Intensive Care Unit Protocol 2014

Index

43	Hyperkalemia
44	Hypomagnesmia
45	Hypermagnesmia
46	Hypophosphatemia
47	Hyperphosphatemia
48	Hypocalcemia
49	Hypercalcemia
	Ventilatory support
50	Indications of ventilatory support
51	Initiation of ventilatory support
52	Ventilator settings
54	Tailoring of ventilatory support
56	Non-invasive ventilation
57	Independent lung ventilation
58	Troubleshooting during ventilatory support
59	Weaning
60	Extubation
61	Prone ventilation
	Nutritional support
62	Nutritional screening
63	Nutritional requirement
64	Enteral nutrition
68	Parenteral nutrition
69	Nutritional assessment
	Infection in intensive care
71	New onset fever
73	Ventilator associated & Hospital acquired & Health care associated pnemonia
74	Catheter related blood strem infection
75	Necrotizing fascitis
76	76. Bacterial meningitis
77	Infective endocarditis
78	Clostridium defficille infection
79	Febrile neutropenia
80	Fungal infection
	Trauma in intensive care
81	ICU management of trauma pt
82	Traumatic brain injury
83	Traumatic spinal cord injury
84	Blunt chest trauma
85	Moderate & severe 85. Thermal injury
86	Smoke inhalation
87	Aortic rupture
88	Submersion injuries
89	Rabdomylosis
90	Unstable pelvic fracture
	Surgical emergencies
91	Acute pancreatitis
92	Intra-abdominal hypertension

	Obstetric emergencies
93	Acute fatty liver of pregnancy
94	Postpartum hge
95	Amniotic fluid embolism
96	Pregnancy induced hypertension
97	Help syndrome
98	Ovarian hyperstimulation syndrome
99	Cardaic arrest
	Drug intoxication
100	General approach to intoxicated patient
102	Organo-phosphorus poisoning
103	**General approach to critical ill patient**
	Cardio-vascular disorders
105	General approach to shocked patient
106	Hypovolemic shock
107	Septic shock
108	Cardiogenic shock
109	Adrenal shock
110	Anaphylactic shock
111	Neurogenic shock
112	Tension pneumothorax
113	Cardiac tamponade
114	Tachycarythmias
115	Braydyarrythmias
116	Hypertensive emergencies
117	Acute decompensated HF
118	General approach to patient with chest pain
120	Acute coronary syndrome
122	Aortic dissection
	Respiratory disorders
123	General approach to a patient with respiratory distress
124	Hypoxemia
125	Acute asthma exacerbation
126	Acute chronic obstructive lung disease exacerbation
127	Acute respiratory distress syndrome
128	Fat embolism syndrome
129	Air embolism
130	Hemoptysis
131	Aspiration pnemonia
	Neurological disorders
132	General approach to a patient with disturbed conscious level
133	New onset seizures
134	Status epilepticus
135	Subarachnoid hemmorrage
136	Brain abscess
137	Viral encephalitis
138	Brain death

Appreviations

Ab	Antibiotic	**Intub.**	**Intubation**
abd.	Abdomen	**ITP**	Idiopathic thrombocytopenic purpura
ABGs	Arterial blood gases	**IV**	Intravenous
ACS	Acute coronary syndrome	**IVIM**	Intravenous immunoglobulin
ACTH	Adrenocortical tropic hormone	**JVP**	Jugular venous pressure
ADH	Antidiuretic hormone	**K**	Potassium
ADHF	cute decompensated heart failure	**L**	Liter
AED(s)	nti-epileptic drug(s)	**LA**	Local anesthetic
AF	Atrial fibrillation	**Lab.**	Laboratory
AFE	Amniotic fluid embolism	**LFT**	Liver function test
AFLP	Acute fatty liver of pregnancy	**LMWH**	Low molecular weight heparin
ALI	Acute lung injury	**LP**	Lumbar puncture
ALS	Advanced life support	**LR**	Lactated ringer
ARDS	Acute respiratory distress syndrome	**LV**	Left ventricle m (s) = month (s)
ATN	Acute tubular necrosis	**m (s)**	
AV	Atrio-ventricular	**MAP**	Mean airway pressure
AVP	Vasopressin	**MDCT**	Multi-detector CT
AVRT	Atrio-ventricular re-entrant tachycardia	**MDRO**	Multi drug resistant organism
BAL	Broncho-alveolar lavage	**Mg**	Magnesium
BCAA	Branched chain aminoacids	**MI**	Myocardial infarction
BE	Base excess	**MILS**	Manual inline stabilization
BIPAP	Bi-level positive airway pressure	**MR**	Mitral regurge
bl pr	Blood pressure	**MRI**	Magnetic resonant imaging
Bl.	blood	**MRSA**	Methecilin resistant staph aeurus
BNP	Natritic peptide	**MV**	Mechanical ventilation
BSL	Blood sugar level	**N&V**	Nausea and vomiting
BVM	Bag valve mask	**N2**	Nitrogen
BW	body weight	**Na**	Sodium
C&S	Culture & senstivity	**NDMR**	Non-depolarizing muscle relaxant
Ca	Calcium	**NGT**	Naso-gastric tube
CABAG	Coronary artery bypass graft	**NIF**	Negative inspiratory force
CAP	Community acquired pnemonia	**NIV**	Non-invasive ventilation
CBC	Complete blood count	**NMB**	Neuro-muscular blocker
CBF	Cerebral blood flow Colony forming unit	**NON-STEMI**	Non-ST segment elevation acute coronary syndrome
CFU	Central	**Ns**	Normal saline
Cent. CHF	Congestive heart failure	**NSAIDS**	Non -steroidal anti-inflammatory drugs
CI	CI = contraindicated	**OHSS**	Ovarian hyperstimulation syndrome
CMV	Cytomegalo-virus	**PCC**	Prothrombin complex concentrate
CNS	Central nervous system	**PCI**	Percutaneous coronary intervention
CO	Carbon monoxide	**PCWPs**	Pulmonary capillary wedge pressure
CO-Hgb	Carboxyhemoglin	**PD**	Peritoneal dialysis
Conc.	Concentration	**PE**	Pulmonary embolism
COP	Cardiac output	**PEEP**	Positive end expiratory pressure

COPD	Chronic obstructive lung disease	**PEFR**	Peak expiratory flow rate
CPAP	Continuous positive airway pressure	**Periph.**	peripheral
CPK	Creatinine phosphokinase	**PFT**	Pulmonary function test
CPP	Cerebral perfusion pressure	**PhE**	Physical examination
CPR	Cardio-Pulmonary resuscitation	**PIH**	Pregnancy induced hypertension
CRBSI	Catheter related blood stream infection	**PND**	Paroxysmal nocturnal dyspnea
CRT	Capillary refill time	**PO**	Post-operative
CS	Cesarean section	**PO4**	Phosphorus
CSF	Cerebro-spinal fluid	**PPT**	Partial thromboplastin time
CT	Computerized tomography	**Pr**	Pressure
CT- PA	Computerized tomography – pulmonary angiography	**PRBC**	Packed red blood cell
CVC	Central venous catheter	**PSVT**	Paroxysmal supra-ventricular tachycardia
CVP	Central venous pressure	**PT**	Prothrombobin time
CXR	Chest X-ray	**Pt(s)**	Patient(S)
d(s)	Day(s)	**ptn**	Protein
DANR	Order of do not attempt resuscitation	**PTS**	Post-traumatic seizures
DC	Discontinue	**RBCs**	Red blood cells
DDAVP	Desmopressin	**Resusc**	Resuscitation
Defib.	Defibrillation	**RF**	Respiratory failure
DIC	Disseminated intravascular coagulation	**RL**	Ringer lactate
Dis.	Disease	**RR**	Respiratory rate
DKA	Diabetic keto-acidosis	**RRT**	Renal replacement therapy
DLT	Double lumen tube	**RSI**	Rapid sequence induction
DVT	Deep venous thrombosis	**RWMAs**	Regional wall motion abnormalities
ECF	Extra-cellular fluids	**s**	Second
ECG	Electrocardiogram	**S**	S yndrome
EDD	Esophageal detector device	**S bl pr**	S ystolic blood pressure
EEG	Electro-encephalogram	**S. aureus**	Staph aureus
EN	Enteral nutrition	**SC**	Subcutaneous
ETT	Endotracheal tube	**SLE**	Systemic lupus erthermatosis
FAST	Focused assessment of sonography of trauma	**ST infection**	Soft tissue infection
FB	FB = foreign body	**STEACS**	ST elevation acute coronary syndrome
FES	Fat embolism syndrome	**STEMI**	ST segment elevation myocardial infarction
FFP	Fresh frozen plasma	**SVT**	Supra-ventricular tachycardia
FiO2	Fractional inspired O2 concentration	**TAD**	Tricyclic antidepressant drug
FOB	Fiberoptic bronchoscope	**TB**	Tuberculosis
FOI	Fiberoptic intubation	**TBI**	Traumatic brain injury
FVC	Forced vital capacity	**TBN**	Total parenteral nutrition
G	Gauge	**TBSA**	Total body surface area
GBS	Guillian barre syndrome	**TCD**	Trans-cranial doppler
GCS	Glasgow coma scale	**TEE**	Trans-eseophgeal eccho
GFR	Glomerular filtration rate	**Temp.**	Temperature
GI	Gastro-intestinal	**TMJ**	Tempo-mandibular joint
HAP	Hospital acquired pneumonia	**TPN**	Total parentral nutrition

HB	Heart block	**TSCI**	Traumatic spinal cord injury
HBO	Hyperbaric oxygen	**TTE**	Trans-thoracic echo
HCAP	Health care associated pneumonia	**TTE**	Tte = transthoracic echo
HD	Hemodialysis	**TTJV U**	Transtracheal jet ventilation Unit
Hge	Hemmorrage	**UF**	Ultra-filtration
HF	Heart failure	**UFH**	Unfractionated heparin
Hgb	Hemoglobin	**UOP**	Urine output
HIT	Heparin induced thrombocytopenia	**US**	Ultrasound
HIV	Human immune-defiency virus	**UTI**	Urinary tract infection
HOB	Head of bed	**VAP**	Ventilator associated pneumonia
HPA	Hypothalmic pituitary axis	**Vent**	Ventilation
HPF	High power field	**VF**	Ventricular fibrillation
Hr (s)	Hour(s)	**VILI**	Ventilator induced lung injury
HR	Heart rate	**VQ**	Ventilation/ Perfusion
HUS	Hemolytic uremic syndrome	**VSD**	Ventricular septal defect
IABP	Intra-aortic balloon counterpulsation	**VTE**	Venous thromboembolism
IAH	Intra-abdominal hypertension	**w (s)**	Week (s)
IAP	Intra-abdominal pressure	**+ ve**	Positive
ICP	Intracranial pressure	**- ve**	Negative
ICU	intensive care unit	**1ry**	Primary
IHD	Ischemic heart disease	**2ndry**	Secondary
IJ	Internal jugular	**1st**	First
ILV	Independent lung ventilation	**2nd**	Second
IM	Intramuscular	**3rd**	Third
Inf.	Infection		
INR	**International normalized ratio**		

Protocol overview

Protocol overview

Rating Scheme for Strength of Evidence

Class I

- Prospective randomized controlled trials

Class II

- Clinical studies in which data was collected prospectively and retrospective analyses that were based on clearly reliable data

Class III

- Studies based on retrospectively collected data.
- Evidence used in this class includes clinical series and database or registry review

- **The protocol based on**
  - **Evidence based practice**
  - Unit specific practice
- This protocol is confined mainly to adult critically ill pts
- The main references of the protocol are
  - ***Uptodate*** "available off line on ICU computer
  - ***ALS***
  - ***ATL***
  - ***Espen&Aspen*** guidelines for nutritional support
  - ***ICU book "paulmarino"***
  - ***AnesthesiadepartmentDA&MVmanual***
  - ***Nice guidelines***
  - ***Surgical critical care net***

- 
  - Means, the resident on call must consult
  - If there is no response or a clear plan, the ICU consultant on call must be informed
- 
  - Means, the resident needs a final decision from the ICU consultant on call or assistant lecturer in some cases
- 
  - **-** Means you should write an **order form**
-
  - **-** Means you should write a **sheet**

Rating Scheme for Strength of Recommendations

Level I

- The recommendation is convincingly justifiable based on the available scientific information alone
- This recommendation is usually based on Class **I** data, however, strong Class **II** evidence may form the basis for a Level I recommendation, especially if the issue does not lend itself to testing in a randomized format
- Conversely, low quality or contradictory Class I data may not be able to support a Level I recommendation

Level II

- The recommendation is reasonably justifiable by available scientific evidence and strongly supported by expert opinion
- This recommendation is usually supported by Class **II** data or a preponderance of Class **III** evidence

Level III

- The recommendation is supported by available data but adequate scientific evidence is lacking
- This recommendation is generally supported by Class **III** data
- This type of recommendation is useful for educational purposes and in guiding future clinical research

ICU rationale

Resident orientation

- Be sticky to**ICU board** for:
  - Consultant on duty rota.
  - Any new events
- Know on call of various departments from the uptaded**on- call file**

Timing
- 3 – 4 ws before rotation beginning

Rotation
- 3 – 4 months
- 3 residents "10 ds duties / month (30 ds month), 11 duties / month (31 ds month) for one of the residents"

Attendance
- At**9.5** am till 12 am of the next d
  - **9.5**-**12**am : ICU round

ICU character
- It is an emergency ICU
- **25** beds 'including 2 for isolation" ----**15** only working at the present time --- nurse/bed 1:2

Pts admission criteria
- **Priority I:**
  - Poly-trauma critically ill pts
  - Peri-operative emergency critically ill pts
- **Priority II:**
  - Any critically ill pts need MV
- **Priority III:**
  - Any pts in need of hemodynamic support
  - *See details in pt admission criteria p4"*

ICU equipment
- **Monitors**
  - ECG including ST segment analysis
  - Pulse oximetry
  - Capnometry
  - Non-invasive bl.pr.
  - Invasive bl. Pr.
  - Non-invasive COP
  - EEG
- **Center station** including recorderfor documentation
- **Ventilators**
  - Bennet 840,7200
  - DraggerEvita 4, safina
  - Semiens I, S, 300. 900
  - Avea
  - High frequency oscillator
- **Resuscitation cart**
  - Resusc. drugs
  - Defibrillator "bi-phasic"
  - Pacing "trans-cutaneous"
  - Airway management including LMAs, and laryngeal tube, ETTs, oropharyngeal airways, and bag valve mask
  - Methods of O2 administration including non-rebreathing mask
  - Interosseous needle, peripheral, and central iv sets, catheters
  - Pressure infusor
  - Resuscitation board
- **Airway management cart**
  - Drugs"Xylocaine gel, spray, IV, Atropine , EP
  - Primary intubation attempts"Mcoy ,Miller, Macintoch 5, oro-pharyngeal and naso-pharyngealairways, stylet, and gum elastic bougie
  - Intubation alternatives "retrograde set, ILMA,airQ"
  - Cannot intubate cannot ventilate " Cricothyrotomy set, standard LMAs, supreme, and I-gel"
  - Bag valve mask, cuff pr manometer, suction catheters, and ETT
- **Portable X-ray**
- **Trans-thoracic ECCHO "TTE"**
- **Hemo-filtration unit**
- **Portable US**with a superficial probe
- **Fiberoptic bronchoscope**
- **ABG analysis**
  - Osmolarity
  - Chloride, Na, Ca ,and k
  - Hematocrit
  - Oxygenation indicies
- **Transport trolley** including
  - Portable ventilator, suction, defib. monitor, oximetry, syringe pump, and airway &resusc. bag

Drugs
- In the refirigrator "Streptokinase, Minirin, Esmeron, succinylcholine, propofol"
- In the resusc. cart "inderal, digoxin, isoptin, cordarone, adrenaline, noradrenaline, dopamine, dobutrex, and nitroglycerine"

Flow sheets
- Admission &Progression sheets
- 2ndrytrauma survey sheet
- Ventilator flow sheets
- Discharge sheet
- Cardiac arrest sheet
- Problem list sheet
- Lab sheet
- ICU &Referal plan & Consultation sheets
- Order form
- Peri-operative sheet

Learning aids
- Android mobile phone with **Medscapeoffline** approach
- **Computrizedbooks** "Text book of ICU, ICU book"
- Off-line**UPTODATE**
- **Handbooks**
  - Text book of ICU
  - ICU book& ICU secrets
  - Booklet of ICU protocols
  - ALS, PELS, ATLS
  - Anesthesia department DA manual
  - MV manual

Assess-ment
- Protocol application
- Continuous knowledge & decision making assessment during ICU rounds
- Practical skills "central line insertion, arterial line insertion, CPR, airway management"
- Communication skills
- History taking & case presentation

Admission principles

Admission criteria

Priority 1:

1. **Trauma pt**

Poly-trauma pt with:

- Hemodynamic instability
- Need for MV for any reason
- In need of O2 therapy "high FiO2"
- Need for airway management for any reason
- Disturbed conscious level

Isolated TBI with the following criteria:

- Severe TBI (GCS < 8)
- Need for MV for any reason
- In need of O2 therapy "high FiO2"
- Hemodynamic instability
- Acute deterioration of conscious level > 2 GCS
- New onset seizure

Isolated chest trauma with criteria:

- Hemodynamic instability
- Need for MV for any reason
- In need of O2 therapy "high FiO2"

Burned patient with;

- Signs of inhalation injury
- Need for MV for any reason
- In need of O2 therapy "high FiO2"
- Hemodynamic instability

Cervical trauma pt with following criteria

- Hemodynamic instability
- Need for MV for any reason
- In need of O2 therapy "high FiO2"

2. Surgical Emergencies emergency and:

Cardiac System

- Acute chest pain including ACS&Shock
- Complex arrhythmias & Acute CHF
- Hypertensive emergencies

Pulmonary System

- Acute RF requiring ventilatory support
- Massive hemoptysis
- Respiratory distress for any cause

Neurologic Disorders

- Acute stroke with altered mental status
- Coma: metabolic, toxic, or anoxic CNS or neuromuscular disorders with deteriorating neurologic or pulmonary function
- Status epilepticus

Drug Ingestion and Drug Overdose

- Hemodynamically unstable drug ingestion
- Drug ingestion with significantly altered mental status with inadequate airway protection
- Seizures following drug ingestion

Gastrointestinal Disorders

- Life threatening GI bleeding
- Fulminant hepatic failure & Severe pancreatitis

Endocrine

- Endocrinal emergencies
- Manifested electrolytes disorders

Surgical

- PO ptsrequiring:Hemodynamic monitoring, Ventilatory support, Extensive nursing care

Miscellaneous

- Septic shock with hemodynamic instability
- Hemodynamic monitoring
- Clinical conditions requiring ICU nursing care
- Environmental injuries (lightning, near drowning, hypo/hyperthermia)
- New/experimental therapies with potential for complication

Priority II

1. Medical pts with:

- Need for MV
- Post cardiac arrest with cardiopulmonary failure

Priority III

1. Acute medical emergencies

- Respiratory distress&Uncontrolled fits&Shock

ICU Admission Policy

- ICU admission is reserved for pts with actual or potential vital organ system failures, which appear reversible with provision of ICU support
- **Organ System Failure**s include RF and cardiovascular instability
- **The ICU support** includes advanced monitoring, invasive procedures and intensive care like MV and vaso-active drugs

ICU Admission Procedure

- The request for admission **must** be made by the referring doctor
- The ICU doctor on-duty must see and assess the referred pt
- Resusc. or admission must not be delayed where the presenting condition is imminently life threatening, (eg profound shock or hypoxia)
- All admissions to ICU mustbe approved by the Consultant ICU on duty
- Pts admitted directly through the ED come under the name of the admitting medical or surgical consultant of the day
- Pts sent to the ICU from the wards must have their beds reserved
- The pt is managed by the ICU staff during their stay in ICU

Admission Protocol

- Organized by the ICU doctor on-duty
- Be sticky to **Admission criteria**
- Inform ICU Charge Nurse to prepare for admission
- Inform the Charge Nurse of the ward currently holding the pt
- On arrival to the ICU, attach monitors and record vital signs of the pt
- Resusc. priorities must follow **ALS** and **ATLS** guidelines
- ICU doctor must discuss management with duty ICU consultant
- ICU doctor must write **Admission doctor's orders**
- The ICU doctor must write all the required **Medications** in new drug charts
- The ICU doctor must complete, **Investigationsrequests**, **Generalconsentform** etc...
- ICU doctor must write a full **Admissionnote** (history, physical exam, assessment and the ICU management) in the progress sheet
- Pt admission out of the 3 priorities or priorities II, III is the ICU consultant on duty

Priority II,III admission is a consultant decision

Discharge criteria

A. When a pt's physiologic status has stabilized and the need for ICU monitoring and care is no longer necessary

- Hemodynamically stable (off vaso-active drugs) for at least **12**hrs
- No evidence of active bleeding
- Oxygen requirement is no more than FiO2 40% with SpO2 >90%
- Acceptable pH
- Extubate for >**6**-**24**hrs no evidence of upper airway obstruction
- Appropriate level of consciousness to protect the airway or has tracheostomy

B. When a pt's physiological status has deteriorated and active interventions are no longer planned, discharge to a lower level of care is appropriate

The ultimate authority for ICU admission, discharge, and triage rests with the ICU Director

Discharge criteria

ICU Discharge Policy

- Pts are discharged from ICU when the need for ttt or advanced monitoring is no longer needed
- The duty ICU consultant must approve ptdischarge

ICU Discharge Procedure

- Inform and discuss with the referring Team
- Inform the ICU nursing staff
- Ensure a ward bed is available
- The appropriate ward will be notified
- Complete the doctor's orders and discharge summary in the pt's notes
- The pt will be informed of the transfer

Discharge Protocol

- The status of pts admitted to an ICU should be revised continuously to identify pts who may no longer need ICU care
- Organized by the ICU doctor on-duty
- Be sticky to **Discharge criteria**
- Inform ICU Charge Nurse to prepare for discharge
- Inform the Charge Nurse of the ward currently receiving the pt
- The ICU doctor must write a full **dischargenote**
- Premature discharge of pt out of policy due to overwhelming of cases is the ICU consultant on duty
- No elective discharge before 9 am

Consult consultant on duty for premature discharge

Medical records *"see appendix5-14"*

Admission sheet

- Document pt**priority**, **ApachII** score, and **IBW**
- Take full recent & remote **History** from pt, relatives, and obtain any previous drug prescription, investigations, or radiology and document
- Pt**presentation** at admission should be clearly documented including invasive devices already inserted before admission
- ICU and referral **Plan** should be clear and written

Order form

- Document the need for vasopressors, inotropes, therapeutic heparin, intense glycemic control, O2 therapy, pnematic compression, specific positioning, EN initiation, C&S

Problem list sheet

The following should be documented in the **Problemlist sheet&**discussed on the morning round:

- 1ry pathology 'surgery, trauma, emergency medical or surgical situations"
- Shock including all varieties
- Metabolic disorders as acidosis, alkalosis, DKA, electrolyte disorders
- Medical emergencies "thyroid storm, hypertensive encephalopathy, etc.,"
- Hypoxemia and the need for O2 therapy
- Organ failure "renal, hepatic, cardiac, or MOF"
- Coagulopathy & hematologic disorders as HIT, HUS, DIC, ITTPHUS, etc.
- Complications of EN 'diarrhea,high GRV, etc.,"
- Complications of PN
- Sepsis, various types of infection "VAP, soft tissue in., CRBSI, UTI, etc"
- Complications of critical illness "stress ulcer, venous thrombo-embolism, critical illness myopathy , or plyneuropathy, and hypoalbuminemia, etc.'
- Ventilatory support, indication "pathology", difficult weaning, and complications "VILI, VAP"
- complications of intub. "subglottic stenosis, etc"
- Cardiac arrest, tachy, or brady-arrythmias
- Procedures "tracheostomy, dialysis, pacing, etc"
- Brain death, fits, major disturbed gcs "drop>2 GCS"

- **In the Morning round**
  - Resident presents every pt. The presentation should include "Pt demography & Remote & recent history & Problem list since admission & Systematic review & Existing problems
  - **ICU plan** should be fulfilled daily by on duty consultant & workup needed as radiology,lab., consultation"

Ventilation initiation &Ventilator flow sheets

- Indication, possible pathology, and objectives, initial settings and the events from intub. or application of NIV mask till the 1st 30 min. should be documented in the **initiation of ventilation sheet**
- Any troubleshooting, setting changes, and weaning plan should be documented on the **ventilator flow sheet**

Cardiac arrest sheet

- Document attendants, timing, pei-arrest events and management during the pei-arrest period

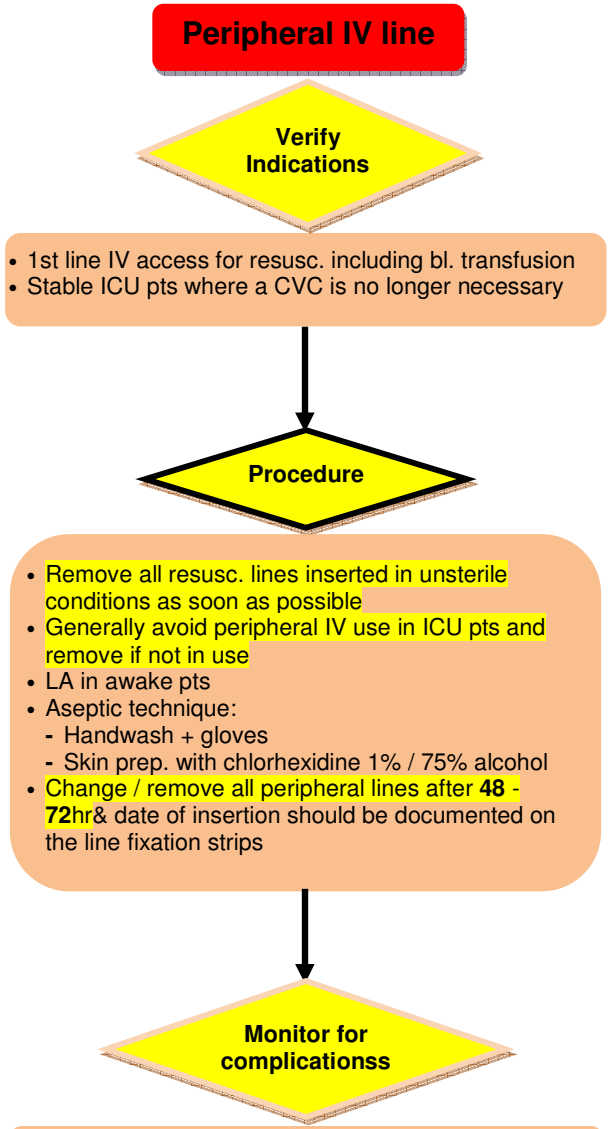
Progression sheet

- All pts must have DVT, GIT ulcer risk assessment reviewed within 24 hr of admission and documented If risk of VTE is identified and prophylaxis withheld, the reason(s) for this must be documented clearly on the progression sheet
- Review /d for adding, change, or DC and document ed
- Ab prescription should follow the unit anibiogram.
- Do not administer ab on admission without clear indication "no prophylactic ab unless indicated"
- Screen daily for change, escalation, or DC and documented
- Start EN within 24 hrs from admission unless CI
- Review daily for change rate "increase or decrease", shift to PN or oral feeding, complications
- Once the pt is intubated, decision of Tracheostomy should be discussed with consultants within the first w and document
- Screen invasive devises as CVC, urinary catheter, or tracheostomy daily for weaning, change and document
- Referral plan should be fulfilled daily by on duty

Other paper work

- Includes ; **2ndry trauma survey, peri-operative, Lab. and consultation sheets**

Clinical procedures


Peripheral IV line
Verify Indications
• 1st line IV access for resusc. including bl. transfusion
• Stable ICU pts where a CVC is no longer necessary
Procedure
• Remove all resusc. lines inserted in unsterile conditions as soon as possible
• Generally avoid peripheral IV use in ICU pts and remove if not in use
• LA in awake pts
• Aseptic technique:
- Handwash + gloves
- Skin prep. with chlorhexidine 1% / 75% alcohol
• Change / remove all peripheral lines after 48 - 72hr& date of insertion should be documented on the line fixation strips
Monitor for complicationss
• Inf., thrombosis, extra-vasation in tissues

Arterial line

Verify Indications

- Should be performed in(**Level I**)
  - Severe hypotension (S bl pr<**80**)
  - During the administration of vasopressor ±inotropic agents
  - Cardiogenic shock
- Useful in when potent vasodilators “Na nitroprusside” given (**Level IIa**)

Procedure

- Remove and replace lines inserted in unsterile conditions as soon as possible
- Aseptic technique
- LA in awake pts
- **Sites**: (order of preference): radial, dorsalis pedis, ulnar, brachial,femoral.Brachial and femoral arterial lines must be changed as soon as radial or dorsalis pedis arteries are available. The femoral artery may be the sole option in the acutely shocked pt
- There is no optimal time for an arterial line to be removed or changed
- Intra-arterial cannulae are changed/removed only in the following settings:
  - 3 ds from insertion
  - Distal ischaemia
  - Mechanical failure (over-damped waveform, inability to aspirate blood)
  - Evidence of unexplained systemic or local inf.
  - Invasive pr measurement or frequent bl sampling is no longer necessary

Measurement of pr

- Transducers ‘zeroed’ to the mid-axillary line
- Maintenance of lumen patency
- Continuous pressurized heparinised saline flush (1u/ml) at 3ml/hr

Monitor for complications

- Inf., thrombosis, digital ischaemia, vessel damage / aneurysm, HITS (2 ndry to heparin infusion)

Central venous catheter

Verify Indications

- Standard IV access in ICU pts:
  - Fluid administration (including elective transfusion)
  - TPN, hypertonic solutions
  - Vasoactive infusions
- Monitoring of right atrial pr (CVP)
- Venous access for:
  - Pulmonary artery catheterisation (
  - CRRT, plasmapheresis.
  - Jugular bulb oximetry
  - Transvenous pacing
- Resusc. "failed 2 peripheral attempts"

Management protocol

Types:

- Standard CVC for ICU is antimicrobial impregnated (rifampicin/minocycline) 20cm triple lumencatheter "*unavailable nowadays in our unit"*

Sites:

- **Subclavian** is the preferred site for routine stable ptsfollowed by internal jugular
- Femoral access is preferable where:
  - Limited IV line (burns, multiple previous CVC's)

Coagulopathic pts:

- Correct coagulopathy
- Avoid subclavian catheterization
- Consider US guided IJ insertion

Procedure

- LA in awake pts
- Strict aseptic technique at insertion
- Seldinger technique only
- Monitor for arrhythmias during insertion
- Suture all lines
- Dressing: non occlusive dressing
- Flush all lumens with heparinised saline
- Check CXR prior to use

CVC insertion in a coagulopathic pt is a consultant decision

Pot-procedure

- At insertion "Arterial puncture
  - Haematoma& Arterial thrombosis/embolism &Neural injury
  - Pneumothorax, haemothorax, chylothorax
- Passage of wire/catheter
  - Arrhythmias
  - Tamponade
- Presence of catheter
  - Thrombosis
  - Air embolism
  - Catheter knotting
  - Catheter inf.

Monitor for complications

- Routine IV administration set change at 5 ds
- Daily inspection of the insertion site and clinical suspicion for inf. irrespective of insertion duration
- Catheters are left in place as long as clinically indicated and changed when:
  - Evidence of systemic inf. "New, unexplained fever, leukocytosis & Deterioration in organ function, +ve bl culture by venipuncture with likely organisms (S. epidermidis, candida spp.), and/or
  - Evidence of local inf.
  - Inflammation or pus at insertion site
- **Guidewireexchanges** are actively discouraged. They may be indicated in the following situation (after discussion with a Consultant):
  - Mechanical problems in a new catheter (leaks &kink)
  - Difficult or limited central access (eg burns)

Catheter exchange over a guide wire in presence of site inf. Is a consultant decision

Epidural catheter

Verify indications

- PO pain relief (usually placed in theatre)
- Analgesia in chest trauma

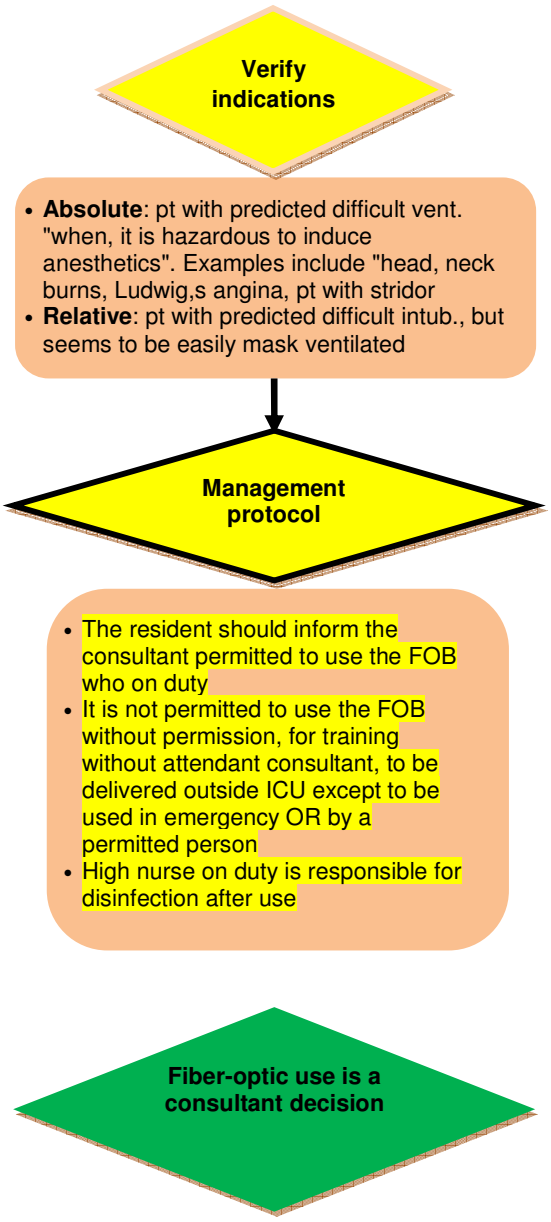
Management protocol

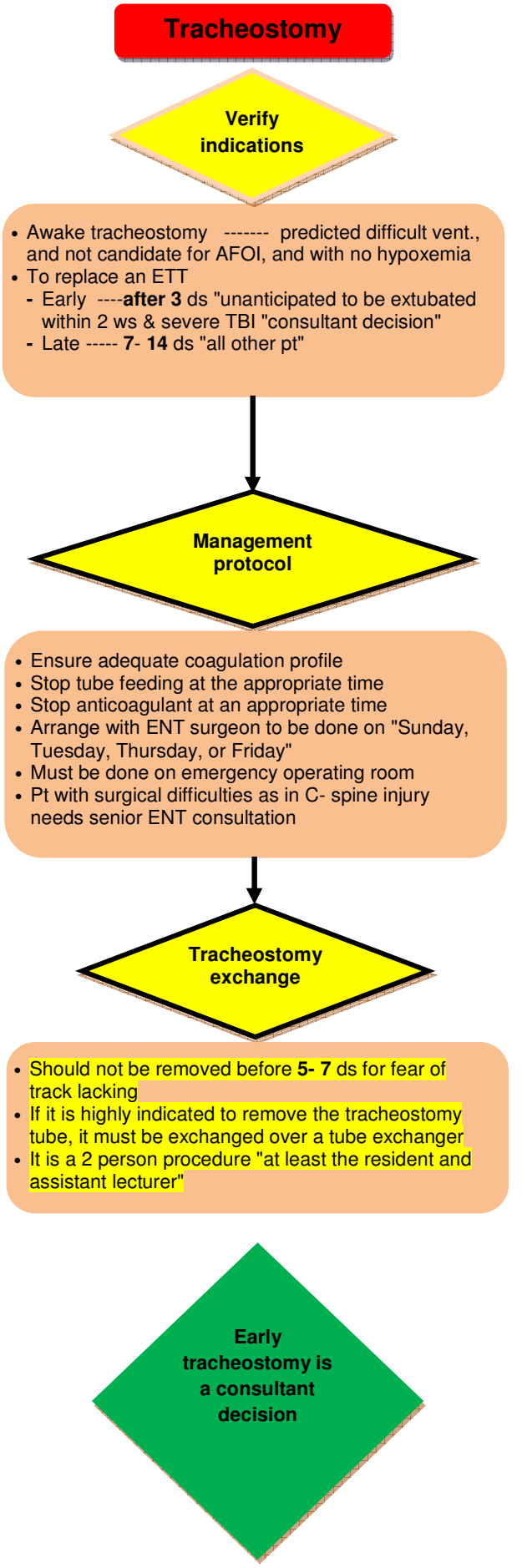
- Strict aseptic technique at insertion
- LA protocol
  - Adequately inserted catheter "tip at center of site to be blocked' ----- consider a boluses of **5**ml 1% Xylocaine with hemodynamic monitoring. If no response after 3 doses, consider failure
  - Followed by infusion**5** – **10** ml/hr**marcine** 0.125% + **fentanyl2** uq /ml
- **Top- up doses protocol**
  - Consider **5** ml **Xylocaine** 1% ------ If no response, consider another 5 ml. If no response, consider failure
  - If there is unilateral anesthesia after 5 ml xylocaine 1%, consider slight withdrawal of the catheter and inject another 5 ml LA. If there is no response, consider failure
  - If there is response after 5-10 ml Xylocaine 1% , re- infuse, and consider increasing the rate
- Daily inspection of the insertion site. The catheter should not be routinely redressed
- After**7** ds ---- weight the risk- benefit for removal of the catheter
- Remove if not in use for > 24 hr or clinical evidence of unexplained sepsis or +ve bl culture by venipuncture with likely organisms (S.epidermidis, candida)
- Insertion &removal of catheter in anti-coagulated pt
  - Prophylactic heparin ----- delay dose for **1** hr after insertion, remove 4 hrs from last dose or 1 hr before next dose
  - Prophylactic LMWH ---- insert **10**-**12** after the last dose
  - Therapeutic LMWH ----- insert **24** after last dose. Re-administer **24** hrs after insertion Remove catheter 2 hr before next dose

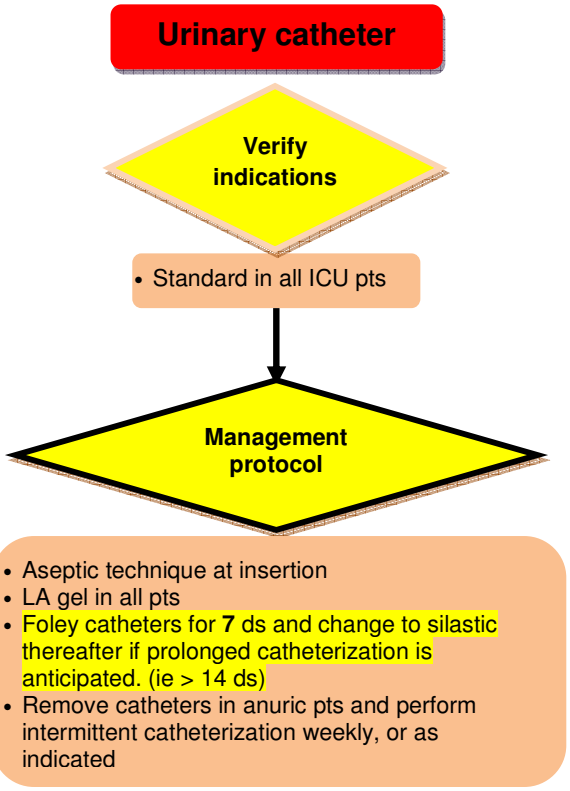
Monitor for complications

- Hypotension from sympathetic blockade / relative hypovolaemia ------ usually responds to adequate IV volume replacement
- Pruritis--if severe, Naloxone **100** uq/10 min "**400** total"
- N & V---- metochloperamide **10** mg /4hr
- Weakness & Numbness --- check catheter migration, stop infusion, re-infuse at a lower rate
- Inf.**:** epidural abscess

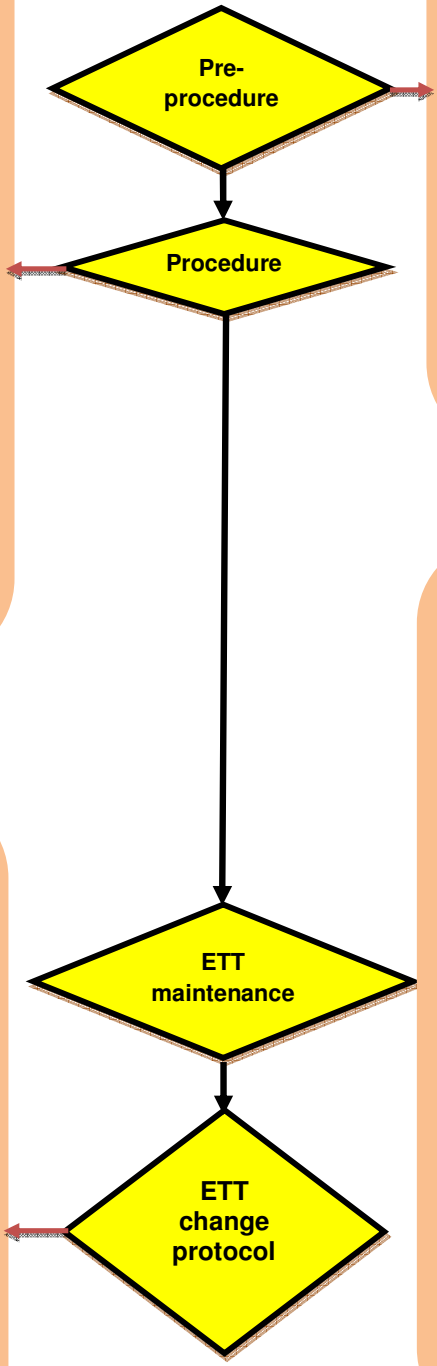
Fiberoptic bronchoscopy




Tracheostomy
Verify indications
• Awake tracheostomy ------- predicted difficult vent., and not candidate for AFOI, and with no hypoxemia
• To replace an ETT
- Early ----after 3 ds "unanticipated to be extubated within 2 ws & severe TBI "consultant decision"
- Late ----- 7- 14 ds "all other pt"
Management protocol
• Ensure adequate coagulation profile
• Stop tube feeding at the appropriate time
• Stop anticoagulant at an appropriate time
• Arrange with ENT surgeon to be done on "Sunday, Tuesday, Thursday, or Friday"
• Must be done on emergency operating room
• Pt with surgical difficulties as in C- spine injury needs senior ENT consultation
Tracheostomy exchange
• Should not be removed before 5- 7 ds for fear of track lacking
• If it is highly indicated to remove the tracheostomy tube, it must be exchanged over a tube exchanger
• It is a 2 person procedure "at least the resident and assistant lecturer"
Early tracheostomy is a consultant decision


Urinary catheter
Verify indications
• Standard in all ICU pts
Management protocol
• Aseptic technique at insertion
• LA gel in all pts
• Foley catheters for 7 ds and change to silastic thereafter if prolonged catheterization is anticipated. (ie > 14 ds)
• Remove catheters in anuric pts and perform intermittent catheterization weekly, or as indicated

Intubation



- Verify **indication**---- if in doubt consult
- Review airway cart , equipment and drugs
- If you are alone --- **Call for help**"1 doctor , and 1-2 nurses
- Be sure that there is at least 1 working **IVline**, and **monitor** attached
- Anticipate physiologic difficulty "consider pathology, appropriate drugs"
- Pts with full stomach with no predicted vent. difficulty ----use**RSI**
- Pts with full stomach with no predicted intub. difficulty ----- use RSI with a backup plan "ILMA, Air-Q"
- Pt with predicted vent. difficulty ---- prepare for awake techniques "intub.or tracheostomy &used by skilled personnel and authorised by the duty consultant "
- Be prepared for initial intub. attempts "2 working laryngoscopes, working lights, bougie on pt ,s chest, working suction, BVM, oro and naso pharyngeal airway, and high flow O2
- prepare surgical airway in failed airway

- Oro-tracheal intubation is the standard method of intubation in our unit
- Naso-tracheal intubation may be indicated where: Fibreoptic intubation is indicated: "Following head and neck surgery, Inability to open the mouth: e.g. inter-maxillary fixation, TMJ trauma, rheumatoid arthritis, etc."
- Standard ETT: low pressure, high volume PVC oral tube.
  - Males **8** mm: secure at **21**-**23cm** to incisors
  - Females **7** mm: secure at **19**-**21cm** to incisors
  - Do not cut tubes to <**26** cm long
- **Double lumen tubes**
  - Unilateral lung isolation for abscess, broncho-pulmonary fistula, or hge
  - These tubes should be inserted as a temporary manoeuver prior to a definitive procedure
  - Allow ILV
- Intubation is a **3**- **4** persons procedures
  - 1 for intub.
  - 1 for drug administration, monitoring
  - 1 for cricoids pr and MILS if needed

- **Confirmplacement** "direct vision, auscultation, capnography, , and CXR"
- **Tapes**
  - Secure tubes with white tape
  - Ensure that loop of tape is snug around back of neck but not too tight to occlude venous drainage. Should allow 2 fingers under tape
  - Re-tape with adhesive tape post CXR
- **Cuffcheck**
  - Volumetric (sufficient air to obtain a seal + 1 ml) tests are done following insertion and whenever a leak is detected with a manual hyperinflation once / nursing shift
  - Assess seal by auscultation over trachea during normal vent.
  - Manometric tests "**25** mmHg, **20** for hypoperfused pt
- **Persistent cuff leaks**
  - Tubes requiring >5ml of air to obtain a seal or if there is a persistent cuff leak must be examined by direct laryngoscopy as soon as possible even if the tube appears to be taped at the correct distance at the teeth
  - Ensure that:
    - Cuff has not herniated above cords
    - Tube has not ballooned inside the oral cavity and “pulled’ the cuff above the cords

- Prepare as for de novo intub.
- Set the FIO2 = 1.0 and controlled vent.
- Ensure sufficient anesthesia ± NMB
- Perform laryngoscopy and carefully identify patency of upper airway after suction, anatomy of larynx, degree of laryngeal exposure and swelling
- Clear view of larynx and no or minimal laryngeal swelling:
  - Application of cricoid pr by assistant and careful, graded extubation under direct vision
  - Maintain laryngoscopy and replace tube under direct vision
- Impaired visualisation of larynx:
  - Place bougie through ETT under direct vision and insert to a length just distal to the end of the ETT
  - Assistant control the bougie so that it does not move duringETT movement
  - Application of cricoid pr by assistant and careful, graded extubation
  - Maintain laryngoscopy,ensure bougie is through cords on extubation
  - Replace tube over bougie and guide through larynx under available vision
- Inflate cuff, check EtCO2, auscultation, VTe and then release cricoid pr
- Secure tube with tape

Lumbar puncture "LP"

The findings on CSF analysis also may help distinguish bacterial meningitis from viral inf of the CNS. However, there is often substantial overlap

Verify indications

Urgent
- Suspected CNS inf. (except brain abscess or a param-eingeal process)
- Suspected SAH in a pt with a -ve CTscan

Non-urgent
- Idiopathic intracranial hypertension
  Carcinomatous & TB meningitis
- Normal pr hydrocephalus
- CNS syphilis & vasculitis

❖ Conditions in which LP is rarely diagnostic but still useful include; Multiple sclerosis, Guillain-Barré syndrome, AND Para-neoplastic syndromes

❖ LP is also required as a therapeutic or diagnostic maneuver in the following situations ; Intra-thecal administration of chemotherapy, Intra-thecal administration of antibiotics, ND Injection of contrast media for myelography or for cisternography

Detect CI

No absolute CI

Caution should be used in pts with:
- Suspected spinal epidural abscess
- Possible raised ICP
  - Order CT scan before LP in pts with altered mentation, focal neurologic signs, papilledema, seizure within previous w, and impaired cellular immunity
- Thrombocytopenia or other bleeding diathesis (including ongoing anticoagulant therapy)
  - Do not perform LP in pts with coagulation defects active bleeding, have severe thrombocytopenia (eg, platelet counts <**50-80**,000/µL), or an INR >**1.4**, without correcting the underlying abnormalities
  - When an LP is considered urgent and essential in a pt with an abnormal INR or platelet count in whom the cause is not obvious, consultation with a hematologist may provide the best advice
  - For elective procedures in a pt receiving systemic anticoagulation ----stop UFH **2-4** hrs, LMWH **12** - **24** hrs, and warfarin **5-7** ds before LP
  - SC heparin administration is not believed to pose a substantial risk for bleeding after LP if the total daily dose <**10**,000 U
  - Aspirin has not been shown to increase the risk of serious bleeding following LP
  - In all cases, the relative risk of performing an LP has to be weighed against the potential benefit (eg, diagnosing meningitis due to an unusual or difficult to treat pathogen)
  - In cases in which LP is considered necessary but the risk of bleeding is considered to be high, it may be useful to perform the procedure under fluoroscopy to reduce the chance of accidental injury to small blood vessels

Management protocol

- **Position**: lateral recumbent ----- allows accurate measurement of the opening pr. The pt assumes a fetal position with neck, back, and limbs held in flexion. The lower lumbar spine should be flexed with the back perfectly perpendicular to the edge of a bed or examining table. The hips and legs should be parallel to each other and perpendicular to the table. Pillows placed under the head and between the knees
- **Insertion site**: L3/4 or L4/5 interspace
- **Procedure**:
  - Antiseptic application ---- surgical drapes ---- needle advancement ---- Once CSF appears and begins to flow through the needle, slowly straighten or extend legs to allow free CSF flow
  - A manometer should then be placed over the hub of the needle and opening pr should be measured
  - Fluid is then serially collected in sterile plastic tubes. A total of **8-15** mL is typically removed, when special studies are required, as cytology or cultures for organisms that grow less readily (eg, fungi or mycobacteria), **40** mL of fluid can safely be removed
  - Aspiration of CSF should not be attempted as it may increase the risk of bleeding
  - The stylet should be replaced before the spinal needle is removed

Monitor for compli-cations

Relativey safe procedure
- Post-LP headache
- Inf.
- Bleeding
- Cerebral herniation
- Minor neurologic symptoms as radicular pain or numbness
- Late onset of epidermoid tumors of thecal sac

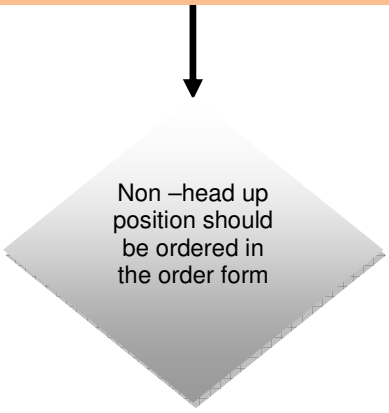
General principles

Drug prescription

- Ideally, drugs should only be prescribed where proven benefit has been demonstrated
- **Drugsprescription** according to Unit protocols and guidelines
  - E.g., steroids, and albumin should not prescribed without clear indications
- Ensure that the drug **Doses** are correct: seek advice if unsure "*See local protocols, medscape, uptodate*"
- The risk and benefit of starting any drug must be carefully considered
  - Critically ill pts have altered pharmacokinetics and pharmacodynamics with the potential for toxicity and drug interactions
- Where possible:
  - Use drugs that can be titrated or prescribed to an easily measured endpoint
  - Use drugs that can be measured to monitor therapeutic drug levels
  - Avoid drugs with narrow therapeutic indices (eg digoxin, theophylline), particularly in pts with associated hepatic or renal dysfunction
  - Cease a drug if there is no apparent benefit
  - If 2 drugs are of equal efficacy, choose the cheaper drug (egpancuroniumvsvecuronium) as the cost of drugs in ICU is significant
- Any **Specificmedications** as manitol, steroids. etc, should be documented and be cleared who prescribe either in the consultation or plan forms
- **O2therapy** with a SPO2 target should be clearly prescribed in the order form
- **Vaopressors** and **Inotropes** with mean arterial pressure target should be clearly prescribed also in the order form
- **Albumin** isindicated in the following settings only
  - Liver cell failure
  - Colloid volume resusc. in septic shock which is not responding to early goal directed therapy"60 gm /d in divided doses"
  - Colloid volume resusc. In septic shock due to spontaneous bacterial peritonitis "1 gm /Kg bolus followed by 1.5 gm /Kg after 2-3 ds
  - ARDS with poor oxygenation despite appropriate protective ventilator strategy "60 gm /d in divided doses"
  - Pts with multi-organ dysfunction syndrome and low albumin "less than 3 gm /L " "60 gm /d in divided doses"
  - Pts with low tolerance to EN and diarrhea with albumin less than 2.5 gm /L "60 gm /d in divided doses"

Patient positioning

- The ideal position for critically pt is **30** - **45**^{0}head of bed "HOB" unless CI
  - Unstable C- spine
  - Hemodynamic instability
- Turning pt /2 hrs according to the nursing protocol
- Prone position mostly for ventilation of the ALI pt should be the responsibility of the on duty consultant *"p61"*

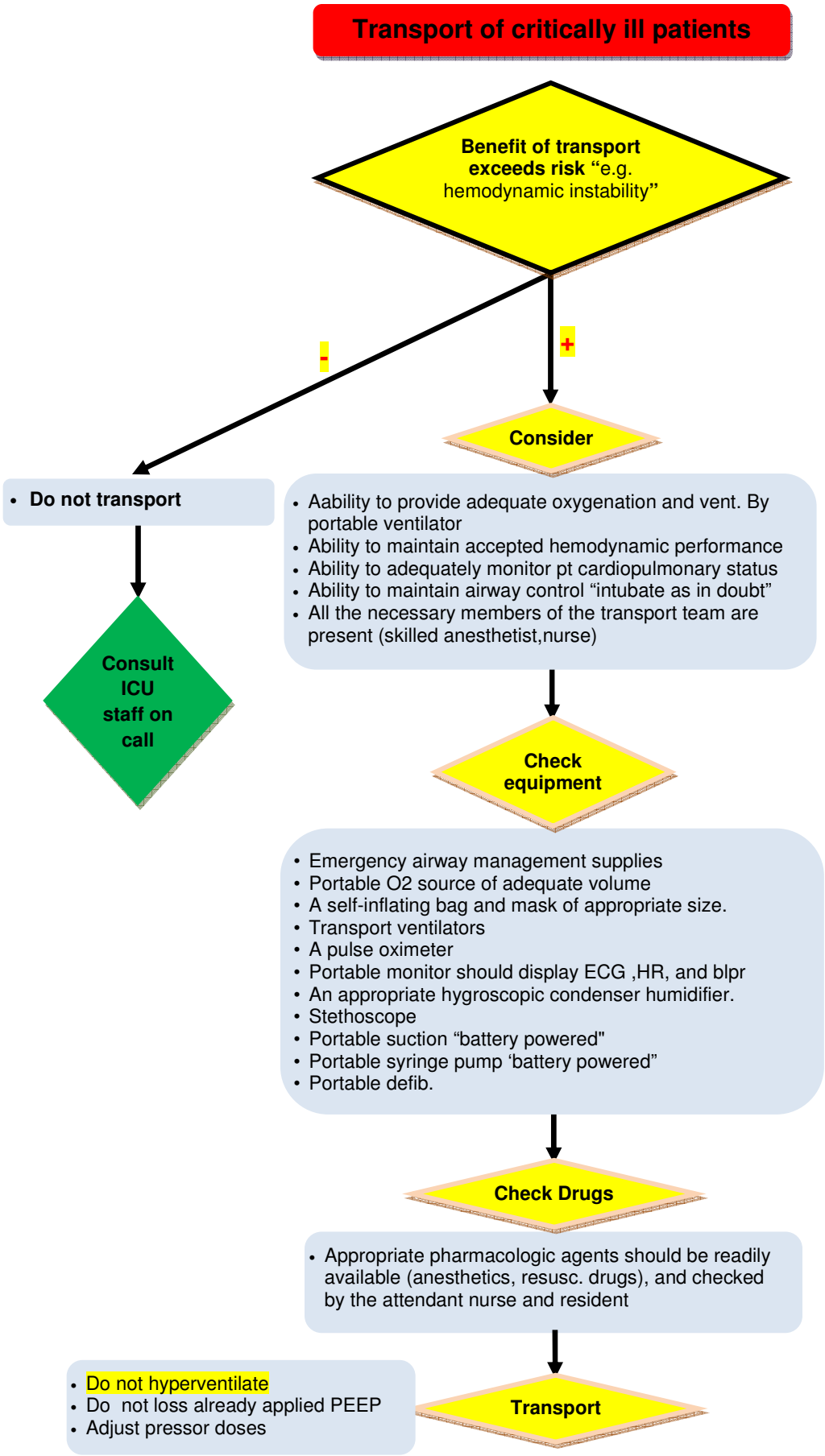


Order of do not attempt resuscitation "DANR"

&

Withdrawal of treatment

- Our unit does not follow the order of do not attempt resusc. and does not withhold ttt from pts with end of life care
- In cases of end of life care as brain dead pts we do not futile therapies
- However, these two issues will need sooner to be clear from both the legal and religious views


Transport of critically ill patients
Benefit of transport exceeds risk "e.g. hemodynamic instability"
-
+
Consider
Do not transport
Consult ICU staff on call
Aability to provide adequate oxygenation and vent. By portable ventilator
Ability to maintain accepted hemodynamic performance
Ability to adequately monitor pt cardiopulmonary status
Ability to maintain airway control "intubate as in doubt"
All the necessary members of the transport team are present (skilled anesthetist,nurse)
Check equipment
Emergency airway management supplies
Portable O2 source of adequate volume
A self-inflating bag and mask of appropriate size.
Transport ventilators
A pulse oximeter
Portable monitor should display ECG ,HR, and blpr
An appropriate hygroscopic condenser humidifier.
Stethoscope
Portable suction "battery powered"
Portable syringe pump 'battery powered"
Portable defib.
Check Drugs
Appropriate pharmacologic agents should be readily available (anesthetics, resusc. drugs), and checked by the attendant nurse and resident
Do not hyperventilate
Do not loss already applied PEEP
Adjust pressor doses
Transport

DVT prophylaxis

- Dose adjustment and/or anti-Xa monitoring for LMWH considered for wt< 45kg, obesity, or renal impairment (CrCl<30 mL/min)
- **Fondaparinux** used where UFH CI as previous HIT

- Prophylaxis should generally not be interrupted for procedures or surgery unless there is a particularly high bleeding risk
- The insertion or removal of epidural catheters should coincide with the nadir of the anticoagulant effect

Daily screen

Surgical

Bleeding risk

IPC& VFP

Medical

CHF, severe respiratory diseases or pt confined to bed + 1 or more **Risk factor**

Bleeding risk

\+

- **LMWH** "Enoxparin 40 mg / 24 hr
- Consider **IPC/VFP** in high risk pt.

