblique
Internal oblique
**T**ransversus abdominus

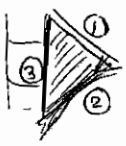
Transversalis fascia
Peritoneal fat
Peritoneum.

* All 3 muscle layer aponeuroses form the ant. rectus sheath & the post. rectus sheath being deficient below the arcuate line.

**Conjoint tendon**
It's Apponeurotic attachment of the "conjoining" of the internal oblique & transversus abdominus to Pubic tubercle.

## * NOTES

- Boundaries of Hesselbach's △ : ① Inf. epigastric vessels.
  ② Inguinal ligament (Poupart's)
  ③ Lateral border of rectus sheath




* Floor consists of internal oblique & the transversus abdominus muscle.

## * Intraop. Notes

* What's the name of subcut. vein that is ligated? Superficial epigastric vein.

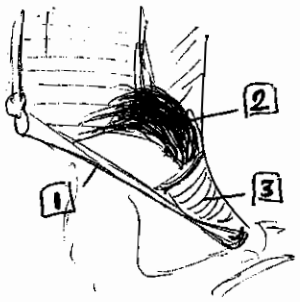
* What happens if you cut the ilioinguinal n.?
  Numbness of inner thigh or lateral scrotum (usually goes away in 6 m.)

* Some surgeons deliberately cut the ilioinguinal n. to remove the risk of entrapment & Postop. Pain.

* Hernia sac ~~made of contents~~ — Direct → Peritoneum ~~(fascia)~~
  Indirect → Patent Processus vaginalis.

## * Inguinal Anatomy

1. Inguinal Ligament
2. Transversus aponeurosis
3. Conjoint tendon.



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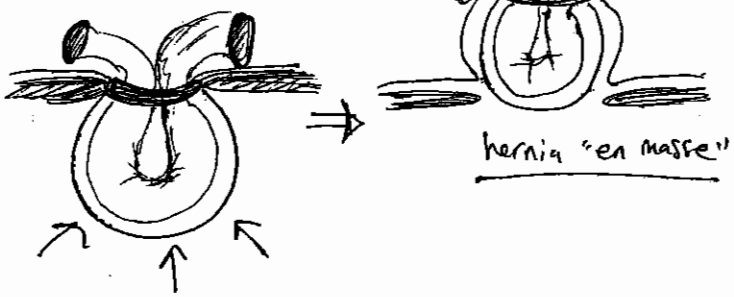
* What is reducing a hernia "en masse"?

It's reducing the hernia contents of hernia sac.



hernia "en masse"

* Paraumbilical Hernia (above umbilicus) causes a crescent umbilicus
* Paraumbilical hernia is at more risk of strangulation than umbilical hernia due to smaller orifice!

The End.

# INGUINAL HERNIA

BUSHRA TBAKHI

## ANATOMIC LOCATION

- Indirect Inguinal Hernia
- Direct Inguinal Hernia
- Femoral Hernia

Inguinal and Femoral Hernias are in the inguinal region

Patients may complain of bulge, swelling, pain, cosmetic apperance.

**Incarceration vs. Strangulation:** Incarcerated: hernia is irreducible, but there are no local signs. Strangulation has local signs of inflammation (redness, induration, warmth, pain)

DDX of Hernia: (Inguinal)

- Hydrocele, Lymph nodes, Saphena Varix, Testicular Torsion, Testicular Tumor, femoral artery aneursym, psoas abscess, varicocele, undescended testis, epidydmo orchitis, lipoma

## INDIRECT INGUINAL HERNIA

- **Most common type** of hernia in males and females
- Protrude at the internal inguinal ring
  - The origin of the hernia sac is located **lateral to the inferior epigastric artery.**
  - Indirect hernias develop **more frequently on the right** in both sexes
  - Most indirect inguinal hernias in adults are **congenital**, even though they may not be clinically apparent in the neonatal period or childhood

patent Processus vaginalis)