\-

- Coagulopathy "Thrombocyto-penia" 50.000"& INR 1.5
- Active bleeding
- Bleeding disorders
- 2 w from thrombotic stroke, 1 w from embolic
- 4 hr from LP, central blockade

Minor procedure + no additional risk factors

Low risk

- No specific measures
- Early mobilizat-on

Minorprocedure+ additional risk factors *OR* **Major procedure + no additional risk factors**

Moderate risk

LDUH/ 12 hr"2 hr before op till7d,ambul-tion or discharge OR **LMWH** 12 hrpreop.± IPC / VFP

Majorprocedure + additional risk factors

High risk

LDUH /8 hr *OR* **LMWH** +/- IPC / VFP

Any procedure with 2 or more risk factors

Very high risk

LDUHq 8 hr + IPC / VFP *OR* **LMWH** + IPC / VFP

Trauma

Bleeding risk

\+

- Intracranial hge
- Incomplete spinal cord injury with associated peri-spinal hematoma
- Ongoing-uncontrolled hge
- Uncorrected coagulopathy

\-

- **LMWH** "Enoxparin 30 mg/12 hrs or 40 mg / 24 hr.
- **IFC** insertion is not recommen-ded for primaryprop hyla-xis

Enoxaparin **20** mg/d if creatinine clearance <30ml/min.

IPC & Graded compression elastic stockings & VFPas the initial prophylaxis Till **LMWH** can be safely used

Risk factors of DVT in trauma pt

- Increasing age & Spinal cord injury
- Lower extremity or pelvic fracture & need for surgery
- Femoral venous line or major venous repair
- Prolonged immobility & duration of hospital stay

Risk factors for DVT in the non-trauma surgical pt

- Prolonged immobility & Age > 40 years
- Stroke & Paralysis
- Previous DVT
- Malignancy and its treatment
- Major surgery (esp., abdomen, pelvis, and lower extremities)
- Obesity &Varicose veins & Cardiac dysfunction
- Indwelling central venous catheters
- Inflammatory bowel disease &Nephrotic syndrome
- Pregnancy or estrogen use
- Congenital and acquired thrombophilic disorders

DVT in the trauma pt

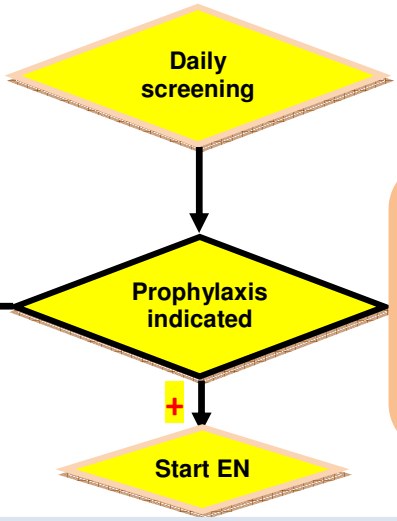
- Presence of TBI without frank hge, complete spinal cord injuries, lacerations or contusions of internal organs, or retroperitoneal hematoma associated with pelvic fracture do not by themselves contraindicate use of LMWH as long as the pt has no evidence of active bleeding
- LMWH can be safely used for prophylaxis in pts with ICH within 24 hr of admission or craniotomy, and surgery is not planned within 12 hrs

- Routine screening for asymptomatic DVT not recommended

- Aspirin &Warafarin should not be used as sole agent

GIT prophylaxis

- **Stress ulcers** are mucosal erosions which primarily occur in the stomach, but can also be found in the distal esophagus or duodenum. They can develop within hrs of a trauma or the onset of a critical illness. Critically ill pts who bleed from these lesions have a 5-fold increase in mortality compared with pts who do not bleed
- Stress ulcers are believed to be caused by an imbalance between gastric acid production and mucosal protection mechanisms. Mucosal ischemia may be an important cause in pts with underlying shock, sepsis, and trauma



- MV>**48**hrs, Coagulopathy" platelet count <**50**,000 / m3, an INR >**1.5**, or a PTT >**2** times the control value", severe TBI, major burns">35%"", and history of GI ulceration or bleeding in last year "**Level I**"
- Poly-trauma, sepsis, and acute renal failure "**Level II**"
- Need of high-dose steroids (>**250** mg hydrocortisone or equivalent / d) "**Level III**"

H2 BLOCKER

- **Rantidine50** mg /8 hrs
- Adjust according to renal function

- Start Early **EN**"contributes to stress ulcer prophylaxis"
- Should not be used alone for the sole purpose of prophylaxis esp., in high risk pts (**Level IIc**)

Select agent

- Administer **OralPPI** rather than any alternative prophylactic agent if EN is feasible (**Level IIb**)
- Administer **IVH2blocker** rather than an IV PPI if EN is not feasible (**Level IIb**). IV H2 blockers are usually much less expensive than IV PPIs and appear to be nearly as efficacious
- If cost is not an issue, and EN is not feasible **IVPPI** is a reasonable choice

DC agent

- Tillweaning from MV or ICU discharge "**Level II**"

Sedation

- Control environment (**Level II**)
- Light turned off at night. Minimize background noise
- Maintain day- night cycle
- The agents of choice are:
  - **Midazolam 2-3** mgat bed time
  - If necessary, **Diphyenylhyramine25 - 50** mg IV

- Facilitate sleep

Indicated

- Agitation / delerium

- Delirium may or may not accompanied by agitation
- Assess using **RASS score**
- Signs of delirium includes:
  - Disorganized thinking, altered consciousness level, and inattention
- Identify and ttt reversible causes
  - Metabolic derangement
  - Pharmacological interaction / side effects
- Control environment

Drug therapy

- The agent of choice for acute delirium is **Midazolam**
- The altenative is **Haloperidol** (**Level III**)
  - **2.5-5** mg bolus followed by **2.5-5** mg / 30 min. till level of sedation is reached
  - Maintain sedation with **5 -10** mg / 4-6 hrs
  - Once controlled, a standing dose of the haloperidol, at 25% of loading dose, is administered / 6 hrs
  - Maximum dose to be administered over a 24 hr period is **40** mg
  - If over sedated hold drug dosage for 1 hr and then resume with 25% reduced dose
  - Give midazolam **1-2** mg / 2-4 hrsfor amnesia and anxiolysis
  - Monitorqt prolongation and arrhythmia (**Level II**)
- **ContinuousMidazolam** is indicated if intermittent doses and Haloperidol did not manage (**Level II**)

- Difficult vent. "bad compliance"
- Reduce O2 demand "severe tachypnea"
- Inverse ratio vent.
- Need for NMB
- TBI

Propofol

- **150-200** mgbolus, then**25-50** mcg/kg/min. and titrate in increments **25**mcg/kg/min. till desired level is achieved
- Check TG after 72 hrs. Watch for lactic acidosis and rhabdomyolysis
- **+**
- **50-100**uq**fentanyl** bolus followed by **50**uq/hr, or **5** mg**morphine** IV then either **2-5** mg / **1**hrprn or continuous infusion 4mg/hr
- **±**
- **Midazolam** "1-2 mglhr"

Richmond agitation sedation "RAS" score

Target	Description
+4	Combative& violent
+3	Remove catheters&agressive
+2	Frequent non-purposeful movement&fight ventilator
+2	Anxious&apprehensive, not aggressive
0	Alert&calm
-1	Awaken to voice 10 ` s
-2	Movement, eye opening, no eye contact
-3	No response to voice&Movemnt, eye opening to physical stimuli
--4	No response to voice
-5	No response to physical stimuli

- Relieve distress
- Reduce anxiety
- Protect against myocardial ischemia

Non-pharmacological measures

- Adequate analgesia before sedation if there is pain (**Level III**)
- The target level of sedation is a calm pt who is easily aroused and has a normal sleep – wake cycle (**RASS0-2**)
- Achieved by either:

Midazolam

- **2** mg bolus followed by **1-2** mg/hr infusion or **1-2** mg q 1 hrprn titrated in increments of 1-2 mg after reassessing ' 30 min. till desired level is achieved
- Concurrent use of narcotics **50-100**uq**fentanyl** bolus followed by **50**uq/hr, or **5** mg **morphine** IV then either **2-5** mg / **1**hrprn or continuous infusion 4mg/hr
- Once adequate level of sedation and analgesia are achieved, the pt is to be evaluated / 4 hrs. If depth of sedation is more than desired, decrease anxiolytics by 25% and analgesic by 25%
- Consider daily interruption

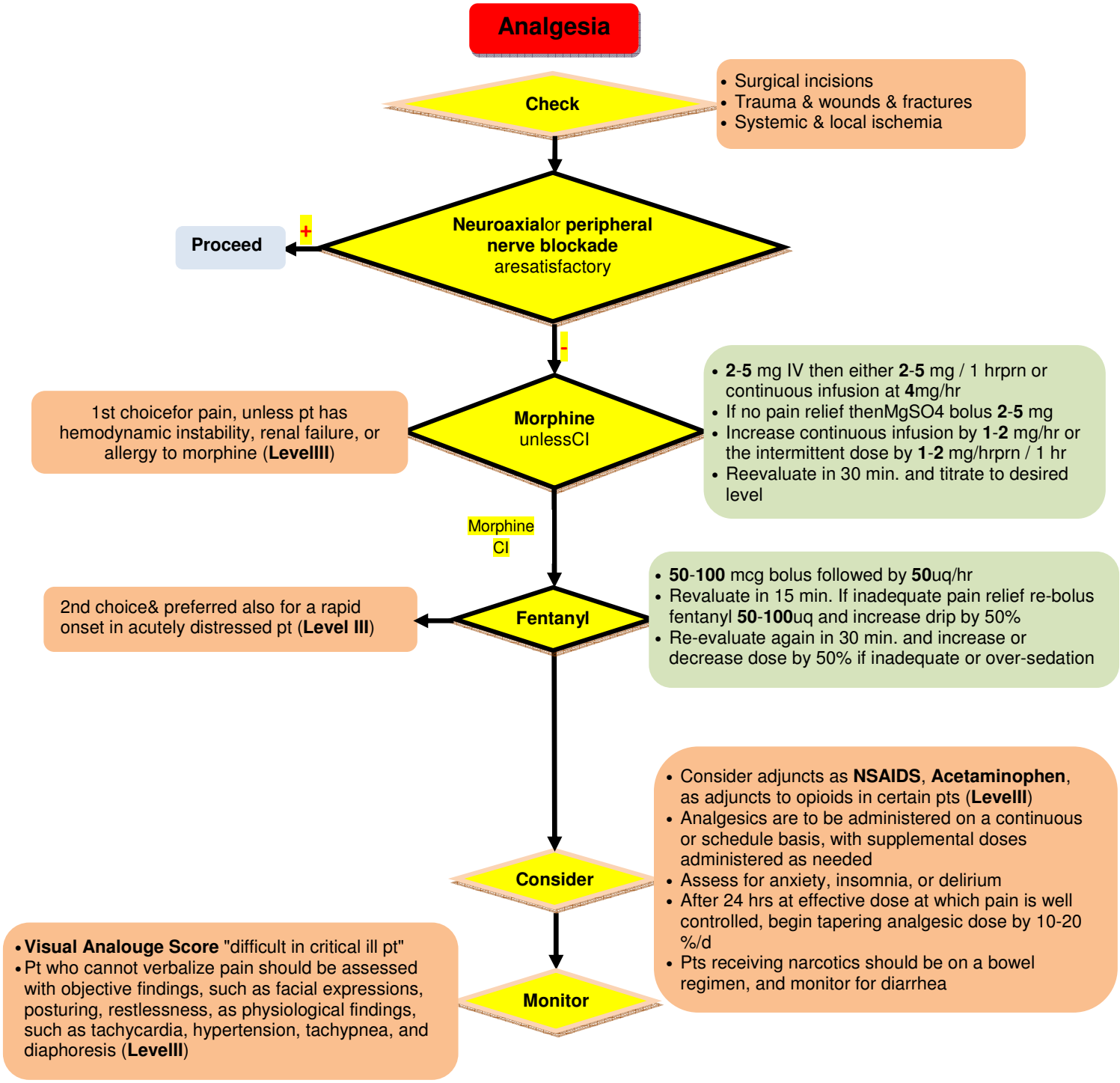
Propofol

- **10-25** mcg/kg/min. titrated in increments of **10-25** mcg/kg/min / 5-10 min. till desired level.
- Indicated if there is failure of conventional sedation or need for rapid neurologic assessment (**Level II**)

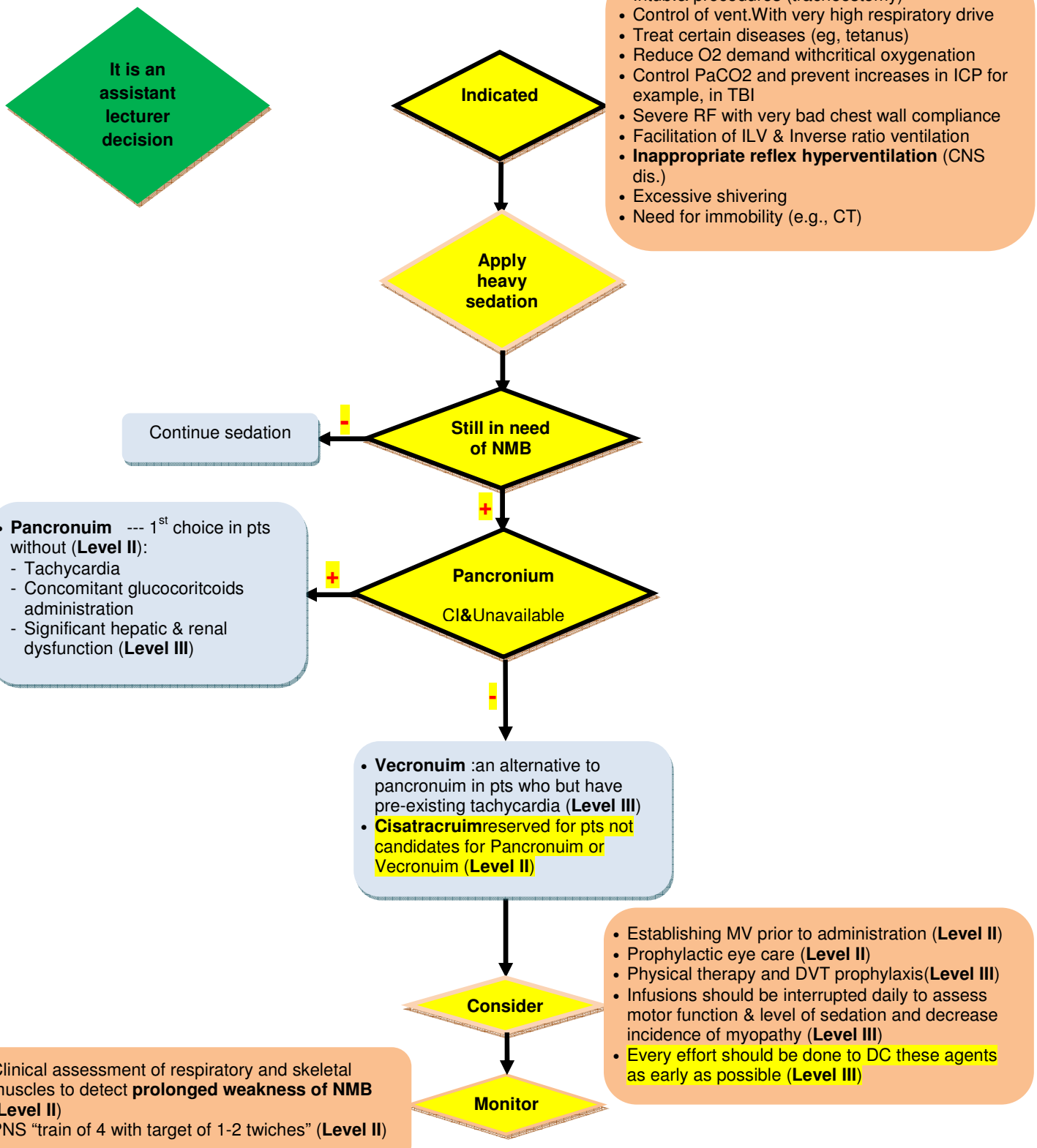
- **BIS** is not yet proven useful in ICU (**LevelIII**)

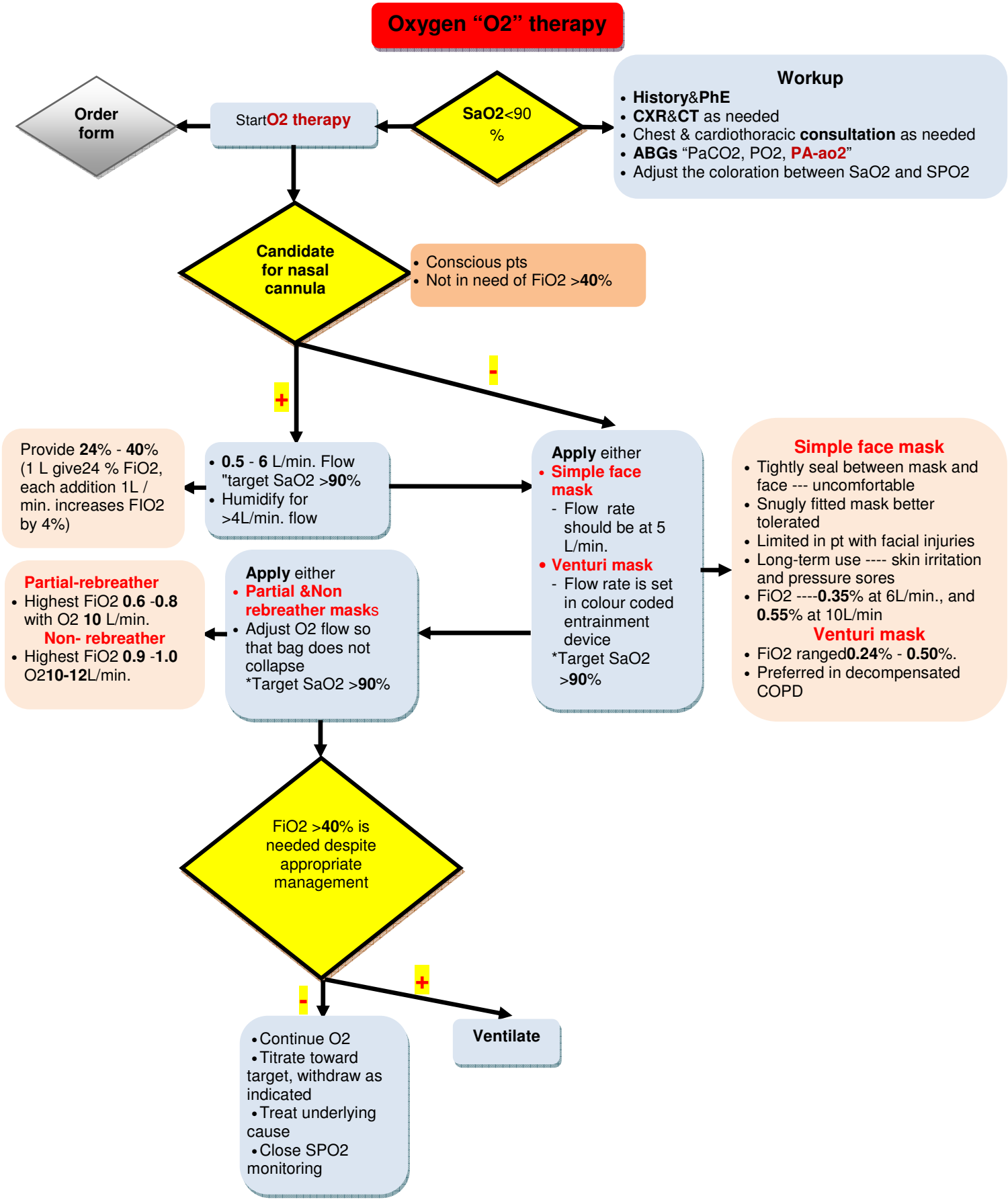
Non- pharmacological measures

- Establish regular sleep-wake cycles
- Reassure & Minimize stimulation during sleep


Analgesia
Check
Surgical incisions
Trauma & wounds & fractures
Systemic & local ischemia
Neuroaxialor peripheral nerve blockade aresatisfactory
+
Proceed
-
Morphine unlessCI
1st choicefor pain, unless pt has hemodynamic instability, renal failure, or allergy to morphine (LevelIII)
2-5 mg IV then either 2-5 mg / 1 hrprn or continuous infusion at 4mg/hr
If no pain relief thenMgSO4 bolus 2-5 mg
Increase continuous infusion by 1-2 mg/hr or the intermittent dose by 1-2 mg/hrprn / 1 hr
Reevaluate in 30 min. and titrate to desired level
Morphine CI
Fentanyl
2nd choice& preferred also for a rapid onset in acutely distressed pt (Level III)
50-100 mcg bolus followed by 50uq/hr
Revaluate in 15 min. If inadequate pain relief re-bolus fentanyl 50-100uq and increase drip by 50%
Re-evaluate again in 30 min. and increase or decrease dose by 50% if inadequate or over-sedation
Consider
Consider adjuncts as NSAIDS, Acetaminophen, as adjuncts to opioids in certain pts (LevelII)
Analgesics are to be administered on a continuous or schedule basis, with supplemental doses administered as needed
Assess for anxiety, insomnia, or delirium
After 24 hrs at effective dose at which pain is well controlled, begin tapering analgesic dose by 10-20 %/d
Pts receiving narcotics should be on a bowel regimen, and monitor for diarrhea
Monitor
Visual Analouge Score "difficult in critical ill pt"
Pt who cannot verbalize pain should be assessed with objective findings, such as facial expressions, posturing, restlessness, as physiological findings, such as tachycardia, hypertension, tachypnea, and diaphoresis (LevelII)

Neuro-muscular blockade"NMB"




Oxygen "O2" therapy
Order form
Start O2 therapy
SaO2<90 %
Workup
History&PhE
CXR&CT as needed
Chest & cardiothoracic consultation as needed
ABGs "PaCO2, PO2, PA-ao2"
Adjust the coloration between SaO2 and SPO2
Candidate for nasal cannula
Conscious pts
Not in need of FiO2 >40%
+
-
Provide 24% - 40% (1 L give24 % FiO2, each addition 1L / min. increases FIO2 by 4%)
0.5 - 6 L/min. Flow "target SaO2 >90%
Humidify for >4L/min. flow
Apply either
Simple face mask
- Flow rate should be at 5 L/min.
Venturi mask
- Flow rate is set in colour coded entrainment device
*Target SaO2 >90%
Simple face mask
Tightly seal between mask and face --- uncomfortable
Snugly fitted mask better tolerated
Limited in pt with facial injuries
Long-term use ---- skin irritation and pressure sores
FiO2 ----0.35% at 6L/min., and 0.55% at 10L/min
Venturi mask
FiO2 ranged0.24% - 0.50%.
Preferred in decompensated COPD
Partial-rebreather
Highest FiO2 0.6 -0.8 with O2 10 L/min.
Non- rebreather
Highest FiO2 0.9 -1.0 O210-12L/min.
Apply either
Partial &Non rebreather masks
Adjust O2 flow so that bag does not collapse
*Target SaO2 >90%
FiO2 >40% is needed despite appropriate management
+
-
Ventilate
Continue O2
Titrate toward target, withdraw as indicated
Treat underlying cause
Close SPO2 monitoring

Renal replacemet therapy

Verify Indications

- Symptomatic ARF: Uncontrolled acidosis, or hyperkalaemia, pulmonary edema
- Symptomatic uremia (urea > 35 mmol/l): Encephalopathy,GIT hge, Pericarditis
- Severe sepsis: Developing oliguric renal failure,removal of cytokines / mediators is an unproven indication
- Diuretic resistant pulmonary edema
- Drug removal: Salicylate, methanol, Theophylline, ethylene glycol, lithium
- Other drug overdoses (eg ecstasy/fantasy) associated with severe hyperpyrexia/ rhabdomyolysis / acidosis

Choose modality

Peritoneal dialysis "PD"

- Is less efficient in altering bl solute composition and fluid removal, can be applied continuously
- The absolute **indication** is the inability to perform any other renal replacement technique. Other relative indications are**:**
  - Hemodynamic instability
  - Bleeding diathesis or hemorrhagic conditions
  - Difficulty in obtaining bl access
  - Removal of high MW toxins (>10 kD)
  - Clinically significant hypo & hyperthermia
  - HF refractory to medical management
- **CI** ------most are relative:
  - Recent abdominal ± cardiothoracic surgery
  - Diaphragmatic peritoneal-pleural connections
  - Severe RF
  - Life-threatening hyperkalemia
  - Extremely high catabolism
  - Severe volume overload in ventilated pt
  - Severe GER disease Low peritoneal clearances
  - Fecal or fungal peritonitis
  - Abdominal wall cellulitis
  - Acute renal failure in pregnancy

Hemodialysis "HD"

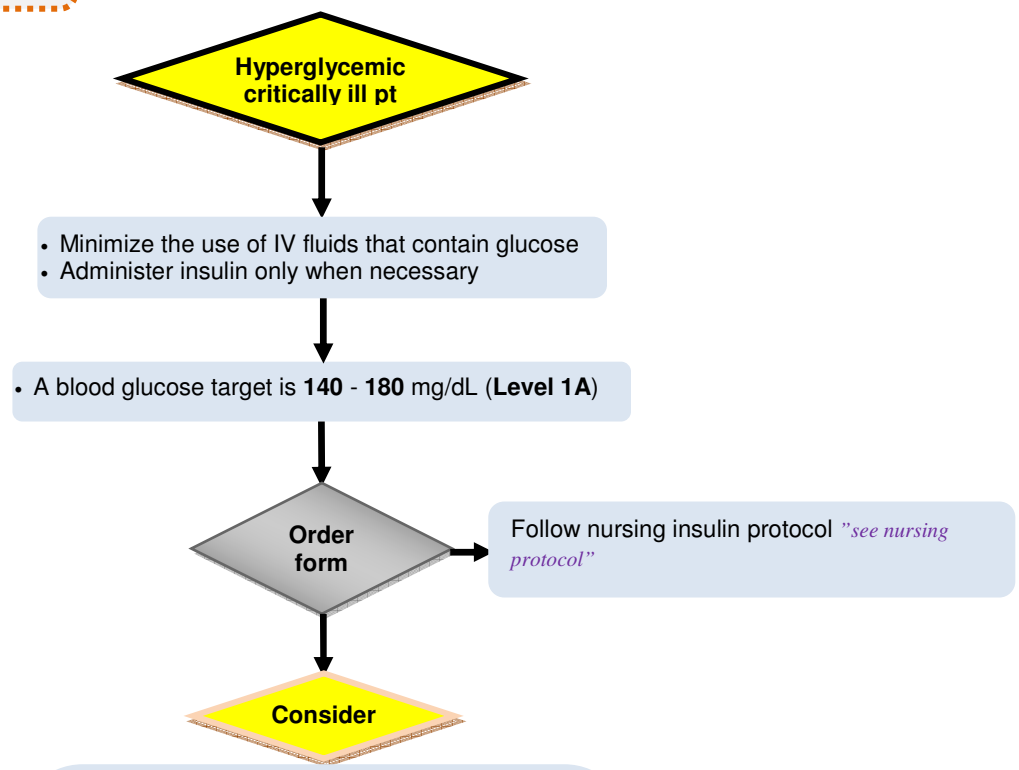
- Rapidly changes plasma solute composition and removes excessive body water compared to the other modalities
- Not tolerated by hemodynamic unstable pt

Continuousrenalreplacementtherapy (CRRT)

- The use of CRRT (**Hemo-filtration**) includes (continuous arterio-venous hemofiltration, continuous VV hemofiltration, continuous AV hemodialysis, and continuous VV hemodialysis)
- The use **VV** circuits rather than **AV** circuits is recommended (**Level 1B**)
- The rate of fluid and solute removal is slow and continuous (better tolerated than HD in pts who are hemodynamic unstable)
- removal of solutes over 24 - 48 hrs is as efficient as HD
- Preferred in pt with sepsis or multiorgan system failure, as it may enhance the removal of cytokines

Glycemic control and intensive insulin therapy

Hyperglycemia is associated with poor clinical outcomes in critically ill pts



Renal protection in critically ill pts

- Pre-existing renal failure
- Hypertensive pts
- Diabetics
- Vasculopaths
- Following crush injury, large contrast media loads
- Diuretic dependent renal function (eg chronic renal failure)

- Close monitoring of serum levels of nephrotoxic drugs
- Adequate fluid repletion in those with hypovolemia
- Aggressive hydration and alkalinization of the urine prior to chemotherapy

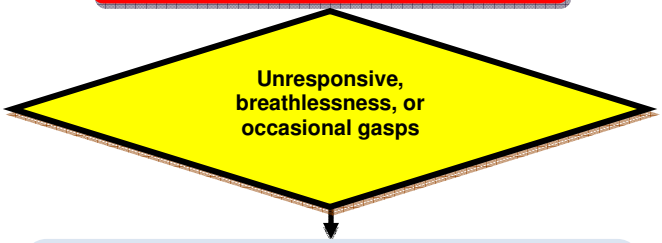
The efficacy of more specific preventive therapy at risk for post-ischemic ATN is unproven

Each of these therapies also is associated with potential toxicity and adverse effects:

- **Mannitol** can result in significant intravascular volume expansion, which with continued renal insufficiency can lead to pulmonary edema, hyponatremia, and acidosis
- **Ototoxicity** is associated with high doses of IV furosemide
- **Low-dose dopamine** associated with tachycardia, arrhythmias & myocardial ischemia
- Although mannitol, furosemide, and low-dose dopamine are sometimes used for prevention of ATN in kidney transplantation recipients, they are not generally recommended in the prevention of ATN (have been evaluated with either inconclusive results or no evidence of benefit)


Advanced life support

ALS universal algorithm



- Check signs of life
- Call for help
- Start CPR **30** compression / **2**vent. “minimize interputtion”
- Attach Defib. monitor

Shockable”VF& pulseless VT”

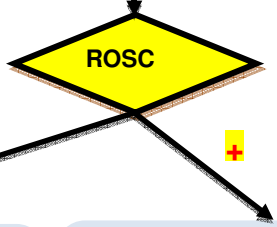
- **1st shock : 150** -**200** bi, **360** monophasic
- **Subsequent shocks : 150** - **360** bi, **360** mono
- If there is doubt about whether the rhythm is asystole or very fine VF, do not attempt defib
- 2 min. **CPR** immediately & minimize interruption”**5** s”
- Pause briefly to check monitor
- **Adrenaline 1** mg is given after 3rd shock, and then / other cycle
- **Amiodarone 300** mg IV after 3rd shock

Non-shockable”Asystole&PEA”

- Immediately resume CPR & minimize interruption
- Adrenaline **1** mg once in hand & IV line in place and / other cycle”3-5 min.” followed by 20 ml NS flush & elevate hand 10- 20 s
- When switching from non-shockable to shockable side, next dose of adrenaline will be given before the 1st or the 2nd shock depend on when adrenaline was last given
- Consider pacing for **Pwave asystole**

During resusc

- Ensure high quality CPR “rate, depth, recoil”
- Consider advanced airway &capnography& O2&No intub.attempt more than 30 s
- Post intubation -- uninterrupted chest compression / ventilation “**100**: **10**”& avoid rapid forceful breath "1 s Ti"
- Consider vascular access “IV or intra-osseous”
- Correct reversible causes “**4H**, **4T**”
  - Hypoxemia, hypovolemia, hypothermia, hypo, hyperkalemia,,and metabolic disorders
  - Thrombosis, cardiac, tamponade, tension pnemothorax,and toxins”



- Consider cessation of resusc. in asystole after 20 min, without reversible cause “esp. if ETCO2 >**10** mmHg”
- Consider resusc, for 1.5 hr after thrombolytic “suspected or proven PE”

- Use ABCDE approach
- Controlled oxygenation & vent.
- 12 lead ECG
- CXR
- Treat precipitating cause

- Ventilate **10** b/min., check pulse / min. in respiratory arrest + pulse

Defib.

- Determine if it is mono or biphasic to detect energy level
- Position: Apico-pericardial"put any paddle on any area df them, do not rely on what is written on the paddles"
- Ensure safety: "No part of any person should make contact with pt directly or indirectly, free O2 1 meter away, dry skin, gloves, and consider a distance of 8 cm from ICD
- Ensure efficacy: shave chest with no delay, minimize PEEP, deliver at end-expiration, increase with emphysema

Tamponade

- Difficult to be diagnosed during cardiac arrest
- Diagnosed just by history, US
- Consider centesis or thoracoomy

Pericordial thumb

- An appropriate therapy for witnessed monitored arrest , and defib. Is not available when several clinicians are present
- Succesful only if performed in the first few seconds of onset
- By a trained health care professional using the ulnar edge of a tightly clenched fist giving a sharp impact over the lower half of the sternum at a height of **20** cm

IV access

- Consider inter-osseous access if failed cannulation for **2** min

Mg

- Give Mg (**2**g IV bolus) for shock-refractory VF if there is any possibility of hypomagnesaemia (e.g., diuretic use)

NaHCO3

- Indicated in
  - Life-threatening hyperkalaemia
  - Tricyclic overdose
  - Arterial pH **< 7.1**
- **50** ml 8.4% cardiac arrest associated with NaHCO3 IV
  - Repeat the dose according to the clinical condition and result of repeated ABG
- Central venous blood gas analysis provides closer estimate of tissue acid base state than arterial one
- Ca solutions & Na HCO3 should not be given simultaneously by the same route

Lidocaine

- Lidocaine**100** mg IV (or 1-1.5 mg kg may be used as an alternative if amiodarone is not available
  - Give an additional bolus of **50** mg if necessary
  - Do not exceed **3** mg kg during 1 sthr
- Do not give if amiodarone has been given already

Ca

- Indicated in PEA caused by
  - Hyperkalemia &Hypermagnesaemia
  - Hypocalcaemia
  - Ca channel blocking overdose
- The initial dose of **10** ml 10% Cacl (6.8 mmolCa) repeat if necessary

K

- **20**mmol/min. for 10 min., then, **10**mmol in 10 min

Fluids

- If hypovolaemiais suspected
- Use 0.9% NS or RL. avoid D5

Pulse check

- Pulse check should be undertaken if organized rhythm is observed
  - If organized rhythm is seen during the 2-min. CPR, do not interrupt CPR for pulse check unless there are signs of life
  - Check ETCO2 if available

Intub.& Vent.

- Pause CPR “**10** S” during tube pass through vocal cord only
- Attempt uninterupted**100**/**10** compression/vent. with supraglotic device unless excess leak is observed ---- shift to **30**:**2**

Airway mangement

Difficult airway "DA"algorithm

Awake techniques
- FOB or direct laryngoscopy & Tracheostomy
- Consider judged sedation **±LA**

Predicted DV
+
-

Plan A
Initial intubation attempt"3"

Succeed

Verify placement
- Visual
- Capnography
- EDD

If you doubt -------Take it out

Failed

Plan B
Intub. alternatives

Succeed

Failed

Consider**awakening**if possible

SPO2 OK
+
-

Consider
- **Awakening** if possible
- Arrange for **awakeintubation**

OK

Plan C
Maintain BVM vent.

Failed

Consider
- **Awakening** if possible
- Arrange for **awakeintub.**
- Intub. through LMA "use FOB, and **aintree** catheter"

- Successful LMA" Max: 2 attempts"
- Consider cricoid release

Plan D
Failed airway

- Temporary **Needle Crico-thyrodotomy**
- Arrange for definitive **surgical crico-thyrodotomy**

Initialintb. attempts
- 1st--- Best look, BURB, Bougie
- 2nd, 3rd --- Better person, Blade change, Better pharmacology

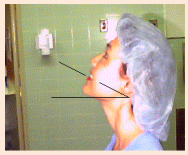
Intub. alternatives
- Options: ILMA, Optical scopes & stylets, videoscopes, etc.
- Choose the available device, and that is you prefer and master

Mask vent.
Attempt mask vent. If oxygenation is not OK at any time

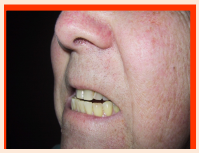
Consider
- Reverse NDMR
- 2 persons mask technique
- Excess head tilt/chin lift if no CI
- Insertion of oro and/or nasopharyngeal airway
- Cricoid pr release
- Mask change(size or type) if seal is an issue
- Ruling out FB
- Rescue devices
- Early attempt at intub.

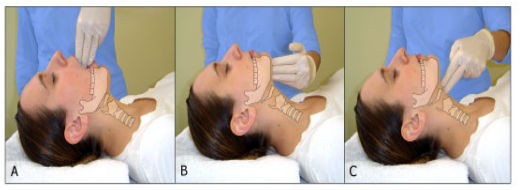
Failed vent.
- Signs of inadequate mask vent. include (but not limited to) absent or inadequate chest movement or breath sounds, auscultatory signs of severe obstruction, cyanosis, gastric air entry or dilatation, decrease or inadequate SpO2, absent or inadequate ETCO2, and hemodynamic changes (*e.g.* tachycardia, arrhythmia)

Neck extension "Quick test"



Anterior jaw subluxation





The spatial relationships depicted here are important determinants of successful direct laryngoscopy. A) The patient can open his/her mouth sufficiently to admit three of his/her own fingers. B) The distance between the mentum and the neck/mandible junction (near the hyoid bone) is the length of three of the patient's fingers. C) The space between superior notch of thyroid cartilage and the neck/mandible junction, near hyoid bone, is the length of 2 of the pt's fingers

5 Predictors of DMV and Oxygenation "OBESE"
- **O**bese(BMI > 26 kg/m2)
- **B**earded
- **E**lderly(>55 y)
- **S**norer
- **E**dentulous

Predictors of DI and laryngoscopy, "Magboul 4 M & Ms"
- **MM** = **MallampatiM** = **Measurements** 3-3-2-1 or1-2-3-3 Fingers, **M** = **Movement** of the Neck "quick test", **M** = **Malformations** of the skull, teeth, obstruction, and Pathology (the Macros and Micros) Which are also summarized in the word (**STOP**) **S** = **Skull**, **T** = **Teeth**, **O** = **Obstruction**, **P** = **Pathology**

Extubating difficult airway (DA)

DA

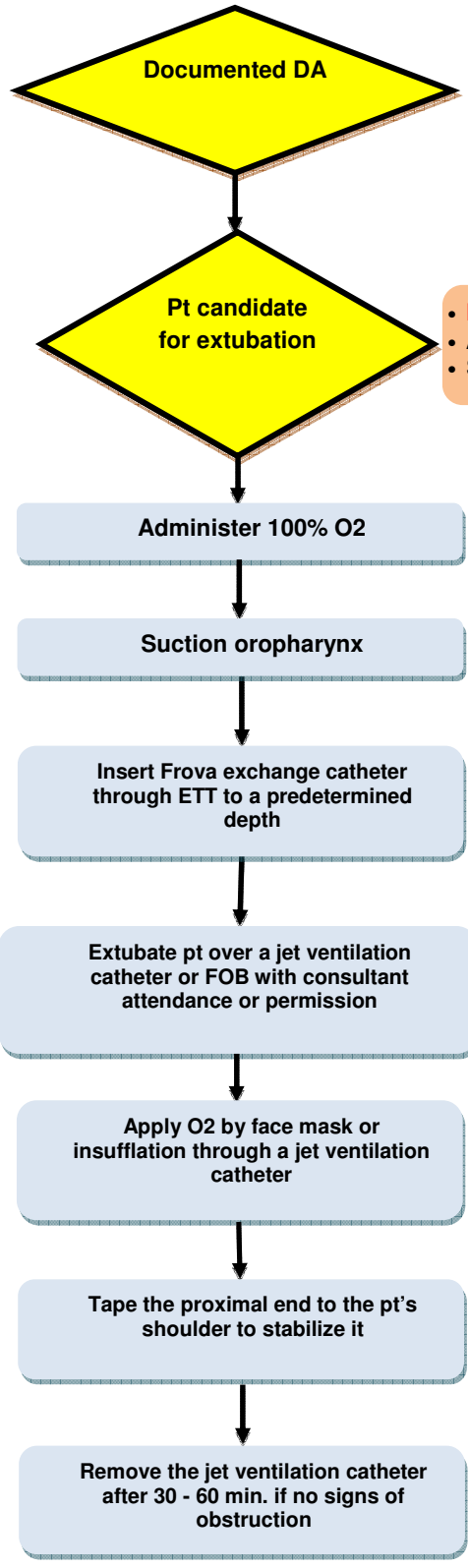
- Clinical situation in which a conventionally trained anesthetist experiences difficulty with B MV of the upper airway, difficulty with tracheal intubation, or both

Difficult BMV

- When it is not possible for the unassisted clinician to maintain the SaO2 > 92% using 100% O2 and positive pr mask ventilation in a pt whose SaO2 was > 90% before clinician intervention

DI

- One that requires multiple attempts, operators, ordevices, excessive lifting force, ELM, or is performed with an inadequate glottic view



Emergent airway management

Pt needs intub.

\+

Pt near death

\+ (left) / \- (down)

Prepare & Oxygenate

O2 ± BVM ± oral airway ± nasal airway

Difficult BVM predicted

\+ (right) / \- (down)

DI predicted

\+ (down) / \- (right)

Pt needs intubation

- To obtain airway "patency"
- To protect airway
  - GCS **< 8**
  - Do not rely on gag reflex
- Failure of oxygenation ± vent. and not candidate for NIV
- Anticipated clinical course "inhalation injury& pre-transport"

Crash induction

- BVM ventilation
- 2 intubation attempts (Use **Succinylcholine 2**mg/Kg in the 2nd, or **Rocronium1** mg/Kg)

Awakelook

- Used when the clinician suspects the airway will be difficult to intubate. Allows practitioner to verify that laryngeal structures are visible, before paralysis
- With light to moderately sedation "**ketamine** ", and intubated using topical anesthesia" not induced nor paralyzed**"**
- Ketamine may not be appropriate with cardiovascular dis. or hypertension

Awake techniques

- Awake FOI
- Awake tracheostomy ±LA
- Retrograde intub.

C- instability

- Consider cervical spine instability even if the initial plain cervical spine x-rays are normal
- **Consider:**
  - C- stabilization, Hard or semi-rigid collar
  - MILS

- **Awake technique** still an option
- **Double setup**"RSI + backup plan "eg, **ILMA**&**AirQ&glides cope**"
- Consider**Awake look**

RSI"Cricoid pressure"

Consider

- Gentle BVM ventilation if de-saturated
- Spontaneous breathing in airway obstruction & trauma, cardiac tamponade.
- Hard collar, **MILS** in **C-instability**
- Possibility of **Difficult physiology**

SPO2 Affected at any time → **BMV ventilation** →

Failed airway

- Attempt **LMA** in parallel with preparations for the **surgicalairway "temporary Needle crico-thyrotomy,** thendefinitive **Surgical crico-thyrodotomy"**

Spinal cord injury

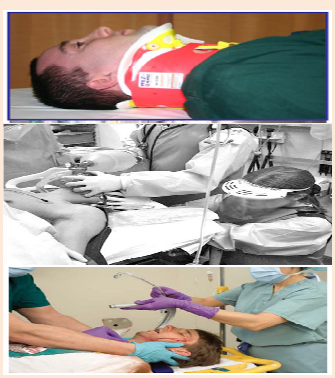
Consider

- MIL

Options

- Awake FOI
- (RSI & Double set-up)

Cervical stability



Morbid obesity

- De-saturate quickly
- Difficult BMV, intub. Laryngoscopy, andcrico-thyrotomy
- Best method for BMV is 2-person technique with oro-pharyngeal and naso-pharyngeal airways, unless CI
- Pre-oxygenate in an upright or semi-upright position and place pt in ramp position (align auditory meatus and sternal notch) if C- spine is ok
- When performing RSI ,use LBW to determine dose of the induction agent (**Level 2C**),IBW to determine the dose of rocuronium (**Level 2C**), total body weight for succinylcholine (**Level 2B**)
- May be prudent to intubate awake

Facio-maxillary trauma

Options

- Awake FOB "remember blood is the enemy of FOB"
- Surgical airway
- (RSI & Double set-up)

Laryngo-tracheal disruption

Consider

- Maintain Spontaneous breathing
- "Do not pressurize proximal to tear"
- Get ETT below tear
- No TTJV
- No LMA

Option

- Awake FOI (laryng, trach)
- Modified RSI (supralaryng)
- Surgical airway (supralaryng)
- Direct tracheal intubation (trach.)
- DLT & CPB (bronch.)

TBI

Consider

- Physiologic difficulty
- C-spine instability

Options

- Awake FOB
- RSI & Double set-up
- Do not crash

Physiologic difficulty

TBI

- Proper assessment of airway may be difficult
- There is often a known or potential C- spine injury
- Intubator is faced with time pressures
- CPP should be maintained
- Intubate the patient with no decrease or increase in blpr using drugs which will decrease the ICP while avoiding any hypoxic episodes and controlling CO2 by adequately ventilating the patient
- Use **Etomidate** for induction of these pts For hypotensive patients, **Etomidate** or **ketamine** may be used
- Ketamine should be avoided in ps with hypertension or if elevated ICP is caused by spontaneous cerebral hge

Ischemic heart disease"IHD"

- Pts may have decreased ventricular function and will manifest an exaggerated response to myocardial depressant agents (pts tend to be older and are also more sensitive to induction agents)
- Maintain a favorable myocardial O2 supply-demand balance
- Avoid Tachycardia and wide swings in bl pr.
- Attenuate the normal cardiovascular physiologic responses to laryngoscopy and intubation

Asthma

- ETT intubation is a potent stimulus for bronchospasm
- In severe cases it can take pr>**50** cm H20 to move even small amounts of air into lungs
- Blunt airway reflexes as possible prior to airway manipulation
- For the hemodynamically stable pt with, induce with **ketamine** or **propofol**. **Etomidate** or **midazolam** are acceptable alternatives
- In hemodynamically unstable pts , induce with **ketamine** or **etomidate.Thiopental** should not be used
- Hypotension is not unusual following intubation
- Avoid excessively advancing ETT
- Allow adequate time for exhalation

Shock states

- In profoundly shocked pts even very small doses of sedatives can have hemodynamic effects of full induction doses in healthy pts
- In very severe hypotension the pt will be obtunded and crash airway can done
- If an awake approach is either CI or deemed not feasible and RSI is elected
- **Ketamine** and **Etomidate** may offer potential advantages in maintaining bl pr.
- A small amnestic dose of **Midazolam** (**0.5-2**mg) may help prevent recall
- Manage post-intubation hypotension with a fluid and short acting pressors

Status epilepticus

- For RSI of pts in status epilepticus, **midazolam** or **thiopental** be used for induction
- Reduced doses should be used in the unusual circumstance of seizure with hypotension
- **Etomidate** may be used when the pt manifests hemodynamic compromise
- **ketamine** not be used because of its stimulant effects

Rapid sequence induction "RSI"

- Virtually simultaneous administration of a sedative and paralytic agent to render a Pt rapidly unconscious and flaccid in order to facilitate emergent intub. and minimize risk of aspiration
- Pre-oxygenation is required to permit a longer period of apnea without clinically significant O2 desaturation
- BMV is avoided between drug administration and intub.

Pre-oxygenatation

- Achieved by high flow O2 via a non-rebreather mask for 3 - 5 min.If time allows
- If time not allow, pt take 8 vital capacity breath/min. or 4 in 1/2 min.
- BMV is unnecessary, even between laryngoscopy attempts. If SPO2 **<90**%, or is unknown, clinicians provide ventilatory support using a **Gentle** bag-mask, with cricoid pr.

Cricoid pr

- Applied immediately following induction and maintained until adequate tube placement
- Light pr before loss of consciousness "**10** N. **3O** Nafter loss of consciousness = comparable to the pr that would feel uncomfortable if applied to the nasal bridg"
- Sometimes it may obscure glottic view, impede passage of ETT, or prevent adequate vent.(if necessary). If so, reduce pr

Time 0 min.

In a pt weighing 75 kg with Sblpr 140

- **Pre-oxygenate** while **preparing**" at least 1, but preferably 2, functioning IV line, cardiac, pulse oximetry, and blpr monitors in place, necessary airway and resusc. equipment are available
- **Position**" airway manager should have easy access to head of bed, and should adjust the height of bed and position of the pt

Time 3min.

- Consider pre-treatment +/-
  - **Lidocaine100** mg (TBI)
  - **ThiopentalFentanyl200** mcg (IHD) +/-
  - **Pancuronium1** mg if using succinylcholine (TBI)

Time 5 min.

- Pharmacologic induction
  - **Propofol150** mg Or
  - **Tiopental250** mg Or
  - **Ketamine120** mg Or
- Followed immediately by Muscle Relaxation
  - **Succinylcholine120** mg*or*
  - **Rocuronium80** mg
- **Cricoid pr** applied when pt rendered unconscious

Time 5 3/4 min.

Failed intubation during RSI

- **-** → **Verify placement**
- **+** → **Call for help**

Succeed

- Try alternative blade
- Limited amount of external laryngeal manipulation possible with cricoid force
- Use bougie
- Do not give further succinylcholine
- No more than 3 attempts
- Maintain
  - BVM Vent.
  - Anesthesia
  - Cricoid prrelease if poor view

Fail

- Maintain cricoid pr
- Insert oral airway or nasopharyngeal airway **+** mask vent. with 100% O2
- 2 handed mask vent.

SpO2 OK

- **+** → **Consider**
  - **Awaken**pt
  - Arrange for **awakeintubation**
- **-** → **Succeed**

Consider

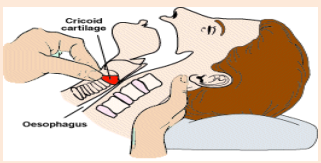
- LMA
- Release cricoid pr. during insertion
- Oxygenate & ventilate

Fail

Failed airway

PlacetheETT withstyletinside

Cricoid pr



Renal &Electrolytes

&

Acid base disorders

Acute kidney injury "AKI"

- Formerly referred to as **Acute renal failure** (ARF)
- Abrupt loss of kidney function that results in retention of urea & other nitrogenous waste products and in the dysregulation of ECF & electrolytes
- The loss of kidney function is most easily detected by measurement of the serum creatinine

The causes of AKI are categorized as follows:

- **Prerenal azotemia** results from either volume depletion (hypovolemia), decrease effective arterial pr (shock), or effective circulating volume (HF)
- **Intrinsic renal disease** includes disorders that involve the renal vascular (arterial or venous thrombosis), glomerular (glomerulonephritis), tubular (ATN), or interstitial pathology
- **Postrenal AKI** is due to bilateral urinary tract obstruction unless there is a solitary kidney.

* Increased abd. pr probably exerts its effect at all above levels: consider decompression if>20 mmHg

Suspect

- Suggestive history 'hypovolemia, sepsis,etc.&Ph E esp., oliguria in susceptible pts

Evaluate

- Detailed history & Ph E can detect specific signs or symptoms for the underlying disorder
- **Urine analysis**
- Serum **Creatinine**to estimate GFR
- **Lab.:**differentiate between prerenal ARF and ATN

	Prerenal ARF	ATN
Urine Na (meq/L)	< 20	> 30
Fractional excretion of Na	< 1%	> 2%
Urine osmolality (mOsm/L)	< 350	> 500
Serum BUN/Cr ratio*	> 20 : 1	< 20 : 1

** Used only in adolescents and older children*

- Renal **US** document presence of 1 or 2 kidneys, delineate renal size, surveys renal parenchyma, and detect urinary tract obstruction and occlusion of major renal vessels
- An IV fluid challenge should be administered to detect pre-renal AKI that may progress to ATN

:I ---- Obvious volume overload or HF

- **Renal biopsy**: mostly in pts with suspected glomerulo-nephritis or those with otherwise unexplained AKI

ATN = acute tubular necrosis, ARF =acute renal failure, BUN = blood urea nitrogen

Classify

STAGe	Serum creatinine criteria	Urine output criteria
1	Increase in serum creatinine of ≥0.3 mg/dL or increase to ≥150 - 200 % (1.5- 2-fold) from baseline	< 0.5 mL/kg / hr for > 6 hrs
2	Increase in serum creatinine to >200 - 300 % (>2- 3fold) from baseline	< 0.5 mL/kg / hr for > 12 hrs
3	Increase in serum creatinine to >300 % (>3-fold) from baseline (or serum creatinine of ≥4.0 mg/dL with an acute increase of at least 0.5 mg/dL	< 0.3 mL/kg / hr for 24 hours or anuria for 12 hrs

* Modified from RIFLE (Risk, Injury, Failure, Loss, and End-stage kidney disease) criteria. The staging system proposed is a highly sensitive interim staging system and is based on recent data indicating that a small change in serum creatinine influences outcome. The diagnostic criteria should only be applied after volume status has been optimized. Only one criterion (creatinine or urine output) has to be fulfilled to qualify for a stage.
200 - 300 % increase = 2- to 3-fold increase.
Given wide variation in indications and timing of initiation of renal replacement therapy (RRT), individuals who receive RRT are considered to have met the criteria for stage 3 irrespective of the stage they are in at the time of RRT

RRT *"p24"*

Manage

General principles

- Avoid life-threatening complications
- ttt underlying cause
- Exclude post-renal obstruction
- Avoid nephrotoxic drugs, esp: Aminoglycosides, Amphotericin, Contrast media, NSAIDs
- Careful prescription and monitoring of gentamicin and vancomycin where indicated
- Prompt ttt of inter-current inf.
- Adequate nutritional support

Maintenance of electrolyte and fluid balance

Hyperkalemia*"p43"* ,**Hyperphosphatemia***"p47"*,**Hpocalcemia***"p48"*&**Acidosis***"p36"*

Intravascular volume

- May be hypovolemic, euvolemic, or hypervolemic (pulmonary edema& HF)
- A pt with a clinical history and pulmonary edemaconsistent with fluid loss, and/or oliguria ------ immediate IV fluid therapy
- If UOP does not increase and renal function fails to improve with the IV fluids ----- invasive monitoring
- An edematous hypertensive pt with a history of oliguria, and/or signs of HF ----- immediate fluid removal and/or fluid restriction (trial of furosemide (**2** - **5** mg/kg / dose) to convert oliguric to non-oliguric RF & should not be continued in an unresponsive pt
- **Low-dose dopamine and mannitol not recommended**
- If a diuresis does not ensue and/or the pt has evidence of fluid overload with pulmonary edema, RRT should be initiated
- Once euvolemia has been obtained, the clinician must pay careful attention to ongoing fluid losses (insensible water loss of approximately 300 - 500 mL/m2 / d in addition to replacement of urine and GI losses) and gains (fluid administered for nutritional and medical requirements)
- Ongoing fluid balance evaluation is aided by daily weights, accurate records of fluid inputs and outputs, and findings on Ph E

Respiratory acidosis

- 1ryincrease in $PaCO_2$
- $PaCO_2$ represents the balance between CO_2 production and CO_2 elimination:
  - CO_2 production is a byproduct of fat and CHO metabolism. Muscle activity, body temp., and thyroid hormone

Suspect

- Pt in RFor impending failure
- Clinically obvious hypo-ventilated pt

Confirm

Draw ABGs

Detect simplicity

Simple

- Plasma HCO_3increases **1** mEq/L /**10** mm Hg increase in $PaCO_2$>**40** mm Hg in **acute** acidosis
- Plasma HCO_3increases **4** mEq/L /**10** mm Hg increase in $PaCO_2$>**40** mm Hg in **chronic** acidosis

- Compensatory response to acute (**6–12 h**) elevations in $PaCO_2$ is limited
- Buffering is primarily provided by Hgb and the exchange of extracellular H^+ for Na^+ and K^+ from bone and the intracellular fluid compartment
- Renal response to retain more HCO3 is very limited acutely
- Full" renal compensation characterizes chronic respiratory acidosis
- Renal compensation is appreciable only after **12–24 h** and may not peak until **3–5 ds**

Detect acuity

Acute

- PH unaccepted

Chronic

- PH accepted "**7.3-7.5**"

Review Causes

ttt

- Reduce**CO_2 production**in specific instances: Dantrolene for malignant hyperthermia, muscle paralysis for tetanus, antithyroid medication for thyroid storm, and reduced caloric intake
- ttt of the **underlying cause:** Bronchodilation, reversal of narcosis, or improve lung compliance "diuresis'
- **Intubate**&**Ventilate** or initiate **NIPPV** as indicated
- In **acute acidosis**; consider
  - Not to increase VT >**8**ml/Kg IBW
  - PEEPi when increasing RR
  - Permissive hypercapnea in ALI&ARDS
- In **chronic** acidosis: consider
  - If acute ventilatory failure developed -- -- return $PaCO_2$ to pt's baselinem. Normalizing $PaCO_2$ will result in respiratory alkalosis

    2 *HCO3 – 8 "*Radwan 96*"
    - CO2 that gives PH "7.35-7.39"
    - If in doubt PaCO2 **60** mmHg
  - O2 therapy must also be carefully controlled" respiratory drive may be dependent on hypoxemia, not $PaCO_2$; or may increase physiological DS.Normal$PaCO_2$ or relative hyperoxia can precipitate severe hypoventilation"

Causes

Alveolar hypoventilation

CNS depression
- Drug-induced Sleep disorders
- Obesity hypoventilation S
- Cerebral ischemia& trauma

Neuromuscular disorders
- Myopathies
- Neuropathies

Chest wall abnormalities
- Flail chest
- Kyphoscoliosis

Pleural abnormalities
- Pneumothorax
- Pleural effusion

Airway obstruction

Upper airway
- FB
- Tumor
- Laryngospasm
- Sleep disorders

Lowerairway
- Severeasthma
- COPD
- Tumor

Parenchymal lung disease
- Pulmonary edema
- Pneumonia Aspiration
- Interstitial lung disease

Ventilator malfunction

Increased CO_2 production
- Large caloric loads
- Malignant hyperthermia
- Intensiveshivering
- Prolonged seizure
- Thyroid storm
- Extensive thermal injury

Respiratory alkalosis

- Primary decrease in **$PaCO_2$**
- The mechanism is usually an inappropriate increase in alveolar ventilation relative to CO_2 production

Suspect

- Pt in repertory failure or impending failure.
- Clinically obvious tachypnea.

Confirm

Draw ABGs

Chronic

Plasma HCO_3decreases**2-5** mEq/L /**10** mm Hg decrease in $PaCO_2$<**40** mm Hg

Acute

Plasma HCO_3decreases**2** mEq/L /**10** mm Hg decrease in $PaCO_2$<**40** mm Hg

Review Causes

Causes

Central stimulation
- Pain
- Anxiety
- Ischemia
- Stroke
- Tumor
- Inf.
- Fever
- Drug-induced
  "SalicylateProgesterone (pregnancy) Analeptics (doxapram)"

Peripheral stimulation
- Hypoxemia
- Highaltitude
- Congestive HF
- PE
- ARDS
- Asthma
- Severe anemia

Unknown mechanism
- Sepsis
- Metabolic encephalopathies

Iatrogenic

ttt

- Correctunderlying process is the only ttt
- Consider :
  - Increase physiological dead space.
  - **Ca^{++}**: if tetany occurred

Metabolic acidosis

- Primary decrease in HCO_3^-
- Mechanisms:
  - Consumption of HCO_3^- by a strong nonvolatile acid Renal or GI wasting of HCO3
  - Rapid dilution of ECF with a HCO3-free fluid
- Pulmonary compensatory in simple acidosis does not reduce $PaCO_2$ to a level that totally normalizes pH but cause hyperventilation

Suspect

- Septic, renal failure, diabetic ketoacidotic pt
- Hemodynamic unstable pts & cardiac arrest
- Shock refractory to pressors and /or inotropes

Confirm

ABGs

- PH <**7.4**
- 1ry↓ in HCO_3^-

Detect simplicity

- Predicted decrease in**$PaCO_2$**

1.5 x HCO_3 + 8 ± 2

- Every **6** mEq change in HCO_3 changes arterial pH **0.1** in same direction

Detect compens.

Compensated
- PH accepted "**7.3-7.5**"

Uncompensated
- PH unaccepted
- PaCO2 accepted "**30-50**"

Partially compensated
- PH unaccepted
- PaCO2 unaccepted

AG& Delta ratio &BE& Standard HCO3

Normal AG

- **Increased GI losses of HCO_3^-**
  - Diarrhea - Anion exchange resins
  - Ingestion of $CaCl_2$, $MgCl_2$ - Fistuls
  - Ureterosigmoidostomy or obstructed ileal loop
- **Increased renal losses of HCO_3^-**
  - Renal tubular acidosis - Carbonic anhydrase inhibitors - Hypoaldosteronism
- **Dilutional**
  - Large amount of HCO3-free fluids
- **TPN (Cl^- salts of amino acids)**
- **Increased intake of CL-containing acids**
  - Ammonium, Lysine,and Arginine hydrochloride

High AG

- **Increased production of endogenous nonvolatile acids**
  - Renal failure
  - Ketoacidosis (Diabetic , Starvation)
  - Lactic acidosis
  - Mixed (Nonketotic hyperosmolar coma, Alcoholic)
- **Inborn errors of metabolism**
- **Ingestion of toxin**
  - SalicylateMethanol - Ethylene glycol - Paraldehyde - Toluene - Sulfur
- **Rhabdomyolysis**

Delta ratio

Increase in AG/ Decrease in HCO3

ratio	Assesment guidelines
< 0.4	Hyperchloraemic normal anion gap acidosis
0.4 - 0.8	Consider combined high AG & normal AG acidosis BUT note that the ratio is often **<1** in acidosis associated with RF
1 - 2	Usual for uncomplicated high-AG acidosis. Lactic acidosis: average value **1.6** DKA have a ratio closer to **1** due to urine ketone loss (esp if pt not dehydrated)
> 2	Suggests a pre-existing elevated HCO_3 level so consider:a concurrent metabolic alkalosis, or a pre-existing compensated respiratory acidosis

Detect associated alkalosis "Delta gap"

ttt

- Controlled ventilation if needed
- **Alkali therapy**
- **Specific therapy "** correct cause"
  - **Diabetic ketoacidosis**--- fluids, insulin
  - **Lacticacidosis**---- restore oxygenation & perfusion
  - **Salicylatepoisoning---**urine alkalinization

Anion gap "AG"

AG = Na + K - Cl + HCO3

- Reference range is **8 - 16 mmol/l**
- Every **1** gm decrease in albumin will decrease AG by **2.5** - **3** mmoles
- **>30 mmol/l**-- metabolic acidosis
- **20-29 mmol/l**, about1/3 of will not have a metabolic acidosis

Alkali therapy "NaHC03"

- **Indications**
  - Hypotension refractory to volume & inotropic support
  - PH <**7.2**" normal AG acidosis"
  - Emergent hyperkalaemia
  - Promote alkalinediuresis(eg to hasten salicylate excretion)
  - No routine use in cardiac arrest and low flow states
- **Dose**
  - (**1** mEq/kg) or **30% bw * BE**
  - Give 50% of calculated dose---serial ABGstoguide therapy
  - Target pH **> 7.25** is sufficient
  - Refractory acidemia ----- HD
- **Molar solution = 84 g/l (8.4 %)**
  - **1 mmol/ml**
- **Undesirable effects of HCO3**
  - Hypokalaemia, hypernatraemia, and hyperosmolality
  - Volume overload
  - Rebound or overshoot alkalosis
  - Impaired O2 unloading
  - Accelerate lactate production
  - Hypercapnia
- **Incompatible with** Dobutamine-Insulin-Mg-Dopamine-Epinephrine

Metabolic alkalosis

- Primary increase in plasma HCO_3^-
- Most cases of can be divided into
  - Those with NaCl deficiency and ECF depletion"**chloride sensitive**"
  - Those with enhanced mineralocorticoid activity "**chloride resistant**"

Suspect

- Hypokalemic, hypoalbuminemicpts
- Pts on diuretics

Confirm

ABGs

- PH **>7.4**
- 1ryin↑ HCO_3^-

Detect simplicity

- Predicted decrease in **$PaCO_2$**

0.8*HCO3+20 + 1.5

- Every **6** mEq change in HCO_3 changes pH by **0.1** in same direction

Detect compens.

Compensated
- PH accepted "**7.3-7.5**"

Uncompensated
- PH unaccepted
- PaCO2 accepted "**30-50**"

Partially compensated
- PH unaccepted
- PaCO2 unaccepted

BE& Standard HCO3

Urinary Cl

Chloride resistant
Increased mineralocorticoid activity
- 1ry hyperaldosteronism
- Edematous disorders (2nd hyperaldosteronism
- Cushing's syndrome
- Licorice ingestion
- Bartter's syndrome

Severe hypokalemia

Chloride sensitive

GI
- Vomiting Gastric drainageChloride diarrhea

Renal
- Diuretics**Posthypercapnic**Low chloride intake

Sweating
- Cystic fibrosis

Search for

-Massive blood transfusion - Alkali therapy
-Hypercalcemia

Detect associated acidosis

ttt

- Correct **underlying disorder**
- Correct any respiratory component contributing to alkalemia "**decreasingVE** to normalize $PaCO_2$"
- AdministerIV **NaCl**&**KCl**for Cl-sensitive metabolic alkalosis

30% BW * (100 – chloride)

- **H_2-blocker therapy** when excessive loss of gastric fluid is a factor
- **Spironolactone** for Alkalosis associated with increased mineralocorticoid activity
- **Acetazolamide**
- When arterial blood pH is >**7.60**
  - IV**hydrochloricacid** (**0.1**mol/L)
  - **HD**should be considered

Base Excess"BE"

- The amount of acid or base that must be added for blood pH to return to **7.40** and $PaCO_2$ to return to 40 mm Hg at full O_2 saturation and 37°C
- +ve value indicates metabolic alkalosis, whereas a -Ve value reveals metabolic acidosis

Standard HCO3

- Similar to BE
- Calculated HC3 concentration of sample corrected to a PCO2 of **40**mmHg
- Raised concentration indicates a metabolic alkalosis whilst a low value indicates a acidosis

Hydrochloric Acid

- Infusion has proven to be safe and effective in correcting severe metabolic alkalosis.
- Needs central venous access because "avoid vein sclerosing
- **Dose**

50% BW * BE

- **Volume (L) of 0.1 "HCL=H^+ Deficit / 100*"**
  - **0.25 N HCL= H^+ Deficit / 250***
  - **Infusion Rate= 0.2 mEq/kg / hr**

Acetazolamide

- Carbonic anhydrase inhibitor
- More acceptable therapeutic option than hydrochloric acid
- Only 1 - 2 doses should be used "**250-500** mg IV"
- Appropriate adjustments in IV fluids and K^+ supplementation are necessary

Pts with COPD

- If the pH is ≥ **7.4**, there must be an element of metabolic alkalosis.In this situation, check $PaCO_2$
- If **$PaCO_2$ is appropriate**(2 x HCO_3 – 8). The cause of metabolic alkalosis is due to high level of HCO_3. " **independent alkalosis** "and usually of the chloride resistant type where urine chloride is > 20 mEq/L
- If **$PaCO_2$<(2 x HCO_3 – 8)**. Urine Cl is usually < **20** mEq/L "**post-hypercapnic metabolic alkalosis**"

Hyponatremia

Normal Serum Na **135** – **145**mEq / L

100 mg/dl of glucose causes 1.6 mmol/L fall in Na

Suspect — Disturbed consciousness ±seizures in susceptible pt

≤ 135 mEq/L — **Serum Na**

Plasma osmolarity

Isotonic "**280**-**295**msom/Kg"
- Hypertriglycedemia

Hypertonic">**295**msom/Kg"
- Hyperglycemia
- Effective osmoles infusion or absorption "mannitol, glycerol"

CSW
- In CNS dis., particularly SAHcharacterized by ECF depletion
- Due to inappropriate Na wasting in urine

Hypotonic" ≤ 280msom/Kg"

Urine osmolarity

<**100**msom/Kg"excess water intake"
- Primary polydypsia
- Malnutrition
- Reset osmostates

>**100**msom/Kg"impaired renal concentrating ability"

ECF

Decreased
Urinary Na >20 mEq/L
- Renal solute loss "diuretics, osmotic diuretics"

Urinary Na <10 mEq/L
- Extra-renal solute loss "skin, GIT"

Increased
Urinary Na >20 mEq/L
- Renal failure

Urinary Na <10 mEq/L
- HF – Cirrhosis - Nephrotic syndrome

Normal
- K depletion
- Hypothyroidism, glucocorticoid deficiency
- **SIAD** *"p39"*

- Azotemia has low effective serum osm, and high measured osm "urea is an ineffective osmoles"

Corrected Osm =measured Osm-Urea/2.8 "2.8 convert mg/dl tommol/dl"

Assess

- **Symptoms** (seizures, coma)
- **Degree** and **rapidity** of decrease
  - **Acute** (< 48 hrs):**≤125mEq/L**"nausea, lethargy, headache,"& **≤115mEq/L** "Seizures & coma"
  - **Chronic** (> 48 hrs):Often asymptomatic

ttt

General principles
- **Formula**desired – measured Na * TBW "Male:0.6 BW Female:0.5 BW old female 0.4'
- **Infusate**
  - Without hypokalemia: Change in seum Na = Infusate Na +infusate K – serum Na/ TBW +1
  - With hypokalemia Change in seum Na = Infusate Na– serum Na/ TBW +1
- Correction should not exceed (to avoid central pontinemylenonecrosis)
  - 10-12 meq/1st 24h
  - 18-20 meq/2nd 48h
- Reset osmostat---- do not correct hyponatremia--- will cause severe thirst

Symptomatic Hyponatermia
Acute < 48 hrs:
- **Hypertonic Saline** 3% **1-2**meq/hr(for 2-3 hrs initially) or symptoms resolves
- **Frusemide**

Chronic > 48 hrs or Unknown:
- **Hypertonic Saline(0.5 -1.0**meq/hr (till 120-130 meq/l)
- **Frusemide**
- Change to water restriction
- Frequent assessment

CSWS
- Volume repletion with NS (remove stimulus to ADH release, resulting in a dilute urine & correct hyponatremia)
- Give mineralocorticoid
- Long-term therapy is not necessary

Fluid (infusate)	Na Content mEq/L	Na Concentration / mL
Lactated Ringers	130	0.13mEq/ml
0.9% NaCl	154	0.154mEq/ml
1.8%	30	0.38mEq/L
3% NaCl	513	0.513mEq/ml
5% NaCl	855	0.855mEq/ml

Asymptomatic Hyponatermia (Chr.)
Hypovolemic hyponatermia
- Replace volume (0.9NaCl)

Euvolemichyponatermia
- Water restriction

Hypervolemichyponatermia
- Salt and water restriction"50 - 60 % of daily requirements" <800 mL/d
- ttt of the cause
- Loop diuretics

SIADH

- Hyponatremia is initially mediated by ADH-induced water retention
- Volume expansion activates 2ndry natriuretic mechanisms, results in Na and H2O loss and restoration of near euvolemiaunless there is a 2ndry problem leading to salt loss (eg, vomiting, diarrhea, or diuretic therapy)
- The net effect is that, with chronic SIADH, Na loss is more prominent than H2O retention
- Hyponatremia may also be associated with K loss. Since K is as osmotically active as Na, the loss of K contributes to the reductions in plasma osmolality and Na concentration

- Urine volume is normally regulated by ADH in response to changes in H2O intake
- When ADH release does not respond to changes in H2O intake, as in SIADH, urine osmolality is relatively fixed and urine volume varies with changes in solute excretion. Increasing solute excretion by giving salt or urea will increase urine volume and tend to raise serum Na

Suspect

Susceptible pts; malignancy as small cell carcinoma, drugs as carbamezapime, surgery, HIV inf., and pulmonary dis. as pnemonia"who developed manifestation of hyponatremia

Serum Na &Serum, urine Osmolarity

- Serum Na <**135** meq/L
- Urine Na>**40** meq /L
- Serum osmolarity <**280** mosm/Kg
- Urine osmolarity >**400** mosm/Kg
- low BUN, uric acid
- Normal thyroid & adrenal functions&K

Principles of ttt

Choose appropriate therapy

Goal

- Depends on severity & presence of symptoms

Symptomatic

- **Severe symptomatic hyponatremia**"seizures, and coma "(**Levella**) ---- urgent regimen (**100** mL 3 % saline IV bolus, should raise serum Na by about**1.5** in men &**2.0**meq/L in women). If neurologic symptoms persist or worsen--- repeat **100** mL bolus 3 % saline 1or 2 more times /10 min
- **Less severe neurologic signs+ serum Na <120 meq/L >48 hrs, a lesser degree of hyponatremia< 48 hrs, or chronic moderate hyponatremia (serum Na 120 - 129 meq/L)**----- therapy depends on severity
  - Pts with confusion and lethargy---- administer hypertonic saline to raise serum Na "**LevelIb**". Target; raise serum Na **1**meq/L / hr for 3-4 hrs. Measure serum Na at 2-3 hrs and adjust subsequent infusion rate to achieve a rate of correction of <**10**meq/L at 24 hrs and <**18**meq/L at 48 hrs
  - Ptswith mild symptoms (eg, gait distur-bance)---- fluid restriction & oral salt tablets
- Maintenance therapy ---- fluid restriction. If serum Na persist <130 meq/L--- add oral salt tablets and, if necessary in pts with a urine osmolality >twice plasma, add loop diuretic (eg, furosemide **20** mg orally /12 hrs)

Asymptomatic pts

- Fluid restriction. Oral salt tablets may be added. Loop diuretic if necessary in pts with a urine osmolality >twice that of the plasma. Do not use tolvaptan, demeclocycline, or lithium

Reset osmostat

- A pattern " variant of the SIADH --- suspect in mild to moderate hyponatremia (125 - 135 meq/L) that is stable over time despite variations in Na and water intake---- only ttt underlying dis.

>**130**meq/L because of the possible presence of suabtle neurologic manifestations and falls when serum Na is 120 - 129 meq/L (**LevelIIb**)

ttt underling cause

Therapies to increase serum Na

1. **Fluid restriction**
   - Target <**800** ml/d
   - SIADH pts with SAH --- hypertonic (3 %) saline to preserve cerebral perfusion and prevent complications from hyponatremia-induced brain swelling ----initial infusion rate of **20** mL/h with subsequent dosing being dependent upon serial measurements of serum Na / 6 hrs
2. **Administration of Nacl**
   - Either as oral salt tablets or IV NS. When using IV NS, the electrolyte concentration of administered fluid mustbe>electrolyte concentration of urine. This requires use of hypertonic saline. NS is infrequently effective and leads to further lowering of serum Na. An initial large rise in serum Na follows hypertonic saline administration that falls over time as the administered Na is excreted
3. **Diuretic therapy**
   - Among pts with a urine osmolality >twice the plasma osmolality (>500 mosmol/kg) --- use loop diuretic to lower urinary concentration, increase water excretion
4. **Vasopressin receptor antagonists**
   - Produce a selective water diuresis without affecting Na and K excretion
   - IV **Conivaptan** (used in hospitalized pts) and **Oraltolvaptan** are available and approved for use in ptswith hyponatremia due to SIADH
   - The utility of **Tolvaptan** therapy is limited by excessive thirst, prohibitive cost, and the potential for overly rapid correction which has led to the necessity for hospitalization for the initiation of therapy
5. **Demeclocycline or lithium**
   - Act on collecting tubule cell to diminish its response to ADH, increaseH2O excretion
   - In ts with bipolar disorder who are treated with chronic lithium therapy, nephrogenicDI develops in up to 20 - 40 %
   - Demeclocycline (**300**-**600** mg /12hrs) is more predictably effective and more often used thanlithium . However, both drugs can be nephrotoxic, demeclocycline can cause nausea, vomiting, and photosensitivity , and lithium has a variety of neuropsychiatric side effects

Hypernatremia

- **Dehydration:** Hypernatremia due to water loss
- **Hypovolemia**, both salt and water are lost

- Plasma tonicity " effective osmolarity" = $\frac{(2 \times Nae + 2 \times Ke)}{TBW}$
- Ineffective osmoles ' Urea, Manitol " moves freely between ECF, and ICF

Changes in plasma osmolality & serum Na conc. associated with osmotic diuresis are complex:

- Pts with DKA or HNKC present with a high Na osmolality due to both hyperglycemia and osmotic diuresis. Serum Na concentration is variable, reflecting the balance between hyperglycemia-induced increase in serum tonicity, lowering serum Na, and glucosuria-induced osmotic diuresis, which will raise serum Na
- Initial rise in serum mannitol will lower serum Na due to osmotic water movement out of cells. In absence of impaired renal function, mannitol is rapidly excreted in urine and associated osmotic diuresis raising serum Na due both to the removal of mannitol from the plasma, which will allow water to move back into the cells, and to osmotic diuresis, which will cause water loss in excess of Na + K
- osmotic diuresis due to urea (during resolution of azotemia) can produce hypernatremia. Because urea is an ineffective osmole, water does not move out of cells,. As urea is excreted in the urine, the loss of water in the urine will raise serum Na conc.

Suspect

- History; water losses in older pts whose losses are not replaced because of impaired mental status
  - Hypothalamic lesion affecting thirst center should be strongly suspected in an alert pt with access to water who has a serum Na concentration **>150** meq/L" **Adipsic diabetes insipidus** "
- Disturbed consciousness in susceptible pt

Serum Na

>145 mEq/L

ECF

Hypovolemia (Unreplaced H2O loss = Hypotonic fluid loss = decreased Total body Na >decreased TBW)

Renal loss "Urinary Na >20&osmolarity>700"

- Post-obstruction
- Osmotic & loop diuretics

Extra-renal loss "Urinary Na <20&osmolarity>700"

- Excess sweat - Burn -Diarrhea - Fistula

Hypervolemia (increased Total body Na >increased TBW "Na gain"**)**

Urinary Na >20

- Hyper-aldosteronism
- Cushing, s syndrome
- Hypertonic dialysis & NaHCO3
- NaCl tablets

Euvolemia (decreased TBW & normal Total body Na)

Renal loss "Urinary Na variable"

- **Diabetes insipidus" osmolarity<700"**
- Insufficent intake "hypodypsia & obtunded bed ridden" "**osmolarity>700**"

Extra-renal loss "Urinary Na variable & osmolarity >700"

- Insesible loss "respiratory, dermal"

Assess severity

- Presence of **symptoms**
- **Degree** and **rapidity** of decrease

ttt

General principles

- **Correction rate**
  - Acute hypernatremia: **1 mmol/L/hr, elderly --- <10** meq/L/d
  - Chronic hypernatremia "**≥**1d" : **0. 5 mmol/L/hr"**0meq/L per day is considered safe"
  - **1/2 deficit in24 hrs**&remaining over 48-72 hrs
  - Add in obligate H2O losses from stool and skin" 40 mL/hr"
  - Add in other ongoing H2O losses from either GIT or urine, if can be estimated
- **Formula**:Water deficit = **plasma Na–140/140 *TBW** "Male:0.6 BW Female:0.5 BW old female 0.4"
- **Infusate**
  - Without hypokalemia: **Change in seum Na = Infusate Na +infusate K – serum Na/ TBW +1**
  - With hypokalemia;**Change in seum Na Infusate Na– serum Na/ TBW +1**
  - Select most hypotonic infusate .The more hypotonic the infusate, the lower the rate required
  - Avoid hyperglycemia when using dextrose

Hypervolemic hypernatremia

- Loop diuretics

Hypovolemic hypernatremia

- Restoring vascular volume quickly
- Then, Replacing water deficit

Isovolemic hypernatremia

- Replacing water deficit (½ NS or free water)

- Pts with secretory diarrheas (cholera, VIPoma) have a Na + K conc. in the diarrheal fluid that is similar to the plasma Na conc. Loss of this fluid will lead to both a fall in ECF volume and K depletion, but will not directly affect the serum Na conc.
- Many viral and bacterial enteritides and osmotic diarrhea induced by lactulose or charcoal-sorbitol are associated with an isosmotic diarrheal fluid that has a Na + K concentration between 40 and 100 meq/L; organic solutes, which do not affect the serum Na conc., make up the remaining osmoles. Unreplaced loss of this fluid will tend to induce hypernatremia, because water is being lost in excess of Na+K. Similar considerations apply to urinary losses during an osmotic diuresis induced by glucose, mannitol, or urea
- Na+K conc. in vomitus and sweat is similar to that in non-secretory forms of diarrhea. Loss of this fluid will tend to raise the serum Na even though the fluid is isosmotic to plasma. Loss of **HCl** has no effect on the serum Na

Diabetes insipidus "DI"

- **Polyuria** is UOP **>3 L/d** in adult& **2 L/m2** in child

- Polyuria in susceptible pt" *see causes below*"

Suspect

- **Central** DI is characterized by decreased release of ADH"vasopressin or AVP", resulting in a variable degree of polyuria. Lack of ADH can be caused by disorders act at sites involved in ADH secretion: the hypothalamic osmoreceptors

DD

- DI(central or nephrogenic) should be differentiated from psychogenic polydipsia, prostatic hypertrophy, or osmotic diuresis (including post-obstructive diuresis)

Polyuria

- Central , nephrogenicDI, and psychogenic polydipsia associated with increased H2O output

Plasma Na & Serum osmolarity

- Normal plasma Na is not helpful in diagnosis but, if associated with a urine osmolality **>600**mosmol/kg ---- excludes a diagnosis of DI
- Low plasma Na (<**137**meq/L) +low urine osmolality (<1/2of plasma) ------**1ry polydipsia**
- High-normal plasma Na (**>142**meq/L, due to H2O loss), esp if urine <plasma osmolality ---**DI**. Plasma Na**>150**meq/L should not occur In pts with no cognitive impairment because initial H2O loss stimulates thirst, increasing intake to match urinary losses except when DI is due to a central lesion impairing thirst, causing adipsia or hypodipsia; here,plasmaNa can be >**160**meq/L

Water restriction test

ADH measurement

- If history and H2O restriction test provide equivocal results, plasma samples collected at baseline and following water deprivation (prior to the administration of ADH)
  - **Nephrogenic DI** excluded if there is appropriate relationship between the rise in urine osmolality and plasma ADH
  - **Central DI** excluded if there is appropriate rise in plasma ADH + rise in plasma Na or osmolality

Confirm

Causes

Central

- Neurosurgery (usually trans-sphenoidal) or traumato hypothalamus and posterior pituitary
- 1ry or 2ndy (most often due to lung cancer,leukemia, or lymphoma) tumors in brain
- Hypoxic encephalopathy or severe ischemia (aswith cardiopulmonary arrest or shock)
- Infiltrative disorders as sarcodosis
- Post-supraventricular tachycardia

Nephrogenic; Drugs(lithium and),Electrolyte disorders (decreased K and increased Ca),Renal disorders (obstetric nephropathy), Miscellaneous (sickle cell anemia)

Water restriction test

Normal physiologic response to H2O restriction

- Raising plasma osmolality, progressive elevation in ADH release and increased urine osmolality
- Once plasma osmolality reaches **295**-**300**mosmol/kg or plasma Na**>145**meq/L, the effect of endogenous ADH on kidney is maximal. At this point, administering desmopressin will not further elevate urine osmolality unless endogenous ADH release is impaired (ie, central DI)

Technique

- Stop drink 2-3 hrs or administer hypertonic saline
- Measureurine volume and osmolality / hr andplasma Naand osmolality / 2 hrs
- Continued tillreaching1 of the following end points:
  - Urine osmolality reaches a clearly normal value (>**600**mosmol/kg) ----- ADH release and effect are intact. Pts with partial DI may have a substantial rise in urine osmolality, but not to this extent
  - Urine osmolality stable on 2-3 successive hourly measurements despite a rising plasma osmolality
  - Plasma osmolality >**295**-**300**mosmol/kg or plasma Na≥**145**meq/L
- In the last 2 settings, desmopressin is administered, and urine osmolality and volume monitored / 30 min. over next 2 hrs

Interpretation

- A submaximal increase in urine osmolality in response to water deprivation (but usually to **≥300**mosmol/kg), with desmopressin resulting in a rise in urine osmolality of >100 % in complete central DI and 15 - 50 % in partial central DI
- Submaximal rise in urine osmolality in response to H2O restriction (but to well <300 mosmol/kg), with desmopressin producing little or no elevation in urine osmolality in complete nephrogenic DI, and a small (<45 %) elevation in urine osmolality with partial nephrogenic DI
- 1ry polydipsia will be associated with rise in urine osmolality, usually >**500**mosmol/kg, and no response toDDAVP(endogenous release is intact)

ttt

Therapeutic options

- Low solute (Na and ptn) diet; desmopressin (dDAVP), an ADH analog; and other drugs (thiazide diuretics)

Choice of therapy

- Varies with the severity of the polyuria. Pts with partial DI and mild to moderate polyuria and nocturia may be adequately controlled with a low solute diet (if acceptable) and, if necessary, a thiazide diuretic. Although these modalities also reduce UOP in pts with marked polyuria and nocturia
- Desmopressin therapy is usually required for symptom control. Desmopressin can also be used in pts with less severe DI who do not want to comply with a low solute diet

Desmopressin

- Long t½: duration 8-12hrs, up to 24hrs
- **1**ug IV/SC /24hrs
- Once nasal mucosa stable can switch to intranasal
- DDAVP:**1**uqIV/SC =**10**uq IN=**0.1** mg oral
- Desmopressin can lead to water retention and hyponatremia if the urine is concentrated for most of the day. This can usually be avoided by giving the minimum required dose to control the polyuria and not administering another dose until the pt has had a period of brisk diuresis, indicating that the effect of the previous dose of desmopressin had waned.
- Serum Na should be checked at 24 hrs after initiation of therapy
- Continued for as long as the pt has central DI

Hypokalemia

Normal K level **3.5**- **5.3mEq/L**

- Severity of hypokalemia proportionate to degree and duration of reduction. Symptoms generally do not become manifest till K**<3.0**meq/L, unless falls rapidly or pthas a potentiating factor, as a predisposition toarrhythmia due to use of digitalis.Symptoms usually resolve with correction

Suspect

- History " diuretic use"
- Arrhythmias esp. if refractory,
- Muscle weakness or paralysis "begins in the lower extremity" including RF, and ileus
- Alkalosis

Serum K

< 3.5 mEq/L

Review**Causes**

Assess &ttt

General principles

- K is given cautiously in redistributive hypokalemia
- Diagnose and correct the underlying cause
- Minimize continuing loses "diuretics"

- Assess muscle strength
- ECG; depresed ST segment,decreased amplitude of the Twave, and increased amplitude of U waves

Severe

- For pts with severe manifestations or unable to take oral medications, ---initiate IV KCL (Level 1B)
- Depending upon severity, IV K may be given at doses ranging from **20**meq / 2-3 hrs to a recommended maximum rate of**10**- **20**meq/hr, and up to**40**meq/hrin life-threatening hypokalemia.Once resolved, slow rate (**10** - **20**meq/h), or changed to only oral
- Infuse above 20 meq/hr into a large central vein or into multiple peripheral veins
- Concentrations of **100-200**meq/L are used commonly
- IVK is most often infused in a peripheral vein at concentrations of **20**-**60**meq/L in a non-dextrose-containing saline solution. Use of an infusion pump is preferred in any IV container with **>40**meq of K or if desired of administration is **>10**meq/h
- Pts should be treated till serum K is persistently above **3.0 - 3.5**meq/L and symptoms or signs have resolved
- Pain and phlebitis can occur during infusion into a peripheral vein. This primarily occurs at rates **>10**meq/hour, but can be seen at lower rates. If pain occurs, reduce rate or, preferably, concentration
- Careful monitoring of physiologic effects of severe hypokalemia (ECG abnormalities, muscle weakness, paralysis) is essential. Continuous ECG monitoring is warranted in pts with significant ECG abnormalities ,IVK repletion at a rate greater than **10**meq/ hr, and patients at risk for rebound hyperkalemia (most often due to thyrotoxic periodic paralysis). Once the hypokalemia is no longer severe, rate of repletion should be reduced or changed to oral therapy
- Pts with hypokalemia-induced rhabdomyolysis ---release of K from muscle cells can mask severity of the underlying hypokalemia or even lead to normal or elevated values at presentation or after K supplementation. If serum K is normal or elevated at baseline, it will not be possible to be certain that underlying hypokalemia was responsible for the rhabdomyolysis and initial K therapy is not warranted and may be dangerous. In pts who present with hypokalemia, K therapy can be initiated with repeated monitoring of the serum K (eg, / 4-6 hrs initially)

Mild - Moderate

- ttt depends upon cause and acid-base status. Pts with gastrointestinal losses are treated with KCL if they have metabolic alkalosis or a normal serum HCO3 concentration, and with KHCO3 (or potassium citrate or acetate) in the presence of metabolic acidosis
- Start with **10** - **20**meq of K given 2-4 times / d (**20** - **80**meq/d), depending upon severity
- Pts who cannot take oral require IV repletion
- Sequential monitor serum K

Causes

Decreased K intake

Increased entry to cell

- Acidosis
- Increased insulin availability
- Elevated adrenergic activity
- Hypothermia
- Hypokalemic periodic paralys
- Marked red cell production

Increased GIT loss

- Vomiting
- Diarrhea
- Tube drainage
- Laxative abuse

Increased urinary losses

- Diuertics
- Primary mineral-corticoid excess
- Dialysis
- Salt wasting nephropathy
- Polyuria
- Plasmapheresis

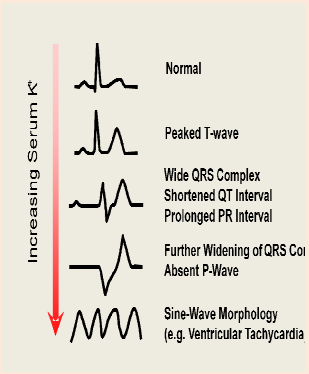
K preparations

- An IV or oral **KCl** preparation is generally preferred over **Kcitrate** or **KHCO3**, esp., with metabolic alkalosis
- K **citrate** or**HCO3** preferred with metabolic acidosis
- Oral **KCl** can be given in crystalline form (salt substitutes), liquid, or in a slow-release tab. or capsule
  - Salt substitutes contain **50 - 65 meq** / level teaspoon ---- safe, well tolerated and much cheaper
- K**Cl** solutions are often unpalatable, and slow-release preparations can rarely cause ulcerative or stenoticGIT lesions
- Increasing intake of K -rich foods is less effective

Hyperkalemia

Normal K level **3.5- 5.3mEq/L**

ECG changes



Suspect — If there is **ECGchanges**, renal failure, acidosis

Serum K — **> 5.5 mEq/L**

- Review**causes**
- Exclude **pseudo-hyperkalemia** "hemolysis while drawing blood"

Assess & ttt

- Continuous cardiac monitoring andserial ECG are warranted who require rapidly actingtherapies. Measure serum K at 1-2 hrs after initiation.Further measurements determined by serum K and response to therapy
- Any extra source of K intake should be eliminated and any potentiating drugs (such as ACEI) should be discontinued
- If hypovolemic, give fluid to enhance urinary K

1- Mild elevation (**5.5-6** mmol l-1)

Remove K from the body with

- **K exchange resins**
- **Sodium polystyrene sulfonate**: Give **15-30** gm orally; pediatric dose is 1 g/kg; do not give within 1 w of surgery; although less preferable, may also be given as retention enema; do not give enema with sorbitol; time to onset is approximately 1-2 hs; may repeat dose after 4 hs based upon repeat serum K
- **Diuretics**: **Furosemide 20 - 40** mg IV; pediatric dose is 1-2 mg/kg IV; higher dose may be required with renal insufficiency; fluid losses must be replaced unless pt is volume expanded
- Consider **dialysis** "HD remove 25 -50 meq K/ hr

2- Moderate elevation (**6 - 6.5** mmol l-1) without ECG changes

Use strategies in 1 + shift K into cells with

- **Dextrose/insulin:** Give IV bolus of regular insulin **10** U with 100 mL of a 50 % glucose solution; give children regular insulin **0.2** U / gm of glucose, give glucose **1** g/kg; monitor fingerstick glucose closely; time to onset is 15-30 min

3- Severe elevation (**>6.5** mmol l-1) without ECG changes

Strategies in 1& 2 + Shift K into cells

- **Salbutamol**: **5** mg nebulized. Several doses may be required (onset in 15-30 min)
- **Sodium bicarbonate**: **50** mmol IV over 5 min if metabolic acidosis present (onset in 15-30 min). HCO3 alone is less effective than glucose + insulin or nebulized salbutamol; it is best used in conjunction with these medications. Pediatric dose is **2 - 3** meq/kg

4- Severe elevation (**>6.5** mmol l-1) with ECG changes

Protect the heart first "Ca"

- (Antagonise toxic effects of hyperkalaemia at the myocardial cell membrane, reducing the risk of VF, but does not lower serum K)
- Give adults **CaCl 500 - 1000** mg (5 - 10 mL of 10 % solution) by IV infusion over 2 - 3 min., preferably via a central line; or give **calcium gluconate 1000** mg (10 mL of 10 % solution) also infused slowly; may be peripherally in large vein. Onset in 1-3 min "CaCL contains 3 times concentration of elemental Ca compared to Ca13.6 vs 4.6 meq in 10 mL of a 10 % solution)
- May be repeated after 5 min. if ECG changes persist; pt must be on cardiac monitor when receiving Ca; Ca can exacerbate digoxin toxicity

+

- K removal & shifting strategies

Causes

Increased cell release

- Metabolic acidosis
- Hyperglycemia –
- Hyperosmolarity

Increased tissue catabolism

- Hyperkalemic periodic paralysis
- B-adrenergic blockade
- Succinylcholine
- Digitalis overdose

Reduced urinary excretion

- Renal failure
- ECF depletion
- Retro-jejeunotomy

Hypomagnesmia

Normal range **1.4** - **1.7** meq/L is equivalent to **0.70** - **0.85** mmol/L and **1.7** - **2.1** mg/d")

- A daily excretion **>10** -**30** mg (in a 24-hr urine specimen) or a fractional excretion of Mg **>2** % in a person with hypomagnesemia and normal renalfunction indicates renal magnesium wasting
- 24-hr urinary Mg excretion **<10** mg or a fractional excretion of Mg**<2**% usually indicates an extrarenal source of Mglosses (typically GI)

Suspect

- Pts with risk factors (eg,chronic diarrhea, PPI therapy,alcoholism, diuretic use)
- Clinical manifestations (eg, unexplained hypo-calcemia, refractory hypokalemia, neuromuscular disturbances, ventricular arrhythmias)

Serum Mg

< 1.4 mEq/L

- ReviewCauses
- Distinct GI from renal losses --- measure24-hr urineMg excretion or fractional excretion of Mg on a random urine specimen
- Checkother electrolyte disorders (hypokalemia, and hypocalcaemia)
- **Normo-magnesmic Mg depletion**, if Mg is normal and Mg depletion is highly suspected

- Considernormo-magnesemic Mg depletion as a cause of refractory hypokalemia or un-explained hypocalcemia in ptsat high risk for Mg loss
- Pts with malnutrition, cirrhosis, diarrhea, or on long-term diuretics have +ve test, whether or notthey have signs or symptoms referable to Mg depletion
- Administer Mg to these pts if theyhave unexplained hypocalcemia ±hypokalemia

Assess Severity

- Presence or absence of **symptoms**
  - Arrhythmias (Atrial tachycardias, AF, SVT, Ventricular arrhythmias, and Torsade de pointes)
  - Neuromuscular (carpo-pedal spasm, fasciculations, tremors, convulsations, muscle cramps and weakness)
- **Degree** and **rapidity** of decrease

ttt

General principles

- Correct Underlying dis.if possible; thiazide or loop diuretics ----- may benefit from adding K-sparing diuretic
- High-risk pts; chronic alcoholics, long term diuretics --- monitor serum Mg regularly& supplement if necessary
- Give Mg daily to ICU pts with normal renal function

Pts with no or minimal symptoms

- Give **240** -**1000** mg oral Mgdaily if available and tolerable**+** normal renal function
- Many pts are given IV rather than oral Mg "unable to take oral or have side effects, even if symptoms are minimal or absent
- If plasma Mg **<1.0** mg/dL ----- give **4**-**8** gm over 12-24 hrs & repeat as needed. If1.0-1.5 mg/dL ---- give **2**-**4** gm over 4-12 hrs. If 1.6-1.9 mg/dL --- give **2** gm over 1-2 hrs
- **Asymptomatic** pts with severe hypomagnesemia **+** moderate decrease kidney function ----1/2 dose of recommended oral preparation for pt with normal renal function ----- Measure plasma Mg before next dose

Pts with severe symptoms

- Give IV therapy **+** continuous cardiac monitoring
- Acutehemodynamic unstable pts (torsade de pointes, hypomagnesemic hypokalemia) -----**1-2** g Mg sulfate initially over 2-15 min
- Hemodynamic stable pts **+** severe symptoms(≤ 1.0 mg/ ----give **1-2** gm Mg sulfate in 50 - 100 mL of 5 % D in water initially over 5 - 60 min followed by an infusion
- A simple infusion regimen for non-emergent repletion ---**4** - **8** g Mg sulfate over 12 - 24 hrs. This dose can be repeated as necessary to maintain plasma Mg above 1.0 mg/dl. In the normo-magnesemic pt with hypocalcemia---- repeat this dose daily for 3-5 ds
  - Pts with moderately reduced kidney function (ie, estimated GFR of 15 - 30 mL/min / 1.73 m^2) and severe hypomagnesemia ----**2 - 4** g IV Mg sulfate over 4 - 12 hrs. Check plasma Mg prior to subsequent doses, and daily if given less frequently
- In children-----slow infusion; **25 - 50** mg/kg with a maximum single dose of **2** g

Causes

- **Redistribution of Mg**
- Refeeding & insulin therapy & Hungry bone S
- Correction of acidosis & Catecholamine excess &Massive blood transfusion

GIT causes

- Reduced intake & Mg free IV fluid
- Reduced absorbtion "malabsorption, chr.diarrhea, intest. resection"

Renal loss

- Diuretics

Renal diseases

- Post-obdtructive nephropathy & Dialysis &Diuretic phase of renal failure

Endocrinal causes

- Hyperper-aldestronism, primary hyperpara-thyrodism, Hyprcalcemia, hyperthyroidism

DM

Alcholism

Normo-magnesemic Mg depletion

24 rs urine

- Excretion/d>**10**-**30** mg with normal renal function indicates renal Mg wasting due to drugs as diuretics, aminoglycosides

Magnesium Retention Test

- Add **24** mmol Mg over 250 ml NS over 1 hr------collect urine from start of infusion in 24 hrs----- urinary Mg excretion < 1/2 of the infused ---- + ve test

Indication

- End point of resusc

Limitation

- Cardiac instability & renal failure

MgSO4"50% "

- Each gm has **8** mEq (4 mmol) of elemental Mg
- MgSO4 solution (500 mg/ml) ----osmolarity of 4000 mOsm/L ---must be diluted to a 10% (100 mg/mL) or 20% (200 mg/mL) solution
- Saline should be used as diluent (Ringer's should not be used)

Hypermagnesmia

Normally, ingested Mgis readily excreted in the urine. On a regular diet, for example, about 3 % of the filtered Mg escapes tubular reabsorption and is excreted.In contrast to most other filtered solutes, most of the filtered Mg is reabsorbed in the cortical aspect of the thick ascending limb of Henle (50 - 60 %), rather than the proximal tubule. Loop reabsorption is appropriately diminished with Mg loading, thereby allowing the excess Mg to be excreted in the urine

Suspect

- ECG changes, CHB, RF, hypotension , and bradycardia, in susceptible pts
- Other electrolytes disorders (hypokalemia, and hypocalcaemia)

Normal range **1.4** - **1.7** meq/L is equivalent to **0.70** - **0.85** mmol/L and **1.7** - **2.1** mg/d")

Serum Mg

>1.4mEq/L

- Review**Causes**
- Check **Ca** level

Hypermagnesemia may inhibit the secretion ofparathyroid hormone, causing transient hypocalcemia

Assess severity

- Presence or absence of **symptoms**
  - ECG changes (as hyperkalemia)
  - CVS (hypotension, bradycardia), RF
- **Degree** and **rapidity** of decrease
  - Plasma Mg **4 - 6** meq/Lnausea, flushing, headache, lethargy, drowsiness, diminished deep tendon reflexes
  - Plasma Mg **6 - 10** meq/Lsomnolence, hypocalcemia, absent deep tendon reflexes, hypotension, bradycardia, ECG changes
  - Plasma Mg **>10** meq/Lmuscle & respiratory paralysis, CHB, and cardiac arrest. In most cases, RF precedes cardiac collapse

ttt

General principles

- Most cases of symptomatic hypermagnesemia can be prevented by anticipation
  - Pts in renal failure should not receive Mg-containing medications and pts receiving parenteral Mg for any reason should be carefully and frequently monitored
  - In normal renal function, cessation therapy allows prompt restoration of normal level

Causes

Redistribution
- Acute acidosis

Excessive intake

Oral
- Antacids Cathartics

Rectal
- Purgation

Parenteral
- Urethral irrigation

Renal failure
- Acute and chronic renal failure
- Rhabdomyolysis

Others
- Lithium therapy
- Familialhypocalciuric hypercalcaemia
- Hypothyroidism
- Addison'sdisease
- Milk alkali syndrome
- Depression

IV fluids
- Dilute extracellular Mg
- With diuretics --- promote urinary excretion
  - NS or RL
  - Adult **1** L IV - Pediatric **20** mL/kg IV initially

Dialysis
- In impaired renal function "PD or HD"
  - HD lowers Mg to nontoxic range in 3-4 hrs

Diuretics
- Furosemide (**Lasix**)
- Adult **20**-**80** mg/dose IV; single dose not >**6** mg/kg Pediatric **1** mg/kg/dose IV /6-12 hr prn