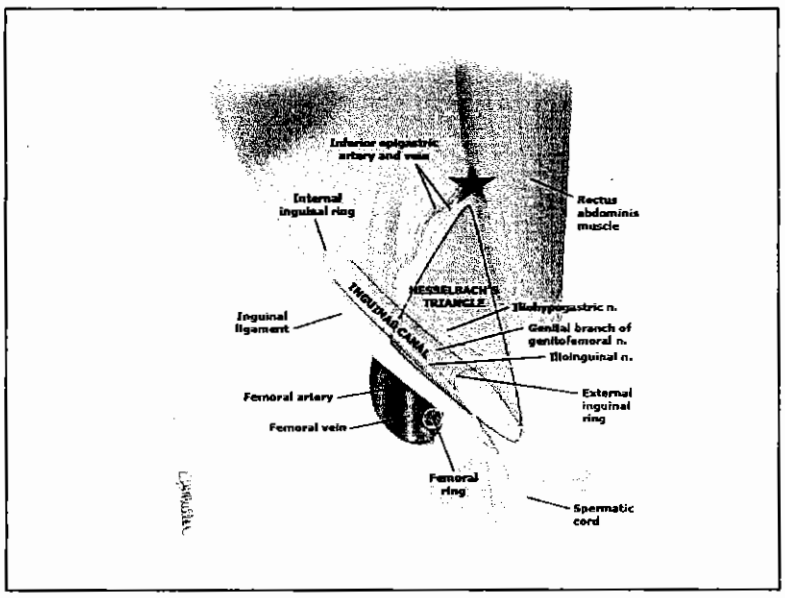
- **Deep (Internal Inguinal) ring** is the site where the spermatic cord in the males and the round ligament in females exit the abdomen. The ring is found **1-2 cm above** the midway point of the **inguinal ligament**. The inguinal ligament stretches between the **ASIS** (anterior superior iliac spine) and **pubic tubercle.**

- ***Note:*** **The midinguinal point is different.** That is from ASIS to pubic **symphysis**, and that is where the femoral pulse is felt.

- **Spermatic cord contents:**
  - **3 arteries:** Cremasteric artery, Artery to VAS, Testicular artery, (CAT)
  - **3 nerves:** genital branch of genitofemoral nerve, sympathetic nerves **note:** Ilioinguinal nerves runs above the cord in the canal but not in the cord itself
  - **3 layers of fascia:** external spermatic, cremasteric, and internal spermatic fascia. Per ext. oblique muscle from internal oblique from transversus abdominis
  - **3 others:** pampiniform plexus of veins, vas deferens, lympatics,

- More hernias on the right: which is thought to be due, in males, to a later descent of the right testicle, and in females, by the asymmetry of the female pelvis.
- They are usually reducible, and if the thumb is kept on the deep inguinal ring and the patient is asked to cough, an indirect hernia will not appear. However, a direct

- Inguinal ligament (Poupart ligament) is derived from external muscle aponeurosis.




A helpful mnemonic to remember **inguinal canal walls: MALT (2M, 2A, 2L, 2T)**
*Starting from superior, moving around in order to posterior:*