Mineral supplements "Ca"
- IV Ca given as a Mg antagonist
- **100** - **200** mg elemental Ca over 5-10 min
- **Cagluconate** (**Kalcinate**)
  - 10% IV solution provides **100** mg/mL of Ca gluconate
  - Adult **100**-**200** mg 10% solution IV,then continuous infusion (**2** - **4** mg/kg/hr)
  - Pediatric **2** mg/kg of elemental Ca IV(about **20** mg/kg of ca gluconate 10%)

Preparation	Elemental Mg
Oral	
Mgcl coated entric tab	64 mg "5.3meq"
Mg oxide tab 400mg	241 mg"19.8meq"
Mg oxide tab. 140 mg	85 mg "6.9meq"
Mg gluconate tab. 500mg	27 mg "2.3meq"
Parentral	
Mg sulphate 50%	500mg "4meq"
G sulphate 12.5%	120mg "1meq"

Hypophosphatemia

Normal range **4.5-6.5**mg/dl

- Hypophosphatemia induced by decreased netintestinal absorption, increased urinary PO4 excretion, or acute movement of extracellular PO4 into cells.
- The normal renal response to PO4 depletion is to increase PO4 re-absorption, leading to decreased PO4 excretion in urine

Suspect

- Arrhythmias, muscle weakness seizures, encephalopathy
- Challenge to wean from ventilator

Serum PO4

≤ 4.5 mEq/L

- Review**Causes**

FEPO4 = [UPO4 x PCr x 100] ÷ [PPO4 x UCr]here U and P refer to the urine and plasma concentrations of PO4 and creatinine (Cr)

PO4 excretion

- 24-hr urine collection
- Fractional excretion of filtered PO4 (**FEPO4**)from a random urine specimen "Daily PO4 excretion **≤ 100** mg and FEPO4**≤ 5** % (N - **20** %) if kidney is responding normally and renal PO4 wasting is not the

The triad of hypercalcemia, hypophosphatemia, and urinary PO4 wasting is usually present in 1ry hyper-parathyroidism. In contrast, hypocalcemia is the major stimulus for hypersecretion of para-thyroid hormone in 2nd dis.Hypophosphatemia and urinary po4 wasting in someone without hypercalcemia should prompt an evaluation for vitamin D deficiency

Causes

Internal redistribution
- Increased insulin secretion, particularly during refeeding
- Acute respiratory alkalosis
- Hungry bone syndrome

Decreased intestinal absorption
- Inadequate intake
- Antacids containing aluminum or Mg
- Steatorrhea & chronic diarrhea
- Vitamin D deficiency or resistance

Increased urinary excretion
- Primary and secondary hyperparathyroidism
- Vitamin D deficiency or resistance
- Hereditary hypophosphatemic rickets
- Oncogenic osteomalacia
- Fanconi syndrome

Assess severity

- **Presence or absence of symptoms:**Symptoms rarely occur unless serum PO4 **<2** mg/dL, serious symptoms as muscle weakness & rhabdomyolysis are not seen until serum PO4**<1** mg/dL
- **Degree and rapidity of decrease**

ttt

General principles

- Most pts will not require therapy other than ttt of underlying cause "GIT loses"
- **10-15** mmol/L / **1000** KcalTPN

Oral Therapy

- If PO4 is **≥1.5** mg/dL ,**1** mmol/kg of elemental PO4 (minimum of **40** mmol & maximum of **80** mmol) can be given in 3-4 divided doses over 24 hrs
- If PO4**<1.5** mg/dL ,**1.3** mmol/kg elemental PO4 (up to maximum **100** mmol) can be given in 3-4 divided doses over 24 hrs
- Severely obese pts -- receive maximal initial doses or adjusted dose based on height & weight
- Pts with reduced GFR should receive approximately 1/2 of suggested initial dose
- Recheck2-12 hrs following last of the divided doses to determine the needed repeated doses -- If so, re-apply the same approach
- Select oral supplement according to its K and Na content and dosed according to mmol of PO4
- Commonly used oral PO4 supplements include 250 mg (8 mmol) of PO4/ tablet

PO4 repletion regimens

- In **asymptomatic** pts with a serum PO4 **< 2.0** mg/dL (0.64 mmol/L) --- give oral PO4 therapy since many of these pts have not clinically apparentmyopathy & weakness
- ttt of **symptomatic** pts varies with severity of hypophosphatemia
  - ttt with oral PO4 if serum PO4 is **1.0 - 1.9** mg/dL (0.32 - 0.63 mmol/L)
  - ttt with IV PO4 if serum PO4 is **<1.0** mg/dL (0.32 mmol/L), and switch to oral replacement when serum PO4 **>1.5** mg/dL (0.48 mmol/L)
  - Monitor /6 hrs. Stop PO4 repletion when the serum PO4 is **≥2.0** mg/dL

IV PO4

- Potentially dangerous---can produce hypo-calcemia due to binding of Ca, renal failure due to CaPO4 precipitation in kidneys, and possibly fatal arrhythmias

In severe symptomatic hypo-phosphatemia or inability to take oral

- If the serum PO4 **≥1.25** mg/dL (0.40 mmol/L) -----give **0.08 - 0.24** mmol/kg over 6 hrs (up tomaximum total dose **30** mmol)
- If the serum PO4 **<1.25** mg/dL (0.40 mmol/L) ----give**0.25 - 0.50** mmol/kg over 8 - 12 hrs (up to max. total dose **80** mmol)

Hyperphosphatemia

Normal range **4.5-6.5**mg/dl

Suspect

- Arrhythmias, muscle weakness seizures, encephalopathy

Serum PO4

>6.5mg/dl

- Review**Causes**
- Check Ca , and tetany
- Exclude Psedudo- hyperphosphatemia

Pseudo-hyperphosphatemia

- Due to interference with analytical methods that may occur in pts with hyperglobulinemia, hyperlipidemia, hemolysis, and hyperbilirubinemia

Assess severity

- Presence or absence of symptoms
- Degree and rapidity of decrease

Causes

Massive acute PO4 load

- Tumor lysis syndrome
- Rhabdomyolysis
- Lactic and ketoacidosis
- Exogenous PO4
- Acute PO4nephropathy

Renal failure

Increased tubular reabsorption of PO4

- Hypoparathyroidism
- Acromegaly
- Bisphosphonates
- Vitamin D toxicity

ttt

Chronic

- Diminishing intestinal PO4absorption by a low PO4diet and PO4binding salts Ca, Mg and aluminum
  - The latter is avoided in renal failure, as aluminum can accumulate (Ca is preferred)

Acute

- Can be life-threatening. if with symptomatic hypocalcemia
- Usually resolves within 6 - 12 hrs if renal function is intact
- PO4excretion can be increased by **NS** infusion------ can further reduce serum Ca by dilution
- **HD**---- in pts with symptomatic hypo-calcemia, particularly if renal function is impaired

Preparation	PO4 Content	Na	K
Oral preparations			
Skim cow's milk	1/L	28 meq/L	38 meq/L
Neutra-Phos	250 mg/packet	7.1 meq/packet	7.1 meq/packet
Phospho-Soda	10mg/mL	4.8 meq/mL	0
Neutra-Phos K	250 mg/capsule	0	14.25 meq/capsule
K-Phos Original	150 mg/capsule	0	3.65 meq/capsule
K-Phos Neutral	250 mg/tablet	13 meq/tablet	1.1 meq/tablet
IV preparations			
Neutral sodium potassium PO4	1.1 mmol/mL	0.2 meq/mL	0.02 meq/mL
Neutral sodium PO4	0.09 mmol/mL	0.2 meq/mL	0
Sodium PO4	3.0 mmol/mL	4.0 meq/mL	0
Potassium PO4	3.0 mmol/mL	0	4.4 meq/mL

Hypocalcemia

Normal total serum Ca 8.5 - 10.5 mg/dL (2.12 - 2.62 mmol/L)

Tetany uncommon unless serum ionized Ca<**4.3** mg/dL(1.1 mmol/L), serum total Ca**7.0 - 7.5** mg/dL (**1.8-1.9**mmol/L)

Suspect

- Carpopedal spasm, tetany, twitches, laryngospasm, seizures
- ECG---- prolonged QT interval

Total Serum Ca

≤ 8.5 mg/dL

Review**Causes**

Ionized Ca, albumin, Mg, PO4

Ionizedcalcium
- **4.65 - 5.25** mg/dL (1.16 - 1.31 mmol/L)

Serum albumin concentration
- Each **1** g/dL reduction in serum albumin will lower total Ca by approximately **0.8** mg/dL (0.2 mmol/L) without affecting the ionized Ca concentration "without producing any symptoms or signs of hypocalcemia"
- If diagnosis is in doubt, either because pt's symptoms are atypical or pt's serum Ca is only slightly low, serum ionized Ca should be measured if reliably available

Serum Mg
- Mg supplementation indicated in unexplained hypocalcemia who are at risk for hypomagnesemia, as malabsorption, alcoholism

PO4
- Persistent hypocalcemia&hyperphosphatemia, in absence of kidney dis. or increased tissue breakdown, virtually diagnostic of hypo-parathyroidism (PTH deficiency) or pseudo-hypoparathyroidism (PTH resistance)
- Low PO4 indicates excess PTH secretion (2nd hyperparathyroidism), or low intake(uncommon)

Assess &ttt

- Dpendsupon absolute level, and rate of decrease ----acute will be symptomatizing at a lower serum Ca than chronic one

Oral Ca

- For acute milder degrees (corrected Ca 7.5 - 8.0 mg/ or a serum ionized Ca> 3.0 - 3.2 mg/) or chronic "typically asymptomatic or mildly symptomatic (eg, oral paresthesias)"
- If symptoms do not improve with oral ---- switch to IV Ca
- **1500** - **2000** mg elemental Ca as **Cacarbonate** or **citrate** daily, in divided doses "eg, Cacarbonate is 40 % elemental Ca --1250 mg Ca carbonate has 500 mg elemental Ca"

IV Ca

- For acute symptoms, prolonged QT interval), rapid and progressive reduction, asymptomatic hypocalcemia with acute decreases to ≤7.5 mg/dL (1.9 mmol/L) who may develop serious complications if untreated, and mild or chronic hypocalcemia who unable to take or absorb oral supplements
- **1** - **2** g of **CaGluconate**, = 90 - 180 mg elemental Ca, in 50 mL of 5 % D5) over 10 - 20 min.(because of risk of serious cardiac dysfunction, including arrest). will raise serum Ca for 2-3 hs "followed by slow infusion in persistent cases"
- **10** % **Cagluconate** (90 mg elemental Ca / 10 ml) "usually preferred "less likely to cause tissue necrosis if extravasated" *or***10** % **Cachloride** (270 mg elemental Ca / 10 ml)
- IV solution containing 1 mg/mL of elemental Cagluconate is prepared by adding 100 mL of 10 % Cagluconate (90 mg elemental Ca / 10 mL for a total of 900 mg elemental Ca) to 1000 mL of 5 % dextrose in water
- Start Infusion 50 mL/h (= 50 mg/h)
- The dose can be adjusted to maintain serum Ca concentration at the lower end of the normal range
- Pts require **0.5 - 1.5** mg/kg elemental Ca / hr
- Infusion considerations:
  - Should be diluted in dextrose and water or saline (Concentrated Ca solutions are irritating to veins)
  - The IV Ca solution should not contain HCO3 or PO4, which can form insoluble Ca salts (If needed, use another IV line (in another limb)

Vitamin D deficiency

- **Ergocalciferol** (vit D2) or **Cholecalciferol** (vit D3)
- Available in several doses orally
- Give daily for ws before full effect
- In hypoparathyroidism ,severe acute hypocalcemia. Rapid onset "hrs"
- Renal failure--**Calcitriol.** Initial **0.25** - **0.5** mcg twice daily. Side effects --- hypercalcemia, hypercalciuria, which, if chronic ---nephrolithiasis, nephrocalcinosis, and renal failure
- Measure urinary Ca excretion periodically, and reduce Ca and vitamin D if elevated (≥**300** mg /d)

Concurrent hypomagnesemia

- Infuse**2** g (16 meq) Mgso4 as 10 % solution over 10 - 20 min., followed by **1** g (8 meq) / 100 mL fluid / hr
- Continue Mg repletion as long as Mg <**0.8**meq/L (1 mg/dL or 0.4 mmol/L)
- Carefully monitor inrenal impairment
- Persistent hypomagnesemia, --- oral Mg, typically **300** - **400** mg daily /8 hrs

hypo-parathyrodism

- Target --- maintain serum Ca in low-normal range"**8.0** - **8.5** mg/dL
- Initial oral dose ---**1.0** - **1.5** g elemental Ca daily, in divided doses
- **Cacarbonate** often used (least price& less well-absorbed in the old)
- Treat with **Cacitrate**
- **Calcitriol**----**0.25**uq/12hrs--- increase/w. Many require **2.0** mcg /d
- Monitor urinary and serum Ca and serum PO4/ weekly initially, until a stable serum Ca,thereafter, / 3-6 ms

Causes

Hypo-parathyrodism
- Genetic
- Post-surgical
- Auto-mmune
- Infiltrative
- Radiation
- HIV

2ndry hyperparathyrodism
- Vitamin D deficiency or resistance
- Parathyroid hormone resistance"hypomagmesmia
- Loss of calcium from the circulation "Hyperphosphatemia, Tumor lysis, Acute pancreatitis, Acute respiratory alkalosis, Sepsis or acute severe illness

Drugs
- Phenytoin

Hypomagnesemia
- Can reduce PTHsecretion or cause PTH resistance and is therefore associated withnormal, low, or high PTH levels

Hypercalcemia

Normal total serum Ca **8.5** - **10.5** mg/dL
(2.12 - 2.62 mmol/L)

Suspect

- Stupor & coma, bradycardia & hypotension, weakness
- ECG : short QT interval

>10.5 mg/dL

High Total Serum Ca

- Review**Causes**
- Repeat result to confirm diagnosis
- **Exclude Factious hypercalcemia**
- **Distinguish malignancy from hyper-parathyrodism**

Factious hypercalcemia

- Increased ptn binding can elevate serum Ca without any rise in serum ionized Ca
- This occurs with hyperalbuminemia due to severe dehydration and multiple myeloma
- In pts with hypo-albuminemia " chronic illness or malnutrition", total serum Ca may be normal when serum ionized Ca is high
- Thus in pts with hyop- or hyper-albuminemia, correct measured Ca for abnormalities in the albumin

Ionized Ca & albumin & PTH

Distinguish malignancy from hyper-parathyrodism

- Malignancy is often evident clinically by the time it causes hypercalcemia, and pts have higher Ca and are more symptomatic from hypercalcemia than individuals with 1ry hyperparathyroidism
- 1ry hyperparathyroidism is often associated with borderline or mild hypercalcemia (serum Ca often <**11** mg/dL [2.75 mmol/L]). Values **>13** mg/dL (3.25 mmol/L) are unusual in 1ry hyperparathyroidism

- Ca <12 mg/dL --- asymptomatic, or nonspecificsymptoms, as constipation, and fatigue
- Ca 12 - 14 mg/dL, chronic---- well-tolerated, acute -----marked symptoms" polyuria, polydipsia,dehydration, weakness, and changes in sensorium
- Ca >14 mg/dL----progression of these symptoms

Assess & ttt

Mild

- Pts with asymptomatic or mildly symptomatic hypercalcemia(eg, constipation) (Ca **<12 mg/dL** [3 mmol/L]) do not require immediate ttt
- Should be advised to avoid factors that can aggravate hypercalcemia, including thiazide diuretic and lithium carbonate therapy, volume depletion, prolonged bed rest or inactivity, and a high calcium diet (>**1000** mg/d)
- Adequate hydration (at least 6-8 glasses of water / d) is recommended to minimize the risk of nephrolithiasis
- Additional therapy depends mostly upon the cause

- A frankly elevated **PTH** or a PTH value in the upper half of the normal range --- 1ry hyperparathyroidism
- Value <**20** pg/mL -----Search for other causes

Severe

- Pts with Ca **>14 mg/dL** (3.5 mmol/L)

Volume expansion

- **NS200 -300** mL/hr according to age,severity, and presence of HF or renal failureinitially--- adjust to maintain UOP 100-150 mL/hr. Stop if develops edema
- In the absence of renal failure or HF, **loop diuretic therapy** is not recommended

Calcitonin

- Increase renal Ca excretion and, decrease bone resorption via interference with osteoclast function
- Give Salmon calcitonin (**4 IU /kg**) IM or SC / 12 hrs ---- increase up to **6 - 8 IU /kg** / 6 hrs
- Rapidly lower serum Ca by a maximum of **1 - 2 mg/dL** beginning within 4-6 hrs
- The efficacy is limited to 1st 48 hrs —-tachyphylaxis
- Of value with hydration in symptomatic pts **+** Ca >**14** mg/L

Bisphosphonates

- Inhibit Ca release by interfer with bone resorption. Effective in treating hypercalcemia resulting from excessive bone resorption of any cause
- Nontoxic compounds. More potent in moderate or severe hypercalcemia
- Administer **zoledronic acid** (**4 mg IV** over 15 min.) or **pamidronate** (**60 - 90 mg** over 2 hs), preferably zoledronic acid (superior to pamidronate in reversing hypercalcemia related to malignancy)
- Effective by 2nd – 4th d "sustained effect"

HD

- If serum Ca **18** - **20** mg/dL **+** neurologic symptoms but a stable circulation, severe malignancy-associated hypercalcemia , renal insufficiency or HF

Moderate

- Chronic symptomatic or mildly symptomatic with moderate hypercalcemia (Ca **12** - **14** mg/dL [3 - 3.5 mmol/L]) ----- no immediate therapy---- follow same precautions of mild one
- Acute --- marked changes in sensorium --- aggressive therapy saline hydration & Bisphosphonates

Causes

PTH-mediated

- 1ry hyperparathyroidism
- Tertiary hyperpara-thyroidism (acute renal failure)
- PTH-independent
- Hypercalcemia of malignancy
- Vitamin D intoxication

Miscellaneous

- Hyperthyroidism
- Acromegaly
- Pheochromocytoma
- Adrenal insufficiency
- PN
- Milk alkali S

Medications

- Thiazide, Lithium, and theophylline toxicity


Ventilatory support

Indications of ventilatory support

Acute respiratory failure
- Inability of the respiratory system to provide adequate gas exchange and is no longer able to meet the metabolic demands of the body
- May be due to:
  - Gas exchange failure manifested by hypoxemia
  - Ventilatory failure manifested by hypercapnea

Impending ventilatory failure
- Worsening respiratory function "more WOB, more tachypnea" and/or worsening ABGs despite appropriate target management to the underlying cause

Failed conventional 02 therapy
- Po2 < 60 mmHg with Fio2 60%*Or*
- PO2 >60 mmHg but with a higher FiO2 "non-rebreathing, or partial rebreathing mask" esp,. if there is no reversible cause

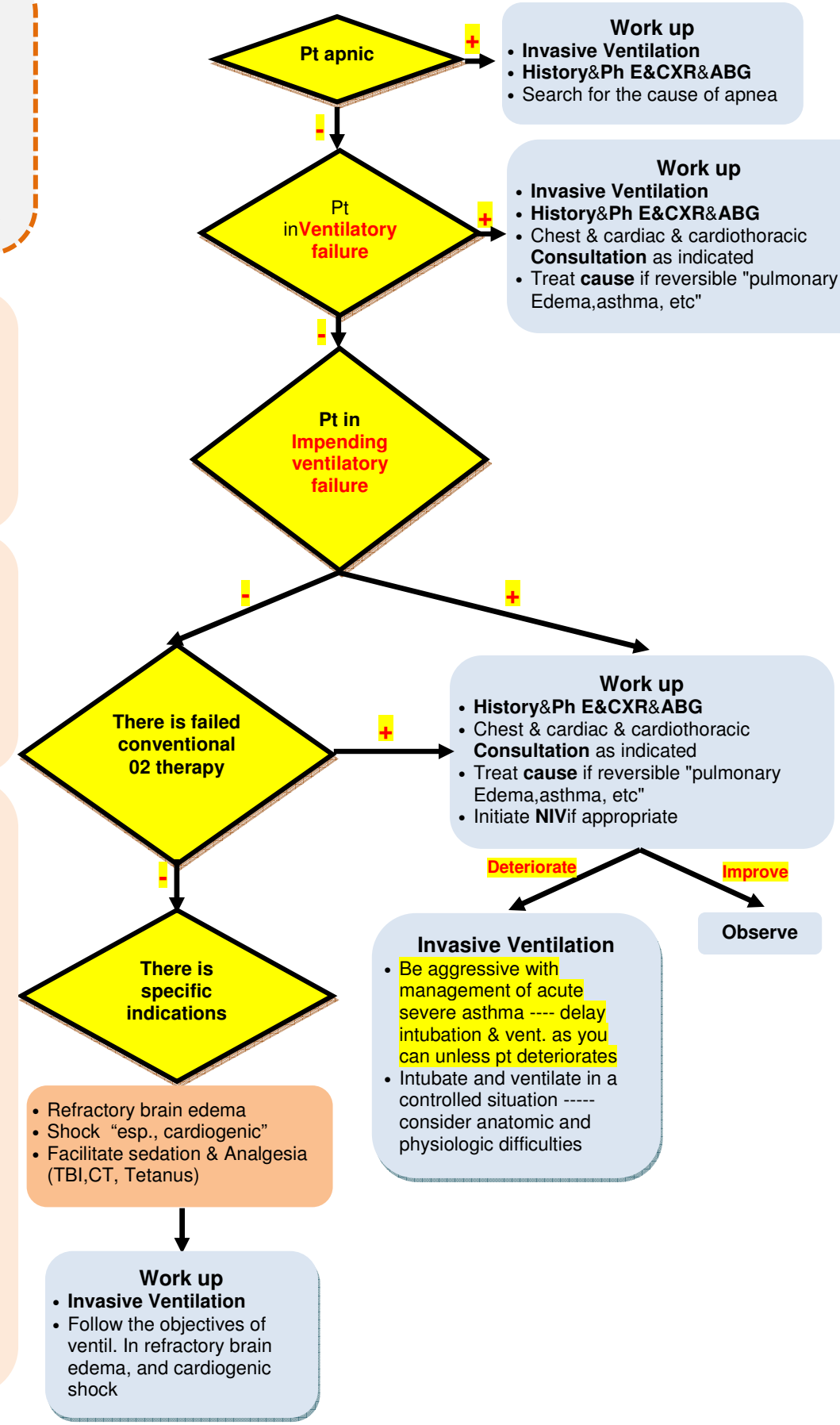
Ventilatory failure

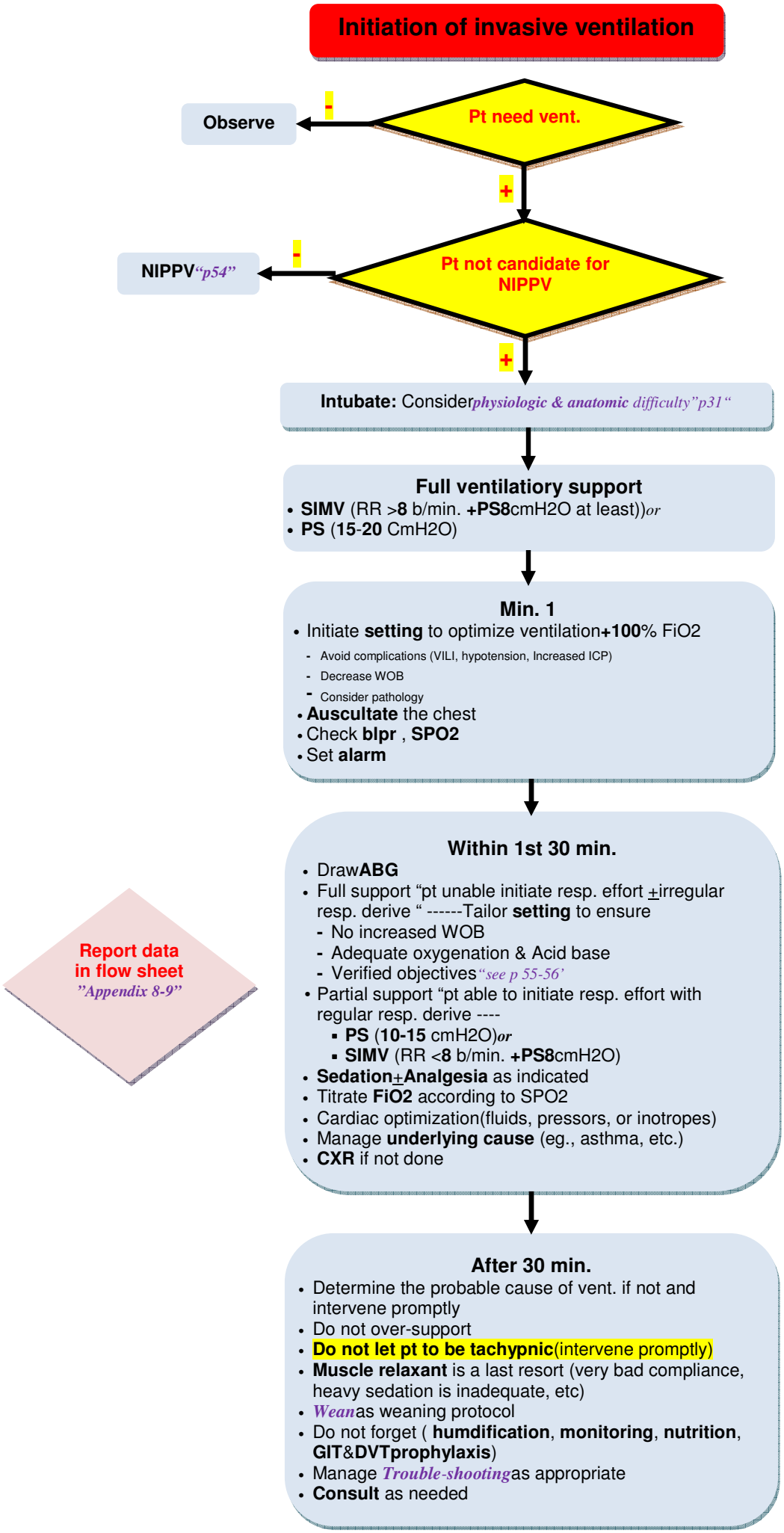
Manifestations:

Respiratory decompensation
- Tachypnea
- Accessory muscle use
- Suprasternal, intercostals, and supraclavicular retractions
- Abdominal paradox

Incraesed sympathetic tone
- Tachycardia
- Hypertension
- Sweating

End-organ hypo-perfusion
- Altered conscious level
- Bradycardia
- Hypotension
- Cyanosis
  - Requires 5 gm reduced Hgb
  - Not obvious in anemic pt
  - Apparent on PO2 40 mmHg and SPO2 75%




Initiation of invasive ventilation
Pt need vent.
-
Observe
+
Pt not candidate for NIPPV
-
NIPPV"p54"
+
Intubate: Considerphysiologic & anatomic difficulty"p31"
Full ventilatiory support
• SIMV (RR >8 b/min. +PS8cmH2O at least))or
• PS (15-20 CmH2O)
Min. 1
• Initiate setting to optimize ventilation+100% FiO2
- Avoid complications (VILI, hypotension, Increased ICP)
- Decrease WOB
- Consider pathology
• Auscultate the chest
• Check blpr , SPO2
• Set alarm
Within 1st 30 min.
• DrawABG
• Full support "pt unable initiate resp. effort ±irregular resp. derive " ------Tailor setting to ensure
- No increased WOB
- Adequate oxygenation & Acid base
- Verified objectives"see p 55-56'
• Partial support "pt able to initiate resp. effort with regular resp. derive ----
▪ PS (10-15 cmH2O)or
▪ SIMV (RR <8 b/min. +PS8cmH2O)
• Sedation±Analgesia as indicated
• Titrate FiO2 according to SPO2
• Cardiac optimization(fluids, pressors, or inotropes)
• Manage underlying cause (eg., asthma, etc.)
• CXR if not done
Report data in flow sheet
"Appendix 8-9"
After 30 min.
• Determine the probable cause of vent. if not and intervene promptly
• Do not over-support
• Do not let pt to be tachypnic(intervene promptly)
• Muscle relaxant is a last resort (very bad compliance, heavy sedation is inadequate, etc)
• Weanas weaning protocol
• Do not forget (humdification, monitoring, nutrition, GIT&DVTprophylaxis)
• Manage Trouble-shootingas appropriate
• Consult as needed

Ventilator setting

FiO2

- After intubation, a **100**% FIO2 should always be used
- Decrease gradually / 20 min. to be below the toxic threshold 60% with saturation 90% or to reach to the least FiO2 with saturation >90%
- Avoid FiO2 > 60% to avoid O2 toxicity by using other techniques to improve oxygenation as PEEP.
- FiO2 should be given for 3 min. before and after suction

RR

- Usually started at **8** breaths/min., may increase according to PaCO2 if the pt is otherwise clinically stable (full ventilatory support)
- < 8 in partial ventilatory support
- Consider higher rates in restrictive lung diseases
- Consider lower rates in chronic respiratory acidosis

Flow Rate

- Pts require a peak flow roughly 4 times that of the VE
- Usually set at **40** - **100** L/min. in volume target ventilation
- High flow rate **90** - **100** L/min in pts with high inspiratory demand, prolonged I:E
- Setslightly higher with the decelerating waveform
- It determines the value of Ti in volume target modes & value of VT in pressure target time cycled modes

VT

- Should be adjusted with R.R. for PaCO2 and PH control (7.30 - 7.45)
- **8** ml/Kg for normal lung (keep Ppl to <**30** cm H2O to limit volu-trauma in pt without IAH)
  - Some ventilators compensate for VT lost in the breathing circuit (keep to **6**ml/Kg)
  - Consider lower VT in asthma, COPD, and ARDS
  - Consider higher VT in neuromuscular diseases

Rising Time

- It is time taken to reach the target pr
- It can affect the total Ti
- The starting value is **0.2** sec
- Alternative to flow (inverse relation)
  - Consider increase if low flow is needed
  - Consider decrease if high flow is needed

Trigger sensitivity

- Should not be too sensitive (avoid auto-cycling)
- Should not be too insensitive (increased WOB)
- Up to**2** L/min. flow &**2** cmH2O pr. is accepted

Ti

- Total Pi is the sum of both inspiratory time and pause time
- Should be set in pr mode
- May be set in volume modes or be the integration of flow, VT or the integration of I:E , respiratory cycle

Pause time

- Delay opening expiratory valve (no flow)
- Is used as a measure of increasing the inspiratory time& to measure Pplat
- May be adjusted in seconds in some ventilators or as a % of

Pressure limit

- To limit increased pressure in volume modes
- A pressure limit of **30** H2O is appropriate in adults

Pi

- Adjust to the value of Ppl during volume targeted ventilation, or the that gives accepted exhaled VT, or ABGs

PS

- Augment the spontaneous breathing by adjustable pr.
- Starting pr "**5 – 8** cmH2O to oppose ETT & circuit resistance
- Increase to the value that decrease the increased WOB

Flow cycling of PS

- Flow cycling % of the peak flow during PS "tailor I:E "
- It is value is inversely proportional to the Ti
  - Consider increased percentage with obstructive diseases
  - Consider decreased percentage with restrictive diseases

I:E

- Normally adjusted to **1:1 - 1:3**.
- ≥ 1:3 ---- to counteract auto PEEP. (COPD pts)
- The following data can be needed to determine I:E ratio
  - Ti, or Ti %
  - Pause time, or %
  - Cycle time "either set directly or calculated by 60/ RR/min
  - I:E ratio can be set directly
- Inversed ratio which is used to improve oxygenation can be done through:
  - Volume targeted mode
    - Increased Ti
    - Decreased flow rate
    - Adding inspiratory pause
  - Pressure targeted modes:
    - Adjusted directly

PEEP

- A positive airway pressure during expiration
- **Indications**
  - ALI
  - Oppose auto PEEP
  - Primary ttt for pulmonary edema
- **Relative contraindications to PEEP:**
  - Unilateral lung disease - Bronchopleural fistula.
  - Hypovolemia**-** Increased ICP
- **Adjustment**
  - In pts with good oxygenation and little pulmonary disease ----- PEEP of **3** - **4** is adequate "**physiologic PEEP"**
  - In pts with bad oxygenation ------- PEEP added in increments of **2** - **5** cm until "**best PEEP"** (The level which provides the highest static compliance "in other words, the lowest Ppl without complications")
- **Consideration**
  - Changes in ABGs for 30 min. May take > 30 min,
  - Circuit should not be broken unless absolutely necessary
  - A PEEP of <**10** cm H2O usually improves PaO2without significant complications
  - PEEP >**20** cm is rarely beneficial and usually results in additional pressure-induced lung injury

Flow Pattern

- In Pressure Control, the flow pattern is decelerating (preset and not adjusted)
- In volume modes "**Bennet** ventilator only", 3 wave patterns can be used

Decelerating flow

- Should be chosen unless there is limitation
- The most effective flow pattern (gives the highest MAP & lowest PIP & improve oxygenation)
- The addition of inspiratory pause with a decelerating flow pattern can simulate pressure mode

Square Wave

- Allows for an adequate I:E ratio with a normal flow rate
- Beneficial if air trapping is a concern

Sine Wave

- Used if high PIP are encountered with square wave

Tailoring of ventilatory support

Neuromuscular Disease

- **NIPPV** is recommended in pts when they present in acute - on-chronic RF & candidate for NIV(**Level III**)
- Use **total** or **partialventilatorysupport** based on the pt's inherent ventilatory muscle strength
  - Pt with a high quadriplegia (e.g., C1-2) lesion needs full ventilatory support whichever mode is used
  - Partial ventilatory support may be appropriate for pts with some ventilatory capability, esp. during recovery
- They are a low risk for barotrauma and frequently prefer large VTs (eg, >**8** ml/kg Ibw) with or without PEEP (**5** - **10** cm H2O)
- Adjust flow rate" PFR "to satisfy pt demands (>**60** L/min)

Post cardiac arrest

- No routine defined period of ventilation, sedation, muscle relaxaton post cardiac arrest
- Adjust ventilaton to maintain normocapnea
  - Post cardiac arrest, hypocapnea induced by hyperventilation causes cerebral ischemia
- **Invasive full ventilatory support**& "**pr** or **volume**"

Cardiogenic pulmonary edema

- When life-threatening hypoxemia accompanies severe CHF ----- consider the potentially beneficial effect of PPV on decreasing venous return and improving oxygenation
- Consider assessment of effect of PPV on hemodynamics
- **CPAP/NIV** is recommended plus standard medical ttt (**Level I**)
- **NIV BIPAP** is preferable in hypercapnic RF (**Level II**)

Cardiogenic shock ,& myocardial Ischemia

- Spontaneous ventilation with increased WOB is likely detrimental
- Choose modes of MV that minimize WOB aiming at lowering systemic and/or myocardial O2 consumption
- **Invasive full ventilatory support**&**pr** or **volume**"

PO Pts

- Ventilate to facilitate heavy sedation ± MR in which agitation, excess movement, or cough will be deleterious
- **Invasivefullventilatorysupport**& "**pr** or **volume**"
- **NIPPV**May be used in pt who develop RF or failure after lung resection or abdominal surgery (**levelII**)

TBI

- There is no evidence to apply prophylactic hyperventilation in pts do not have raised ICP
- The increase in MAP associated with a dramatic increase in VE may cause a paradoxic increase in ICP
- PEEP up to 10 cm H2O is accepted with HOB 45 ^{0}
- Ventilatory support may be indicated to facilitate sedation and analgesia as an initial management of increased ICP
- Adjust ventilaton to maintain normocapnea"Hypocapnea causes cerebral ischemia"
- Other application is in refractory brain edema (refractory to positioning,sedation, dehydrating measures, and CSF aspiration in absences of surgically corrected causes) ---- ----Do not decrease PaCO2 below 30 mmHG
- **Invasivefullventilatorysupport**& "**pr** or **volume**"

Bronchopleural Fistula (BPF)

- Some are amenable to direct surgical repair, but mostly, resolution depends on resolution of primary process
- Chest tube suction is necessary to evacuate continued gas leak "degree of suction exerts a variable effect on flow through fistula"
- To facilitate closure
  - **a.** Use the lowest VT that allows adequate ventilation
  - **b.** Use a ventilatory mode & settings that minimize PIP &Pplt necessary to maintain adequate ventilation
  - **c.** Consider permissive hypercapnia
  - **d.** Minimize **PEEP**
- No single effective ventilatory mode or approach
  - Ventilation of pts with BPF is usually adequate
  - Consider **ILV***"p53"*or **HFJV** in cases where a large air leak produces inability to inflate lung or failure to adequately oxygenate/ventilate

ARDS & ALI

- The main 2 **Objectives** are to Prevent lung injury mainly volutrauma by applying low VT and keeping Ppl<**30**. Better **28**cm H2O, and atelectrauma by applying PEEP" **Lung protective strategy"**, and to Improve oxygenation
- **Invasive vs noninvasive**
  - Use invasive MV rather than an initial trial of NPPV (**Level 2C**)
- **Mode selection**
  - **Volumelimited** and **prlimited** modes are both acceptable; "no evidence that pr limited approaches confer additional benefit"
  - **Fully supported modes** are favored (**Level 2C**)
- **Lung protective strategies (LTVV)"ARMA trial"** (**Level 1B**)
  - Set mode to volume assist-control
  - Set initial VT to **8** ml/kg IBW
  - Reduce VT to **7** and then to **6** ml/kg over 1-3 hr
  - Set initial RR ≤**35** b/min. to match baseline VE
  - Subsequent VT adjustment Pplt goal ≤**30** cmH2O
    - Check inspiratory Pplt with 0.5 S inspiratory pause at least / 4 hrs and after each change in PEEP or VT
    - IfPplt>30 cmH2O, decrease VT in 1 ml/kg PBW steps to 5 or if necessary to 4 mL/kg PBW
    - If Pplt<25 cmH2O and VT <6 ml/kg, increase VT by 1 mL/kg PBW until Pplt>25 cmH2O or VT = 6 ml/kg
    - If breath stacking (PEEPi) ± severe dyspnea--- VT may be increased to 7 or 8 mL/kg IBW if Pplt remains ≤30 cmH2O
- Oxygenation goal PaO2 **55**-**80** mmHg or SpO2 **88**-**95** %
  - Use FiO2/PEEP combinations to achieve oxygenation goal:

FiO2	0.3	0.4	0.5	0.6	0.7	0.8	0.9	1.0
PEEP	5	5-8	8-10	10	10-14	14	14-18	22

Consider using high PEEP rather thanincreasingFiO2 > 60%

- **Refractory hypoxemia:** Consider; IRV, prone ventilation, HFV

- **HFV** provides VT below that of the anatomic dead space at RR >60 b / min. Potential benefits include reduced barotrauma, improved V/Q matching, and less risk of hemodynamic compromise.Complications include inspissation of mucus, airway damage, air trapping, should be reserved as salvage therapy for pt failing conventional ventilation

COPD & Asthma

NIV

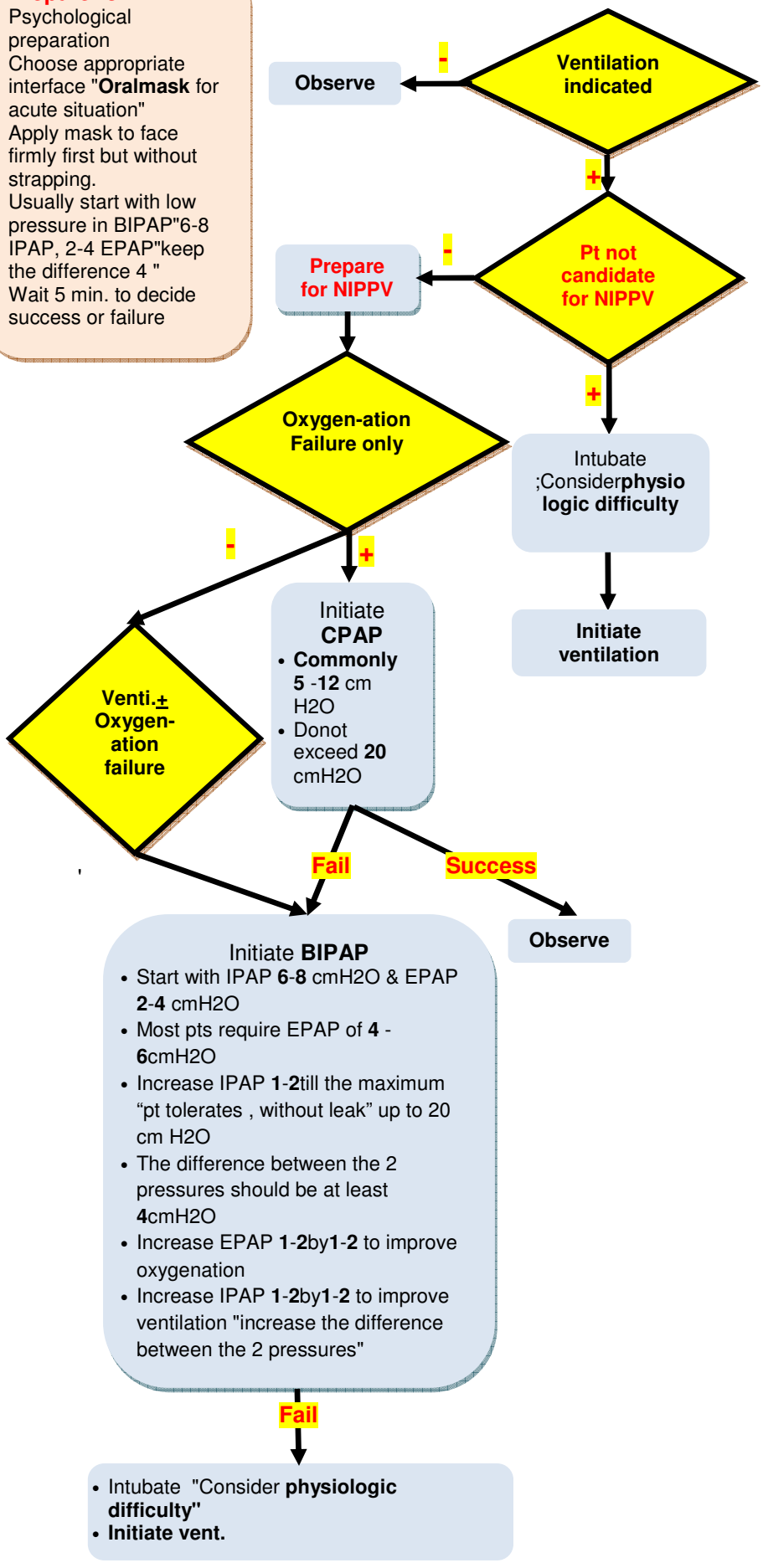
- **BIPAP** is the preferred mode of NPPV for COPD pt**.** Consider with standard medical therapy, when they present in acute exacerbation (PH < 7.35, PaCO2 > 45mmHg) (**Level I**). Pts with relatively mild exacerbation of COPD (PH>7.35) may not benefit from NIV (**Level II**). However, it does not cause harm
- No routine use in asthma exacerbation (**Level III**)
  - Try in acute severe asthma who fail to respond quickly to medical ttt and have no CI (**Level II**)

Invasive full ventilator support

- Indicated in severe RF or in which NIV is CI
- No evidence that one mode is better than another ---- Choose a pr or volume modes (**pr**targeted modes may not apply well in asthma "requires a high pr")
- Maintain Pplt<**30** cm H2O
- Sedation ± paralysis "last resort" may be necessary in somepts if the ventilation mode cannot be matched to pt's needs (i.e., patient "**fighting**" the ventilator)
- Adjust PaCO2 to the basal not the normal in COPD
  - That gives accepted PH
  - **2*HCO3 -8 "*Redwan 96*"**
  - If confused "keep 60 mmHg"
- Monitor for & minimize **PEEPi** by:
  - **Change ventilator settings**
    - Increase expiratory time, decrease RR&VT
  - **Reduce ventilatory demand**
    - Reduce anxiety, pain, fever, shivering, reduce dead space, give sedatives and paralytics
  - **Reduce flow resistance**
    - Use large-bore ET, suction frequently, give bronchodilators
  - **Counterbalance expiratory flow limitation**
    - **External PEEP**
      - 75-85% of measured PEEPi"
      - Till WOB decreased
      - Till PIP and Pplt start to increase

Non-invasive ventilatorysupport"NPPV"

Prepare for NIPPV
- Psychological preparation
- Choose appropriate interface "**Oralmask** for acute situation"
- Apply mask to face firmly first but without strapping.
- Usually start with low pressure in BIPAP"6-8 IPAP, 2-4 EPAP"keep the difference 4 "
- Wait 5 min. to decide success or failure



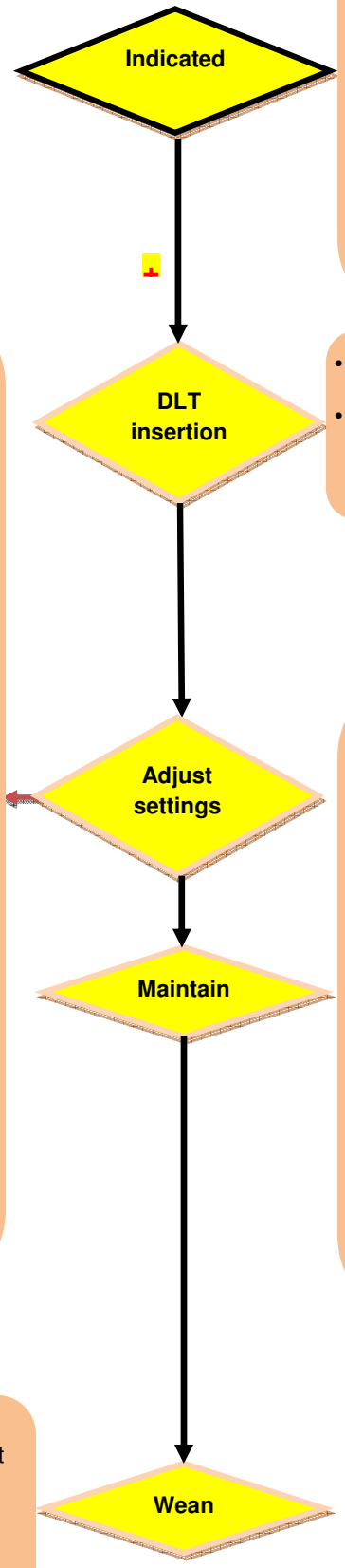
Pt not candidate for NIPPV
- Respiratory arrest
- Need for immediate intubation
- Inability to protect airway
- Intractable vomiting
- Inability to clear secretion
- Inability to cooperate or tolerate mask vent.
- Hypotension (S bl.pr < 90mmHg)
- Uncontrolled arrhythmia
- Upper airway obstruction
- Relative CI --- Morbid obesity, bronchospasm

CPAP uses
- Lung contusion, chest trauma with flail chest
- Plus standard medical ttt in cardiogenic pulmonary edema (**LevelI**)
- May be used with great caution in cases of ALI (**Level III**)

BIPAP uses
- NIV is not recommended for routine use of asthma exacerbation. (**Level III**). NIV may be tried in ICU in pts of acute severe asthma who fail to respond quickly to medical ttt and have no CI
- Respiratory distress or RF after lung resection or abdominal surgery
- Neuromuscular disease and chest wall deformity when they present in acute - on-chronic RF if the pt can handle secretions
- Obesity hypoventilation syndrome (Central alveolar hypoventilation syndrome) with acute RF
- Hypercapnic cardiogenic pulmonary edema

Independent lung ventilation "ILV"

- **ILV is a rare and technically demanding procedure for managing unilaterallung dis. or injury in pts who have failed conventional modes of MV**
- **Nocontrolled clinical trials of ILV exist and there are no clear-cut indications for its use**
- **In select critically illpts, ILV can significantly improve aeration of collapsed alveolar segments, increase systemicoxygenation, reduce hypoventilation, and reduce intrapulmonary shunt fraction**
- **While the need to applyILV is rare, it is a skill with which all physicians who manage the critically ill should be familiar**



- Pat with radiographically apparent unilateral lungdis. and 1 or more of "**Level III**":
  - Hypoxemia refractory to high FiO2 and PEEP
  - PEEP-induced deterioration in oxygenation or shunt fraction
  - Over-inflation of the noninvolved lung with or without collapse of the involved lung
  - Significant deterioration in circulatory status in response to PEEP
- Pt with BPFwith1 or more of "**Level III**":
  - Air leak exceeding 50% of the delivered VT
  - Hypercapnic respiratory acidosis (pH<7.30)
  - Refractory hypoxemia particularly in pts in whom increases in PEEP exacerbate air leak
  - Persistent lung collapse despite optimum catheter drainage

- Proper placement and positioning isessential to the success of ILV
- The volume of air necessary to seal cuffs of a DLT is relatively small; inflation with largervolumes can lead to mucosal injury and even bronchial rupture

Synchronous ILV

- The RR applied to each lung is the same,but the VT, flow, PEEP, and FiO_2 are selectively titrated to optimize oxygenation andventilation while minimizing the potential for VILI in each lung
- Requires 2 ventilators with special software that are synchronized using anexternal cable

AsynchronousILV

- RR, VT, flow, PEEP, FiO_2, andeven mode of support (controlled MV, intermittent mandatory ventilation, pressurecontrol ventilation, high frequency oscillatory ventilation, CPAP, etc…) candiffer from one lung to the other
- Does not require specialized software packages andis considered to be less complicated than synchronous ILV

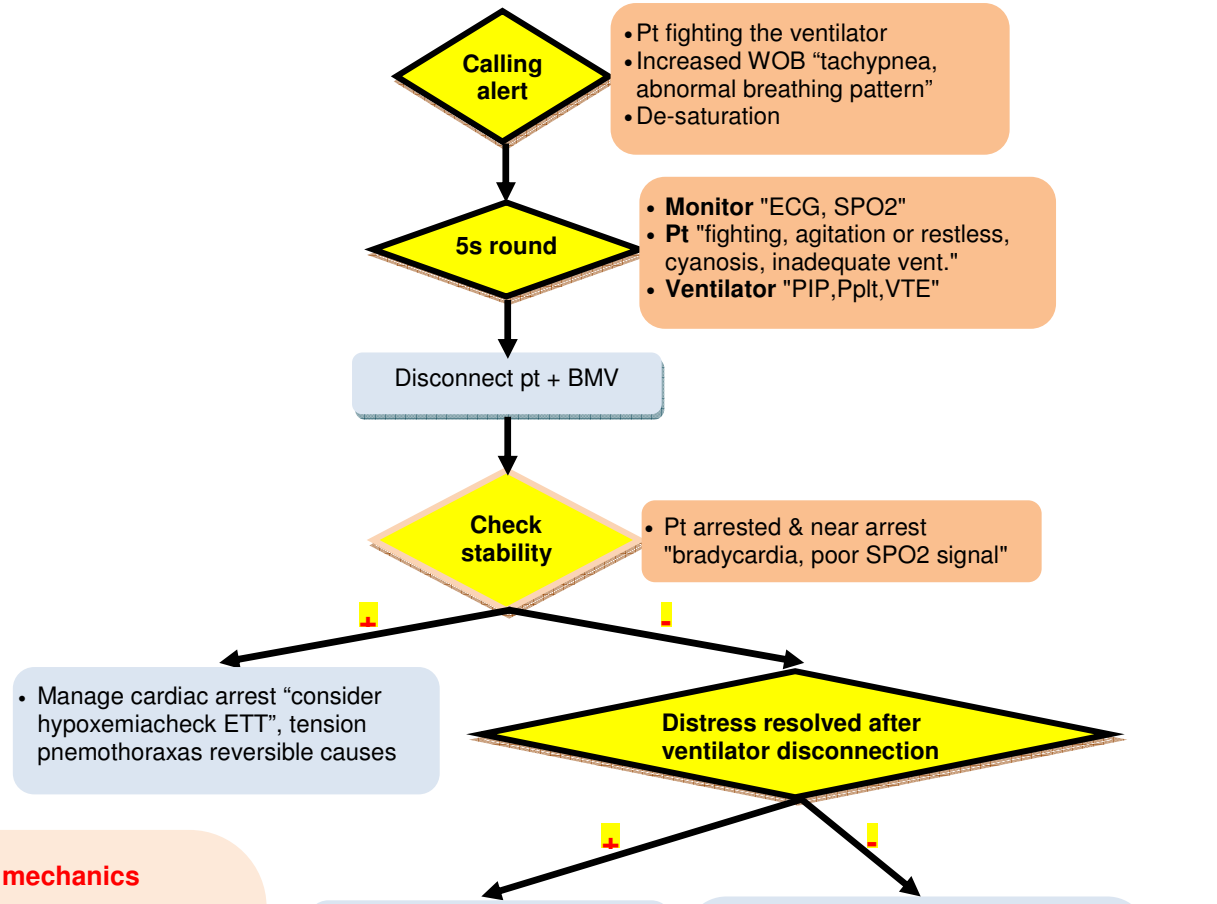
Starting settings

- VT of **5** mL/kg in the normallung and **2** mL/kg in the injured lung, titrated to achieve adequate ventilation while maintaining Pplats below **26** cm H_2O, which has been demonstrated to optimize PaO_2/FiO_2 and compliance
  RR, flow, PEEP, and FiO_2 should be adjusted to optimize oxygenation and CO2 excretion while, in the pt with a BPF, simultaneously minimizing air leak