**Superior wall (roof): 2 Muscles:**
internal oblique Muscle
transverse abdominus Muscle

**Anterior wall: 2 Aponeuroses:**
Aponeurosis of external oblique
Aponeurosis of internal oblique

**Lower wall (floor): 2 Ligaments:**
inguinal Ligament
lacunar Ligament

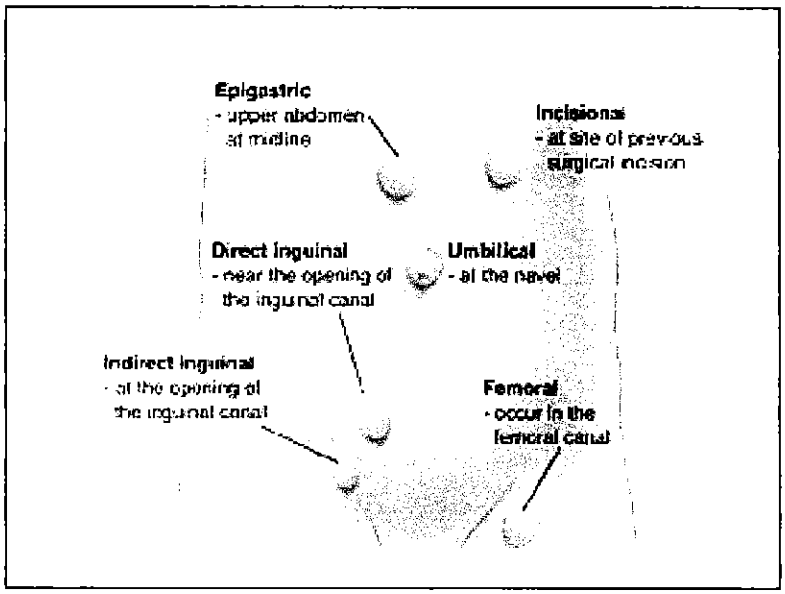
**PosTerior wall: 2Ts:**
Transversalis fascia
conjoint Tendon

## DIRECT INGUINAL HERNIA

- Direct inguinal hernias protrude **medial** to the inferior epigastric vessels within **Hesselbach's triangle**
- Direct inguinal hernias occur as a result of a weakness in the floor of the inguinal canal.

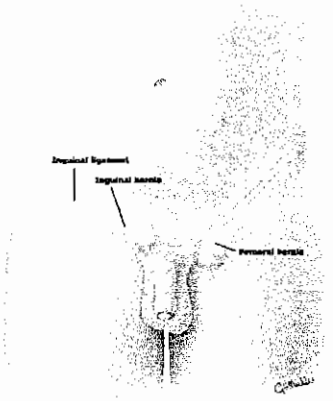
  This weakness appears to be due to connective tissue abnormalities in many cases, although some may occur due to deficiencies in the abdominal musculature resulting from chronic overstretching or injury.

- **Hesselbach's triangle borders: Formed by the inguinal ligament inferiorly, the inferior epigastric vessels laterally, and the rectus abdominus muscle medially**
- **Direct hernias are usually bilateral.**
- **If they can be reduced, they reappear with cough impulse if the thumb is kept on the deep inguinal ring.**




-Paraumbilical hernias produce a crescent-like or smiley-face umbilcius shape

# FEMORAL HERNIAS




RF
- ♀
- Pregnancy
- exertion

-Femoral Hernias appear below the inguinal ligament, and are more common in females than males..
NOTE: Overall, inguinal hernias are more common in BOTH females and males. So the Most Common hernia in females is still inguinal hernia. But, pts with femoral hernias are more likely to be female.

**-Femoral canal:**
superiorly: inguinal ligament,
medially: lacunar ligament,
base: pectineal ligament,
laterally: femoral vein

FeMoral (FM) ≡ Femoral hernia is Medial to Femoral vessels

* 85% are ♀

-Femoral hernias are more likely to have complications than inguinal hernias, because the borders of the femoral canal are rigid. (only the femoral vein is a compressible wall of the canal, the rest of the borders are ligaments)

* The repair of femoral hernia is usually McVay (Cooper's ligament repair)

## MANAGEMENT

- "Asymptomatic" hernias can be managed with watchful waiting in pts who are high risk for surgery. Young patients should be repaired, and infants should be repaired immediately.
- Trusses are the only non-surgical option, strongly discouraged
- **Surgical Indications:**
  1. **Elective Surgery: Uncomplicated Hernia** but symptomatic (painful, cosmetic appearance bothersome, interrupting lifestyle..)
  2. **Emergent Surgery: Complicated Hernia** (Strangulated, Bowel Obstruction..)

-Watchful waiting vs. Repair to prevent complications: individualized according to patient
-No truly "asymptomatic" hernias. Unless high risk for operations, all hernias should be repaired to prevent complications.
-Trusses (Hernia belts) is a device with a metal/hard plastic plug that is positioned over the hernial defect. Not enough data to prove its benefit, but may potentially lead to harm if it impinges on hernia contents. Proglonged tissue pressure can lead to atrophy of the spermatic cord or fusion to the hernia sac. Atrophy of the fascial margins can occur which complicate surgical repair.