- If DLT displacement is suspected, tube position should immediately be confirmedvia bronchoscopy with repositioning as necessary.
- The small lumens of a DLT can make bronchial hygiene, suction, and bronchoscopy difficult
- Even small movements of the tube, as may occur during routinept care, may compromise lung separation and ILV. Sedation and neuromuscular paralysis are commonly necessary
- **Monitor For complications**
  - Laryngeal trauma, bronchial trauma, and obstruction of the DLT lumens
  - The small lumens of a DLT can makebronchial hygiene and suctioning as well as bronchoscopy difficult if not impossible
- Invasive hemodynamic monitoring is advisable to monitor the effect of ILV on cardiac output and intrapulmonary shunt

- ILV shouldbe continued as long as is necessary to allow sufficient healing of the injured lung such that the VT and compliance of the 2 lungs differs by less than **100** mL and **20**% respectively
- At thattime, conventional mechanical ventilation using a single-lumen ETTcan generally bereinstated and pt weaned as tolerated

Troubleshooting during ventilatory support



Lung mechanics

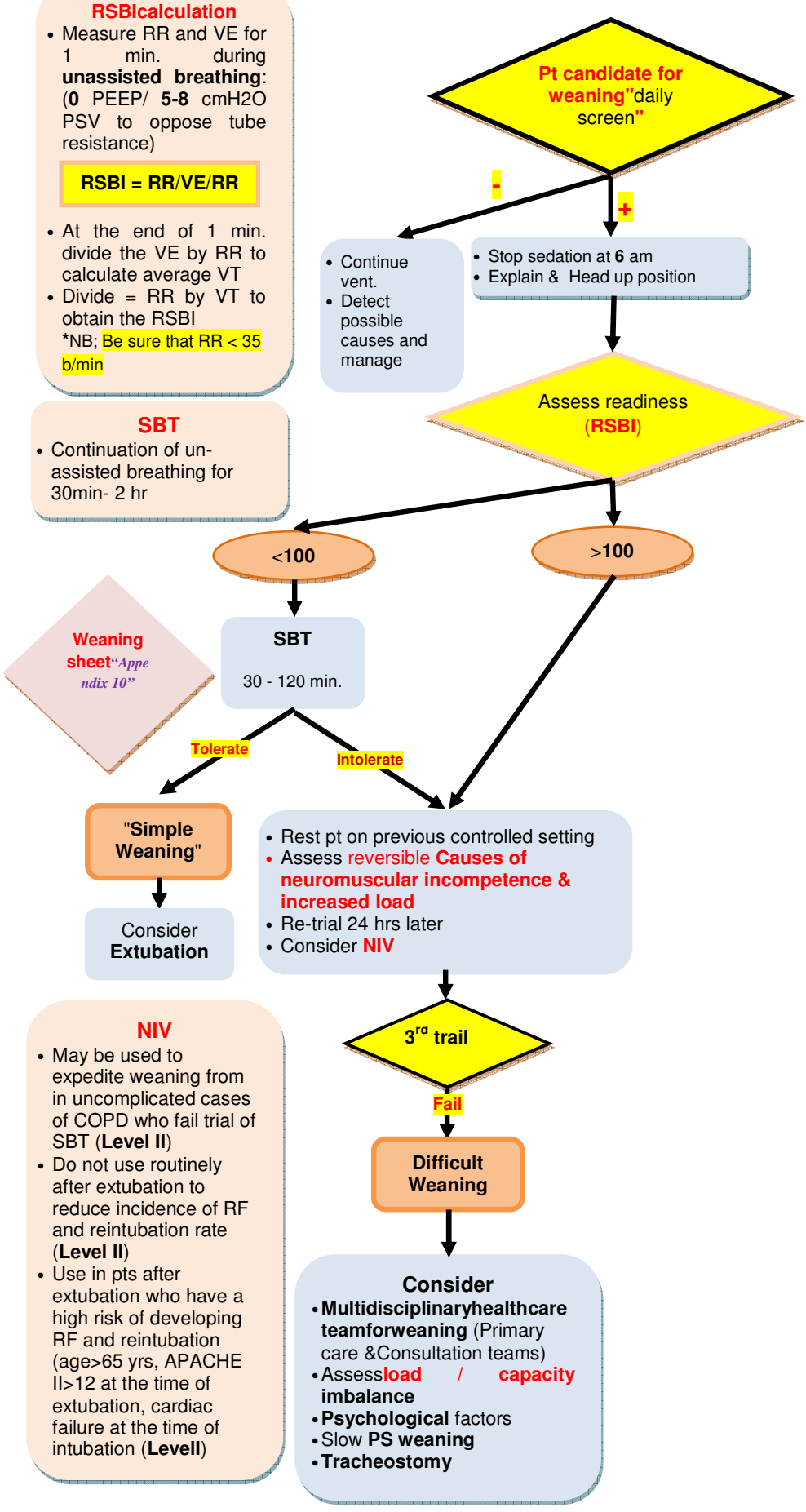
Low PIP
- Disconnection & cuff leak
- Hyperventilation "pain, anxiety"

Normal PIP
- PE
- Extra-thoracic cause

High PIP + High Pplt "Decreased compliance "
- PEEPi
- Atelectasis
- Abdominal distension
- Large pleural effusion
- Stiff lung "pneumonia, ALI, pulmonary edema"

High PIP + NormalPplt "Increased resistance"
- ETT obstruction"mucous plug, kink"
- Acute bronchospasm
- Aspiration of gastric content

Weaning& Discontinuation



RSBIcalculation

- Measure RR and VE for 1 min. during **unassisted breathing**: (**0** PEEP/ **5-8** cmH2O PSV to oppose tube resistance)

RSBI = RR/VE/RR

- At the end of 1 min. divide the VE by RR to calculate average VT
- Divide = RR by VT to obtain the RSBI

*NB; Be sure that RR < 35 b/min

SBT

- Continuation of un-assisted breathing for 30min- 2 hr

Pt candidate for weaning

- An improvement in or some resolution of the underlying **cause**
- **Pao2/Fio2>150 - 200**
- Can initiate **respiratoryeffort**
- **Hemodynamically** stable (Absence of active ischemia & S bl pr> 90 mmHg, with minimal pressors &inotropes (<5ug/Kg /min. dobutamine, dopamime)
- No need for heavy sedation

Signs of SBT tolerance

- RR<**35**/min.
- SaO2 >**90**% with up to 10% increase in FiO2
- HR & bl pr stability within 20% of baseline
- ICP <**20** mmHg
- Absence of marked use of accessory muscles, abdominal paradox, diaphoresis, excessive dyspnea, decreased consciousness,and agitation attributed to increased WOB
- Spontaneous VT>**5**ml /Kg IBW

Increased load

Chest wall
- Abdominal distension & ascitis
- Pnemothorax & Rib fracture

Lung
- Alveolar edema &PEEPi
- Atelectasis & infection

Minute ventilation
- Sepsis &PE
- Excess calories

Resistive load
- Bronchospasm “asthma & COPD”
- Obstructive sleep apnea
- Upper airway obstruction “croup”
- Edema & secretion of airway

Neuro-muscular incompetence

Decreased respiratory drive
- Hypothyrodism & Drug overdose
- Brain stem lesion
- Sleep-disorded brathing

Impaired neuro-transmission
- Aminoglycosides
- Guillian barre s & Myasthenia Gravis
- Phernic nerve palsy
- Spinal cord lesion
- Neuro-muscular blockers

Muscle weakness
- Electrolytes disorders&Malnutrition
- Hypoperfusion&Myopathy
- Disuse atrophy
- Incomplete rest between trails

NIV

- May be used to expedite weaning from in uncomplicated cases of COPD who fail trial of SBT (**Level II**)
- Do not use routinely after extubation to reduce incidence of RF and reintubation rate (**Level II**)
- Use in pts after extubation who have a high risk of developing RF and reintubation (age>65 yrs, APACHE II>12 at the time of extubation, cardiac failure at the time of intubation (**Levell**)

Extubation

- Delay extubation (**Level 2C**)

Pt not candidate for extubation

+

- Pts whose cough strength is weak, whose GCS is < 8, or who require suctioning >/ 2-3 hr

-

Determine pt at stridor risk

-

≥1 of the following:
- Traumatic intubation
- Prolonged intubation (>7 ds)
- Traumatic injury
- Oversized ETTs
- Self extubation

+

Cuff leak test

- Cuff leak " Leak of the administered VT upon deflation of the ETT" cuff test is an adequate test to assess for laryngeal edema(**Level II**)

+ve

Extubation likely successful (Level II)

-ve

Extubation unlikely successful

Administer **Steroids**

Consider
- **Extubation** if cuff leak test is +ve
- **ENT consultation** if still -ve

Extubate
- Pt is placed in upright position and both oral cavity & ETT are suctioned
- Pt to take a deep breath and exhale
- During exhalation, the cuff is deflated and ETT is removed in a single, smooth motion
- Oro-gastric tubes are typically removed simultaneously
- Following extubation, oral cavity is again suctioned and supplemental O2is administered by facemask
- The SPO2, HR, RR, and blpr are monitored throughout the extubation process. Pts with increased secretions may require more frequent or nasotracheal suctioning

- Humidified **O2**
- Monitor SPO2
- Observe for stridor
- Early aggressive management can prevent re-intubation" suctioning, bronchodilator therapy, diuresis, or **NPPV"**

Steroids

- Decreases post-extubation stridor & re-intubation in pts at increased risk for extubation failure due to edema (**Level I**)
- Administered >**6** hrs prior to extubation to be effective in reducing edema (**Level I**)
- **Dexamethasone4**mgIV/6 hrs **(Level III**)

Cuff leak test

- **Qualitative** ---- deflate cuff and listening for air movement around the ETT using a stethoscope over upper trachea
- **Quantitative** --- deflate ETT cuff and measure difference between VTi & VTe of ventilator-delivered breaths during volume-cycled MV
- The lowest 3 VTEs obtained over 6 breaths are averaged and subtracted from VTI to give the cuff leak volume
- Cuff leak volumes <**110** mL or <**12** - **24** % of the delivered VT have been suggested as thresholds for determining whether airway patency may be diminished

NPPV

- Routine use of NIV after extubation for reducing incidence of RF and reintubation rate is not recommended (**Level II**)

Prone ventilation

- MV with the pt lying in the prone position
- It improves oxygenation in most pts with ARDS, although the mechanism and clinical benefit of this effect are uncertain

Consultant decision

Indicated

- Used as early rescue therapy for refractory hypoxemia after conventional ventilatory strategies has been failed
- No CI-- spinal & hemodynamic instability, thoracic, abdominalsurgeries"relative", facial, pelvic fractures, increased ICP, life-threatening arrhythmias
- We do not use prone ventilation as routine for pts with ARDS

+

Consider

- Possible adverse effects of positioning on chest tube drainage
- Confirm from a recent CXR that tip of the ETT is located 2-4 cm above main carina
- Inspect and confirm that the ETT and all central and large bore peripheral catheters are firmly secured
- Consider exactly how pt's head, neck, and shoulder girdle will be supported after turning prone
- Stop EF, check for residual, fully evacuate stomach, and cap or clamp the feeding and gastric tubes
- Prepare endotracheal suctioning equipment, and review what the process will be if copious airway secretions abruptly interfere with ventilation
- Decide whether the turn will be rightward or leftward
- Prepare all IV tubing and other catheters and tubing for connection when pt is prone
  - Assure sufficient tubing length
  - Relocate all drainage bags on the opposite side of the bed
  - Move chest tube drains between legs
  - Reposition IV tubing toward patient's head, on opposite side of bed

Apply

- Place one (or more) people on both sides of the bed (to be responsible for the turning processes) and another at the head of the bed (to assure the central lines and the ETT do not become dislodged or kinked)
- Increase the FiO2 to 1.0 and note the mode of ventilation, the VT, the VE, and PIP &Pplt
- Pull the pt to the edge of the bed furthest from whichever lateral decubitus position will be used while turning
- Place a new draw sheet on side of bed that the pt will face when in this lateral decubitus position. Leave most of the sheet hanging
- Turn the pt to the lateral decubitus position with the dependent arm tucked slightly under the thorax. As the turning progresses the non-dependent arm can be raised in a cocked position over the pt's head. Alternatively, the turn can progress using a log-rolling procedure
- Remove ECG leads and patches. Suction the airway, mouth, and nasal passages if necessary
- Continue turning to the prone position
- Reposition in bed center using new draw sheet
- If the pt is on a standard hospital bed, turn his/her face toward the ventilator. Assure that the airway is not kinked and has not migrated during turning. Suction the airway if necessary
- Support the face and shoulders appropriately avoiding any contact of supporting padding with orbits or eyes.
- Position the arms for pt comfort. If the pt cannot communicate, avoid any type of arm extension that might result in a brachial plexus injury
- Auscultate chest to check right mainstem intubation. Reassess the VT and VE
- Adjust all tubing and reassess connections and function
- Reattach ECG patches & leads to back.
- Tilt the pt into reverse trendelenburg. Slight, intermittent lateral repositioning (20-30º) should also be used, changing sides at least / 2 hr
- Document a thorough skin assessment every shift, specifically inspecting weight bearing, ventral surfaces

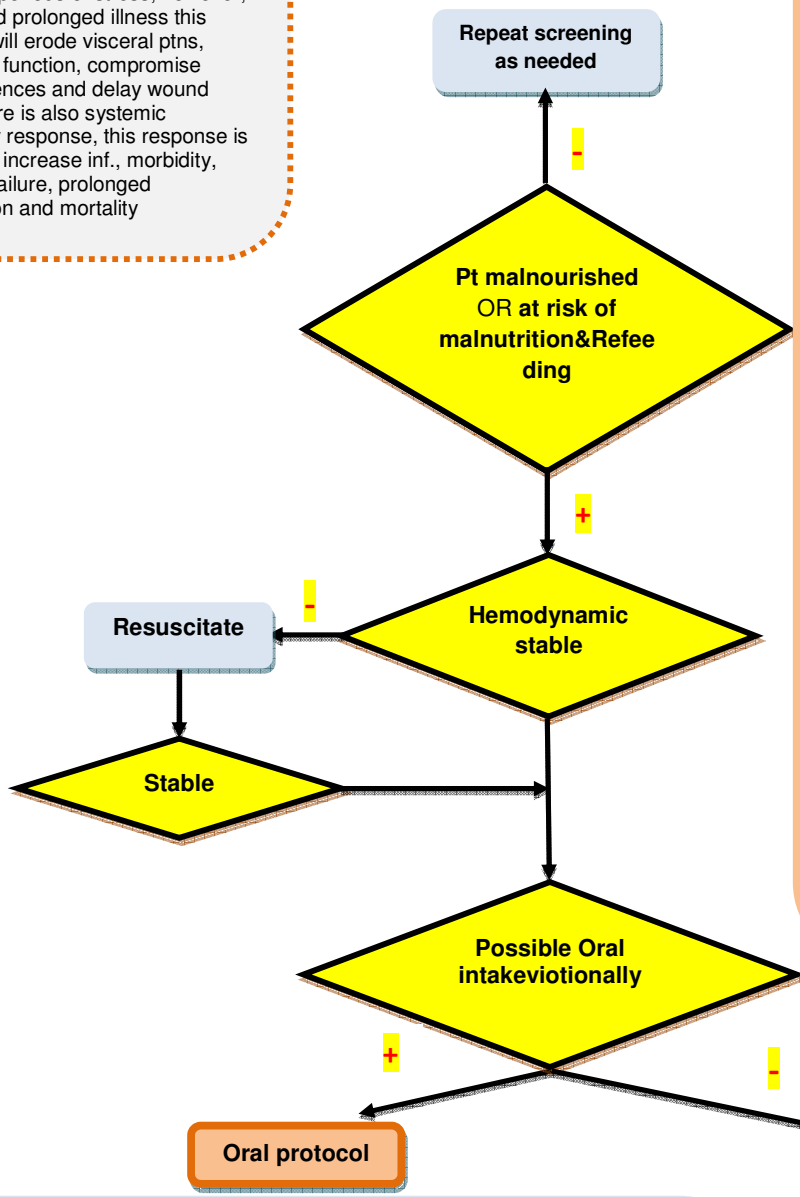
Post procedure

- PIP &Pplt tend to increase immediately after a pt is placed in the prone position, but typically decline with time
- Paralysis is not required and potentially harmful
- No need for additional monitoring, although the need for endotracheal suctioning should be assessed with increased frequency after being prone
- All involved staff should know how to quickly put the pt back into the supine position for CPR
- Maintain prone ventilation for as long as it is effective, with position changes only as needed for nursing care
- Transient hemodynamic instability and de-saturation can be minimized by providing adequate sedation and pre-oxygenating with 1.0 FiO2
- Skin breakdown over pressure points and dependent facial edema can occur. These adverse events can be minimized by frequent repositioning and soft padding
- Rate of EN should be reduced

Nutritional support

Screening for nutrition support

- Critical illness is associated with an increased metabolic rate, breakdown of skeletal muscle, gastrointestinal mucosa and other tissues. This tissue breakdown may provide adequate nutrients for organ function and integrity during short periods of stress, however, in severe and prolonged illness this breakdown will erode visceral ptns, impair organ function, compromise immune defences and delay wound healing. There is also systemic inflammatory response, this response is coupled with increase inf., morbidity, multi organ failure, prolonged hospitalization and mortality



Malnutrition

- **BMI<18.5** Kg/m^2
- Unintentional weight loss >10% within last 3 – 6 ms
- BMI <**20** Kg/m2 and unintentional weight loss >5 % within last 3 – 6 ms

At risk of malnutrition

A.

- Critically ill with expected stay >3 ds
- Eaten little or nothing for >3-5 ds and/or likely to eat little or nothing for the next 5 ds or longer
- Poor absorptive capacity, and/or high nutrient losses "mal-absorption, fistula, short bowel S" ± increased nutritional needs from catabolism "burn, trauma, and sepsis"*or*

B.

- **Nutrition risk score**

Re-feeding risk

- Life threatenening condition encompassing acute micronutrient deficiencies, fluid , electrolyte imbalances, and disturbances of organ function and metabolic regulation that may result from over-rapid or unbalanced nutrition support provision to malnourished pt
- **NICE2006criteria** to determine risk :

≥ 1 of	≥2 of
BMI <16 Kg/m^2	BMI <18.5 Kg/m^2
Unintentional weight loss >15% in the last 3-6 ms	Unintentional weight loss >10% in the last 3-6 ms
Little or no nutritional intake for >10 ds	Little or no nutritional intake for >5 days
Low level of K, Ph, Mg prior to feeding	History of alchol abuse or drug as insulin, diuretics, chemotherapy, antacids

- Consider oral nutrition support to improve nutritional intake for people who can swallow safely
- Overall nutrient intake should offer a balanced dmixture of ptn, energy, fiber,electrolytes, vitamins, and minerals
- If there is a concern about the adequacy of mico-nutrient intake, a complete oral vitamins, and mineral supplement providing the reference nutrient intake for all vitamins and trace elements should be considered
- Oral nutrition support should be stopped when the patient is established on an adequate oral intake
- Peri-operative oral nutrition support should be considered for surgical patients who can swallow safely
- Consider some oral intake within 24 hrs post CS or gynaecological surgeries
- Consider some oral intake within 24 hrs post abdominal surgeries in pts who can swallow and without specific concerns about gut function ,with monitoring for any signs of N&V

Nutrient requirement

IBW

Male
- Height **150** cm, weight **48** kg
- Every **2.5** cm above **150** cm of height, **add** 2.7 kg

Female
- Height **150** cm, weight **45** kg
- Every **2.5** cm above **150** cm of height, add **2.3** kg

Nutritional goals

Should focus on
- The ptn, calories and micronutrients needs
- The potential response of pts to different nutrients
- The composition of specialized formulas

Energy

- **25** Kcal/Kg "Espen 2009" During acute and initial phases of critical illness an exogenous energy supply in excess of **20**–**25** kcal/kg BW/d should be avoided, whereas, during recovery, the aim should be to provide values of **25**–**30** total kcal/ kg BW/d
- Consider **Hypocaloricfeeding** in critically ill obese BMI >**30** Kg/m^2 **(60** -**70** % of total energy requirement or **11** -**14** Kcal/Kg actual body weight or **22** -**25** Kcal/Kg IBW "Aspen 2009")
  - Re-feeding syndrome
  - Severe malnutrition
  - Trauma pts following shock resuscitation
  - Hemodynamic instability
  - ARDS or COPD
  - SIRS or sepsis

❖ Use actual body weight if weight is < IBW

Ptn

- Contain **4** Kcal /g
- **1.2** - **2** g/Kg if BMI <30 Kcal/Kg, and may likely be even higher in burn or multi-trauma pts (**Level E**)
  - **2** g/Kg if BMI 30 -40 Kcal/Kg
  - **2**.5 g/Kg if BMI >40 Kcal/Kg "Aspen 2009"
- **15** -**20** %"mild to severe stress" of total calories
- Non-ptn calorie/ g of nitrogen ratio for critically ill = **100**:**1** "**150** : **1** in mild stress to severe stress"

Fat/Lipid*

- Fat contain **9** Kcal /g
- Should provide **30** -**40**% of total calories
  - Diabetic: **60** % fat & CHO **40**%
  - Pulmonary: **50** -**60** % fat & CHO **40** -**50** %
- **0.7** – **1.5** g/Kg "Espen 2006"Caution with use of fats in stressed & trauma pts:
  - High fat feedings (esp LCT) cause immno-suppression
  - New formulas focus on Omega 3 Fatty Acids.
- Intolerance sings:
  - Hyperlipidemia
  - Impaired Immune function
  - Hypoxemia

❖Consider all lipid sources as propofol, or glucose sources as glucose infusions when calculating energy

Glucose *

- Contain **4** kcal /g
- Should provide **60**-**70**% of total calories

NB
- **100** g glucose is needed to prevent ketosis
- Maximal oxidation rate **4** -**7** mg/Kg /min./24 hrs
- Ideally keep <**4** -**7** mg/Kg /min./ 24 hrs "Espen 2009"
- Minimum **2** g /Kg
- Blood glucose levels should be monitored and nutrition regimen and insulin adjusted to maintain glucose between **110**-**150** mg/dl

Fluid Needs

- **30** - **40** ml/Kg
- **1** ml/kcal baseline+ cover Additional losses – (ie. fever, diarrhea, GI output, tachypnea)
- Fluid restriction – CHF, renal failure, hepatic failure, CNS injury, and electrolyte abnormality

Potential source of fluid loss & excess

Intake	Output
• Maintainace IV fluids • Medications given via IV drip • Water flushes with crushed medications • Water flushes to keep tube patent • Water contained in tube feedings or PN	• Chest tubes • Percutaneous drains - Billary& pancreatic • Wound drainage • Ostomies/stool/ urine • Naso/Orogastric suction • Excess drolling&siallorrhea • Fistulas • Insensible losses - Burn –Tracheostomy -Fever

Electrolytes & Vitamins & Trace elements

Nutrient	Daily requirements/day
Sodium	**2**mmol/Kg
Potassium	**1**mmol/Kg "depends on renal function"
Magnesium	**0.2**mmol/Kg "depends on renal function"
Phosphate	**0.3**mmol/Kg "depends on renal function"
Calcium	According to clinicalcondition
Trace elements	Zinc, copper,chromium, manganese, selenium, iodide
Multivitamins	B group;b12,folate, A,D,E
Vitamin K	Weekly unless on warafarin

Enteral nutrition "EN"

- Critical illness is associated with gut mucosal atrophy and increased permeability and a failure in the "barrier" function of the mucosa to the translocation of bacteria or endotoxin
- Poor gut perfusion implicated as a key element in ongoing organ dysfunction in critically ill
- Delivery of essential nutrients to gut mucosa and improved gut perfusion has been attributed to the use of the enteral route for feeding
- Early EN associated with less gut permeability, diminished activation and release of inflammatory cytokinase

EN CI

- Bowel obstruction & ischemia, gastroparesis, intractable vomiting& diarrhea, active gastrointestinal bleeding " **absolute**"
- Enteric fistula, imminent bowel resection & endoscopy "**relative**"
- Acute pancreatitis & Enteric anastomosis may still option for **Elementalfeeds**

\+ / -

No CI to gastric feeding

- Gastroparesis or severely delayed gastric emptying &acute/chronic gastro-esophgeal reflux& major intra-abdominal surgery
- Gastric feed intolerance "GRV >**250**ml despite prokinetics"
- **High risk pulmonary aspiration**"**Level III**"
- Acute severe pancreatitis

PN protocol*"p 68"*

Consider post-pyloric feeding

Initiate

Timing
- Within the 1 st 24 hrs of admission

Access
- Insert Naso-entric tubes surgically or endoscopically or manually
- Obtain direct small bowel access in pt undergoing laparotomy for blunt & penetrating abdominal injuries (via nasojejunal feeding tube, gastrojejunal feeding tube, or feeding jejunostomy) and EN begun as soon as is feasible following resusc.**(Level III)**

Delivery
Determine energy & nutrient requirement
- Deliver continuously over 16–24 hrs
- Start standard **20** ml**Polymeric feeding**"**1Kcal/ml**, unless **Specialized formula** is indicated
- Increase **20 ml**/4 hrs till target

Maintain

- Do not aspirate jeujenal feeds
- Medications in liquid form (not crushed tablets)
- Reflux into the stomach is still possible ----- check GRV via an NGT or gastric port of PEG/PEJ tube / 6 hrs x 24hrs then discontinue if GRV contains enteral feeding material and is consistently < 300 ml. If > 400 ml and contains enteral feeding material, continue to check GRV or use continuous gastric suctioning if gastric access is present "NGT". High NG output ">800 ml ----- hold feeding & X-ray to verify placement
- Observe clinically for abdominal distension, emesis, cramping etc. If any signs of intolerance, hold feeds and check KUB
- Monitor for **Complications**

Consider gastric feeding

Initiate

Timing
- Within the 1 st 24 hrs of admission
- Commences in surgical patients without waiting for flatus & bowel motion "**LevelII**"

Access
- No recommendation of gastrostomy over NGT
- When possible, use a large-bore feeding tube for the 1st 1-2 ds of EN **"Level I"**
- Consider gastrostomy for long-term (>4 ws) EN
- Percutaneous endoscopic gastrostomy (**PEG**) tubes can be used 4 hrs after insertion

Delivery
Determine energy & nutrient requirement

Intermittent feeding
- Bolus feeding **125** ml / feed, increase by **125** ml till target with 3 hrs interval at least
- Volume should not be > **400** ml / feeding which includes flushing tube with **30- 50** ml water before and after feeding
- Do not allow feeding funnel to run empty
- Observe pt during feeding for signs of respiratory distress or vomiting

Continuous feeding
- For pts with risk for aspiration "**Level IV**" & GI intolerance
- Deliver continuously over 16–24 hrs
- Start standard **20** ml **Polymeric feeding** "**1Kcal/ml**, unless **Specialized formula** is indicated
- Increase **20 ml**/4 hrs till target

Polymeric preparations
- Contains intact ptn, complex CHO, and fats in addition to the trace elements, vitamins, minerals, and fiber
- Fiber is an important constituent of such feeds as it maintains the structural integrity of the gut enterocytes
- These feeds are usually made lactose free as lactose intolerance is common in critically ill pts
- Should be used unless the pt demonstrates intolerance, GI complications "short bowel syndrome & pancreatitis"

Constipation

"Difficult passing & no motion for **>3** ds"

- Rule out dehydration
- Increase free water content
- Rectal exam to exclude impaction
- Ensure adequate bowel regimen
- **Lactulose** "NG **20** ml/ 12hrs

Emesis

- Review medications that may cause N&V
- Exclude ileus & obstruction
- Reduce rate by half x 2 hrs and observe clinically. If emesis continues, hold feeds x 6 hrs, place NGT/OGT if not already in place and apply low continuous wall suction, examine abdomen and check KUB. Consider anti-emetic therapy. If emesis resolves, restart feeds at rate 25 mL/hr< previous rate (minimum10mL/hr)
- Consider
  - HOB elevation
  - Anti-emetics & gastric motility agent
  - Elemental formula in malabsorbtion
  - TPN if intractable

Abdominal distension&Cramp

- Review medications that cause cramping
- **Mild** ------ Check for constipation, maintain rate, re-examine in 6 hrs, if remains mild---maintain rate
- **Moderate** ----- Check small bowel obstruction abdominal X-ray series". If present --- stop feeding, replace existing tube, consider checking bladder pr, CBC, electrolytes with Mg&PO4, ABG with lactate, and consider changing formula to low f fiber if present

Diarrhea

- **Mild** "**1-2** /shift or 100-200/12 hrs" ----- Evaluate medications & Maintain rate & Increase to goal
- **Moderate** "**3-4** /shift or 200-300/12 hrs" ---- Evaluate medications & Maintain rate,& Examine in 6 hrs, if mild or moderate ---- increase to goal
- **Severe"> 4** /shift or >300/12 hrs" ----
- Consider
  - Manage CDI suspected
  - Distended, tympanic, or painful abdomen ---- DC EN
  - Medical/surgical history consistent with diarrhea "short bowel, pancreatic insufficiency " ------ medical intervention
  - Risk of stool impaction ----- Rectal check; manual disimpaction if +ve.Obtain abdominal x-ray to rule out more proximal impaction as indicated
  - Change all oral liquid medications to tablet or parenteral alternative;change oral electrolyte solutions to parenteral; DC all known cathartics if possible. rule out other potential drug related cause of diarrhea "Metochloperamide , , Mg & PO4, Sorbitol , Erythromycin"
  - If All above –ve;
    - consider, Elemental, Isotonic, Fiber-containing ,and high MCT content FormulaS (in fat mal-absorption)
    - Use low fat or skim yoghurt in place of feed to stop (Mix each 500 ml of water with 1 can of yoghurt)
    - Anti-diarrheal
    - Decrease rate by 50 %. If persists, consider PN

Maintain

- Follow **General principles of tube feeding**
- Follow **Recommendations of administering medications**
- Evaluate for risk of aspiration **"Level I"**
- Elevate HOB **30 -45**^{O} unless CI
- Aims to achieve **80-100** % of the goal rate within 48 -72 hrs ---consider prokinetics, postpyloric feeding --- If not, consider supplemented PN
- If 100% reached for a minimum of 24 hrs---- insert fine bore tube feeding
- Withhold EN if hemodynamically unstable---- Restart once resuscitated
- Check gastric residuals / 4 hrs during the 1 st 48 hrs. After target rate is achieved ----check/ 6-8 hrs in non-critically ill pts for another 48 hrs**"Level III**"&/4-hr measurements are prudent in critically ill pts**"Level II"**
- Monitor for**GRV**
- **Consider When to stop tube feeding**

Monitor for complications

Assessment

- See assessment *"p69"*

Wean

- Start oral, as pt is alert & able to manage mechanics of chewing & swallowing
- Hold EN 1 hr before scheduled meals to stimulate appetite
- If oral intake approaches 50% of the nutrients requirements for > 2 - 3 consecutive ds, rate can be:
  - Reduced in infusion rate *Or*
  - Reduced in the number of feeding

Regurgitation
- Effortless passage of gastric contents into the nasopharynx

Reflux
- Simple passage of gastric contents into the esophagus

Emesis/ vomiting
- Passage of gastric contents into the nasopharynx that is associated with retrograde peristalsis and abdominal muscle contractions

Penetration
- Entry of material into the larynx above the true vocal cords

Aspiration
- Inhalation of material into the airway below the level of the true vocal cords

Micro aspiration
- Aspiration of small volume that is usually asymptomatic and clinically undetected

Macro aspiration
- Aspiration of large volume that is usually witnessed or detected by clinical observation

Silent aspiration
- Aspiration occurring in the observed acute symptoms

Symptomatic aspiration
- Aspiration accompanied by acute clinical symptoms of coughing, chocking, dyspnea or respiratory distress

When to hold tube feeding

- ½ hr-- procedure s require trendlenberg position
- 1 hr-- upper GIT endoscope “place NGT to suction”
- 6 hrs--GA for non-intubated pt
- 6 hrs--intubated ptundergoe airway surgery including tracheostomy & planned intubation ‘thoracotomy”
- Midnight -- intubated pt with planned GIT surgery
- At the time of depature to the OR -- all other intubated pt including those who will be prone or those will be extubatedpo “flush & aspirate gastric tube”

N.B.

- Stop insulin infusion before transport to OR
- Alert anesthetist to perform acuocheck in OR if SC insulin is given within 2 hrs
- Restart feeding PO unless orders to hold

- Adhere to the ICU approved extubation NPO periods:
  - Pre-extubation: NPO 1 hr(place NG to suction)
  - Post-extubation: resume EN at last tolerated rate in 4 hrs (unless CI)
  - Post-extubation: resume oral intake (sips/clear fluids) in 6 hrs (unless CI)
  - Prior to extubation, assess if EN is required. If required, ensure the pt has the appropriate feeding access. Unless CI, small bore NG tube is preferred
- For pt on BiPAP EN is initiated/continued unless CI:
  - <4 hrs post-extubation
  - Impending intubation
  - High risk aspiration (i.e. Hx GERD; elevated GRV; inability to protect airway – decreased LOC)

General principles of tube feeding

- Feeding should be at room temperature, and should not be hanged more than 12 hrs. Supply at one time
- Use closed entral feed systems
- Change administration sets as per manufacture guidelines &“open systems /24 hrs”
- Use sterile water for flushing tube
- Use sterile liquid feeds
- Hang closed system formulas for 24 hrs& ” powder reconstituted feeds for “4 hrs”
- Store unopened liquid formula as manufacture guidelines & use before expiraydate
- Avoid use of food dyes to assist in identifying regurgitation or aspiration
- Flushing feeding tubes with 10-30 ml of water / 4 hrs during continuous feeding
- Flushing the feeding with 30 ml of water after residual volume measurements
- Blood sugar control “/6 hrs keep between **140** -**180**& use insulin infusion s needed
- Consider additional micronutrient supplementation
- TTT of tube occlusion ----- flush with warm water, replace tube if necessary

Specialized feeds

Immune modulating formula

- Formula”enriched with **arginine**, **nucleotides**, **omega3fattyacids**
- At least deliver 50% - 65% energy need ‘**Level III**”.
- Superior to standard enteric formula in:
  - Elective upper GIT surgical pts “**Level I**”
  - Mild sepsis “APACHE II <15 “not recommended in pts with severe sepsis “**Level II**”
  - Trauma “**Level I**”
  - ARDS “formula containing omega 3 fatty acids, antioxidants” “**Level II**”
- Insuffient data in burn
- ICU pts with very severe illness who do not tolerate >700 ml enteral feeds / d should not receive immune modulating formulas “**Level II**”

High lipid, low CHO formula

- In difficult to wean pt

Elementalformula

- The various macronutrients are present in a readily absorbable form e.g. ptn“ peptides or AA”, fats “ medium chain TG”, and CHO“ mono or disaccharides’
- For pts with severe form of mal-absorption

Solublefiber

- Beneficial for fully resuscitated, hemodynamically stable critically ill pt who develops diarrhea. Insoluble fiber should be avoided in all critically ill ps. Both should be avoided in pts at high risk for bowel ischemia or severe dysmotility (**Level III**)

Renalfeeds

- For patient with AKI not on CRRT, or CKD with electrolytes abnormalities “**2 Kcal/ml**”, low PH & K

The addition of enteral Glutamine

- Considered in trauma, and burn (**Level II**)
- Insufficient data in surgical, heterogeneous critical ill pts

AdministrationofProbioticagents

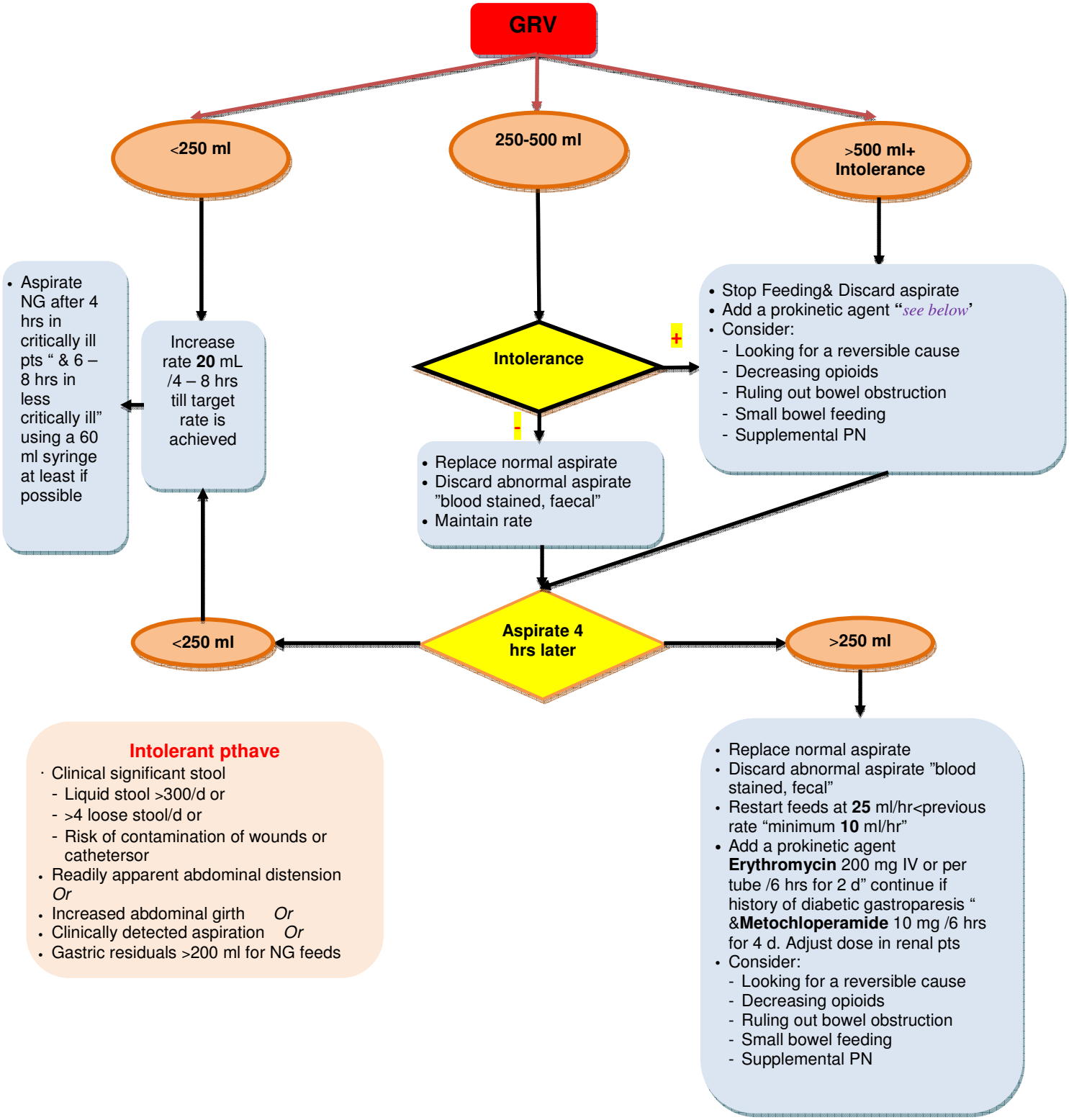
- Improve outcome in specific critically ill pt populations involving transplantation, major abdominal surgery, and severe trauma (**LevelIII**).
- No recommendation for general ICU population

Antioxidant & Vitamins & trace minerals Should be provided to all critically ill pts receiving specialized nutrition therapy (**Level II**)

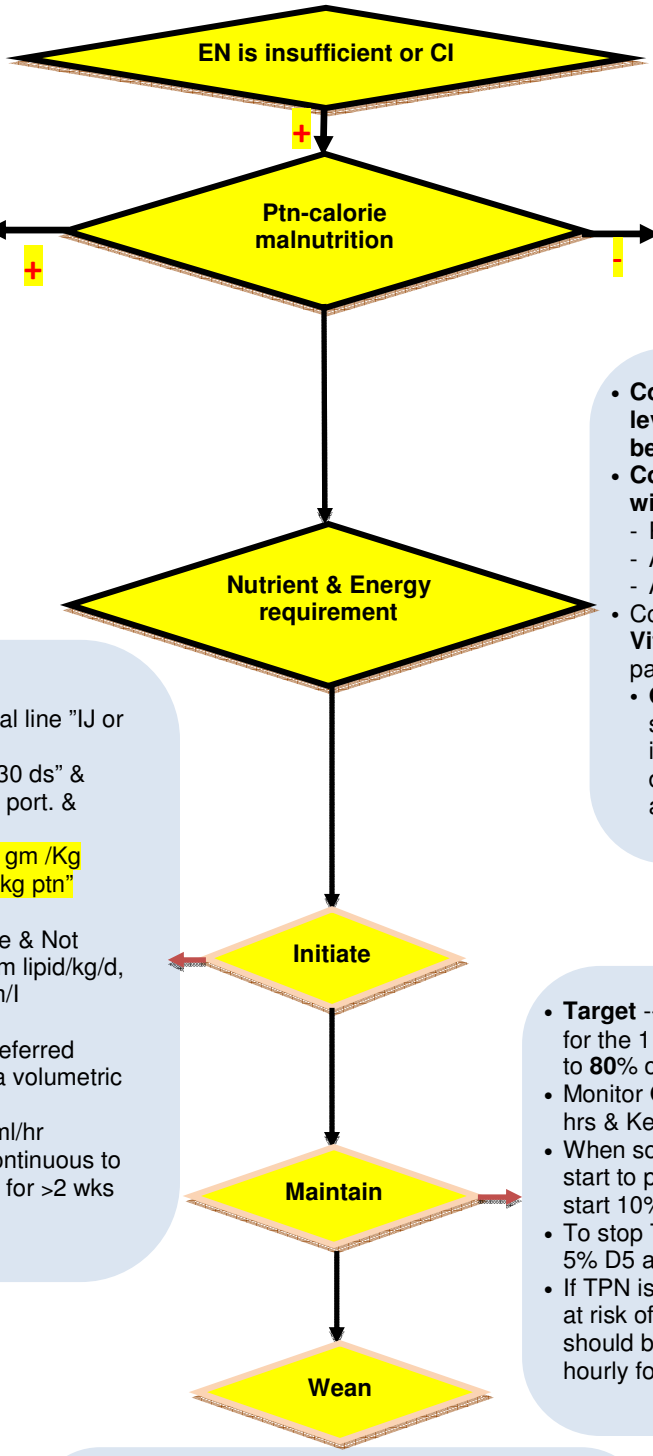
- Burn --- higher rate of **Traceelements** “**Level I**”

For medications administration

- Each medication should be administered separately through an appropriate access. Liquid dosage forms should be used when available and if appropriate. Only immediate-release solid dosage forms may be substituted. Grind simple compressed tablets to a fine powder and mix with sterile water. Open hard gelatin capsules and mix powder with sterile water**(Level II)**
- Prior to administering medication, stop the feeding and flush the tube with at least 15 mL water. Dilute the solid or liquid medication as appropriate and administer using a clean oral syringe (≥ 30 mL in size). Flush the tube again with at least 15 mL water taking into account pt’s volume status. Repeat with the next medication (if appropriate). Flush the tube one final time with at least 15 mL water. Assess fluid restrictedptsindividually


GRV
<250 ml
250-500 ml
>500 ml+ Intolerance
Aspirate NG after 4 hrs in critically ill pts " & 6 – 8 hrs in less critically ill" using a 60 ml syringe at least if possible
Increase rate 20 mL /4 – 8 hrs till target rate is achieved
Intolerance
+
-
Stop Feeding& Discard aspirate
Add a prokinetic agent "see below"
Consider:
- Looking for a reversible cause
- Decreasing opioids
- Ruling out bowel obstruction
- Small bowel feeding
- Supplemental PN
Replace normal aspirate
Discard abnormal aspirate "blood stained, faecal"
Maintain rate
<250 ml
Aspirate 4 hrs later
>250 ml
Intolerant pthave
Clinical significant stool
- Liquid stool >300/d or
- >4 loose stool/d or
- Risk of contamination of wounds or cathetersor
Readily apparent abdominal distension
Or
Increased abdominal girth Or
Clinically detected aspiration Or
Gastric residuals >200 ml for NG feeds
Replace normal aspirate
Discard abnormal aspirate "blood stained, fecal"
Restart feeds at 25 ml/hr<previous rate "minimum 10 ml/hr"
Add a prokinetic agent Erythromycin 200 mg IV or per tube /6 hrs for 2 d" continue if history of diabetic gastroparesis " &Metochloperamide 10 mg /6 hrs for 4 d. Adjust dose in renal pts
Consider:
- Looking for a reversible cause
- Decreasing opioids
- Ruling out bowel obstruction
- Small bowel feeding
- Supplemental PN

Parentral nutrition "PN"



- **Consider full required energy level & IV lipids if PN expected to be >10 ds**
- **Consider hypocaloric PN & withhold lipids if**
  - PN expected to be <10 ds
  - After resusc.
  - At risk of re-feeding
- Commence daily IV **Vitamins**&**Traceelements** if not part of PN regimen
  - **Glutamine** supplemented PN or supplement with IV glutamine infusion "**LevelIII**". Parenteral doses **0.5** g L-glutamine/kg/d **2** and **0.2** - **0.4** g L-glutamine/kg/d **7**

Access

- Central PN ----- Via separate Central line "IJ or Subclavian" & tunneled catheter is recommended for long term use ">30 ds" & Separate catheter or exclusive one port. & tubing changed daily
  - Infusion rates not exceed /hr "**0.5** gm /Kg glucose, **0.15** gm/Kg fat, **0.1** gm/ kg ptn"
  - Avoiding femoral venous access
- Peripheral PN ---- for short term use & Not >**10**% D &≤**10**% AA solution &≤**2** gm lipid/kg/d, not >60% of total kcal &<**900** mosm/l

Delivery

- Continuous administration is the preferred method ----- Infuse over **24** hrs "via volumetric pumps"
- Solution is generally started at **25** ml/hr
- Consider a gradual change from continuous to cyclical delivery in pts requiring PN for >2 wks

- **Target** -----No >50% of estimated needs for the 1 st 24–48 hrs& Increase gradually to **80**% of target at **72** hrs
- Monitor **Glycemic control** "Measure /4 hrs & Keep between **140** -**180** mg/dl
- When solution is discontinued, taper like start to prevent rebound hypoglycemia or start 10% D5
- To stop TPN for surgery, replace TPN with 5% D5 at the same infusion rate
- If TPN is stopped suddenly, the patient is at risk of hypoglycaemia. Insulin infusions should be ceased and BSLs monitored hourly for 4 hrs

- Reassess daily for EN eligibility
- Begin tapering PN when enteral tube feedings are providing 33 to 50% of the total nutrient requirements
- When enteral tube feedings are well tolerated and provided >60 -75% of nutrient requirements, PN can be discontinued
- Reduce PN/EN by ½ of the goal
- Reduce by ½ of goal "DC lipids, decrease D5
- EN can be cycled to 12 nighttime cycled to encourage appetite during the day
- When EN are well tolerated and provided >60-75% of nutrient requirements, PN can be DC

Nutritional assessment

Parameter	Frequency	Rationale	Interpretation
Sodium, potassium, urea, creatinine	Baseline Daily until stable Then 1 or 2 times a week	Assessment of renal function, fluid status, and Na and K status	Interpret with knowledge of fluid balance and medication Urinary sodium may be helpful in complex cases with gastrointestinal fluid loss
Glucose	Baseline 1 or 2 times a day (or more if needed) until stable Then weekly	Glucose intolerance is common	Good glycaemic control is necessary
Magnesium, phosphate	Baseline Daily if risk of refeeding syndrome Three times a week until stable Then weekly	Depletion is common and under recognised	Low concentrations indicate poor status
Liver function tests including International Normalised Ratio (INR)	Baseline Twice weekly until stable Then weekly	Abnormalities common during parenteral nutrition	Complex. May be due to sepsis, other disease or nutritional intake
Calcium, albumin	Baseline Then weekly	Hypocalcaemia or hypercalcaemia may occur	Correct measured serum calcium concentration for albumin Hypocalcaemia may be secondary to Mg deficiency Low albumin reflects disease not protein status
C-reactive protein	Baseline Then 2 or 3 times a week until stable	Assists interpretation of protein, trace element and vitamin results	To assess the presence of an acute phase reaction (APR). The trend of results is important
Zinc, copper	Baseline Then every 2–4 weeks, depending on results	Deficiency common, especially when increased losses	People most at risk when anabolic APR causes Zn decrease and Cu increase
Selenium[a]	Baseline if risk of depletion Further testing dependent on baseline	Se deficiency likely in severe illness and sepsis, or long-term nutrition support	APR causes Se decrease Long-term status better assessed by glutathione peroxidase
Full blood count and MCV	Baseline 1 or 2 times a week until stable	Anaemia due to iron or folate deficiency is	Effects of sepsis may be important

Assess nitrogen balance

- Reliable only when creatinine clearance stable and above 50 ml/min.
- **Method**
  - Obtain 24 hr timed urine urea nitrogen in grams / 24hrs- Multiply by 0.85 to correct for non-urea urine nitrogen losses
  - Add 2 - 4 gms for correct for insensible nitrogen losses
  - Multiply by 6.25 to determine ptn intake required for equilibrium
- **Interpretation**:
  - Compare result to patient's protein intake from all sources: oral, enteral and parenteral
  - Intake > output: "positive" = anabolism exceeds catabolism
  - Intake < output: "negative" = catabolism exceeds anabolism
  - Intake = output: "zero" = in equilibrium between catabolism and anabolism
- **Goal**:
  - Achieve a positive to zero nitrogen balance. May not be feasible in severely hypercatabolic patients for several weeks

Metabolicgasanalysis

- Indirect caliometry and 24 hrs urine urea N2 should be utilized to determine the caloric needs for mechanically ventilated pts for more than 7 ds

Traditional nutrition assessment tools are not validated in critical care (albumin, prealbumin, and anthropometry). Before initiation of feedings, assessment should include evaluation of weight loss and previous nutrient intake before admission, level of disease severity, comorbid conditions, and function of the GIT (**LevelE**)

Assess visceral protein stores

- Note that these parameters are unreliable if creatinine clearance is under 50 ml/min. or if patient is in fulminant renal or hepatic failure.
- Serum albumin is invalid as an indicator of protein nutrition after fluid resuscitation has been initiated and acute stress response has occurred.
  - Start to measure once acute inflammatory phase begin to improve & patient received EN at goal at goal for more than 72 hrs.
- Laboratory values may differ slightly by institution and assay used

	Adequate stores mg/dl	Mild depletion Mg/dl	Moderate depletion Mg/dl	Severe depletion Mgl/dl
Albumin	3.5 - 5.0	2.8 - 3.4	2.1 - 2.7	≤ 2.1
Transferin	212 - 360	150 - 211	100 - 149	≤ 100
Prealbumin	18 - 45	15 - 17	11 - 14	≤ 10

Infections in ICU

New onset fever

- Suspect **pnemonia** ------ **Pneumoniaprotocol** *"p73 "*
- Suspect **CRBSI**--- **CRBSIprotocol** *"p74"*
- Suspect**UTI** ---- **Evaluation of suspected UTI**
- Suspect **soft tissue inf.**-----**Soft tissue inf. Protocol** *"p 75 "*
- **Suspect abdominal sepsis** (tenderness, distension, in tolerate EN) ----- abd. CT &US
- Suspect **C defficilediarrhea** ----- **CDI protocol** *" p78 "*
- Suspected **infectedFB&devices** ----- remove
- **Suspect CNSinf.** ---- **CNSinf. protocol** *"p 76 "*
- Suspect **infectedFB&devices** ---remove
- **POfever** -------
  - likely after 72 hrs "exceptions are erysiplelas, clostridialmyonecrosis or toxic shock S
  - If there is suspicion of inf. --- open wound & no need for culture if there is no inf. (**Level II**) & Obtain gram –ve stain & cultures from any expressed purulence from levels within incisions consistent with a deep incisional or surgical site inf., tissue biopsies & aspirates preferable to swab(**Level III**)

Fever "temp. >**38.3**^{c} twice **48**hrs post-admission

- **Temp. measurementProtocol**
- **Antipyretics, Cooling** as indicated

+

-

Obvious source

+

-

- Suspect **non-infectious causes** ---- **Procalcitonin** if in doubt
- If **community acquired infection** is clinically suspected ---- **bl.& sputum & urine cultures**&culture from possible site ----start empiric ab
- **In the 1 st 72 hrs PO**
  - A high level of suspicion should be maintained for superficial thrombophlebitis, and VTE
  - CXR not indicated if fever is the only indication "exception:possible peri-operative aspiration, community acquired process as Legionella pneumonia"
  - Urine analysis & culture not indicated if fever is only indication

Temp. Measurement protocol

- Accurately measured by intravascular, esophageal, bladder thermistor, then rectal, oral, and tympanic membrane
- Axillary should not be used **(Level II)**

- Considernon-infectiouscauses **----Procalcitonin** to confirm diagnosis (**LevelIII**)
- **Bl.& sputum & urine culture** if not done
- **Empiric ab**
- Pt in **septicshock** ---- ***Septic shock protocol"p107"***
- **Ptneutropenic ----** ***Neutropenia protocol"p79"***
- **Central line >2 ds----**draw bl. Culture centrally
- If**Sinusitis**is clinically suspected "esp., if there isnasal tube" -----
  - Remove tube&obtainCT or US (**LevelII**)
  - ENT consult ----- puncture & aspiration of sinuses under sterile conditions (**LevelII**), Gram stained & cultured (**LevelII**)

De-esclateab according to C&S

Evaluate response after **48**- **72**hrs**&**

Good response

Bad response

- **Culture +ve**----- Continue till after 48 hrs from apyrexia – consider, procalcitinin for follow up&Consider longer course in infective endocarditis, deep seated inf., MRSA, and pseudomonas inf.
- **Culture -ve**------ review **Organism**& repeat culture & consider **non-infctious causes**

- **Culture +ve**"review**Drug**&**Organism**
- **Culture-ve**---- Consider**non-infctious causes**
  - Venography&Abd. image
  - **Drug fever:** Consider new medications & bl. products. If possible, re-challengepts with drug to confirm diagnosis. If suspected drug can be stopped, do so. If drug cannot be stopped, consider a comparable substitute **(Level II)**

---- Review **Organism**

Antipyretic & Cooling

- Do not use routinely
  - Antipyretics preferred to cooling
- Lower the body temp.e in:
  - Temp.>**41**·**1**°C- Limited cardiorespiratory reserve
  - Recent stroke, TBI - Pregnancy

Pt who is hypothermic or euthermic may have a life-threatening inf. Unexplained hypotension, tachypnea tachycardia, rigors, confusion, skin lesions, acidosis, leukocytosis, leukopenia, oliguria or thrombocytopenia might mandate a comprehensive search for inf., and aggressive, immediate empiric therapy

Empiric ab therapy

- Helpful information guide
  - **Localization**
  - Source " **Nosocomial/community"**
  - **Presence of MDRO(Level II)**
  - Patterns of resistance, organisms prevalent in ICU environment "Up to date **Unit Antibiograms**"
- Consider adequate dose, intervals & duration
- Combination therapy for MDR pathogens
  - G-ve coverage typically involves a β-lactam, fluoroquinolone or aminoglycoside "Quinolones better lung penetration & less renal toxicity"
- Consider **Antibioticcycling**
  - Classes of drugs are used for empiric therapy for 3-4 ms "3 gen**Cephalosporins, Flouroquinolones**,**Piperacillin – tazobactam, Carbapemnems"**

Site	Suggested treatment
UTI& Severe **acute pyelonephritis**	**Community acquired** • Ciprofloxacin 400 mg /12 hrs **Hospital acquired** • Indwelling catheter related infection • Piperacilin –tazobactam 4.5 g /8 hrs
Intra-abdominal sepsis "for 7-14 ds"	• **Cefipime 2**gm IV /12 hrs*or* • **Piperacillin –tazobactam4.5** g IV /8hrs*or* • **Imipenem 500** mg IV /6 hrs
Diabetic foot "severe"	• **Piperacillin –tazobactam4.5** g IV /8hrs*or* • **Imipenem 500** mg IV /6 hrs **+** • **Vancomycin 15** mg /Kg IV12 hrs
Pnemonia	**See cap &VAP& HCAP** *"p73"*
CRBSI & Septic thrombo-phelbitis	**See CRBSI** *"p74"*
CNS inf.	**Community acquired meningitis** • Ceftriaxone 2 gm IV /12 hrs**+** • Vancomycin 15 mg /Kg /8 hrs "max. 2 gm" **+** - Adjust according drug level • Ampicilin 2 gm /4 hrs "immune-suppresedpts/more than 50 years" *Simultaneously with* • Dexamethazone 10 mg /6 hrsfor 4ds "suspected or known streptococcal inf." **Hospital acquired meningitis** • Cefipime 2 gm IV /8 hrs or • Meropenem 2 gm IV /8 hrs + • Vancomycin 15 mg /Kg /8 hrs "max. 2 gm" + *Simultaneously with* • Dexamethazone 10 mg /6 hrs for 4 ds **In prescence of shunt** • Rifampacin 600 mg /24 hrs for 4 ds **Herpetic meningo-encephalitis** • Acyclovir 10 mg /Kg / 8 hrs
ST inf.	**Cellulitis** • **Amoxicilin/clauvinic acid 1.2** gm /8 hrs **Necrotizing fasciitis** • See*Necrotizing fasciitis* *"p75"*

MDRO risk factors

- Recent hospitalization ± MV
- Residency in a healthcare-associated facility

Bl. culture

- Obtain before initiation of ab therapy **(Level II)**
- For pts with no vascular catheter, obtain at least 2 cultures 10 min. apart from peripheral sites by separate venipunctures (**Level II)**
- If venipuncture is difficult to perform and if there is an intravascular device in place, 2nd culture can be drawn from the device at the time of peripheral one. In most cases, when true bacteremia or fungemia exists, the 2 cultures will be identical. Most of discordant results, culture drawn through device will be positive and by venipuncture will be negative; in such case, organism is more likely to be a contaminant not pathogen"
- If CVC is suspected to be a potential source, drawn a bl. culture through the catheter
- Obtain no >3 cultures within first 24 hrs of onset of fever
- Obtaining bl. culture more than / 24 hr is rarely helpful
- Additional bl. cultures should be drawn thereafter only when there is clinical suspicion of continuing or recurrent bacteremia or fungemia or for test of cure or prolonged bactermia "staph Inf."
- Additional cultures should paired **(Level II)**
- For cutaneous disinfection, 2% chlorhexidinegluconate in 70% isopropyl alcohol is preferred, but tincture of iodine is equally effective. Both require ≥30 s of drying before proceeding with the culture procedure
  - Povidone iodine is an acceptable alternative, but must be allowed to dry >2 min. **(Level I)**
- Wipe injection port of bl. culture bottles with 70 - 90% alcohol before injecting sample into the bottle **(Level III)**
- Draw **10**-**20** up to **30** mL bl. / culture **(Level II)**
- Label blood culture with the exact time, date, and anatomic site **(Level II)**

Evaluation of suspected UTI

- Collect urine from sampling port not drainage bag **(Level II)**
- Transport urine to lab. within 1 hr. If delayed, specimen should be refrigerated **(Level II)**
- Bacteria & WBCs are a normal finding in catheterized pt. Cultures >**10^3**cfu/mL represent bacteriuria or candiduria, but neither higher counts nor presence of pyuria alone are of much value in determining if catheter-associated bacteriuria or candiduria is the cause of fever; mostly, it is not the cause of fever **(Level I)**
- UTI in catheterisedpt is defined as:
  - >**105**bacteria **+** +ve culture ,**+**
  - >**500** WBC/HPF.
- Abs will notclear colonization & indicated if it is thought that the pt is systemically unwell from this source. Catheter removal only effective. Bladder wash-out may be beneficial

HAP & VAP & HCAP

CAP
Start **Empiric ab**--- Culture---- Descalateab

< 48hrs

Clinical suspicion

HAP
- Pneumonia > 48 hrs after admission, in pt with no ETT at admission

VAP
- Early >48–96 hrs post intub.
- Late >96 hrs post intub.

HCAP
- Includes hospitalized pt in an acute care hospital for >2 ds within 90 ds of inf.; resided in a nursing home or long-term care facility; received recent IV ab therapy, chemotherapy, or wound care within the past 30 ds of current inf.; or attended a hospital or hemodialysis clinic
- Need therapy for **MDRO**

Empiric ab
- For severe **CAP** "need ICU admission" **& Early VAP** ----- Choose effective empiric ab if MDRO is not suspected
  - Levofloxacin 750 mg IV/24 hrs**+ Ceftriaxone 2** gm IV /24 hrs
- **Late VAP** &**MDR pathogen** &**pseudomonas** ----- initiate the following for **7** -**14** ds
  1- Broad spectrum B-lactam "select one"
  - **Cefipime 2** gm IV /12 hrs
  - **Piperacillin–tazobactam 4.5** g IV /8hrs
  - **Imipenem 500** mg IV /6 hrs**+**
  2- Anti-pseudomonal quinolones "select one"
  - **Levofloxacin 750** mg/24 hrs
  - **Ciprofloxacin400** mg/ 8 hrs**+**
  3- If MRSA risk factors are present "select one"
  - **Vancomycin15** mg/Kg IV /12 hrs
  - **Linezolid 600** mg IV /12 hrs

A. New persistent or progressive infiltrate
B. Respiratory "purulent trachea-bronchial secretion, declined pulmonary function
C. Inflammatory "fever, leucocytosis, new onset delirium"

A + more than one of **B**, or **C**

Search for other source

> 48hrs

CPIS&Others

<6

>6*or*<6+ highclinicalprobabili

- **Empiric ab"** Consider**MDRO"**

Trach.aspirate or bronchoscopicBAL

+veculture

Clinical improvement

\+ **DC ab**

\- Search for **otherpathogens** "mycobat., virus, fungi, &**repeatculture**, consider **non-infctious cause**&**complicatio ns** "empyma, lung abscess" &**othersite** of inf.

\+ **De-escalate ab.**

Clinical improvement 2-3 ds later"reduced CPIS"

\- Adjust ab therapy
- Search for **otherpathogens** "mycobat., virus, fungi, & repeat culture & consider **non-infctious cause**&**complications** "empyma, lung abscess" &**othersite** of inf.

\+
- **De-escalate**ab
- ttt selected pts for **7-10** ds
  - Longer therapy for **psedumonas**, **acentobacter**
- Reassess

CPIS

Factor	Score
Temperature	
• 36.5–38.4 C	**0**
• 38.5–38.9	**1**
• < 36 or > 39	**2**
Oxygenation "PaO2/FiO2"	
• PaO2/FiO2 >240 or ARDS	**0**
• PaO2/FiO2 < 240 and no evidence of ARDS	**2**
Blood leukocytes (cells/µL)	
• 4000–11000	**0**
• < 4000 or > 11000	**1**
• > 500 band forms -2 points: moderate or greater growth	**2**
Tracheal secretions (score)	
• Rare	**0**
• Abundant	**1**
• Abundant &purulent	**2**
Culture of tracheal aspirate	
• -ve	**0**
• +ve	**2**
Pulmonary radiography	
• No infiltrate	**0**
• diffuse or patchy infiltrates	**1**
• localized infiltrate	**2**

- +ve BAL = >**105 CFU/mL**
- If BAL counts of **104-105** CFU/mL----- repeat bronchoscopy if clinical course still suggests pneumonia

BAL
- Ensure sufficient sedation
- Place pt on 100% O2
- Select lobe to be lavaged
- LA gel is CI
- If possible, do not suction through scope prior to lavage Pass scope directly into the selected lobe
- Wedge scope as far as possible
- Lavage with 4-6 x 20-40 ml aliquot's of sterile NS
- Discard first aliquot
- Aspirate between aliquot's and label aliquot's accordingly
- Send aspirates immediately for quantitative culture

Others
- **Pleural fluid cultures**, if effusion is large and cause is uncertain
- **Legionella urinary antigen**
- **Nasopharyngeal or BAL for respiratory virus and *Legionella* detection**
- Obtain **CRP** and **procalcitonin** "High levels ---- more severe pneumonia"

Non-infctious causes
Atelectasis, CHF, VTE, drug fever pancreatitis, chemical pneumonitis from aspiration,pulmonary hge,or proliferative ARDS

Catheter related blood stream infection "CRBSI"

- Bloodstream inf. in a pt with a CVC in situ, where the catheter is proven to be the source of the septicaemia
- **CVC**:any catheter which terminates in the great veins or right atrium includes femoral lines and peripherally inserted central catheters

Catheter tip culture

- Quantitative or semi-quantitative culture of catheter is recommended
- Culture tip not the SC segment
- Growth of >**15** cfu from a 5-cm segment of tip by semi-quantitative culture or>10^2 cfu by quantitative broth culture reflects colonization
- Semi-quantitative growth of <15 colony-forming U/plate of the same microbe from both insertion site culture and catheter hub culture strongly suggests, it is not CRBSI

Suspect

- New onset fever >**39** c or Hypothermia
- New onset leucocytosis or leucopnea**+**
- Central line >**72** hrs**+**
- No obvious source rather than CRBSI

Catheter site inf.

-

- Obtain bl culture"cent. &periph."
- **Empiricalab.** --- De-escalate if +ve. If –ve --- consiser non-infectious causes & other source & repeat culture if highly suspect

+

Bl culture

- A paired bl. samples from catheter & a peripheral vein should be cultured before initiation of ab **(A-II).** Clean catheter hub with either alcohol or tincture of iodine or alcoholic chlorhexidine (>0.5%) and allow adequate drying time to mitigate bl. culture contamination **(A-I).** For quantitative bl. cultures, a colony count of microbes grown from obtained through catheter hub that at least **3**-fold >that from bl. samples obtained from a peripheral vein best defines CRBSI **(A-II)**
- If a bl. sample cannot be drawn from a peripheral vein, ≥2 bl. samples should be obtained through different catheter lumens **(B-III)**. Colony count for bl. sample drawn through one lumen is at least **3**-fold >colony count for bl. sample obtained from 2nd lumen should be considered to indicate possible CRBSI **(B-II)**
- Difficult peripheral cultures ----- use external jugular, brachial artery, or US –guided venepuncture

- Obtain bl culture"cent. & periph."
- Remove catheter
- Send **tip for culture**
- Expressed purulence from catheter site Gram stained & cultured (**LevelII**)
- It is not routinely necessary to culture infusate, unless there is strong epidemiologic evidence (**Level**i)
- Insert at a new site
- Consider catheter change over guide wire if there is technical difficulty with reinsertion and there is no catheter site infection sings. Repeat cultures after an interval of 2-3 days should be considered to ensure the new catheter has not become infected
- **Empiricalab**
- Exclude vascular compromise, and embolic phenomena (**LevelII**)

Empiric management

- Suspect **G-ve bacilli** if they are critically ill, have sepsis, neutropenic, have a femoral catheter, or if they have a known focus of G-ve bacillary inf. **(A-II).** Empirical coverage should be based on local antimicrobial susceptibility and severity (e.g., a **4th-generation cephalosporin, carbapenem, or β-lactam/β-lactamase combination±aminoglycoside**) **(A-II)**
- **Vancomycin** if **MRSA** suspected. **Linezolid** should not be used for empirical therapy **(A-I)**
- Empirical **Antifungal**therapy for suspected **Candidemia** should be used for septic pts with associated risk factors for fungal inf. **(B-II)** *"see p. "*

CVC culture +ve

- **Catheter inf. unlikely**

+

Bl culture +ve

- **Catheter colonization**
- ab not required

+

CRBSI
- **ab**according to C&Sconsider ab lock therapy

+ **Persistent bactermia & fungemia**

ab management

- **Coagulase negative staph-------** ab for **5**-**7** ds
- **Staph aureus** ------ ab for **14** ds & TEE "to exclude vegetations" ---- if TEE +ve --ab for **4**-**6** ws**(A-II)**
- **Gram-negative rods** except pseudomonas, and enterococci---- ab **7**-**14** ds
- **Pseudomonas ---** "antipseudomonal ab for **14** ds
- **Candida** ----**systemic antifungal** for **14** ds after last +ve bl. culture and signs or symptoms of inf.Have been resolved

Consider

Infective endocarditis

- TEE for pts with: prosthetic valve, pacemaker, or ICD, S. aureus CRBSI
- Exclude suppuration **(B-II)**
- Catheter withdrawal is required **(A-II)**

Suppuration

- Confirm by radiology (CT, US) **(A-II)**
- Surgical consultation
- Role of heparin use unresolved **(C-III)**
- 4-6 ws of antimicrobial therapy **(B-III)**

Necrotizing fascitis

Deep seated inf. of SC tissue results in progressive destruction of fascia & fat, but may spare skin

Clinical
- Fever, malaise, myalgias, diarrhea, and anorexia may be present during 1st d
- Hypotension may be present initially or develop over time
- Signs & symptoms of inf. are not initially apparent in pts with postsurgical inf., gunshot , knife wounds, or diabetes "due to peripheral neuropathy "

Lab
- Nonspecific. Blood tests demonstrate a leukocytosis with a marked left shift, coagulopathy, and elevations in the serum lactate, creatine kinase and creatinine

Imagingstudies
- Soft tissue x-rays, CT scan and MRI are most helpful if there is gas in the tissue “type I necrotizing fasciitis or gas gangrene caused by clostridia”
- Emergent non-contrast CT to assess presence of air along the fascial planes is the most expedient approach. MRI may not be adequate to delineate findings of air along the fascial planes. However, gas is highly specific but not very sensitive
- Imaging studies show only soft tissue swelling, which is not unusual in pts with trauma, postsurgical or postpartum. Direct surgical exploration will determine if necrotizing fasciitis or myonecrosis is present in such cases
- Absence of evidence of deeper tissue involvement by CT scan or MRI can be useful in cases in which the diagnosis of necrotizing fasciitis has been raised but is considered clinically unlikely. MRI can be overly sensitive, because it tends to overestimate deep tissue involvement, and does not distinguish necrotizing fasciitis from cellulitis or inflammation

Suspect

- Unexplained **pain** increases rapidly over time “may be 1stsign”
- Diffuse or local **erythema** within 1st 2 ds, darken to a reddish-purple color, frequently with **blisters** and **bullae**; bullae can also develop in normal appearing skin, and initially filled with clear fluid but rapidly take on a blue or maroon appearance
- **Crepitus** is present in about 10 %
- Locations — feet (in diabetics), head and neck, and perineum

Confirm

- **Type I necrotizing fasciitis***:* mixed inf. caused by aerobic and anaerobic bacteria and occurs most commonly after surgical procedures and in pts with diabetes and peripheral vascular disease
- **Typell necrotizing fasciitis**refers to a mono-microbial in. caused by group A streptococcus (GAS, Streptococcus pyogenes)
  - Predisposing factors include a history of blunt trauma, varicella (chickenpox), injection drug use, a penetrating injury as laceration, surgical procedures, and childbirth

ConsultSurgeon

- To establish diagnosis and distinguish from gas gangrene, pyomyositis, and myositis
- In necrotizing fasciitis, to perform aggressive surgical debridement of the involved fascia, and obtaining material for appropriate cultures
- Re-exploration should be performed in 24 hrs; repeat explorations and debridement may be necessary on a daily base till all necrotic tissue has been removed

Cervical necrotizing fasciitis
- Careful & expedited management is essential because of proximity to vital structures of neck
- Determine depth and extent of the inf. and to separate necrotizing fasciitis from more benign conditions using CT scan or MRI but should not delay emergent surgical consultation in pts with systemic toxicity or those with obvious rapid extension of inf. clinically

Fournier's gangrene
- Is a surgical emergency, and early aggressive drainage or debridement is essential
- Affected pts may require cystostomy, colostomy, or orchiectomy

ttt

Embiricab
- Cover aerobes& anaerobes
- **Ampicillin** or **ampicillin-sulbactam+clindamycin** or **metronidazole**
- For pts who have been hospitalized previously---- gram-ve coverage by substituting **ticarcillin-clavulanate**, **piperacillin-tazobactam**, 3 rd generation **cephalosporin**, a **carbapenem**, **fluoroquinolone**, or an **aminoglycoside** for ampicillin or ampicillin-sulbactam
- If group A streptococcal is suspected, combine **clindamycin** and **penicillin**
- Ab therapy should be narrowed based upon operative culture results and susceptibility pattern

Reusc.
- Massive IV fluids (10 - 20 L/d) are often necessary to maintain tissue perfusion even though anasarca is a predicted complication. blpr may improve with fluids alone
- **Dopamine** is pressor of choice if needed

Bacterial meningitis

- The major causes of community-acquired meningitis are Streptococcus pneumoniae, Neisseria meningitidis, and, primarily in pts over age 50 - 60 years or those who have deficiencies in cell-mediated immunity, Listeria monocytogenes
- The major causes of healthcare-associated bacterial meningitis are (usually staphylococci and aerobic gram--ve bacilli) and, in cases occurring after neurosurgery, may vary with whether or not antimicrobial prophylaxis has been given to prevent surgical site inf. Healthcare-associated bacterial meningitis may also occur in pts with internal or external ventricular drains, or following trauma (ie, cranial trauma or after basilar skull fracture with or without clinical evidence of leak of CSF

Suspect

- Fever & disturbed conscious level in a quite ill pt

PhE

- Sometimes, hypothermia insteadof fever
- Neurologic sequale; seizures, focal deficits, and papilledema ----- may be present early as in **Listeria** meningitis or later in the course
- **N. meningitides**---- characteristic skin lesions, as petechiae and palpable purpura
- Neck rigidity

LP

- CT scan brain before LP should be performed in
  - Immuno-compromised state (eg, HIV inf.)
  - History of CNS dis. (mass lesion, stroke)
  - New onset seizure (within 1 w)
  - Papilledema
  - Abnormal level of consciousness
  - Focal neurologic deficit
- If delayed or deferred, Obtain 2 sets of **blcultures** "about 50 - 90 % have +ve cultures"
- The usual CSF findings are ; High opening pr, WBCs**1000**-**5000**/microL (range of <100 - >10,000) with a % of neutrophils usually >**80**%, ptn of **100**-**500** mg/dL, and glucose <**40** mg/dL (with a CSF:serum glucose ratio of ≤**0.4**)
- Absence of ≥ typical findings is of little value
- A Gram stain should be obtained whenever there is suspicion of bacterial meningitis
- No absolute CI to LP. Be caution with evidence of raised ICP, thrombocytopenia or another bleedingdiathesis, or spinal epidural abscess

Confirm

ttt

- ***Dexamethasone***(**0.15** mg/kg IV / 6 hrs) before or at the same time as the ab. Continue if CSF Gram stain and/or the CSF or bl cultures reveal S. pneumoniae.
- ***Rifampin*** is added under certain circumstances
- Once the CSF Gram stain results are available, ab regimen should be tailored to cover the most likely pathogen. If CSF findings are consistent with diagnosis, but Gram stain is - ve, empiric ab should be continued. The ab regimen should be modified further, when indicated, based on the CSF culture and susceptibility results

Empiric abs

- Just after CSF or bl culture

No known immunodeficiency

- ***Ceftriaxone*** — **2** g IV / 12 hrs*or*
- ***Cefotaxime*** — **2** g IV / 4-6 hrs**+**
- ***Vancomycin*** — **15 - 20** mg/kg IV / 8-12hrs (not to exceed 2 g / dose or a total daily dose of 60 mg/kg; adjust dose to achieve vancomycin serum trough concentrations of 15 - 20 mcg/mL) **+**
- In pts>50 years, ***ampicillin*** — 2 g IV /4 hrs

Impaired cell-mediated immunity

- ***Vancomycin*** — **15 - 20** mg/kg IV / 8 - 12 hrs (not to exceed 2 g / dose or a total daily dose of 60 mg/kg) **+**
- ***Ampicillin*** — **2** g IV /4 hrs + EITHER
- ***Cefepime*** — **2** g IV /8 hrsOr
- ***Meropenem*** — **2** g IV /8 hrs

Healthcare-associated meningitis

- ***Vancomycin*** **+**
- ***Ceftazidime*** — **2** g IV /8 hrsOR
- ***Cefepime*** — **2** g IV /8 hrsOR
- ***Meropenem*** — **2** g IV /8 hrs

- **Allergy to beta-lactams**
- ***Vancomycin*** **+**
- Moxifloxacin — 400 mg IV once daily +
- If Listeria coverage is required (in pts>50 years of age and/or in those with defects in cell-mediated immunity), trimethoprim-sulfamethoxazole – 5 mg/kg (of the trimethoprim component) IV / 6 - 12 hrs

- **Pleocytosis** — a false-+ve elevation of the CSF WBC count can be found after traumatic LP, or in pts with intracerebral or SAH in which both RBCs and WBCs are introduced into the subarachnoid space ---- used this formula to correlate true WBC count in the presence of CSF RBC:

True WBC in CSF = WBC in bl x RBC in CSF - Actual WBC in CSF x RBC in bl

- Generalized seizures may induce transient pleocytosis (primarily neutrophilic)

- Normal CT scan does not always mean that performance of an LP is safe and that certain clinical signs of impending herniation (ie, deteriorating level of consciousness, particularly a GCS<11; brainstem signs including pupillary changes, posturing, or irregular respirations; or a very recent seizure) may be predictive of ps in whom an LP should be delayed

- Normal CSF values are <**50** mg/dL of ptn, a CSF/serum glucose >**0.6**, <**5**WBCs/microL, and a lactate concentration <**3.5**meq/L

Infective endocarditis "IE"

False +ve cultures

- Occasionally occur despite use of the most exacting techniques for collection and processing. When organisms as Propionibacteriumspp, Corynebacteriumspp, Bacillus spp, and coagulase-negative staphylococci are recovered from a single bl culture or a minority of bl culture bottles, the result is probably falsely +ve. However, since all of these organisms are capable of causing endocarditis, it is important to determine if the bacteremia is persistent
- The definition of persistent bacteremia varies with the likelihood that the organism is a cause of endocarditis:For an organism likely to cause endocarditis (eg, S. aureus, viridans streptococci), +ve samples collected more than 12 hrs apart. For an organism that is more commonly a skin contaminant, **3** or a majority of 4 or more separate bl cultures are +ve and the 1st and last samples are collected at least 1 hr apart

Suspect

- **Predisposing factor** as structural heart dis."eg., rheumatic heart dis."& prosthetic valves &HD , HIV, and previous IE pts, and IVdrug use**+**
- Recent **source of bacteremia**"CVC insertion"**+**
- Evidence of HF or neurologic deficit

Confirm

PhE

- Signs of new regurgitant murmurs or HF**&**focal neurologic impairment &embolic manifestations "renal, splenic infarcts" & cutaneous or muco-cutaneous lesions include petechiae "commenest", splinter hges, Janeway lesions "macular, non-painful, erythematous lesions on the palms and soles", Osler's nodes 'painful, violaceous nodules found in the pulp of fingers and toes", and Roth spots" exudative, edematous hemorrhagic lesions of the retina

Bl cultures

- A minimum of 3 at any time prior to giving ab
- If subacute and pt is not critically ill--- delay therapy for 1-3 ds while awaiting results. If pt is acutely ill----- obtain over 1-hr time span before giving ab
- Culturing for anaerobes is rarely necessary
- A minimum of **10** mL (best 20 mL) of bl from adults & 0.5 - 5 mL from infants and children
- Obtained from separate venipuncture sites, not from pre-existing vascular catheter

The following organisms as "typical causes" of IE: Staphylococcus aureus, Viridansstreptococci,Streptococcusbovis, Enterococci, HACEK group organisms; staphylococci account for the majority of healthcare-associated IE., staphylococci and streptococci occur in roughly equal proportions ofps with community-acquired IE.

Baseline ECG & CXR

TTE

- Confirm diagnosis (persistent bacteremia without a known source or high clinical suspicion with -ve cultures)
- Detect vegetations, valvular dysfunction, shunts or abscessesand assess hemodynamic severity
- TEE — Has a higher spatial resolution & more sensitive for detection of endocarditis, vegetations, prosthetic valve endocarditis, abscess, and assessment of embolic risk
- Perform TTE as the 1st diagnostic test & begin with TEE in selected settings: Limited transthoracic windows (eg, due to obesity, or MV), prosthetic valves, esp., aortic or mitral, prior valvular abnormality (including previous endocarditis). S. aureus, and , bacteremia due to an organism known to be a common cause of IE as viridans streptococci
- For pts with a normal TTE ---perform TEE in: A high clinical suspicion of IE (persistently +vebl cultures and/or multiple minor criteria for endocarditis), a technically limited TTE study

Cardiac CT

- ECCHO more sensitive for detecting vegetations , less cost and no radiation exposure than CT and MRI

Consult

- Early cardiac surgeon consultation for cases
- Specialists in infectious dis. and/or cardiology

ttt

Native Valve endocarditis "NVE"

- **Vancomycin** (**30** mg/kg / 24 h IV in 2 divided doses)
- Optimal ab regimen depends upon causative organism and in vitro susceptibility results
- Duration: Short **2**wsin carefully selected pts with right-sided endocarditis and endocarditis due to highly susceptible viridans streptococci. Most ps are treated for **4** or **6**ws. longer 6wttt regimens are used in pts with highly virulent or more resistant pathogens, those with 2ndry cardiac or extracardiac complications, and in pts with inf. of long duration prior to diagnosis
- Careful regular clinical follow-up includes serial PhE&bl cultures to insure antimicrobial efficacy
- Indications of surgery; valve dysfunction leading to HF (Level 1B), inf. with difficult to ttt pathogens (Level 1C). asymptomatic individuals with valve destruction resulting in severe regurgitation with hemodynamic evidence of high LV end-diastolic or left atrial pr(Level 1C), persistent infection, including perivalvular abscess (Level 1C), embolic events while on an appropriate antibiotic regimen OR associated with a large vegetation (Level 2C), and severe regurgitation or stenosis and vegetation >10 mm with no coexisting major embolic stroke (severe regurgitation or stenosis and vegetation >10 mm with no coexisting major embolic stroke (Level2B)

Prosthetic valve endocarditis"PVE"

- More difficult ttt&may require surgery
- abregimens as for NVE (Grade 1B). An exception is staphylococcal endocarditis---ttt with 3 agents in combination, with one of these being rifampin (Level 1B)
- Duration: at least **6** ws (Level1C)
- ttt choices for staphylococcal PVE are the same regardless of whether the pathogen is coagulase-negative staphylococcus or S. aureus. The primary consideration in choosing therapy hinges upon whether or not the organism is sensitive to methicillin
- ttt regimen for enterococcal PVE---- penicillin,ampicillin, or vancomycin and an aminoglycoside

Clostridium difficile infection"CDI "

- C. difficile accounts for **10**% - **25**% of all cases of ab-associated diarrhea and virtually all of the cases of ab associated pseudo-membranous colitis
- Other organisms that can cause fever & diarrhea include Salmonella, Shigella, Campylobacter jejuni, Aeromonas, Yersinia, E. coli, Entamoebahistolytica, and multiple viruses that are not usually identified by standard techniques
  - In general, these are community-acquired diseases and only rarely cause infectious diarrhea acquired after a pt has been admitted to ICU

Suspect

- Pt with **fever** and mild to moderate **diarrhea**">2 stools / d"who received an antibacterial agent or chemotherapy within 3 wsesp. if there isno other source of inf. *or*there is no response to initial ab therapy

- Majority of infected hospital inpatients are asymptomatic, sometimes,presented with lower **abd. cramping**
- Rare pts, esp those who are PO, may present with **ileus** or toxic megacolon without diarrhea
- Severe **colitis** without pseudo-membrane formation may occur with profuse, debilitating diarrhea,

Confirm diagnosis

Sigmoidoscopy

- Direct visualization of pseudo-membranes is highly specific for *C. difficile*colitis
- In terms of sensitivity, in pts with severe disease, only **71**% of pts had pseudo-membranes documented by direct visualization, while pseudo-membranes were present in only **23**% of pts with mild disease
- Concerns about cost, risk of perforation during examination, and relative ease of cytotoxin assays have removed flexible sigmoidoscopy and colonoscopy from the forefront of diagnosis.
  - However, a role for direct visualization may exist in cases requiring rapid diagnosis if laboratory results will be delayed or if false negative C. difficile toxin assays are suspected
- In our unit, we treat such pts empirically rather than perform sigmiodoscopy or colonoscopy

- Testing for C. difficile or its toxins should be performed only on diarrheal stool
- Testing stool from asymptomatic pts is not clinically useful
- **Stool assay**for toxins A or B "Toxin enzyme-linked immunosorbent assay (**ELISA**) tests"
  - Up to 2 stool specimens should be examined for **leukocytes**&Neutophilia and toxin ELISA test (**LevelII**)
- Should the ELISA be negative and a high index of suspicion for C difficile exist, the following are recommended:
  - **Sigmoidoscopy**(**LevelIII**), and/or
  - **Cytotoxicity assay**, and/or
  - CT scan abd. looking for thickened colonic wall
- Stool cultures for other enteric pathogens are rarely indicated in a pt who did not present to the hospital with diarrhea or in pts who are not HIV infected (**LevelII**)

ttt

- DC therapy with inciting antimicrobial agent(s) as soon as possible (**A-II**)
- Avoid anti-peristaltic agents "may obscure symptoms and precipitate toxic megacolon" (**C-III**)
- When severe or complicated CDI is suspected, initiate empirical ttt as soon as the diagnosis is suspected while awaiting diagnostic studies (**C-III**)
- Empiric therapy is not generally recommended if 2 stool evaluations are -ve using a reliable assay
- **Metronidazole** is the drug of choice for the initial episode of mild-to-moderate CDI. The dosage is **500** mg orally 3 times / d for **10** -**14** ds (**A-I**)
- **Vancomycin** administered orally (and per rectum, if ileus is present) with or without IV administered metronidazole is the regimen of choice for the ttt of severe, complicated CDI
  - Dosage is **125** mg orally 4 times / d for **10** -**14** ds (**B-I**) and **500** mg in approximately 100 mL NS / rectum / 6 hrs as a retention enema
  - Metronidazole dosage is **500** mg IV / 8 hrs (**C-III**)
    - Consider colectomy for severely ill pts
    - Do not use metronidazole beyond the 1st recurrence of CDI or for long-term chronic therapy

Stool assay for toxins A or B

- Slightly less sensitive than tissue culture assay, but technically more easy, gives answer within 2-3 hrs
- Sensitivity is **72**% for 1 st sample and **84**% for 2 nd sample
- Once diagnosis is made and therapy commences, repeat toxin assays should not be done to assess response to therapy or used as criteria to DC enteric precautions, as many pts will harbor toxin in a carrier state without any manifestations of colitis

Fecal leukocyte exam

- Sensitive for identifying inflammatory diarrhea & non-specific
- If fecal leukocytes are demonstrated by methylene blue stain, the sensitivity is **40**%; using lactoferrin latex agglutination test increases sensitivity to **75**%

Cytotoxicity assay

- Gold standard test
- Has a high sensitivity (**94** - **100**%) and specificity (**99**%).
- Disadvantages ---high expense & time needed to complete assay (-3 ds) ---- Not routinely performed
- Cultures may be useful in the setting of nosocomial outbreaks when isolates are needed for strain typing for epidemiologic purposes

Febrile neutropenia

- **PhE**should focus on the skin, ocular fundus, sinuses, CNS, pelvis, and peri-rectal area.Pneumonias may only be evident by rales
- **Empiric ab therapy**
  - Start initial combined ; **Amikin+** either **Ceftazidime**, or **Ceftriaxone**, or **piperacillin/tazobactam**
  - Add **Vancomycin** if MRSA & CRBSI, or soft tissue inf. suspected
  - Consider local prevalence & susceptibility patterns
  - Consider inf. site
- **Bl. Culture** "2 peripheral & 1 central if CRBSI is suspected"
- **Sputum** ,**urinecultures**&Culture from possible site
- **Cytotoxic assay** if CDI suspected
- **CXR**
- **LP** is not routinely needed

Suspect

- A single temp. >**38.3**°C (or a temp. >**38**.0°C for >1 hr) in pts with an absolute neutrophil count <**500**/ µL (or <**1000**/µL expected to decrease to <**500**/µL)

Workup

Daily assess

- Risk status
- Potential site of infection
- Culture results " consider organism for de-escalation"

Assess clinical instability

- Presence of shock
- Fever persistence

Unstable

Culture +ve

+

- **De-escalate**according to culture
  - Continue after 48 hrs of apyrexia

Fever persists

+

- Obtain a **2nd bl. culture**
- Review**Drug**
  - Dosing & interval"
  - Shift to another B-lactam "Pipracilin, tozobactam to Imepenem"
  - Add **Vancomycin** if indicated
- Review **Organism** "fungi, virus"
- Consider **Non-infectious causes**
- Thorough clinical examination to exclude **Uncontrolled inf.**,"peritonitis, endocarditis" & bacterial samples
- Remove persistent fever source "catheter"

-

- Obtain a **2nd bl. Culture**
- Review**Drug**
  - Dosing & interval"
  - Shift to another B-lactam "Pipracilin, tozobactam to Imepenem"
  - Add **Vancomycin** if indicated
- Review **Organism** "fungi, virus"
- Consider **Non-infectious causes**
- Thorough clinical examination to exclude **Uncontrolled inf.**,"peritonitis, endocarditis" & bacterial samples
- Remove persistent fever

Stable

Culture +ve

+

- **De-escalate**according to culture
  - Continue after 48 hrs of apyrexia
- Withdraw Vancomycin if no resistant G +Ve bacteria identified

Fever Recurs

-

- Continue initial ab
  - Continue after 48 hrs of apyrexia

Fungal infection

Suspect

- Fever despite appropriate empiric / therapeutic therapy + 2 **Major risk factors**

No therapy (-)

- Culture bl. & Urine & Sputum
- Start empiric **systemic Anti-fungal** "**Fluconazole**, or**Amphotercin** if there is previous azole therapy

+ve bl. culture (+)

- Fundoscopic exam
- Change CVC if there is significant CFU& rule out venous thrombosis
- Start **systemic Antifungal**
  - **Amphotercin**in
    - Previous azole therapy
    - C,glabrata&C.krueisi&C. luistania
  - **Fluconazole** in
    - no previous azole therapy
    - C. albican & C.tropicalis & C. parapisolosis
  - For C. albican & C.tropicalis & C. parapisolosis ------- use**Caspofungin**

>2 colonization sites "sputum, urine, CVC, vaginitis" (-)

- Start **systemic Anti-fungal**
- Change urinary catheter in candiduria --- if persists --- start **Fluconazole**

(+) Repeat bl. Cultures after **3-5** ds to assess organism clearance

(+) Continue for at least **14** ds from the last +ve culture

+ve peritoneal culture (-)

Start systemic **Ani-fungal** (+)

Repeat bl. Cultures after **3-5** ds to assess organism clearance

Continue for at least **14** dsfrom the last +ve culture

+ve thrush (-)

No therapy

(+) Start **Diflucan** suspension **200** mg /d

Major risk factors

- A leading risk factors are colonization of non-sterile sites 'risk increases with the number of sites affected"& prior use of broad spectrum ab >5 ds
- Others: Severity of illness, presence of CVC, TPN, diabetes, renal support and gastrointestinal perforation, burn, systemic steroids. immunosuppression, abd. surgery, intra-abd. abscess,

- Particularly with Candida spices spp, differentiating between colonization and inf. is often difficult
- There are few recognized standards for significant colony counts
  - >**10.000** CFU c/m3 in urine is significant for inf.
  - Significant colony count for intra-cutaneous segment, tissue, or sputum cultures -- not been well defined
  - +ve cultures from sterile sites as bl. should be considered an inf.

- Non-neutropenic pts with isolation of Candida species from pulmonary samples (tracheal aspirates, bronchoscopic or blind sampling methods), even in high concentrations, are unlikely to have invasive candidiasis
  - Initiation of antifungal in these pts should be based on histologic evidence or identification from sterile specimens

Amphotercin

- Infusion related reactions include hypotension, fever, and tachycardia, and doses should be preceeded by 1 mg test dose followed by infusion over 4 hrs
- Premedicate with Hydrocortisone -- -- minimize changes in symptomatic pt
- Dose
  - Stable pt: **0.5** – **0.7** mg Kg/d cont. infusion with total of **6-8** mg/Kg
  - Unstable pt: **0.7** – **1** mg /Kg /d cont. infusion with total of **10** mg/Kg
  - Aspergillus: **1-1.5** mg/Kg/d with the total ofd**10-30** mg/Kg
- Nephrotoxicity develop in up to 30% of pts, and minimized by Na+ loading with 500 ml NS before and after dosing. Avoid other nephrotoxic agents as possible

Caspofungin

- Dose
  - **70** mg /Kg IV**,** then **50** mg/Kg/d
- Suitable for pt with renal failure, oronamiodarone
- Doses are normal for hepatic function

Fluconazole

- Duse
  - **800** mg bolus 1 then **400** mg /24hrs
- Should be given enterally if possible , if not, give IV
- Doses are normal for renal function

Trauma in ICU

ICU management of trauma patient

- Pts should be evaluated by an emergency physician and/or a general or trauma surgeon prior to transfer to the ICU
- However, pts remain at risk for deterioration due to unrecognized injuries, iatrogenic complications of initial diagnostic studies and therapy, and general complications of ICU

1ry survey

ABCDE approach

- Evaluate breath sounds, and, if pt is intubated, position of ETT should be assessed to insure that it did not dislodge during transport
- The pt's cardiovascular status should be evaluated, the patency of peripheral and central intravascular catheters confirmed, and total volume of IV fluids administered since presentation established
- Fully document pt's neurologic status Pt should be completely exposed, at least briefly, to evaluate external injuries

2ndry survey

- Take past medical history
  - Intentional drug overdose prior to injury should also be considered
- Perform a repeat head-to-toe trauma survey on all pts within 24 hr of admission
  - Potentially life-threatening injuries may have been missed on initial evaluation include **Intracranialhge, Traumatic aortic disruption, Intra-abdominal injuries**, and Rhabdomyolysis
- Consider early femoral fracture fixation

Support

- **GIT, DVT prophylaxis**
- **Nutrition, ab**if indicated

Fulfill sheet *"Appendixp12"*

Intra-cranial injuries

- Serious intra-cranial injuries may remain undetected due to a failure to obtain an indicated CT, reliance upon an in-experienced interpreter, or because the injury was not apparent on initial scan
- Initial CT may be normal when epidural or subdural hematomas result from venous disruption
- Development of intra-cerebral hematomas hrs or ds following trauma and after an initial, normal CT has been reported
- Follow up CT for intra-cerebellar contusions in 1st 24 hr
- The course of all pts with potential intra-cranial injuries should be reviewed upon admission to the ICU to insure that a head CT has been performed, if indicated
- If the pt's neurologic condition deteriorates or fails to improve as expected, a repeat scan should be obtained despite an initially normal study

Intra-abdominal injury

- A missed intra-abdominal injury should be suspected with blunt or penetrating trauma and evidence of ongoing unexplained blood loss
- Abdominal CT scanning is non-invasive and can both localize and quantify the injury
  - Has sensitivity 88 % and specificity 97 %
  - May miss pancreatic diaphragmatic, bladder, and intestinal injuries
  - Only appropriate in hemodynamically stable pt
- Serially monitor bladder pr
- **US**is fast, noninvasive,for hemodynamically unstable pt."sensitivity and specifity ranging from 88% - 100 %"
- **FAST** evaluation looks at per-ihepatic, peri-splenic, pelvic, and pericardial views, rapid, easy for hemodynamically unstable pt
  - Has a sensitivity 80-100% and specifity 88% - 100 %

Pulmonary contusion

- Pulmonary contusion is easily missed initially because clinical findings may not develop until several hrs after the initial injury and may occur in absence of any visible chest wall injury, esp., in children
- **Radiographic** findings may not develop until 4-6 hrs post-injury
- Homogeneous opacification that does not conform to segmental or lobar anatomy is frequently seen and may take up to 10 ds to resolve
- CT scans of the chest have a higher sensitivity for diagnosing contusion than plain radiographs but are significantly more expensive and rarely affect ttt decisions
- Fractured ribs may or may not be present; contusions resulting from fractured ribs have a more localized pattern, whereas in the absence of rib fractures the pattern is more diffuse

Traumatic brain injury "TBI"

Suspect

- History of trauma
- Ph E 'GCS, Laterlization "

ABCD

- Intubate as indicated 'onsider physiologic difficulty"
- BP should be monitored and hypotension (systolic BP <90 mm Hg) avoided (**Level II**)
- Oxygenation should be monitored and hypoxia (PaO_2<60 mm Hg or O_2 saturation <90%) avoided

Neuro-surgeon Consultation

Perform CT brain

Avoid increasein ICP

Analgesics, & sedatives

- Do not use prophylactic **Barbiturates** to induce burst suppression EEG (**Level II**)
- Use high-dose **Barbiturate** to control high ICP refractory to maximum standard medical and surgical ttt "Hemodynamic stability is essential before and during therapy (**Level II**)"
- **Use Propofol** to control high ICP, but not to improve mortality or morbidity (**Level II**)"

Anti-seizure prophylaxis

- Prophylactic use of **Phenytoin** is not recommended for preventing late post-traumatic seizures (**Level II**)
- Anticonvulsants are indicated to decrease incidence of early PTS (within 7 ds of injury)" early PTS is not associated with worse outcomes (**Level II**)"

Hyperventilation

- Prophylactic hyperventilation ($PaCO_2$ ` 25 mm Hg) is not recommended (**Level II**)

Prophylactic hypothermia

- Not significantly associated with decreased mortality "greater decrease observed when target temp. are maintained for ` 48 hr (**Level III**)
- Still not experimented in our ICU

Cerebral perfusion thresholds

- Aggressive attempts to maintain CPP >70 mm Hg with fluids and pressors should be avoided because of the risk of ARDS (**Level II**)
- CPP of <**50** mm Hg should be avoided **(Level III**)
- CPP target :**50–70** mm Hg "Pts with intact pr auto-regulation tolerate higher CPP values **(Level III**)"

ICP monitoring

- In all salvageable pts with severe TBI; after resuscitation) and an abnormal CT scan 'hematomas, contusions, swelling, herniation, or compressed basal cisterns" (**Level II**)
- In pts with severe TBI with a normal CT scan if >2 of the following are noted at admission: age over 40 years, unilateral or bilateral motor posturing, or syst bl pr <90 mm Hg (**Level III**)
- TTT should be initiated with ICP thresholds >**20** mm Hg (**Level I**)

Monitor

Brain oxygen monitoring and thresholds

- Jugular venous saturation thresholds <**50**% & brain tissue O2 tension thresholds <**15** mm Hg **(Level III)**

Steroids

- Not used to improve outcome or reduce ICP (**Level I**)

Hyperventilation

- Temporary to reduce high ICP (**Level III**)
- Avoided during first 24 hr after injury (**Level III)**
- Do not decrease <**30** without measuring CPP

Control high ICP

Hyperosmolar therapy" Mannitol"

- Control raised ICP"**0.25** - **1** g/kg". Avoid hpotension (syst. bl pr <90 mm Hg) (**Level II**)
- Use prior to ICP monitoring in herniation or progressive neurological deterioration not due to extracranial causes (**Level III**)
- Keep osmolarity <**320** mosmol/l

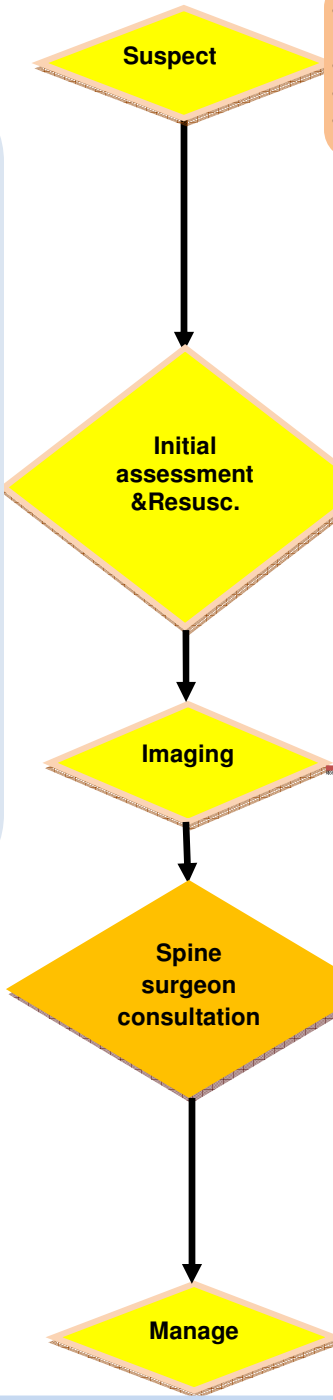
Analgesia& Sedation

- Ensure adequate analgesia if there is pain
- Ensure sedation: midazolam with no high ICP, compromised CPP. Propofol with high ICP & exclude metabolic causes, and drug effects

Further manage

- **Nutrition**: Initiate in the 1 st 24 hr & Full caloric requirement by d 7 (**Level II**) -**DVTprophylaxis- Inf prophylaxis**: Pre-procedural ab for intub.reduces pneumonia - **Earlytracheostomy** does not decrease mortality nor pneumonia (**Level II**)

Traumatic spinal cord injury "TSCI"



- Significant distracting injury & injury above clavicles
- Altered consciousness for any reason (TBI, etc)
- Neck pain or tenderness
- Abnormal neurological signs or symptoms

A&B

- High cervical cord injury may require airway suction, intub. , or vent.
- RSI with MILS is preferred when airway is urgently required. If time is not an issue, intubate over a FOB
- SPO2 should be monitored and O2 supplemented as needed
- Till spinal injury has been ruled out, neck and body immobilization must be maintained using cervical collar, straps, tape, and blocks" Hard collars allow up to 73% of normal flexion and extension – and so still need appropriate spinal care even if in place". Soft collars donotprovide effective C-spine immobilization
- Consider log-roll movements and a backboard for transfer

C

- **Manage shocked pt**

D

- Neurologic examination"mental status & cranial nerve function should be included & completed as soon as possible"
- Check bladder distension by palpation or US. Catheter should be inserted as soon as possible

Plain X- ray

- Antero-posterior, lateral, and open-mouth odontoid views
- Oblique views if suspects a lateral mass or facet injury or damage
- All C- vertebrae & top of T1 must be visualized
- In muscular males with a neck injury, pulling shoulders down by pulling down on wrists in a straight line and downward towards feet may better allow visualization of lower C- vertebrae

CT

- Pts suspected to have a spinal injury & normal plain films should undergo CT
- A **swimmer's view** if the lower cervical levels and the top of T1 are not adequately visualized
- Better than plain films in assessing patency of spinal canal
- Provides some assessment of the para-vertebral soft tissues and perhaps spinal cord, but is inferior to MRI

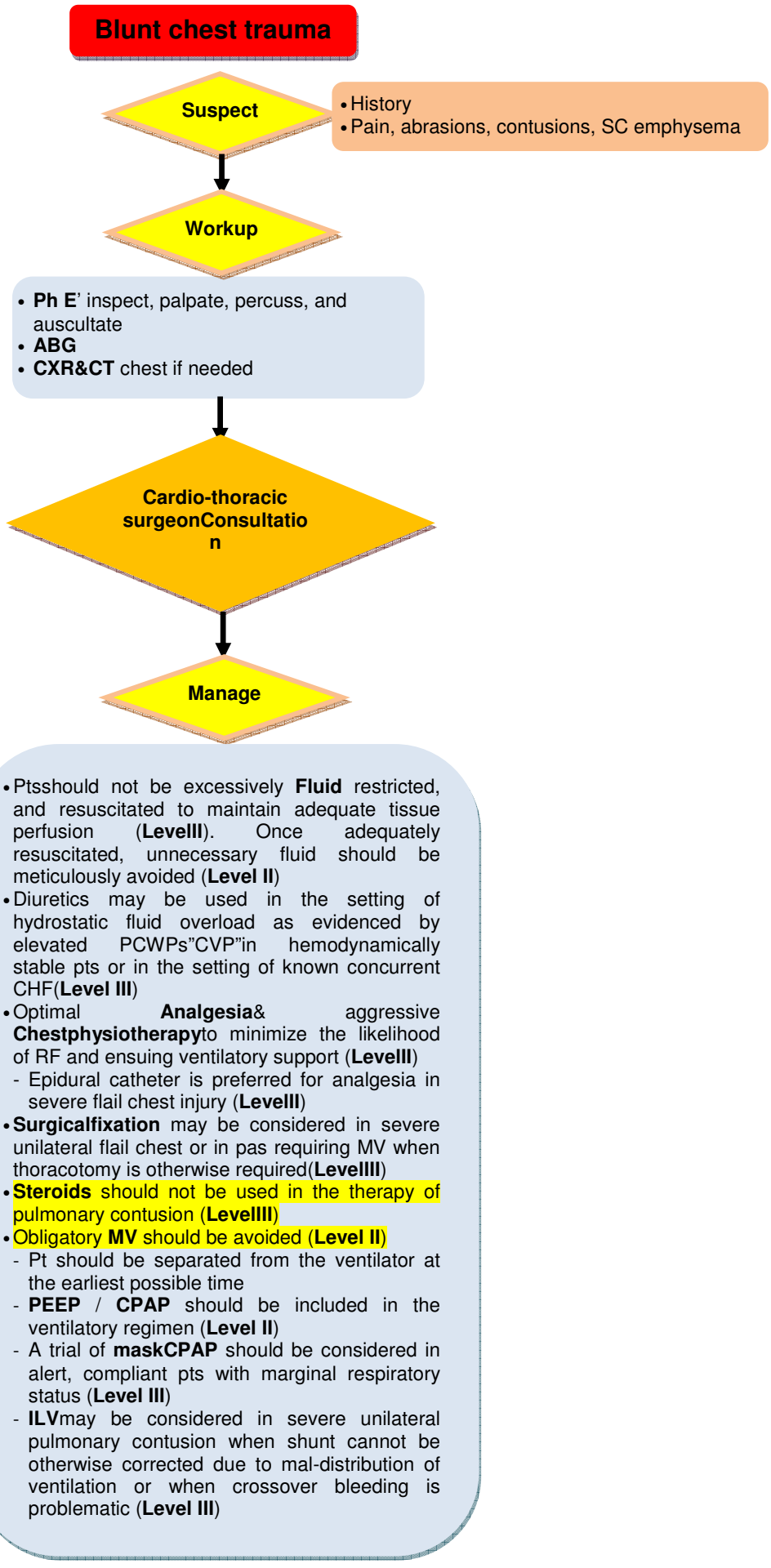
MRI

- Provides a detailed image of spinal cord as spinal ligaments, inter-vertebral discs, and para-spinal soft tissues " superior to CT & more sensitive for detecting epidural hematoma"
- Indicated in pts with negative CT scan who are suspected to have TSCI, in order to detect occult ligamentous or disc injury or epidural hematoma
- Inferior than CT in assessing bony structures
- CI in the setting of a cardiac pacemaker and metallic FBs
- In absence of cord transection or intramedullary hemorrhage, MRI is not perfectly sensitive to cord damage in the earliest stages of TSCI