## HERNIA REPAIR

- 1. **Herniotomy**: ligating and cutting the sac. Only done in peds patients, because they have a patent processus vaginalis AKA: High ligation
- 2. **Herniorapphy**: hernia repair by approximation and suturing of ligaments and tissues. These produce **tension**, and alter anatomy. Largely abandoned due to high reccurence.
- 3. **Hernioplasty**: hernia repair using a mesh. Using a mesh allows for repairs to be tension free, as tissues do not need to be approximated. May be done Open or Lap (-plasty refers to use of prosthesis)

- **In Peds patients it is due to a patent processcus vaginalis, so the sac must be cut and ligated. It is an emergent surgery in pediatric patients due to high risk of complications.**

## HERNIA REPAIR

1) **OPEN Repair:**
   a) Non-Mesh Primary Tissue Approximation
      Examples: Bassini, McVay, Shouldice
      These 3 have been largely abandened, due to the higher reccurence rates and pain. (Herniorapphy)
   **b) Tension-Free Repair With Mesh**
      Examples: **Lichtenstein**, Plug and Patch (Plug instead of mesh)

2) **LAPAROSCOPIC Repair (both with mesh)**
   1) **TEP** (Totally ExtraPeritoneal)
   2) **TAPP** (TransAbdominal ProPeritoneal)

- **Tension Free Repair with mesh**: although hernia surgeries are classically considered "clean" (classification 1), the use of Mesh, a foreign body, makes it class 1d, and prophylactic antibiotics should be given 1 hour pre-op, because the control of infection with prosthesis, such as mesh, is difficult, and sometimes may necessitate removal of prosthesis.
- Tension free repair with mesh and Laparoscopic repairs are all considered hernioplasties.
- **LAP Repair indications:**
  - Bilateral Hernia
  - Recurring Hernia
  - Minimal post-op time needed

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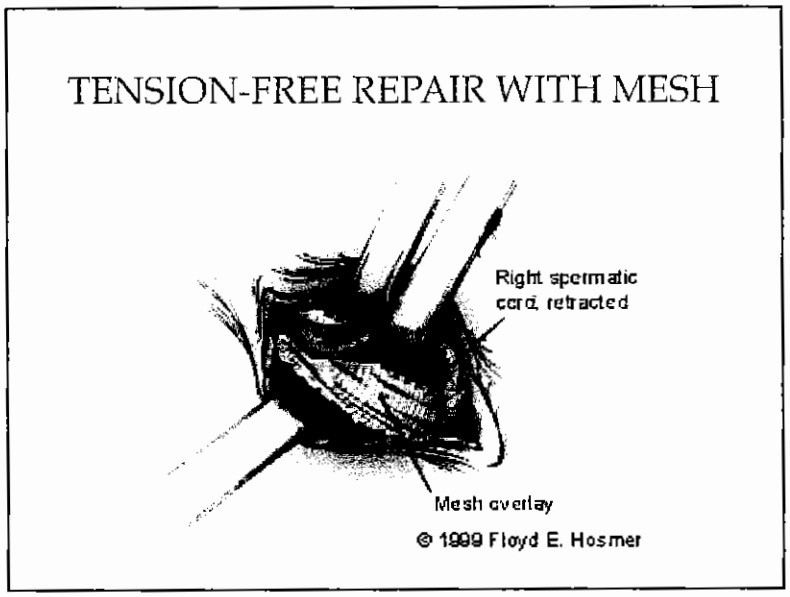


Open mesh repair. Mesh is used to reconstruct the inguinal canal. Minimal tension is used to bring tissues together.