Glucocorticoids "Methyl-prednisolone"

- **Methylprednisolone** only ttt that improve outcomes in acute, non-penetrating TSCI" evidence is limited, best in 1st 8 hrs"
- Indication ---- acute spinal injury with neurological loss within **8 hrs**of injury
- Loading dose: **30**mg/kg methylprednisolone over 15 min.
- Infusion**: 5.4** mg/kg/hr 45 minutes after completion of loading dose for:
  - 23 hrs if started within 3 hrs of injury
  - 47 hrsif started 3-8 hrs after injury

- **Ventilatory support** when indicated
- Early **Chest physiotherapy**& frequent **Suction**
- **Consider early Tracheostomy**
- **DVT prophylaxis**
- **Pain control**
- **Pressure sores**
  - After spinal stabilization, pt should be turned side to side (log-rolled) / 3 hr
  - Rotating beds should be used , if available
- **Urinary catheterization**
  - Initially to avoid bladder distension
  - 3-4 ds later, intermittent catheterization
- **Gastrointestinal stress ulceration**
  - PPI upon admission for 4ws
- **Glucocorticoids "Methylprednisolone"**


Blunt chest trauma
Suspect
History
Pain, abrasions, contusions, SC emphysema
Workup
Ph E' inspect, palpate, percuss, and auscultate
ABG
CXR&CT chest if needed
Cardio-thoracic surgeonConsultation
Manage
Ptsshould not be excessively Fluid restricted, and resuscitated to maintain adequate tissue perfusion (LevelII). Once adequately resuscitated, unnecessary fluid should be meticulously avoided (Level II)
Diuretics may be used in the setting of hydrostatic fluid overload as evidenced by elevated PCWPs"CVP"in hemodynamically stable pts or in the setting of known concurrent CHF(Level III)
Optimal Analgesia& aggressive Chestphysiotherapyto minimize the likelihood of RF and ensuing ventilatory support (LevelII)
- Epidural catheter is preferred for analgesia in severe flail chest injury (LevelII)
Surgicalfixation may be considered in severe unilateral flail chest or in pas requiring MV when thoracotomy is otherwise required(LevelIII)
Steroids should not be used in the therapy of pulmonary contusion (LevelIII)
Obligatory MV should be avoided (Level II)
- Pt should be separated from the ventilator at the earliest possible time
- PEEP / CPAP should be included in the ventilatory regimen (Level II)
- A trial of maskCPAP should be considered in alert, compliant pts with marginal respiratory status (Level III)
- ILVmay be considered in severe unilateral pulmonary contusion when shunt cannot be otherwise corrected due to mal-distribution of ventilation or when crossover bleeding is problematic (Level III)

Moderate and severe thermal injury

1ry survey

ABCDE approach

- Early tracheal **Intubation** if inhalation injury or as indicated. Consider the need for awake intubation
- Support oxygenation & vent.
- Consider *Lungprotection*
- Venous **Line**& Initial **Fluid resusc.**
- **Stop burning process**

2ndry survey& Exclude Cyanide, CO toxicity

- History&Head to toe exam

Estimate**TBSA & depth**

Fluid & bl. resusc

General management& Wound care

Manage **Chemical &Electrical & Circumferential extremity burns**

Fluid resusc

- Rapidly, aggressivelyreplete fluid
- Major burns --- insert 2 large-bore IV lines through non-burned skin, "orbe inserted , or may be CVC through burned tissue if necessary"
- Monitor blpr invasively; UOP to determine adequacy of resusc.
- Over-resusc. correlate with increased intra-abdominal pr& compartment S
- Initially, administer IV **LR** solution
- **Hypertonic saline**has no better outcomes
- **Colloids** do not improve survival compared with crystalloids
- After adequate resusc. &ptstabilization, change fluids to **5 % D in 1/2 NS** (ie, 0.45 % Nacl) with 20 m EqKcl / L
- Any change to infusion rate is made as gradually as possible

Estimated fluid needed "Parkland"

- During the initial 24 hr is **2-4** mL/kg of bw for each % of TBSAburned"*appendix p1*"
- Superficial burns are excluded
- 1/2 in first 8 hr& remaining 1/2 over the subsequent 16 hr. This formula for starting target rate---- then amount of fluids adjusted to UOP target "**0.5** ml/Kg for adults, **1**ml/Kg for children <**30** Kg. Should UOP fall <**0.5** mL/kg/hr or other clinical parameters suggest inadequate resusc, additional fluid is infused (bolus of IV crystalloid (average 500 mL - 1000 mL) is given & crystalloid increaseby approximately **20**-**30** %)
- Children require maintenance fluid in addition to calculated fluid resusc. Volumes.. Children <**10** Kg need D5 in the resusc. fluid

Circumferential extremity burn

- Aware of signs of compartment S. If there is concern, measure compartment pr (insert a needle connected to pr tubing "arterial or CVP monitor" into the compartment. If pr >30 mmHg, **Escharotomy** is indicated "usually not needed in 1st 6 hrs from burn"
- Copartment S may also present with cicumferencial chest and abdominal burns, leading to increased PIP--- Perform chest and abdominal esachrotomies down anterior axillary line with a cross incision at junction of thorax and abd.
- Remove all jewelry on extremity
- Assess status of distal circulation
- Fasciotomy is seldom required , but may be needed to restore circulation in skeletal trauma, crush injury, and high voltage electrical injury

Stop burning process

- Remove carefully chemically burned clothes. Do not peel off adherent ones
- Synthetic fabrics can ignite, burn rapidly at high temp.and melt into hot residue that continue to burn the pt
- Brush dry chemical powders from wound & avoid direct contact with chemical. Then rinse involved BSA with copious amountsof warm tap water**,** then covered with warm, clean linen to prevent

General management

Tetanus immunization&DVT prophylaxis

No role for prophylactic IV ab

Gastrointestinal interventions

- Insert NGT with burns >20 % TBSA
- High-risk pts receive medication to reduce gastric acid secretion
- Early EN "within 24 hr"
- Avoid overfeeding

Pain management

- Manage hypoxemia, hypovolemia
- Simply covering wounds
- Use IV **Morphine** for significant burn. Extremely large doses or other opioids may be required
- Give pts with significant burns

Benzodiazepines to relief anxiety

Wound care

- Partial thickness burns are painful when air current passes over --- Gently coverwith clean sheets
- Do not break blisters or apply any anti-septic agents
- Any applied medications should be removed before applying appropriate anti-bacterial topical agents
- Do not apply cold water to burn >10%

Bl. Transfusion

- If no risk for an ACS, threshold **8** g/dL"**2**U of RBCs"; for pts at risk for ACS, a transfusion threshold of **10** g/dL

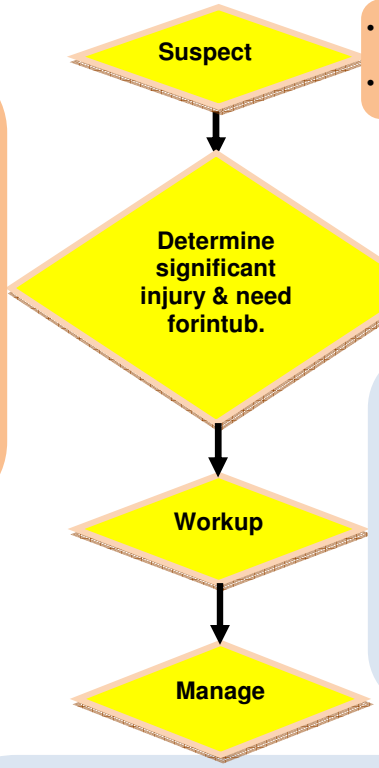
Electrical burn

- Attention should be directed to airway, breathing, and venous lineinsertion
- Bladder catheterization&ECG. If there are no arrhythmias in 1st few hrs--- long term monitoring is not necessary
- Assess for skeletal and muscular damageincluding spinal injuries
- Pts frequently needs fasciotomies

Chemical burn

- Alkali burns are more dangerous than acid ones "alkalies penetrate deeply'
- Rapidly remove chemicals and immediate wound care
- Brush dry powder if before water irrigation. Otherwise, immediately flush away with large amount of water for 20-30 min. using hose. alkalie burn require longer irrigation. Neutralizing agents offer no advantage over water lavage
- Alkalie burn to eye requires continuous irrigation during 1st 8 hrs. A small –caliber cannula can be fixed in the papebral sicus for irrigation

Smoke inhalation injury



- Coma, death of another victim, facial burns or soot in the nares or mouth, and wheezes & crackles
- Closed space fires

- Persistent cough, stridor, or wheezing
- Hoarseness
- Deep facial or circumferential neck burns
- Nares with inflammation or singed hair
- Carbonaceous sputum or burnt matter in mouth or nose
- Blistering or edema of the oropharynx
- Depressed mental status, including evidence of drug or alcohol use
- Respiratory distress (may not develop for several hrs) & Hypoxia or hypercapnia

- **ABG&CXR&EtCO2**
- The oro-pharynx should be examined for erythema; if present ----perform **Laryngoscopy**
  - If edema or blistering of the upper airway is appreciated ---- intubate without delay
  - In absence of such findings, close observation is warranted for 24 hrs
  - The appearance of the sub-glottic airways does not definitively affect management & unreliable in predicting need for ventilation
- Suspect & investigate for ***CO poisoning***

- The clinical manifestations of inhalation injury may be subtle, and frequently do not appear in the 1st 24 hrs
- If the provider waits for the X-ray evidence of pulmonary injury or changes in blood gas determinations, airway edema can preclude intubation, and a surgical airway may be required

- If a full thickness burn of the anterior and lateral chest wall will lead to severe restriction of chest wall motion, even in absence of a circumferencial burn, chest wall escharotomy may be required

- Bronchospasm ------ **O2 &Bronchodilators**
- **HOB 30^0-- reduce neck and chest wall edema**
- **Corticosteroid should not be used** (increaserisk of bacterial inf.)
- Severe inhalation injury or respiratory distress ---- ----- **Intubation** should not be delayed
- Judge fluid resusc.------ can exacerbate laryngeal swelling, increasing breathing difficulty)
- **Succinylcholine** may be used as part of a RSI during the first 72 hrafter severe burn, but not thereafter ' risk of severe hyperkalemia"
- If the pt is comatose or has COPD and is a suspected CO2-retainer ------ immediate intubate to deliver highest possible FIO2 & prevent progressive CO2 retention
- Consider large lumen **ETT**------ enable optimal management of secretions
- Humidify O2 ----- avoid inspissations
- ETT should be left in place until resolution of upper airway edema has been documented (3 - 5 ds) ----- Changing the ETT in the presence of upper airway edema is dangerous, and
- **PrimaryTracheostomy** has same mortality aspts who endotracheally intubated
- **ElectiveTracheostomy** in anterior neck burn should be delayed till 5 - 7 ds after skin grafting

Carbon monoxide poisoning

- Pts with CO level <**20**% usually have no physical symptoms
- Higher levels can result in
  - Headache & nausea "**20-30**%"
  - Confusion "**30-40**%"
  - Coma "**40-60**%"
  - Death "more than **60**%"
- Cherry –red skin colour is rare, and may only be seen in moribund pt

Suspect

- Altered conscious level, headache, N&V
- Coma, death of another victim
- Fire in a closed space

- Initiate of 100% **NBOtherapy**

Workup

- CO-Hgb level
- ECG
- ABGs
- CBC, Chemistry

Determine need for HBO

- Unconscious at scene or hospital
- New neurologic deficit or mental status change
- Obtain **CO –Hgb** level
  - CO-Hgb ` 40%
  - Pregnant women with CO-Hgb ` 20 %
- ECG ischemic changes
- PH < 7.1

+ → **Consider HBO** → **Need transfer to a specialized center**

-

Headache, N&V, blurred vision, CO-Hgb>10%

+ → **100**% **NBO** therapy till **CO-Hgb** ` 10% and asymptomatic

- → **Discharge**

Aortic rupture

- Among those who arrive in the ED, as many as 40 % die within the 1st 24 hr unless the injuries are detected and surgically repaired

- Selection of diagnostic studies to exclude or establish diagnosis of aortic disruption based on stability of pt, clinical probability of a serious aortic injury, and skills and experience of local personnel
- When clinical suspicion is high but results of a given diagnostic study are equivocal, it may be necessary to resort to a different modality

Suspect

- Rapid deceleration injury following a motor vehicle accident or a fall from a height

Confirm

- Pts may present with few or non-specific symptoms, as chest or mid-scapular pain, dyspnea, hoarseness, or dysphagia
- **Ph E---** may be non-contributory. Up to 50 % of pts may have no external signs of chest trauma
- Chest CT is only suitable for hemo-dynamically stable pts. It does not yield detailed information on vascular anatomy and can miss small arterial tears
- **TEE** offers the advantage of portability; can be performed at the bedside in unstable pts
  - Have a comparable sensitivity to CT in diagnosing aortic disruption
  - Ooperator-dependent, requires that the pt be sedated, does not have high resolution of the distal ascending aorta and proximal arch, and is difficult to interpret in the setting of athero-sclerotic disease
- **Angiography** is considered the gold standard for assessing aortic injuries and has the highest resolution of vascular detail
  - Invasive and time-consuming
  - It is associated with a 1 - 10 % complication rate and is +ve in only 10 % of pts with a widened mediastinum on chest radiograph
- **MRI** has seen infrequent clinical use in the diagnosis of traumatic aortic rupture, but has shown promise in several pilot studies

Cardiothoracic consultation

Submersion injuries

- dA process resulting in primary respiratory impairment from submersion or immersion in a liquid medium
- Drowning and near-drowning typically begins with a period of panic, loss of normal breathing pattern, breath-holding, air hunger, and a struggle by the victim to stay above the water
- Reflex inspiratory efforts eventually occur, leading to hypoxemia by means of either aspiration or reflex laryngospasm that occurs when water contacts the lower respiratory tract
- Both types of near-drowning "salt and fresh water "result in decreased lung compliance, ventilation-perfusion mismatching, and intrapulmonary shunting, leading to hypoxemia that causes diffuse organ dysfunction
- Hypoxemia in turn affects every organ system, with the major component of morbidity and mortality being related to cerebral hypoxia

ABCDE

- **Intubate** as indicated
- **O2** therapy ---- maintain SPO2 >94%
- Cervical spinal cord injury is uncommon in near-drowning victims, unless there are clinical signs of injury or a concerning mechanism
- **Ventilate** as indicated
  - If pt candidate for NIV ---- NIV either CPAP, or BIPAP
  - If not ---- intubate & insert oro-gastic tube & consider **lung protection**
- **Circulatory support**
  - Consider volume repletion in case of hypovolemia "cold diuresis"
  - Inotropic support *& CVC in case of hypoxic cardiomyopathy
- **Remote wet clothing andinitiate rewarming**

Subsequent management

- Frequent vital sign measurements and clinical reassessment, monitoring of symptomatic pt should include continuous O2 saturation, Et CO2, and bedside glucose measurement

Organ support

Neurologic

- Prevent 2nd neurologic injuries due to ongoing ischemia, cerebral edema, hypoxemia, fluid and electrolyte imbalances, acidosis, and seizures
- Maintain: Normocapnea or temporarily mild hyperventilation "**30**-**35** mmHg, normo-thermia,normo-glycemia, elevated HOB, diuretics without volume depletion, aggressive control of seizures, NMB better avoided "mask neurologic signs"

Respiratory

- CXR may not reflect the severity of pulmonary involvement and should be performed only when indicated by symptoms
- Bronchospasm ---- treat similarly to acute asthma; most cases rapidly improve with inhaled b-adrenergic agonists
- There is no good evidence to support the routine use of glucocorticoids or prophylactic ab. Ab should be used only in cases of clinical pulmonary inf. or if the victim was submerged in grossly contaminated water. If pneumonia follows near-drowning, a high suspicion for water-borne pathogens, as aeromonas, Pseudomonas, and

Radbdomyolysis

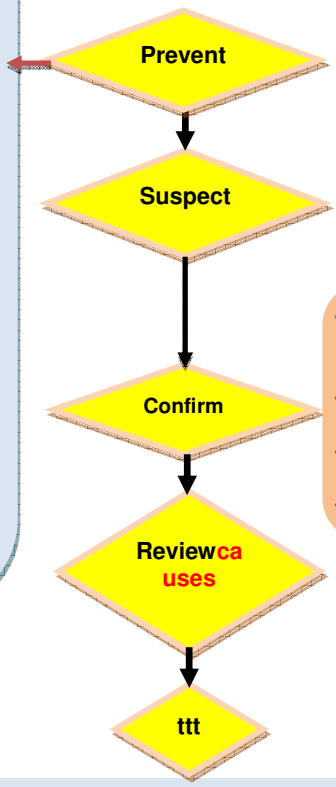
- **Rhabdomyolysis** is a syndrome characterized by muscle necrosis due to either traumatic or non-traumatic muscle injury & release of intracellular muscle constituents into circulation, causing heme pigment-associated **ATN**, resulting in abrupt rise in serum creatinine, or **AKI**
- **Crush syndrome**
  - **Crush injury** complicated by **AKI**
  - May include hypovolemic shock, sepsis, electrolyte disturbances (esp, hyperkalemia), HF, arrhythmias, ARDS,DIC, bleeding, and heme pigment-induced **ATN**
  - Occurs in 30- 50 % of traumatic **rhabdomyolysis**

Administer high fluid rate (**Level 1B**)
- Starting prior to extrication of the victim whenever possible (**Level 2B**)
- Give **NS** rather than an isotonic alkaline solution (**Level 2C**)
- Administer fluid at **1 L/hr** initially
- Avoid IV solutions containing k, as LR

After the victim has been removed from rubble & UOP is documented, switch to HCO3 isotonic solution (**Level 2C**)
- **1** L of NS alternating with **1 L** of 1/2 NS + 50 mEq **NaHCO3**"**200-300** meq in 1st d may be given"
- Close monitoring of serum HCO3, Ca, K, , serum and urine PO4, volume overload
- Goal urine pH **> 6.5**
- Discontinuing the HCO3-containing solution is recommended (but continuing to replete volume with NS) if the arterial pH B, the serum HCO3 exceeds 31 mEq/L, or the pt develops symptomatic hypocalcemia
- **Ca** should be given only for symptomatic hypocalcemia, or severe hyperkalemia

If UOP >20 mL/hr among victims removed from the rubble, add Mannitol to the IV alkaline solution (**Level 2C**)
- Add **50** mL of 20 % mannitol (**1**- **2** g/kg /d [total,120 g], given at rate of 5 g/hr)
- Maximum rate of fluid is **500** mL/hr
- CI ----- pts with oligo-anuria
- Discontinue if the **Desired diuresis**cannot be achieved (**Level 1B**)

Loop diuretics have no impact on AKI outcome
- May worsen already existing hypocalcemia
- Judicious use may be justified in elderly pts, esp., if volume overloaded



ATN
- Due to heme pigment-induced AKI
- Characterized by initial oliguric period followed by polyuria, starts within 1-3 ws may present with a non-oliguric course

- **Compartment S**"swollen muscles"
- Traumatic ---blunt or penetrating trauma
- Crushing ----Myalgia & AKI & dark urine & hypovolemia

- Plasma creatinine concentration rises more rapidly with rhabdomyolysis (up to **2.5** mg/dL or /d) than with other AKI causes
- Hyper "kalemia ,phosphatemia", and hypocalcemia
- Marked elevation in the plasma CK level"above **100,000** IU/L.
- Myoglobinuria "late cource"

Causes
Traumatic or compression
- Multiple trauma & Crush injuries
- Vascular or orthopedic surgery
- Coma & Immobilization

on-traumatic
ɛrtional
Normal muscle
- Extreme exertion
- Environmental heat illness
- Sickle cell trait
- Seizures
- Hyperkinetic states

Abnormal muscle
- Metabolic myopathies
- Mitochondrial myopathies
- Malignant hyperthermia
- Neuroleptic malignant syndrome

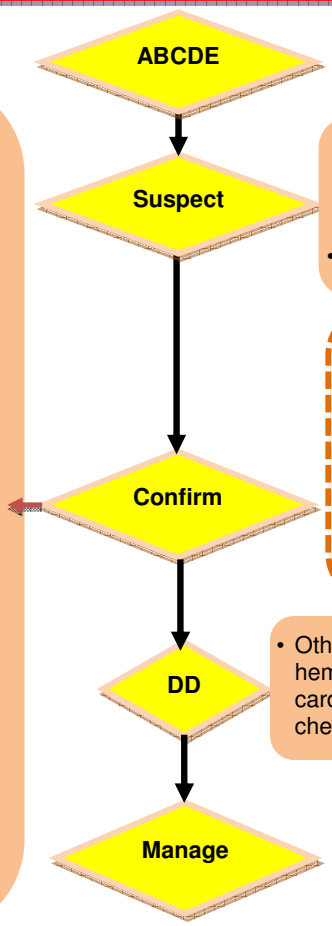
ɲ-exertional
- Alcoholism & Drugs and toxins
- Inf. (including HIV)
- Electrolyte abnormalities
- Endocrinopathies
- Inflammatory myopathies

- **Fasciotomy** if the compartment pr is within 30 mmHg of diastolic pr
- Cause of rhabdomyolysis should be identified & specifically treated
- Initiate **Dialysis** for usual indications

Compartment S
- Severe pain, weakness, paresthesia, paresis or paralysis and pallor in affected extremities
- Distal pulses may be absent when intra-compartmental pr. is very high "increased pr. may be present even when distal pulses are palpable"
- Compartment pr. should be monitored
- Normal tissue pr. is approximately 0 mmHg and that the ischemic threshold of normal muscle is reached when the intra-compartment pr rises to within 20 mmHg of diastolic pr or 30 mmHg of mean pr

UOP goal
- Adjust administration of IV fluid to maintain the UOP at **200** - **300** ml/hr
- If goal is achieved, this fluid regimen should be administered until the disappearance of myoglobinuria (either clinically or biochemically)
- This usually requires several ds
- If the desired diuresis is not established, place CVP catheter. And closely monitor input and all losses (urinary volume + other losses together) of the previous d
- Abandon forced diuresis if CVP above accepted threshold "**15** cm H2O"
- Therapy should be based on CVP measurements, biochemical analysis, close monitoring of fluid intake and output, and body weight
- If the pt is anuric and catabolic, a stable body weight may be deceptive ----- Administer **500** - **1000** mL fluid in excess of all losses of the previous d

Unstable pelvic fracture



- Pelvic injury is indicative of a high energy trauma and pts with pelvic fractures frequently have severe associated injuries “rectal, bladder, uretheral, vaginal. And iliac vessels

- History of trauma
- Pt without pelvic X-ray during 1ry survey
  - Pelvic pain or tenderness on palpation
  - Overlying abrasions or contusions
- Hemodynamically unstable pt

Pelvic fractures can be described as mechanically stable or unstable. 2 or more breaks in the pelvic ring are needed to create an unstable pelvic fracture. Pelvic fractures are classified as rotationally unstable, vertically unstable, or both. The hemodynamic status of the pt is not necessarily related to the type of mechanical stability of the pelvis. Fractures that create soft tissue defects are termed “**open**” fractures and these associated injuriescomplicate management

- Other life-threatening injuries that causes hemodynamic instability such as hemothorax, cardiac tamponade, orhemoperitoneum, using chest radiograph, pelvic radiograph, and FAST

PhE

- “**Rocking**” the pelvis by pressing down on the iliac crest from front to back and side to side should **not** be used “it risks “re-opening” an unstable pelvis, potentially disrupting any pelvic hematoma leading to vascular injury

Imaging

- **X-ray pelvis** if not done during 1ry survey
- **CT pelvis**
- CT scan, particularly 3-D reconstruction, helps define management strategy and helps in planning definitive fracture repair by defining geometry of the pelvic fracture, identifying occult fractures not seen on plain film, quantifying pelvic displacement, and evaluating integrity of ligaments. Pelvic bleeding may also be seen as contrast extravasation but this finding should not be used in isolation as an indication for arteriography
- Transportation of the hypotensive pt ispotentially unsafe. Hemodynamically stablepts undergoing further imaging should be accompanied by a qualified intensivist who should be prepared to abort the study and transport the pt directly to the operating room if pt becomes hemodynamically unstable
- **FAST scan** is used in the initialevaluation of abdominal trauma

Control of retroperitoneal bleeding

- Hemodynamically unstable pts with indications for surgery (eg, positive FAST scan) are taken directly to the operating room to identify and manage abdominal hge. If ongoing hemodynamic instability following exploration is thought to be due to retroperitoneal bleeding from pelvic fracture---do **external fracture fixation** and **pre-peritoneal pelvic packin**or **angio-embolization**
- Fracture stabilization decreases pelvic volume, promotes tamponade of venous bleeding and prevents shifting of the bony elements which can lead to 2ndryhge
- **Pre-peritoneal pelvic packing** is a surgical procedure in which laparotomy sponges are placed in the pre-peritoneal space to tamponade bleeding and reduce the available volume of the retroperitoneal space
- Following an interval of ongoing resusc. and management of other urgent injuries, **internal fracture fixation** is performed, if indicated
- Management in operating room also allows examination of rectum and vagina and washout of any open wounds
- **Arteriography** is appropriate for pts with persistent bleeding who are responsive to fluid resusc. and do **not** have other urgent indications for surgery. If arteriography identifies arterial bleeding, **angio-embolizatio**n can be used. Selective embolization is preferred to minimize the potential for tissue ischemia or necrosis. disadvantage ---- addresses arterial hge, however, the majority of bleeding due to pelvic fractures is venous in origin. Only a small % of pts who undergo arteriography can have their bleeding sites successfully

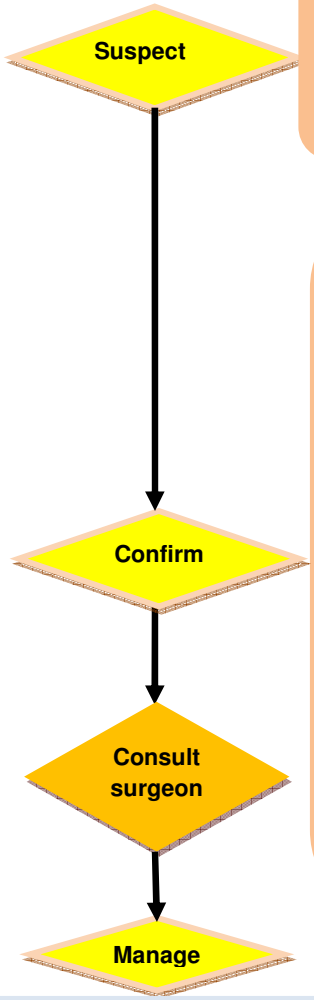
Resusc. & Care

- Acute resusc.encompasses the 1st 12 hrs following injury and combines optimizing tissue perfusion, restoring normo-thermia, and restoring normal coagulation
- Determines when the pt can return to operating room to replace pelvic packing or definitively manage other injuries
- Specific goals of resusc. include a base deficit <6, normal coagulation indices, and a core temperature >37º C
- Fluid management ------- May require infusion volumes of 10 L. Early colloid administration is appealing, but evidence to date does not support this concept in pts who do not demonstrate acute traumatic coagulopathy ----- Managing crystalloid remains a challenging “ balancing optimal cardiac performance against excessive tissue edema which can contribute to pulmonary edema, ACS, and poor wound healing”-----Use a goal-directed resusc. approach consisting of initial volume loading, followed by the judicious use of inotropic agents or vasopressors
- Maintain Hg >**10** g/dLduring initial resusc ---- Thereafter, transfusion for Hg <7 g/dL (euvolemic pt) limits the adverse inflammatory effects of stored RBCs
- Use OF PACs remains controversial “ cardiac function, metabolic status), may be useful in managing multiply-injured pts who require advanced inotropic support”
- Give (mechanical or pharmacologic)prophylaxis for DVT, unless CI ----- Use of removable IVC filter is a good choice

Surgical disorders

Acute pancreatitis

Acute inflammatory process of the pancreas



- Acute upper abdominal **Pain** at the onset "steady and may be in the mid-epigastrium, right upper quadrant, diffuse, or, infrequently, confined to the left side"
- **Systemic features**: fever, tachycardia, and, in severe cases, shock and coma

Clinical

- **History** of gallstones, alcohol use, hyper-triglyceridemia or hypercalcemia, family history of pancreatic disease, prescription & non-prescription drug history, trauma,and autoimmune diseases
- In mild disease, epigastrium may be minimally tender "severe episodes are often associated with abdominal distention, esp. in epigastrium, and guarding"
- May shallow breathing "diaphragmatic irritation"
- Dyspnea may occur "if there is pleural effusion"
- Ecchymotic discoloration in the flank (**Grey-Turner's sign**) or the peri-umbilical region (**Cullen's sign**) "in 1 % of "not diagnostic"

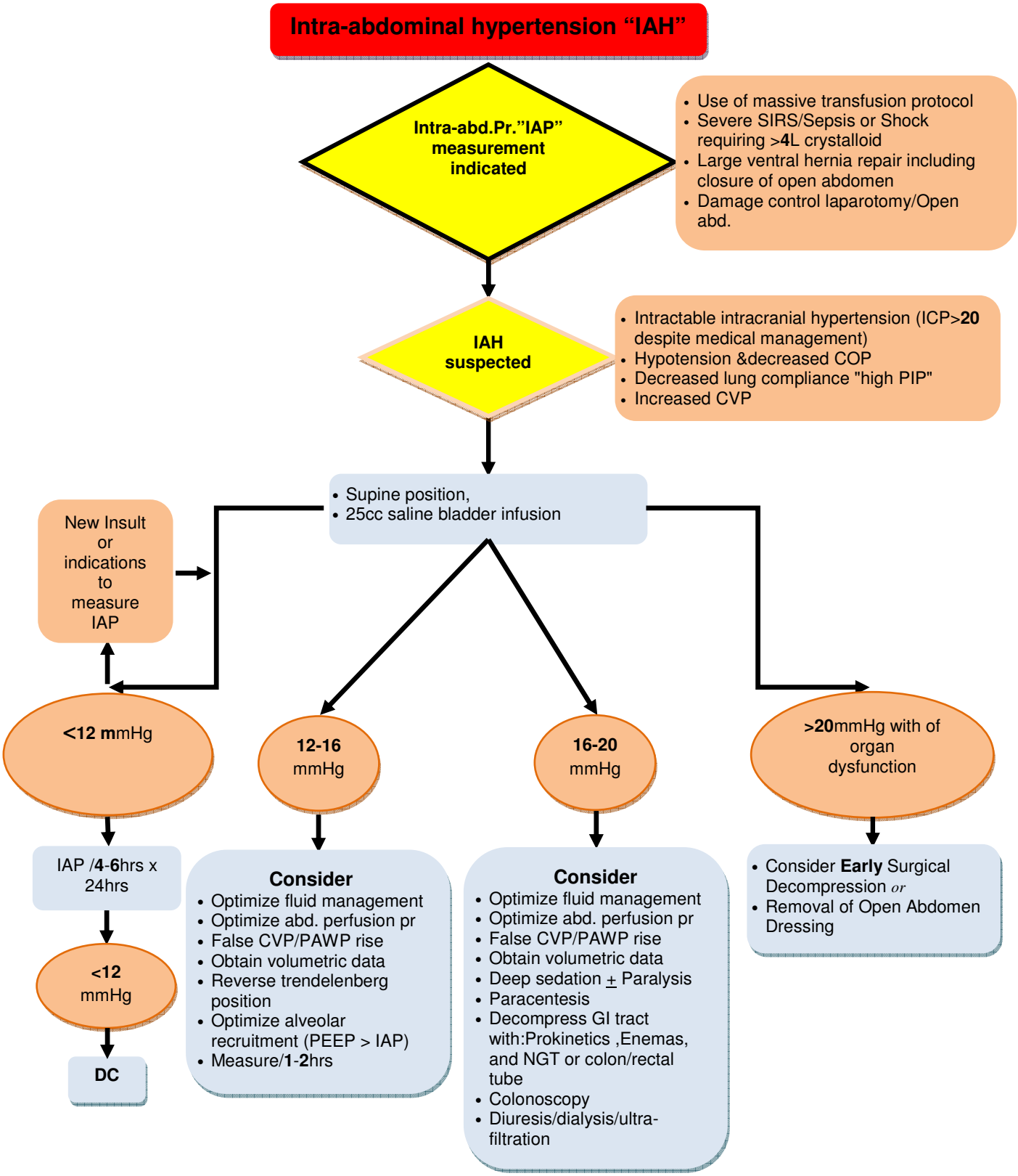
Lab

- Elevated serum **Amylase**&**Lipase**

Radiology

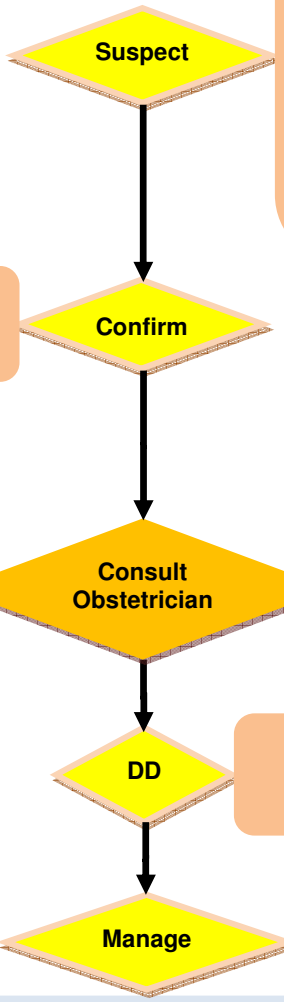
- Abd. **Plainfilm**& US
- **CT** abdomen
- **MRCP**
- **CXR** "pleural effusion"

- Correct any **Underlying predisposing** factorsas ERCP in gallstone pancreatitis
- **Supportive ttt**
  - **Volume repletion"250-300** cc of IV fluids / hr are typically required for 48 hr if cardiac status permits**"**
  - **O2** administered to maintain SPO2 of >**95** %
  - **DVTprophylaxis** "pnemattic compression"
  - **Pain relief** "mepridine , or fentanyl"
- **Prevent inf. in acute necrotizing form**
  - **EN**
  - **Selectivedigestivedecontamination**
  - Prophylactic abs "**Imepenem** for **14** ds'
- **ttt of pancreatic necrosis**
  - Minimally invasive approaches or surgical debridement in stable pts with infected pancreatitis necrosis who do not improve with abs (**Level2B**)
  - The choice should be based upon the available expertise and whether the collection is localized or diffuse


Intra-abdominal hypertension "IAH"
Intra-abd.Pr."IAP" measurement indicated
Use of massive transfusion protocol
Severe SIRS/Sepsis or Shock requiring >4L crystalloid
Large ventral hernia repair including closure of open abdomen
Damage control laparotomy/Open abd.
IAH suspected
Intractable intracranial hypertension (ICP>20 despite medical management)
Hypotension &decreased COP
Decreased lung compliance "high PIP"
Increased CVP
Supine position,
25cc saline bladder infusion
New Insult or indications to measure IAP
<12 mmHg
12-16 mmHg
16-20 mmHg
>20mmHg with of organ dysfunction
IAP /4-6hrs x 24hrs
<12 mmHg
DC
Consider
Optimize fluid management
Optimize abd. perfusion pr
False CVP/PAWP rise
Obtain volumetric data
Reverse trendelenberg position
Optimize alveolar recruitment (PEEP > IAP)
Measure/1-2hrs
Consider
Optimize fluid management
Optimize abd. perfusion pr
False CVP/PAWP rise
Obtain volumetric data
Deep sedation ± Paralysis
Paracentesis
Decompress GI tract with:Prokinetics ,Enemas, and NGT or colon/rectal tube
Colonoscopy
Diuresis/dialysis/ultra-filtration
Consider Early Surgical Decompression or
Removal of Open Abdomen Dressing

Obstetric disorders

Acute fatty liver of pregnancy"AFL"



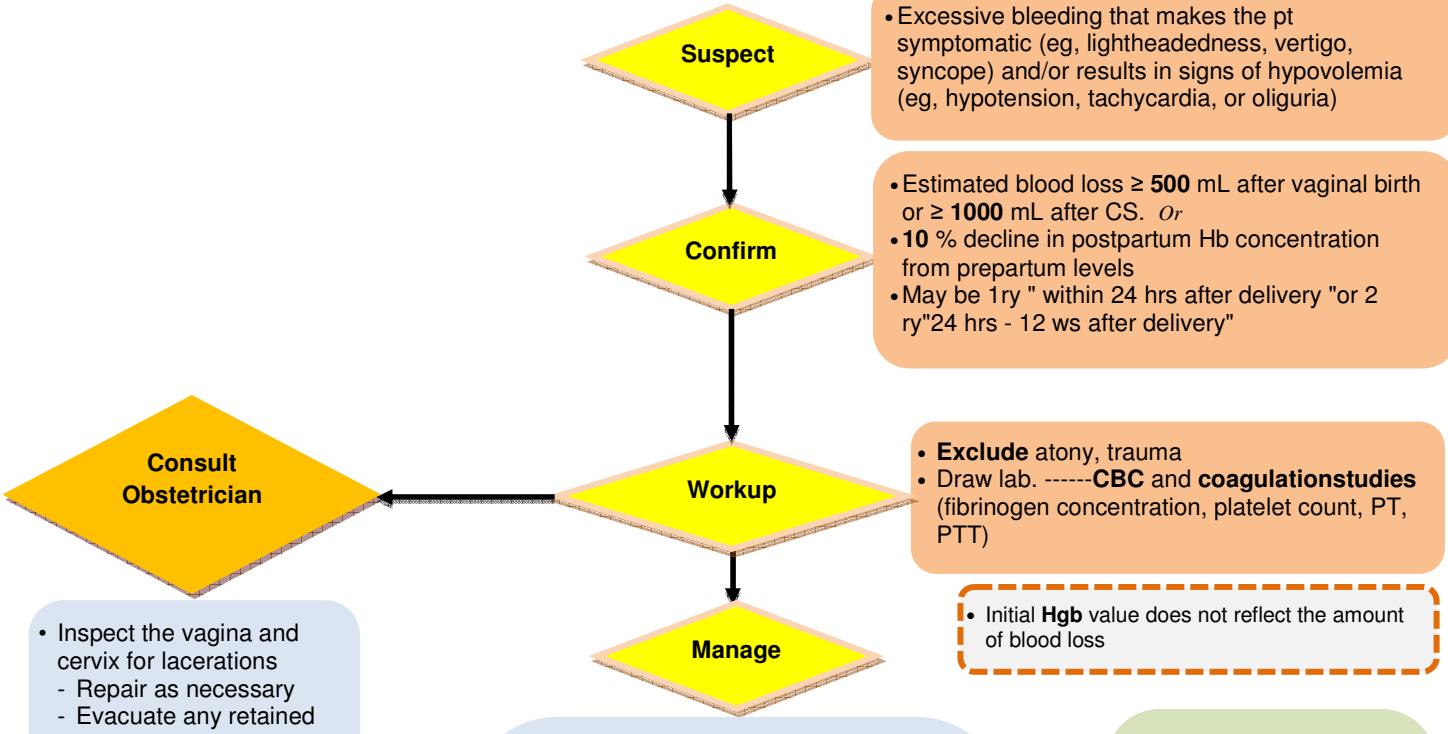
- **GIT troubles**: N&V (75 %), abdominal pain (50 %), anorexia
- Signs of **Preeclampsia**"About 50 % at presentation or during course of illness"
- **Timing**: In = 2nd half of pregnancy, usually in the 3rd trimester (not always diagnosed until after delivery)
- Jaundice
- Transient polyuria & polydipsia (central DI)
- Resistant hypoglycemia
- Coagulopathy
- **Lab**.: Abnormal liver tests, thrombocytopenia, renal failure

- **Lab**. : DIC& renal failure
- **Radiology** to exclude hepatic infarct or hematoma
- **Liverbiobsy** is diagnostic

Differentiate from **HELPsyndrome** (difficult "hypoglycemia & encephalopathy with AFL of pregnancy")

- Prompt **delivery**, usually emergently, after maternal stabilization
- **Supportive** management (MV,TBN,dialysis, blood component therapy)

Postpartum hemorrhage



- Excessive bleeding that makes the pt symptomatic (eg, lightheadedness, vertigo, syncope) and/or results in signs of hypovolemia (eg, hypotension, tachycardia, or oliguria)

- Estimated blood loss ≥ **500** mL after vaginal birth or ≥ **1000** mL after CS. *Or*
- **10** % decline in postpartum Hb concentration from prepartum levels
- May be 1ry " within 24 hrs after delivery "or 2 ry"24 hrs - 12 ws after delivery"

- **Exclude** atony, trauma
- Draw lab. ------**CBC** and **coagulationstudies** (fibrinogen concentration, platelet count, PT, PTT)

- Initial **Hgb** value does not reflect the amount of blood loss

- Inspect the vagina and cervix for lacerations
  - Repair as necessary
  - Evacuate any retained products of conception
- **Uterine massage**

Establish 2 large bore IV line "at least 18 G"

Utero-tonic drugs:

- **Oxytocin** (**10**-**40** U in 1 L NS IV infusion; 80 U in 1 L NS may be given for short time)
- **Methergine** (**0.2**mgIM/2-4 hrs) if no hypertension
- **Carboprosttromethamine (Hemabate)** (**250** mcg IM / 15 - 90 min., as needed, to a total dose of **2** mg) if no asthma
- **Misoprostol** (**800** - **1000** mcg rectally) for women with hypertension or asthma

Fluid resuscitation and transfusion

- Crystalloid (target S blpr 90 mmHg)
  - A typical ratio is 3:1
  - maintain UOP at >30 mL/hr
- Before lab. are available, transfuse **2** U packed RBCs if hemodynamics do not improve after 2 - 3 L NS and continued bleeding is likely
- Plasma& platelets transfusion
  - *See hypovolemic shock p106 "*
- Monitoring & end points of resusc.
  - *See hypovolemic shockp 106 "*

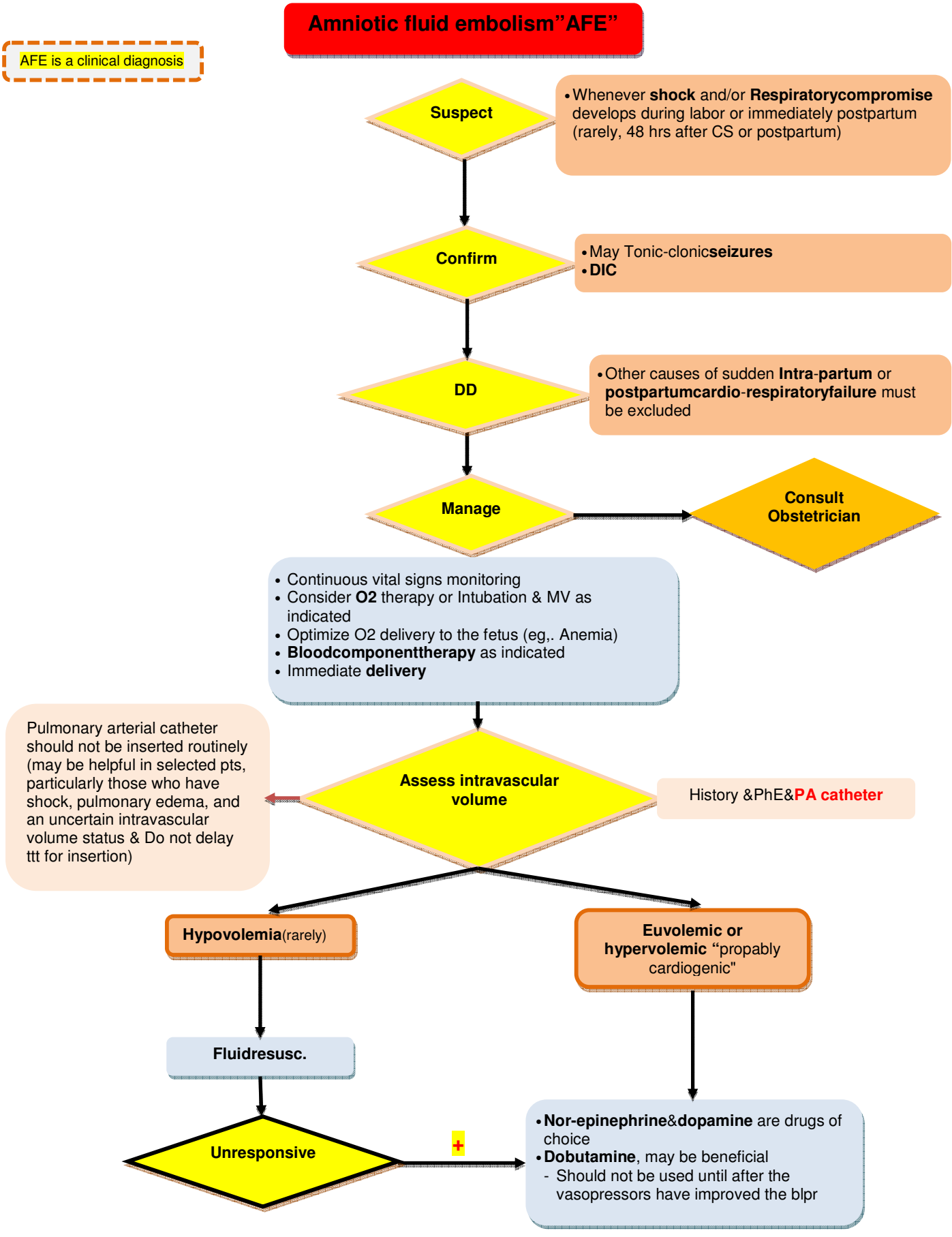
Recombinant activated factor VIIa

Recombinant activated factor VIIa

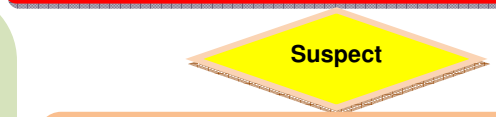
- Doses of **16.7** - **120** mcg/kg as a single bolus injection over a few min, / 2 hrs until hemostasis is achieved have been effective, and usually control bleeding within 10 - 40 min. of the 1st dose

Amniotic fluid embolism"AFE"

AFE is a clinical diagnosis



Pregnancy induced hypertension "PIH"



- **1**- New onset of **Hypertension** (S pr **140** mmHg, D pr **90** mmHg) after 20 ws of gestation in a previously normo-tensive woman
- **2**- Grand mal **Seizures** in a pregnant

Search for proteinuria

- **1 + Proteinuria** (> 0.3 gm in a 24-hr urine specimen) ---- diagnose **Pre-eclampsia**
- **1 +2 + Proteinuria -------** diagnose **Eclampsia**
  - May be with gestational hypertension
  - Seizures not be attributable to another cause

- Exclude other disorders characterized by hypertension and proteinuria

- Mild or severe (there are no criteria for moderate preeclampsia)

- **Well-being** & **growth**
- Consider ante-natal **corticosteroids**

Mg sulphate therapy

- **Indications**
  - Prevent eclampsia in severe preeclampsia (**Level 1A**) & mild pre-eclampsia (**Level 2B**)
- **Loading 4** - **6**g IV 15 - 20 min.
- **Maintenance 1** - **3** g / hr.
  - Therapeutic level: **4**-**6** mg/dl. "Sub-therapeutic" Mg levels (<**4** mg/dL) more with a 4 g loading
  - Following serum Mg levels is not required if woman's closely monitored clinically
  - Maintenance phase is given only if patellar reflex is present, RR> 12 / min., and UOP > 100 mL / 4 hrs
  - Dosing should be adjusted with renal insufficiency (serum creatinine **>1.0** mg/dL)
  - Give a standard loading dose, then reduced maintenance dose (**1** g / hr or no maintenance dose if the serum creatinine is **> 2.5** mg/dL) with close monitoring of their serum Mg / 6 hr
- Continuedfor 24 hr postpartum (12 hr in mild pre-eclampsia, may 48 in severe one)
  - Adjust dose down if there is clinical evidence of toxicity
- **CI** in myasthenia gravis ---- use diazepam
- Concurrent Ca channel blockers may cause hypotension

Preeclampsia superimposed upon chronic hypertension

- When a woman with preexisting hypertension develops new onset proteinuria >**20** ws gestation
- Women with both preexisting hypertension and proteinuria are considered pre-eclamptic if there is an exacerbation of bl pr systolic **≥160** mmHg or diastolic **≥110** mmHg in the last half of pregnancy, especially if with increased liver enzymes or thrombocytopenia

Chronic hypertension

- Refers to systolic pr **≥140** mmHg, diastolic pr ≥ **90** mmHg, or both, that antedates pregnancy, is present <**20th** week pregnancy, or persists >**12** ws postpartum

Gestational hypertension

- Hypertension without proteinuria (or other signs of preeclampsia) in the latter part of pregnancy
- Bl pr should be elevated on at least 2 occasions at least 6 hrs apart
- Should resolve by **12** w postpartum

Indications of delivery

- Deliver mild preeclampsia by **40** W gestation
- Progression to eclampsia
- Severe preeclampsia &eclampsia regardless of gestational age

Seizures

- If is witnessed ---maintain airway & prevent aspiration
- Roll gravida onto left side
- A bed with raised, padded side rails protect from trauma
- Supplemental **O2**
- **Mg sulphate therapy**

Antihypertensive therapy

- Control mildly elevated bl pr in PIH does not alter the course of the disease, nor to diminish peri-natal morbidity or mortality
- Consider aggressive therapy for sustained D pr >**105** - **110** or S ≥**160** mmHg
- Target –S pr1**40** -**155**& D pr **90** -**105** mmHg
- Options for tt include
  - **Hydralazine** (5 mg IV, followed by **5** - **10** mg boluses as necessary / 20 min.) or
  - **Labetalol** (**10** or **20** mg IV followed by doubling dose at 10-min. intervals up to **80** mg for a maximum total cumulative dose of **220** - **230** mg [eg, 20 – 40 – 80 - 80 mg or 10 – 20 – 40 – 80 - 80 mg])

Invasive monitoring

- You can manage without these tools and avoid risks associated with arterial and CVC (Useful in complicated pt "severe cardiac&renal disease, oliguria, refractory hypertension, or pulmonary edema")

HELP syndrome

- HELLP syndrome (hemolysis with a microangiopathic blood smear, elevated liver enzymes, and a low platelet count) develops in approximately **1** of **1000** pregnancies overall and **10** - **20** % of pregnancies with severe PIH

Suspect

- Abdominal **pain** and tenderness in the mid-epigastrium, right upper quadrant, or sub-sternal, typically in 3rd trimester, but may 2nd trimester or postpartum
- **Hypertension** and **proteinuria** are present in approximately **85** % of cases" may be absent in with otherwise severe HELLP syndrome"
- **Lab**. : hemolysis, elevated liver enzymes, and thrombocytopenia

Clinical

- Many pts also have N &V, and malaise
- DIC, abruptio placentae, acute renal failure, pulmonary edema, sub-capsular liver hematoma, and retinal detachment
- Jaundice and ascites may be present. Bleeding due to thrombocytopenia is uncommon
- Some pts are asymptomatic

Lab

- Micro-angiopathic hemolytic anemia with characteristic schistocytes (also called **helmet cells**) on blood smear
- Elevated LDH or indirect bilirubin and a low serum Hgb (≤25 mg/dL)
- Platelet count ≤**100,000** cells/microL
- Serum LDH ≥ **600** IU/L or total bilirubin ≥ **1.2** mg/dL
- Serum AST ≥**70** IU/L. Some obtain ALT instead of, or in addition to, AST levels. An advantage of the AST is that it is a single test that reflects both hepatocellular necrosis and red cell hemolysis
- Women who do not meet all of the above laboratory abnormalities are considered to have **partial HELLP syndrome**. These pts may progress to complete expression of HELLP S

Imaging

- Particularly CT or MRI, are useful when complications such as hepatic infarction, hematoma, or rupture are suspected

Confirm → **Consult Obstetrician**

DD

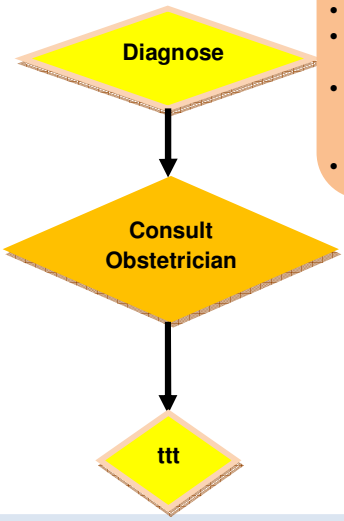
Acute fatty liver of pregnancy, gastroenteritis, hepatitis, appendicitis, gallbladder disease, ITP, lupus flare, anti-phospholipid syndrome, hemolytic-uremic syndrome, or thrombotic thrombocytopenic purpura

ttt

- Severe maternal disease (eg, multiorgan dysfunction, DIC, liver infarction or hge, renal failure, abruptio placenta) or non-reassuring fetal status ---- prompt **Delivery** regardless of gestational age
- ≥**34** ws gestation ----- **Delivery** rather than expectant management is recommended (**Level 1C**)
- <**34** ws gestation in which maternal and fetal status is reassuring ----- **Delivery** after a course of glucocorticoids rather than expectant management or prompt delivery is recommended **(Level 2C**)
- <**30** - **32** ws with an unfavorable cervix ----- **CS**
- Give **Dexamethasone** (**Level 1B**). Does not accelerate resolution of lab. abnormalities or reduce risk of complications
- **Platelet transfusion** in maternal bleeding (spontaneous or from surgical incisions), or if platelet <20,000 cells/microL. If CS is planned, transfusion is recommended to achieve a preoperative count ≥**40,000** - **50,000** cells/microL