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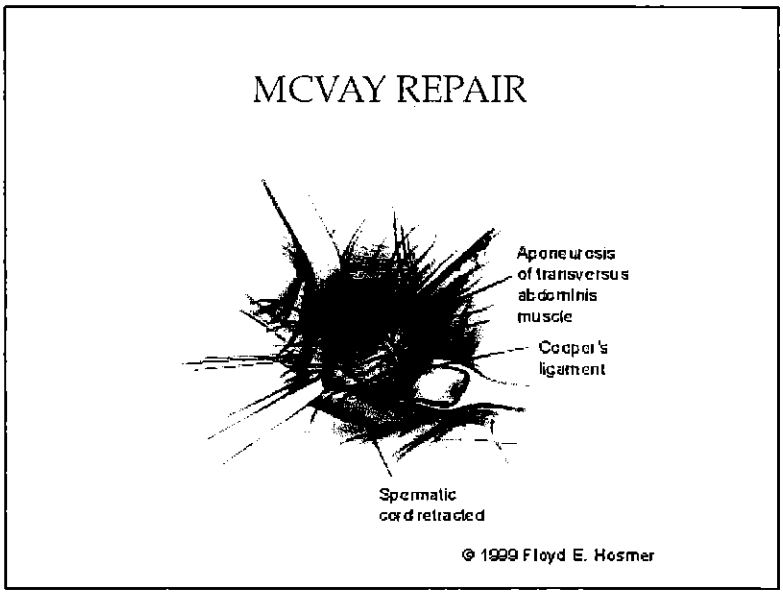


McVay
NOT DONE ANYMORE Open anterior repair. This repair reconstructs the inguinal canal without using a mesh prosthesis.

# INGUINAL HERNIA

## Physical exam



✓ Exposure : from umbilicus to knees

✓ Start when patient is standing to asses true size

✓ Know your objectives :

1. Confirm Lump is a hernia
2. Differentiate inguinal from femoral hernia
3. If inguinal : direct / indirect?

> **Note :**
> - inguinal hernia : Above & medial the pubic tubercle
> - femoral hernia : below & lateral to pubic tubercle

### Inspection

- Look for scars, skin changes
- If hernia is obvious : start examining it :
- If not : Ask patient to cough & observe for expansile cough impulse.
- Ask patient to reduce hernia/or attempt to reduce it by applying gentle pressure in direction of inguinal canal, if it's irreducible, then its ← not a hernia
  ← incarcerated hernia
- comment if lump is confined to inguinal region or descends to scrotum

### Palpation

- Begin by defining anatomy
  - Note : * **mid-inguinal point** : point halfway along aline joining ASIS and midline (pubic symphysis) = location of femoral a.
    * **midpoint of inguinal ligament** : halfway along inguinal ligament
      - inguinal ligament : between pubic tubercle & ASIS
      - = Deep inguinal ring

So,
* Femoral pulse is located medial to deep to inguinal ring.

- palpate the pubic tubercle & ASIS

** How to locate the pubic tubercle?

Palpate midline downward from umbilicus until reaching symphysis pubis then move laterally until reaching pubic tubercle

- place one finger on each landmark & demonstrate that inguinal lig run between the two
- Ask patient to cough
  - → show that hernia is above this line → Inguinal hernia & medial to pubic tubercle
  - → demonstrate that it has a palpable cough impulse

ISS → Direct / indirect?
- If hernia is reducible, reduce it back
- Place **1** finger above midpoint of inguinal ligament
- ask pt to cough
- If hernia does not appear ⇒ INDirect hernia
- If hernia reappears ⇒ Direct hernia

* complete exam by examining scrotum ± contralateral groin

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**Notes!**

- DDX for lump in groin:
  L-SHAPE
  Lymph node / Lipoma of cord
  Saphena Varix / skin lesion
  Hernia: Inguinal / femoral
  Aneurysm of femoral a.
  Psoas abscess or bursa
  Ectopic / undescended testicle

- to differentiate a hernia from a scrotal lump ⇒ examine upper edge
  - If you can get above it (i.e feel upper edge using your thumb and index finger) ⇒ scrotal swelling
  - If you <u>cannot</u> get above it ⇒ hernia

<u>But</u>: in peds: it might also be an encysted hydrocele of cord.

The End