Ovarian hyperstimulation syndrome "OHSS'

- A serious complications of excessive ovarian stimulation
- Refers to a combination of ovarian enlargement due to multiple ovarian cysts and an acute fluid shift out of the intravascular space.
- It is a potentially life-threatening complication of ovulation induction



- Respiratory distress & hypoxemia , may pneumonia, ALI, and VTE
- Hemodynamic instability& oliguria
- Ascitis " Women with severe OHSS can gain as much as 15 - 20 kg over 5 - 10 ds".
- Thromboembolic complications "reported in the internal jugular, subclavian, axillary, and mesenteric vessels"
- Stroke

General measures

- **Intercourse** is restricted in all grades of OHSS because of risk of cyst rupture
- Avoid impact-type activities or strenuous exertion

Grade I hyperstimulation "supportive"

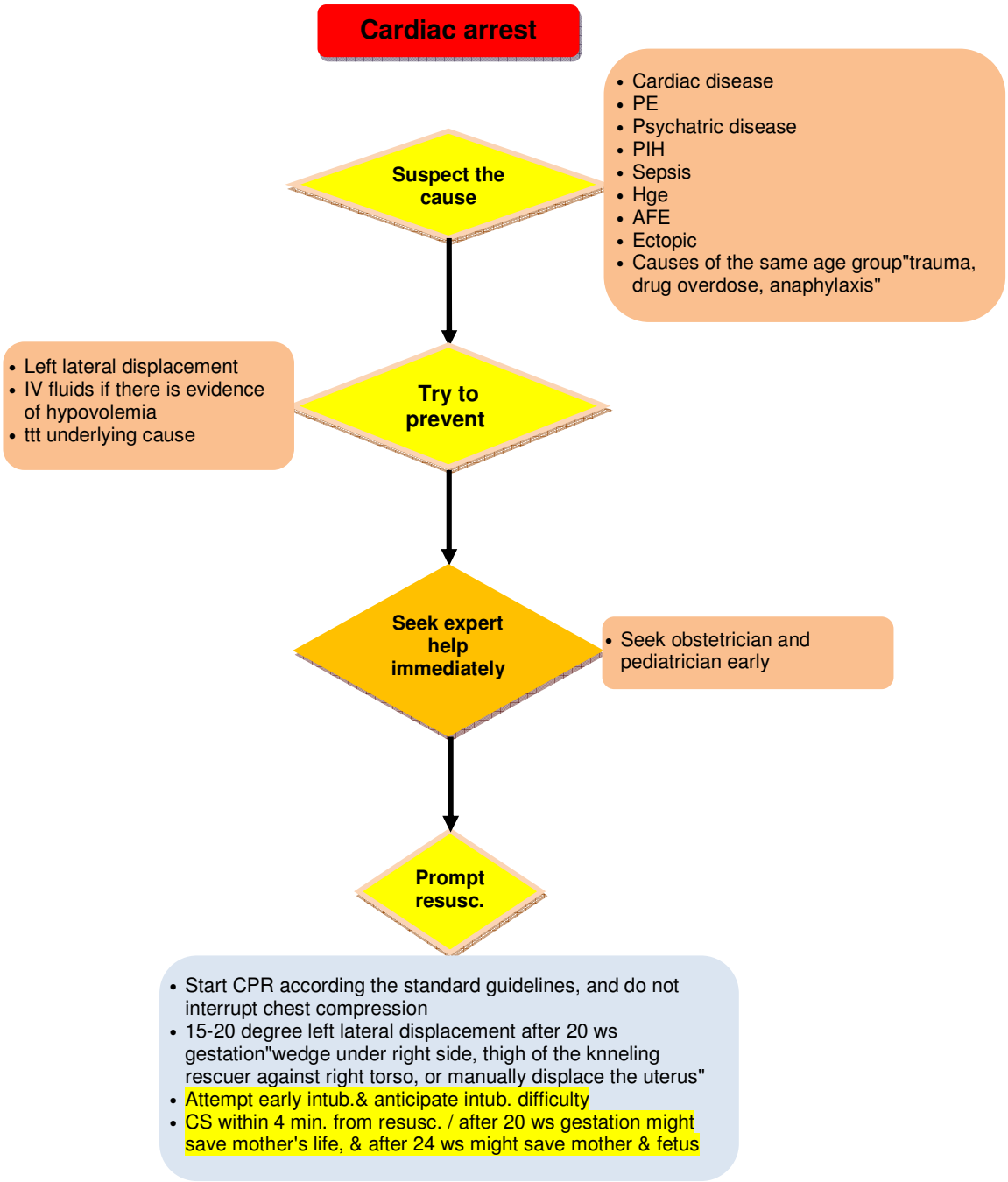
- Observe for enlarging abdominal girth, acute weight gain, and abdominal discomfort on an ambulatory basis for at least 2 ws or until the appearance of menstrual bleeding

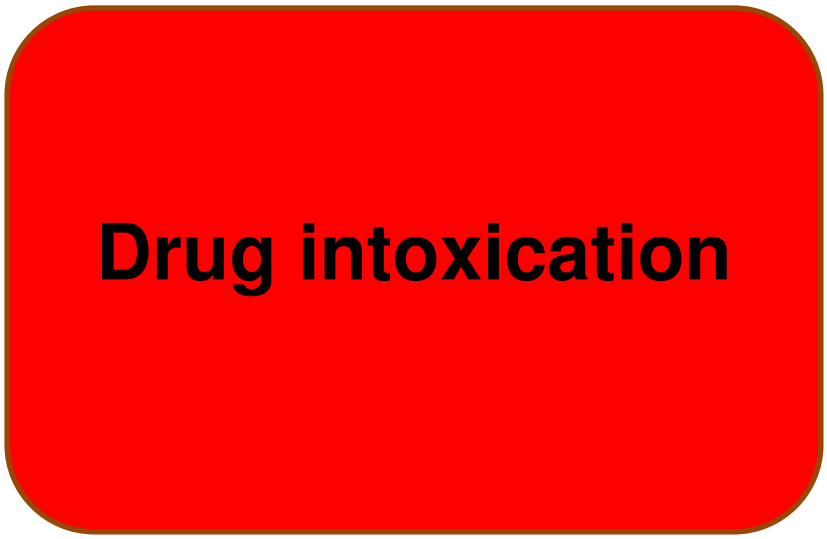
Grade II hyperstimulation

- Observation, bed rest, adequate fluids
- Sono-graphic monitoring of cyst size " Initiation of resolution is apparent when the cysts become smaller on 2 consecutive US"
- Obtain serum electrolytes, ht, and creatinine
- Monitor fluid balance --- A discrepancy in fluid balance >1000 mL/d would be of concern

Grade III hyperstimulation

- Maintaining blood volume while correcting the disturbed **Fluid and electrolyte balance**
  - **1**-**2** L of NS in 1st hr---- restore tissue perfusion
  - Further fluids -------while monitoring CVP
  - Fluid repletion ----- continue at the initial rapid rate as long as the cardiac filling pressures and the systemic blpr remain low
  - Check vital signs & intake/output& weight &abd. girth
  - CVC----- in hemodynamically unstable pt
  - Plasma expanders as dextran, human albumin (**200** mL of 25 % albumin over 4 hrs), and plasma (**500**-**1000** mL over 24 hrs) supplemented with appropriate electrolytes should be administered early & repeated as needed
  - Ht / 4 hrs; stop plasma expanders when <38 %
- Oral **Indomethacin**" blocks prostaglandin synthesis and reduces capillary permeability"
- **Diuretic** agents are not recommended
- **Pleural effusions** ------Drainto relieve dyspnea
- **Massive ascites** --- Cautious Us guided **paracentecesis**
- Preventing thromboembolic phenomena
- **Laparotomy**----- catastrophic complications" ovarian torsion or rupture and internal hge"


Cardiac arrest
Suspect the cause
Cardiac disease
PE
Psychatric disease
PIH
Sepsis
Hge
AFE
Ectopic
Causes of the same age group"trauma, drug overdose, anaphylaxis"
Try to prevent
Left lateral displacement
IV fluids if there is evidence of hypovolemia
ttt underlying cause
Seek expert help immediately
Seek obstetrician and pediatrician early
Prompt resusc.
Start CPR according the standard guidelines, and do not interrupt chest compression
15-20 degree left lateral displacement after 20 ws gestation"wedge under right side, thigh of the knneling rescuer against right torso, or manually displace the uterus"
Attempt early intub.& anticipate intub. difficulty
CS within 4 min. from resusc. / after 20 ws gestation might save mother's life, & after 24 ws might save mother & fetus


Drug intoxication

General approach to intoxicated patient

ABCDE

Suspect

Disturbed conscious level

DD

Other metabolic % central causes of disturbed conscious level

Take history

- Often unreliable when provided by a pt following intentional ingestion
- Confirmed and correlated with clinical, and laboratory the expecteddata
- If history is unreliable, take information from paramedics, police, family, friends
- A thorough search of the exposure environment should be conducted for pill bottles or a suicide note, drugs prescribed for pt, pt's family or friends
- Unknown pills or chemicals may be identified by consultation with toxicology department

Ph E

- Mental status & Vital signs
- Pupil &Odour
- Respiratory rate & pattern

See up-todatefor details

Work up

- **ECG** forsymptomatizing pt who exposed to potentially cardiotoxic agent
- **Radiology** not required in every pt
- Symptomatic pts , those with an unreliable or unknown history should undergo **urinalysis**, Serum **electrolytes**, **BUN**, **creatinine**, and **glucose**
- Serum **osmolality**, **ketones**, **creatinekinase**, **liverfunctiontests**, **amylase**, **Ca**, and **Mg**in most significantly ill pt
- **Urine pregnancy test** in childbearing women
- **ABGs**, **co-oximetry**, and serum **lactate** in pts with acid-base, cardiovascular, neurologic, or respiratory disturbances
- In acid-base disorders, rule out increased serum **Osmolalgap,** toxic etiology

See up-todatefor details

Toxin Screen

- Rarely necessary when pt with a non-intentional ingestion is asymptomatic or have clinical findings consistent with medical history
- Screen for **Acetaminophen**&**Salicylates** for pt with uncertain history or intentional poison
- "**Drugsofabuse**" immunoassay screens can be used to detect **Opiates**, **Benzodiazepines**, **Cocaine** metabolites, **Barbiturates**, TAD, and phencyclidine in urine " inexpensive and provide rapid results, usually within 1 hr'
- **Comprehensivetoxicscreening** should be performed on blood and urine only in pts with severe or unexplained toxicity
- **Quantitativeassays**--- Useful in guiding management of certain intoxications when interpreted in conjunction with clinical status and the timing of poisoning

- Optimal management depends on specific poison(s) involved, presenting and predicted severity of illness, and elapsed time between exposure and presentation
- The sooner the decontamination, the more effective it is at preventing poison absorption

Decontaminate

Dermal exposure

- Initial ttt is immediate decontamination
- Manually remove clothing and particulate matter and exposed surfaces copiously irrigated with NS, RL, or tap water. forat least 20 min., and longer for corrosives. Wash with soap and water is more effective than water in **Organophosphate** or **Carbamate** insecticides if skin is intact)
- Water irrigation is absolutely CI in certain elemental and reactive metal compounds (eg, Ca oxide, cesium, lithium, Mg, PO4, K)

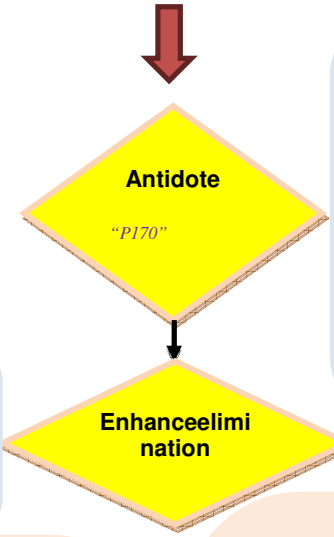
- Copious water or NS irrigation for topical exposures and administration of **Activatedcharcoal**for ingestions are preferred methods
- Other methods may be warranted **asyrup of ipecac, gastric lavage, whole bowel irrigation, endoscopy, surgery, dilution,** and**cathartics**

Inhalation exposure

- **CO** is responsible for the bulk of them
- Victims should be rapidly removed from exposure environment and treated with fresh air or O2

Occular exposure

- Immediately decontaminate
- Carefully remove particulate with a cotton tipped swab and irrigation performed for at least of 30 min. with copious amounts of NS, RL solution, or tap water. Continued till pH of ocular fluid is between 6 and 8 in pt with acid or alkali exposures



- Antidote may be eliminated more rapidly than the ingested substance
  - Somnolence and respiratory depression due to ingested opiates reversed with **Naloxone** but recur in approximately 1/3 of cases because elimination 1/2life only 60 - 90 min.
  - In certain situations antidotes may require repeated administration or infusion
- Routine administration of **Flumazenil**suspected of benzodiazepine overdose may precipitate seizures and worsen clinical course if TCA have been co-ingested

- **Urinary alkalinization**
- **Multi-dose activated charcoal**
- **HD &hemoperfusion**
- **Hemofiltration**
- **Exchange transfusion**

Gastric lavage

Technique
- Performed with the pt in the left lateral decubitus position with the head in a 15º Trendelenburg position
- Intubation is not necessary in an awake cooperative pt
- Gravitational instillation & drainage of multiple sequential aliquots of **200** - **300** mL of warmed NS or tap water
- Continued till effluent is relatively clear "**5** L sufficient"

Efficacy
- Decreases as the time between ingestion &ttt increases
- Large amounts of unabsorbed drug removed in minority
- Less than activated charcoal & equivalent to ipecac
- More effective when combined with activated charcoal

Indications
- Its routine use is no longer recommended
- Acceptable if pt ingested potentially toxic substance and the procedure can be performed within 1 hr of ingestion
- Its use is not excluded in pt who present > 1 hr, particularly when they are highly toxic

CI
- A corrosive agent
- A depressed mental status &pt not intubated
- Risk for hge or perforation

Complications
- Increased risk of aspiration
- Laryngospasm, hypoxia and hypercapnia, esophageal and gastric erosions, bleeding, perforation, inadvertent tracheal insertion, arrhythmias, ischemia, pneumothorax, fluid and electrolyte imbalances, and hypothermia

Activated charcoal

- The preferred means of GI decontamination

Agents not well adsorbed by activated charcoal
Heavy metals Arsenic Lead Mercury Iron Zinc Cadmium Inorganic ions Lithium Sodium Calcium Potassium Mg Fluoride Iodide Boric acid Corrosives Acids Alkali Hydrocarbons Alcohols Acetone Ethanol Ethylene glycol Isopropanol Methanol Essential oils

- 90 % of an intoxicant adsorbed when the activated charcoal / intoxicant is ≥10:1
- Larger doses (single or multiple), are more effective

Dose
- (**1** g/kg with at least a 10::1 activated charcoal to intoxicant); the usual single adult dose is **25** - **100** g with water and administered as a slurry by mouth or NGT
- Doses >**100** g are not recommended in obtunded pt

Efficacy
- Greatest when administered within 1 hr from ingestion

Indications
- Administered even with equivocal exposure history
- For greatest benefit ----administerwithin 1 hr

CI
- Absolutely CI in pt with bowel obstruction or perforation and should not be given to pt with a depressed level of consciousness until the airway is secured by ETT
- Not recommended in pt who have ingested non-adsorbable acidic or alkaline corrosives and who require endoscopy "will obstruct view of the endoscopist"

Complications
- N&V, cramps, diarrhea, constipation & bowel obstruction
- Aspiration , pneumonitis or, rarely airway obstruction

Urinary alkalinization

- **Indications and efficacy**

Agents for which alkaline diuresis may enhance elimination
2,4-D chlorphenoxyacetic acid (herbicide) Chlorpropamide Salicylates Diflunisal Fluoride Methotrexate Barbiturates Phenobarbital

- Urinary alkalinization is the most effective single method short of HD to enhance **Salicylate** excretion

Technique
- Targeturine pH ≥**7.5** while a serum pH no >**7.55 - 7.60**
- Administer IV bolus of **1**-**2**mEq/kg of 8.4 % NaHCO3, followed by continuous infusion "mixed by placing **150**mEq of NaHCO3 into **1** L of 5 % D5 in water "at 200 - 250 cc/hr".
- Prior to initiation ,perform baseline measurements of electrolytes, BUN, serum creatinine, glucose, systemic pH, urinary pH, and serum drug conc.
  - Place of a foley catheter to accurately measure UOP

Syrup of ipecac

- Induces emesis in > 90 % of overdose pts with a mean time of onset of 20 min.
- The adult dose is **30** mL by mouth + **240** mL water

Indications
- Should not be administered routinely
- Alert, conscious pt within 1 hr.

CI
- Depressed mental status (eg, coma, seizure)
- Substance compromise airway protective reflexes in 1 hr
- Corrosive agents

Complications
- Protracted vomiting in a substantial number of pt
- May delay giving activated charcoal and oral antidotes

Organo-phoshrus poisoning

Acute Toxicity

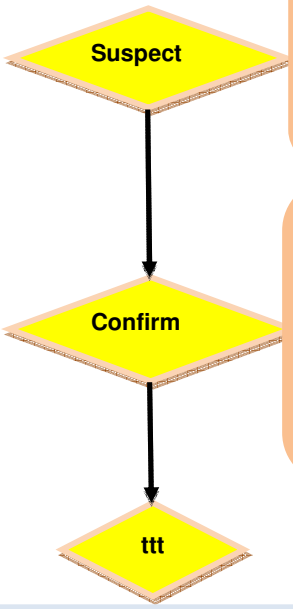
- Generally manifests in min. to hrs
- Evidence of cholinergic excess
- Respiratory insufficiency "result from muscle weakness, decreased central drive, increased secretions, and bronchospasm"

Intermediate Syndrome

- Occurs 24-96 hrs after exposure
- Bulbar, respiratory, and proximal muscle weakness are prominent features
- Generally resolves in 1-3 ws

Organophosphorous Agent-Induced Delayed Peripheral Neuropathy (OPIDN)

- Usually occurs several ws after exposure
- Primarily motor involvement
- May resolve spontaneously, but can result in permanent neurologic dysfunction



- History
- Evidence of cholinergic symptoms
  - **SLUDGE** = **S**alivation, **L**acrimation, **U**rination, **D**efecation, **G**astric **E**mptying
  - **BBB** = **B**radycardia, **B**ronchorrhea, **B**ronchospasm

- **Atropinechallenge** if diagnosis is in doubt (**1** mg IV in adults, **0.01**-**0.02** mg/kg in pediatric)
  - Absence of anti-cholinergicsigns and symptoms (tachycardia, mydriasis, decreased bowel sounds, or dry skin) strongly suggests poisoning with organophosphate or carbamate
- Draw blood sample for measurement of RBC **acetyl-cholinesteraseactivity**

- Deliver 100 % **O2**
- Strongly consider **Intubation**
- **Atropine2-5** mg IV bolus (**0.05** mg/kg IV in children)
  - Escalate (double) dose / 3-5 min. till bronchial secretions & wheezing stop
  - Tachycardia and mydriasis are not CI to atropine use; Hundreds of mg may be needed over several ds in severe poisonings
- **Pralidoxime** (2-PAM) **2** g (**25**-**50** mg/kg in children) IV over 30 min.
  - Continuous infusion at **8** mg/kg/hr in adults (**10**-**20** mg/kg/hr in pediatric)
- **Benzodiazepine** therapy

Approach to critical ill patient

General approach to critical ill patient

General principles

- UseAirway, Breathing, Circulation, Disability, and Exposure approach "**ABCDE** approach"
- Initially assess and reassess regularly
- Treat life-threatening problems before moving to next step
- Assess effects of ttt.It can take a few min.
- Aim of initial ttt is to keep pt alive &achieve some clinical improvement (buy time)
- Call for help early
- Assessment, attaching monitors, IV line under taken simultaneously
- Communicate effectively

First steps

- Ensure personal **Safety** (eg., gloves)
- **Firstlook** at the pt in general to see if the pt appears unwell
- If the pt is awake, ask "How are you?"
  - If pt unconscious or has collapsed, shake him and ask "Are you alright?" If he responds normally he has patent airway, is breathing and has brain perfusion
  - If speaks in short sentences, he may have breathing problems
  - Failure to respond indicates critical illness
- Monitor the **Vitalsigns** early
  - Attach pulse oximeter, ECG monitor and non-invasive blpr monitor rapidly
- Insert an IV **Cannula** as soon as possible
  - Take bloods for investigation
- Take **History** as possible

Airway

- Look for the signs of airway obstruction

- Treat airway obstruction as a medical emergency:
  - Basic airway management, adjuncts, or advanced a
- Give **O2** at high concentration
- Stabilize C- spine if in doubt

Breathing

Data in A +

- Look, listen and feel for signs of respiratory distress
- Count the **RR** (tachypnea is a marker of illness)
- Assess the depth of each breath, pattern and equality
- Note any chest deformity, raised JVP (e.g., in acute severe asthma or a tension pneumothorax); note presence and patency of any chest drains
- Record the FIO2 given to pt and SpO2
- Listen to breath sounds a short distance from his face:
  - Rattling air way noises indicate airway secretions
  - Stridor or wheeze suggests partial, but significant, airway obstruction
- **Percuss** the chest:
  - Hyper -resonance with pneumothorax; dullness with consolidation
- **Auscultate** the chest:
  - Bronchial breathing indicates lung consolidation Absent or reduced sounds with a pneumothorax or pleural fluid or consolidation
- Check the position of trachea in suprasternal notch:
  - Deviation with mediastinal shift (pneumothorax, lung fibrosis , pleural fluid)
- Feel chest wall for surgical emphysema or crepitus
- Order **CXR**, **BNP**, **D-dimer**, **ABGs**, or **PFT** as appropriate

- Diagnose &immediately ttt**Life-threateningconditions** as acute severe asthma, PE, tension pneumothorax
- The specific ttt depends upon the cause
  - All critically ill pts should be given O2
  - In COPD PaO2 of 60 mmHg or SpO2 of **90**-**92**% is a suitable target
- If depth or RR is judged to beinadequate, or absent, use non-invasive, BVM or ETT ventilation to improve oxygenation and ventilation

Circulation

Data in A + B +

- Suspect shock in disturbed conscious level , tachycardic, pt with cold skin
- In most medical and surgical emergencies, consider hypovolaemia as primary cause, till prove otherwise
- In surgical pt, rapidly exclude hge (overt & hidden)
- Breathing problems, as a tension pneumothorax, can compromise a pt's circulatory state
- Look thoroughly for external hge from woundsor drains or evidence of concealed hge (e.g., thoracic, intra-peritoneal, retroperitoneal)
- Look at the colour of the hands and digits:
  - Are they blue, pink, pale or mottled?
- Assess the limb temperature by feeling pt' s hands
  - Are they cool or warm?
- Measure the capillary refill time (CRT)
  - A prolonged CRT suggests poor peripheral per fusion. Other factors (e.g., cold surroundings, poor lighting, old age) can prolong CRT
- Assess state of veins (underfilled or collapsed with hypovolaemia)
- Count pulse rate (or preferably HR)
- Palpate peripheral and central pulses, assessing for presence, rate, quality, regularity , equality
  - Barely palpable central pulses suggest poor COP & bounding pulse may indicate sepsis
- Measurebl pr.
  - Even in shock, blpr may be normal
  - Low diastolic pr suggests vasodilation (eg., sepsis)
  - A narrowed pulse pr (normally 35-45 mmHg) suggests arterial vasoconstriction (cardiogenic shock or hypovolaemia) or tachyarrhythmia
- Look for other signs of a poor COP, such as reduced conscious level and, if the pt has a urinary catheter, oliguria (urine volume < 0.5 ml kg /hr)
- Auscultate the heart
  - Is there a murmur or pericardial rub?
  - Are the heart sounds difficult to hear?
  - Does the audible HR correspond to the pulse rate?

- The specific ttt of cardiovascular collapse depends on the cause, but should be directed at fluid replacement, hge control and restoration of tissue perfusion
- Seek signs of conditions that are immediately life threatening, e.g., cardiac tamponade, massive or continuing hge, septicaemic shock, and ttt them urgently
- Insert one or more large short, wide-bore (14 or 16 G) IV line
- Take blood from the cannula for routine haematological, biochemical, coagulation and microbiological investigations, and cross-matching, before infusing IV fluid
- Give rapid fluid challenge (over 5-10 min) 500 ml of warmed crystalloid solution if normotensive. 1 L, if hypotensive
  - Use smaller volumes (e.g., 250 ml) for pts with known cardiac failure and use closer monitoring (listen to the chest for crackles after each bolus, consider a CVP line)
- Reassess HR and BP regularly (/ 5 min), aiming for pt's normal BP or, if this is unknown, a target > 100 mmHg systolic
- If pt does not improve, repeat fluid challenge.
- If symptoms and signs of cardiac failure (dyspnoea, increased HR, raised JVP, a third heart sound and pulmonar y crackles on auscultation) occur, decrease the fluid infusion rate or stop the fluids altogether
  - Seek alternative means of improving tissue perfusion (e.g. inotropes or vasopressors)
- If the pt has primary chest pain and a suspected ACS record a 12-lead ECG early, and ttt initially with O2, aspirin, nitroglycerine, and morphine

Disability

Data in A + B + C+

- **Causes** of unconsciousness include:metabolic causes "profound hypoxia, hypercapnia, cerebral hypo-perfusion, recent administration of sedatives or analgesic drugs" or central causes
- Check the pt's drug chart for reversible drug-induced causes of depressed consciousness
- Examine the **Pupils** (size, equality and reaction)
- Make rapid initial assessment of conscious level using the **AVPU score** "Alert, responds to Vocal stimuli, responds to Painful stimuli or Unresponsive
  - Alternatively use the GCS score
- Detect **Seizures,** or signs of **Lateralization**
- Measure the blood glucose, CBC, thyroid, adrenal functions, screening for TTP, DIC if appropriate
- CT or MRI brain or LP if central causes suspected

- ttt metabolic causes
- Give an antagonist where appropriate (e.g., naloxone for opioid toxicity)
- ttt ABCD, exclude & treat hypoxia and hypotension
- If bl glucose below **50** mg/dl, give **50** ml of 10% glucose solution IV
- ttt definite seizures with **Phenytoin** orequivalent
- Nurse unconscious pt in the lateral position if their airway is not protected

Exposure

Data in A + B + C+ D+

- Appropriate exposure individually
- Consider head to toe examination in poly-trauma pt
- Search for bleeding, suction drain, bed sores, edema, etc,.

- Prevent hypothermia
- Keep dignity of the pt

Cardiovascular disorders

SHOCK "see drug summary p165"

Physiologic state characterized by significant reduction of systemic tissue perfusion, result in decreased tissue O2 delivery

Suspect

- Suggestive **History**
  - (trauma, septic focus, fluid loss, MI, long term steroids,trigger anaphylactic agentetc)
- Suggestive **Examination**
  - Altered conscious level, tachycardia, cold clammy skin

Confirm

PhE

Cardinal findings

- **Hypotension** (**Absolute** (eg, S blpr< 90 mmHg) or **Relative** "eg, drop in S blpr> 40 mmHg "pt may in shock with a high or normal BP").
- **Oliguria**
- **Change in mental state**
- **Cold clammy skin** (early distributive or terminal shock ----- may flushed, hyperemic)
- **Metabolic acidosis**

Suggestive findings (neither sensitive nor specific)

Lab.

- **CBC** (anemia in hypovolemia, or leucocytosis in sepsis
- **Chemistry** tests (Na, K, CL, serum HCO3)
- **BUN, creatinine, liver function tests, amylase, lipase**
- **PT** or **PTT**, **fibrinogen**, fibrin split products or dimer, cardiac enzymes
- **ABG**, **toxicologyscreen**&**lactate**
- **Urinalysis**
- **Gramstain** of material from sites of possible infection (sputum)

ECG

- Ischemia, MI, and arrhythmias

Radiology

- A **CXR** for pulmonary congestion or pulmonary edema, abdominal radiograph for intestinal obstruction
- **FAST** examination in stable or unstable pt in traumatic abd. injury "**CT** scan typically is performed in stable pt

ECHO

- Assess ventricular function; detect tamponade, severe MR, vent. septal rupture, proximal aortic dissection)
- **TTE** is usually performed (difficult to obtain adequate image in ICU pts) "TEE is not 1st choice "some risk in non -intubated pt"

Intra-arterial pr

- Should be performed (**Level I**) in
  - Severe hypotension (sys Pr< 80)
  - During the administration of vasopressor ± inotropic agents
  - Cardiogenic shock
- Useful in when potent vasodilators "Na nitroprusside" given (**Level IIa**)

Pulmonary art. Catheterization

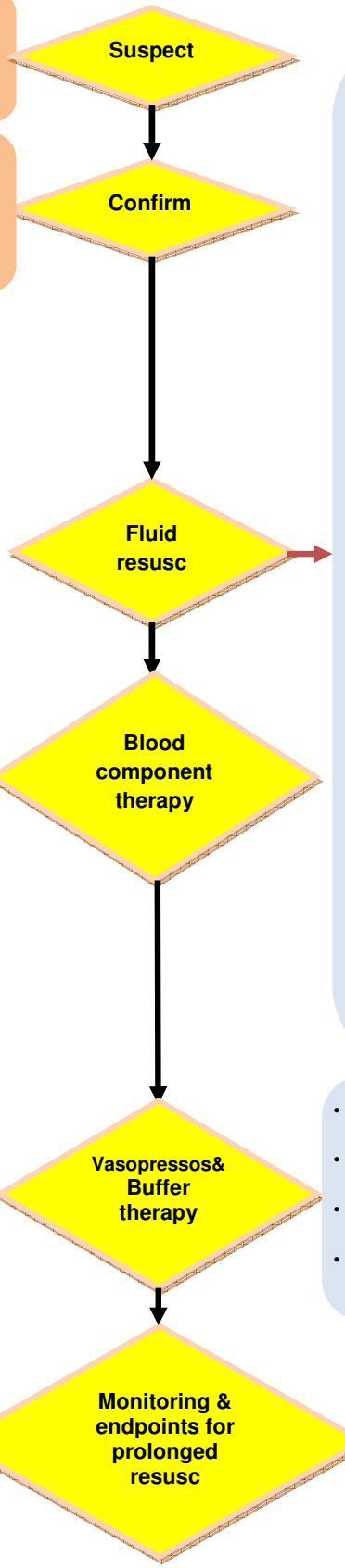
Should be performed in

- Progressive hypotension when no response or CIto fluids
- Suspected mechanical complications of STEMI (ventricular septal rupture, papillary muscle rupture) if echo is not performed

Considered useful (Level IIa) in:

- Hypotension without pulmonary congestion who has not responded to initial fluid resusc.
- Cardiogenic shock
- Severe & progressive HF or pulmonary edema that does not respondrapidly totherapy
- Persistent signs of hypoperfusion with no hypotension or lung congestion

Hypovolemic shock



- Positive history; hematemesis, hematochezia, melena, vomiting
- Evidence of blunt or penetrating trauma, or operation

- **PE**; Decreased skin turgor, dry skin, axillae, tongue, or dry oral mucos, postural hypotension, or diminished CVP
- **CBC**&**Na& ABGs & Lactate**
- **FAST**

4+ bl. In the floor

- **4=** Intapertoneal, Retropertoneal&pelvi, Chest, Long bones
- **Bl. In the floor =** External he

- Initial fluidresusc. for traumatic hemorrhagic shock ------**2** L of **NS** (**Level 2C)**
- Change to **LR** after the initial resuscitation (ie, once 3 L or 50 mL/kg of NS has been infused) if require additional IV fluid
- Given as rapidly as possible via short, large (>16) peripheral lines
- Blpr, UOP, mental status, and peripheral perfusion, are adequate to guide resusc.
- Peripheral edema is often due to dilutionalhypoalbuminemia and not used as a marker for adequate resusc.
- Place arterial line in all pts who fail to respond promptly to initial resusc." monitoring of CVP can direct therapy"
- Measure PCWP if pt has underlying cardiopulmonary dis.
- In traumatic shock, further transfusions are based upon response to initial transfusion
  - MAP **65** mmHg or a Sblpr 90 mmHg is a reasonable in penetrating trauma
  - In blunt trauma pt, particularly those with possible TBI, a MAP >**105** mmHg or a SBP >**120** mmHg is reasonable
  - These goals adjusted upward in ptwith a history of uncontrolled hypertension
  - During resusc., do not allow initial favorable response to fluids to distract from possible severe, occult injury "**4 + bl. In the floor**" **+** Consult as indicated
  - **Delayed fluid resusc.**
    - Targets early fluid resusc. to a S blprof **70** mmHg" Controlled hypotension
    - Beneficial in hemorrhagic shock due to torso injuries(gunshot or stab wound)
    - May be detrimental to blunt trauma pts with brain injury
    - Do not implement unless emergent surgical exploration can be performed

RBCs transfusion (Hgic)

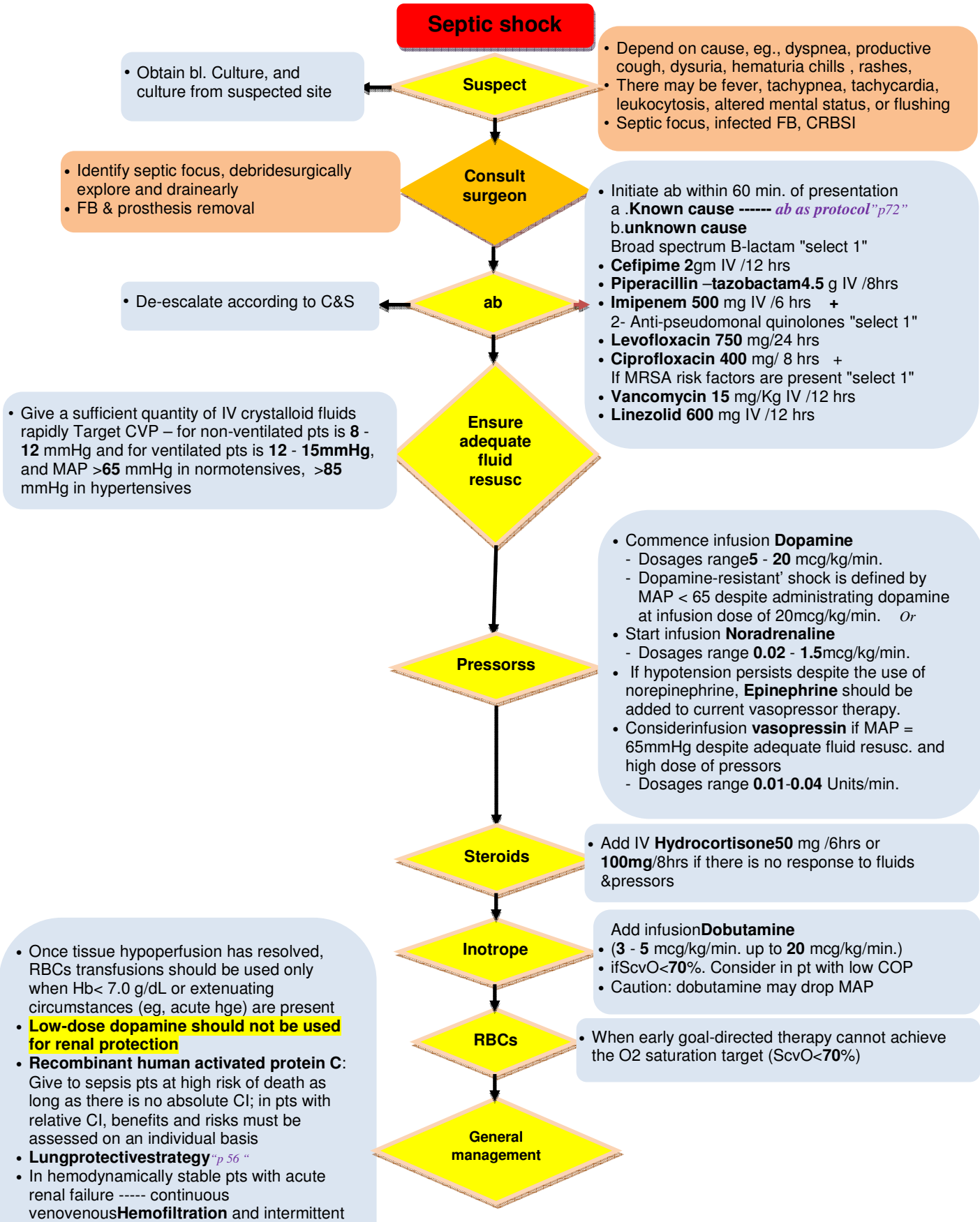
- **2** U of PRBC transfused if hemodynamics fail to improve after administration of 2-3 L (or > 50 mL/kg) of crystalloid (**Level** 2C)
- Immediate transfusion is needed with exsanguination imminent
- Typed and cross-matched PRBCs are best, but can require considerable time to prepare. If the pt's condition warrants, transfuse immediately using O Rh-positive for males and type O Rh-negative for girls and women of child-bearing age, until type-specific or typed and cross-matched blood is available

Plasma & platelets transfusion (hgic)

- For pts with severe ongoing bleeding who have received **4**U of PRBCs, give **1** U of FFP / **1**U of PRBCs
- Give **6** U of platelets once **6**U of PRBCs transfused

- Do not administer pressors (do not correct the primary problem & reduce tissue perfusion)
- Na HCO3 can be added to the replacement fluid if pH < 7.10
- Efficacy of alkali therapy in lactic acidosis remains controversial
- Optimal monitoring of acid-base may require measurement of mixed venous as ABGs

- **Blpr**: MAP >**65** mmHg for penetrating, and >**105** mmHg for blunt trauma
- **HR**: **60** -**100** b/ min. **SPO2**: maintain >**94UOP**: >**0.5** mL/kg/hr**CVP**: **8** - **12** mmHg
- **Lactate** and **base deficit**: serum lactate and serum HCO3 / 4 hrs
- **SVO2**: / 4 hrs; "target >**70** %"
- Transfuse bl. products in pts without massive bleeding undergoing prolonged resusc. of trauma-related shock using the following guidelines:
  - **Hb**: **2** U **PRBCs**if Hgb falls <**8** g/dL for pts without risk for **ACS**, or <**10** g/dL for pts at risk for ACS
  - **Platelets**: **6** U if <**50**,000/microL
  - **INR**: **2**U of FFP if INR rises > 2
  - **Fibrinogen**: **10** U of **cryoglobulin** if <**100** mg/d


Septic shock
Suspect
• Depend on cause, eg., dyspnea, productive cough, dysuria, hematuria chills , rashes,
• There may be fever, tachypnea, tachycardia, leukocytosis, altered mental status, or flushing
• Septic focus, infected FB, CRBSI
• Obtain bl. Culture, and culture from suspected site
Consult surgeon
• Identify septic focus, debridesurgically explore and drainearly
• FB & prosthesis removal
ab
• Initiate ab within 60 min. of presentation
a .Known cause ------ ab as protocol"p72"
b.unknown cause
Broad spectrum B-lactam "select 1"
• Cefipime 2gm IV /12 hrs
• Piperacillin –tazobactam4.5 g IV /8hrs
• Imipenem 500 mg IV /6 hrs +
2- Anti-pseudomonal quinolones "select 1"
• Levofloxacin 750 mg/24 hrs
• Ciprofloxacin 400 mg/ 8 hrs +
If MRSA risk factors are present "select 1"
• Vancomycin 15 mg/Kg IV /12 hrs
• Linezolid 600 mg IV /12 hrs
• De-escalate according to C&S
Ensure adequate fluid resusc
• Give a sufficient quantity of IV crystalloid fluids rapidly Target CVP – for non-ventilated pts is 8 - 12 mmHg and for ventilated pts is 12 - 15mmHg, and MAP >65 mmHg in normotensives, >85 mmHg in hypertensives
Pressorss
• Commence infusion Dopamine
- Dosages range5 - 20 mcg/kg/min.
- Dopamine-resistant' shock is defined by MAP < 65 despite administrating dopamine at infusion dose of 20mcg/kg/min. Or
• Start infusion Noradrenaline
- Dosages range 0.02 - 1.5mcg/kg/min.
• If hypotension persists despite the use of norepinephrine, Epinephrine should be added to current vasopressor therapy.
• Considerinfusion vasopressin if MAP = 65mmHg despite adequate fluid resusc. and high dose of pressors
- Dosages range 0.01-0.04 Units/min.
Steroids
• Add IV Hydrocortisone50 mg /6hrs or 100mg/8hrs if there is no response to fluids &pressors
Inotrope
Add infusionDobutamine
• (3 - 5 mcg/kg/min. up to 20 mcg/kg/min.)
• ifScvO<70%. Consider in pt with low COP
• Caution: dobutamine may drop MAP
RBCs
• When early goal-directed therapy cannot achieve the O2 saturation target (ScvO<70%)
General management
• Once tissue hypoperfusion has resolved, RBCs transfusions should be used only when Hb< 7.0 g/dL or extenuating circumstances (eg, acute hge) are present
• Low-dose dopamine should not be used for renal protection
• Recombinant human activated protein C: Give to sepsis pts at high risk of death as long as there is no absolute CI; in pts with relative CI, benefits and risks must be assessed on an individual basis
• Lungprotectivestrategy"p 56 "
• In hemodynamically stable pts with acute renal failure ----- continuous venovenousHemofiltration and intermittent hemodialysis are equally effective. Inhemodynamically unstable pats ------ continuous hemofiltation provides easier management of fluid balance
• Na HCO3 only for severe acidosis (arterial pH < 7.10 -7.15)
• Tight glycemic control"p25"

Cardiogenic Shock

Suspect

- Positive history “eg.;chest trauma, cardiac pt”
- Symptoms; dyspnea, chest pain, or palpitations

Confirm

- Ph E; crackles , a new murmur ,high JVP, PCWP, and CVP” may be normal or borderline elevated PCWP initially, then becomes clearly elevated after fluid challenge”
- Right vent. MI ------ (jugular venous distension with no lung congestion)
- ECCHO, angiography, ECG, and **Hemodynamic profiles**

Optimize intra-vascular volume

- Guided by ------ PCWP,SaO2, systemic blpr, and COP
- pts with suspected cardiogenic shock when there is no evidence of pulmonary congestion “clinical, chest x-ray” and pt is not in distressed ------ Give empiric challenge of **250** mL of NS prior to right heart catheterization in
- Right ventricular shock due to a right ventricular MI ------High filling pr are required to maximize flow to left ventricle
- Volume overload and pulmonary edema without hypotension ------ Give diuretics, morphine, O2, and vasodilators for

Vent.

- To correct hypoxemia and, in part, acidosis

Circulatory support

Reperfusion in MI

Tachy& Brady Arrhythmias *‘P 114-115”*

Late shock

- May be due to recurrent ischemia or re-infarction or rupture of ventricular septum or papillary muscle
- Do emergency ECHO is extremely important to rule out "mechanical" causes
- The only role for thrombolysis administered > 12 - 24 hrsif late shock is thought to be due to recurrent coronary artery occlusion and if urgent angiography and revascularization cannot be performed; prompt transfer to a tertiary center with revascularization facilities should be arranged, unless impossible

Pharmacologic agents

Dopamine

- In alpha dose (>**15** µg/kg /min.)
- Minimum required dose should be used
- It has positive inotropic effects, which may be beneficial but produces an undesirable elevation in PCWP

Nor-epinephrine

- Give when the response to dopamine is inadequate, profound hypotension or persists despite use of an IABP, or if there is marked tachycardia
- Increased SVR limit improvement in COP, increase cardiac work, raise PCWP

Dobutamine&Milrinone

- Produce vasodilation, do not reverse the hypotension
- Dobutamine should not be used as 1 st-line single therapy when hypotension is present, but can be given to less sick pts with a low CI and high PCWP but no hypotension
- An additive effect can be achieved by combining moderate dose dobutamine with dopamine
- Milder cases (ie, non-hypotensive pts in low output state) can be treated with dobutamine combined with vasodilators (IV nitroglycerin) “reduce afterload & preload”

Devices “IABP”

- Produce rapid, temporary, stabilization of the shocked pt
- In many pts, IABP reverses end-organ hypoperfusion
- Use IABP as a stabilizing measure in an acute MI in pts When cardiogenic shock not reversed with drug therapy

Coronary angiography

- Assess coronary anatomy in shock who are candidates for PCI & CABAG “Emergency PCI while patient in shock should be performed in centers with appropriate facilities & personnel

Fibrinolysis

- Fibrinolysis superior compared to placebo, but PCI or CABG are superior compared to fibrinolysis
- very high risk pts when primary PCI is not available----givefibrinolytics within 3 hr of onset, then urgent transfer
- **Usewith IABP**
  - If an IABP is to be used, fibrinolytic therapy should not be delayed once diagnosis of MI is established
  - Femoral artery IABP should be inserted by experienced one
  - **Choice of agent :** Not different from pts without cardiogenic shock

PrimaryPCI

- Preferred for shock complicating AMI “Should be performed with IABP”

CABAG

- For pts with MI who have left main or 3 vessel disease
- Underutilized in our community

Hemodynamic profile

- Pts with acute MI and LVF with evidence of peripheral hypoperfusion divided into 2 **Subset 1** "**pre-shock**" PCWP >15 mmHg, systolic pr>100 mmHg, and a cardiac index < 2.5 L/min / m2
- **Subset 2”** PCWP > 15 mmHg, systolic pr< 90 mmHg, and CI < 2.5 L/min / m2
- Useful in assessing degree of VC or VD and in identifying pts with low left-sided filling pressures due to inadequate intravascular volume or to rt vent. Infarct “Some have vasodilatory shock after MI’

Adrenal shock

Suspect

- Positive history “eg., long term steroids use”
- Critically ill shocked pt “eg., septic pt” unresponsive to standard measures

Confirm

- In normal subjects serum cortisol higher in early morning (about 6 AM), “**10-20** µg/dL”, than at other times of the day
  - Early morning low serum cortisol (< 3 mcg/dL) provides clear evidence that pt has adrenal insufficiency, and value <10 mcg/dL suggests diagnosis
  - Normal serum cortisol “3 **-** 10 µg/dL’ (85 **-** 275 nmol/L) at 4 PM, and concentrations are lowest, < 5 µg/dL
- Normal response to high-dose (IV 250 mcg) ACTH stimulation test is a rise in serum cortisol concentration after 30 or 60 min. to a peak of 18 - 20 mcg/dL (500 - 550 nmol/L) or mor
- Low-dose (1 mcg iv bolus) ACTH stimulation test criteria for a normal cortisol response after 20 or 30 min. are more variable: 17 - 22.5 mcg/d
- For both tests, a subnormal response confirms diagnosis of adrenal insufficiency
  - A normal response to the high-dose (250 mcg) ACTH stimulation test excludes primary adrenal insufficiency, and most patients with secondary adrenal insufficiency

Emergency measures

- Establish large-gauge IV access
- Draw bl. for stat serum electrolytes and glucose and plasma cortisol and ACTH “Do not wait for lab results”
- Infuse **2** - **3** L of **NS** or 5 % dextrose in isotonic saline as quickly as possible
- Monitor for signs of fluid overload by measuring central or peripheral venous pr. and listening for pulmonary räles
- Reduce infusion rate if indicated
- Inject **4** mg **dexamethasone** IV. IV **hydrocortisone** (**100** mg immediately and / 6 h thereafter) may also be used, but will interfere with measurement of plasma cortisol during the short ACTH stimulation test
- Use cortisol assays nothydrocortisone if biochemical testing is performed
- For pts with a previously known diagnosis of adrenal insufficiency, any IV glucocorticoid preparation may be used “diagnostic testing not necessary”
- Mineralocorticoids are unnecessary
- Use supportive measures as needed

Sub-acute measures

- Continue IV NS at slower rate for 24 - 48 hrs
- Search for and treat possible infectious precipitating causes of the adrenal crisis
- Perform a short ACTH stimulation test to confirm diagnosis of adrenal insufficiency, if pt does not have known adrenal insufficiency
- Determine the type of adrenal insufficiency and its cause if not already known
- Taper **Glucocorticoids** to maintenance dose over 1-3 d, if precipitating illness permits
- Begin **Mineralocorticoid** replacement with fludrocortisone (0.1 mg by mouth daily) when saline infusion is stopped

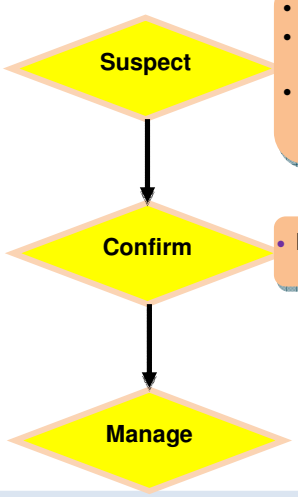
Anaphlactic shock

- Positive history “eg., trigger agent” followed by sudden collapse ± respiratory insufficiency ± upper airway obstruction

- Signs of upper airway obstruction
- Auscultatory wheezes
- Signs of circulatory collapse
- Generalized rashes

- Remove **trigger**
- **Lie** pt flat, raise leg
- Airway stabilization "consider early **Intubation**& consider awake techniques, surgical airway" + high flow **O2**.
- **Adrenaline0.5** mg 1/1000 IM "antero-lateral middle thigh" repeat 5 min. Apart, or 50 uq 1/10-000 IV titration
- Rapid fluid challenge "1/2 -1 L" **crystalloids**, repeat as needed
- Anti-histaminc" **chlorphenamine'10** mg IM or slow IV
- **Hydrocortisone200** – **500** mg IM or slow IV
- ttt bronchospasm as appropriate

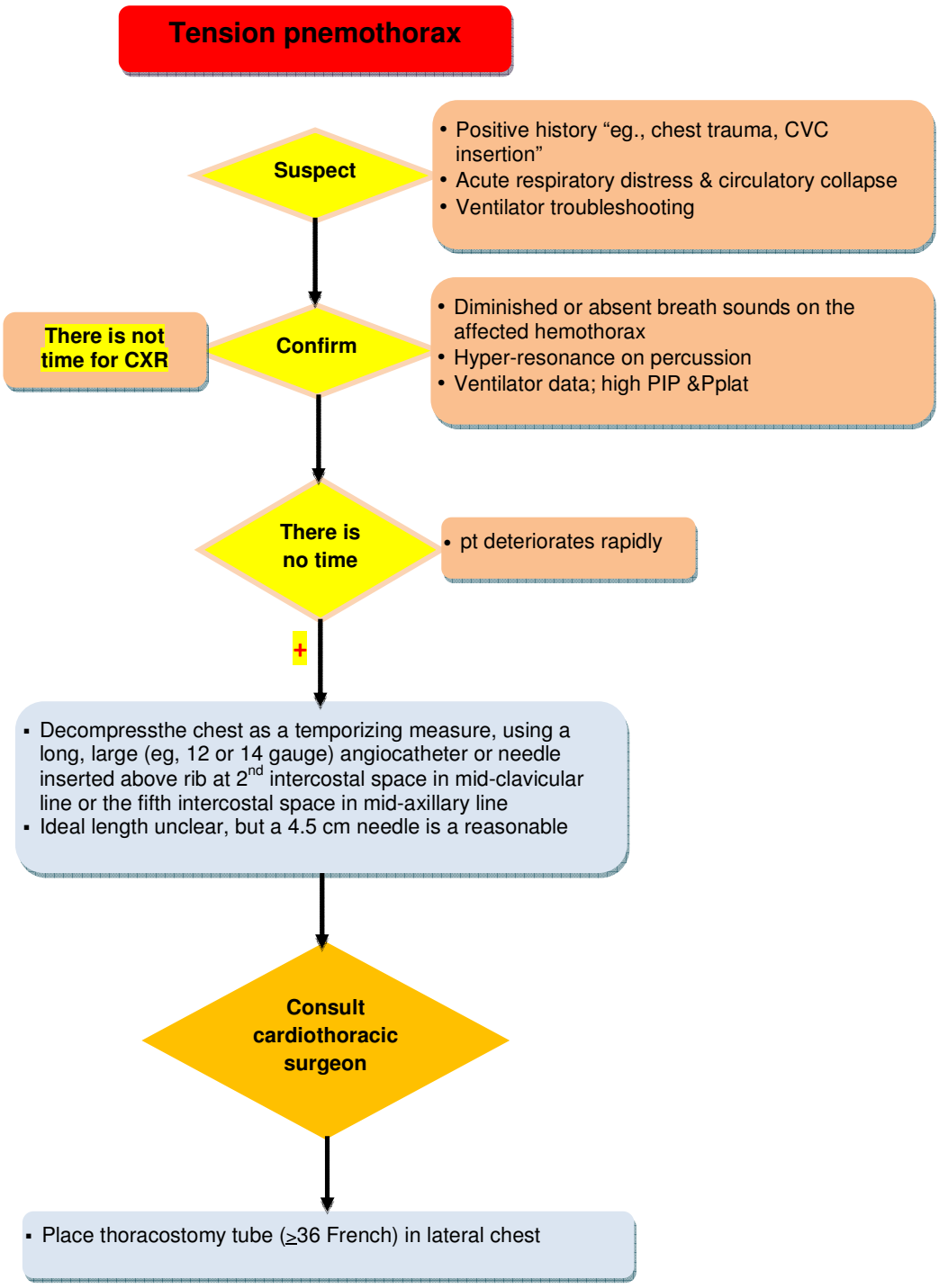
Neurogenic shock



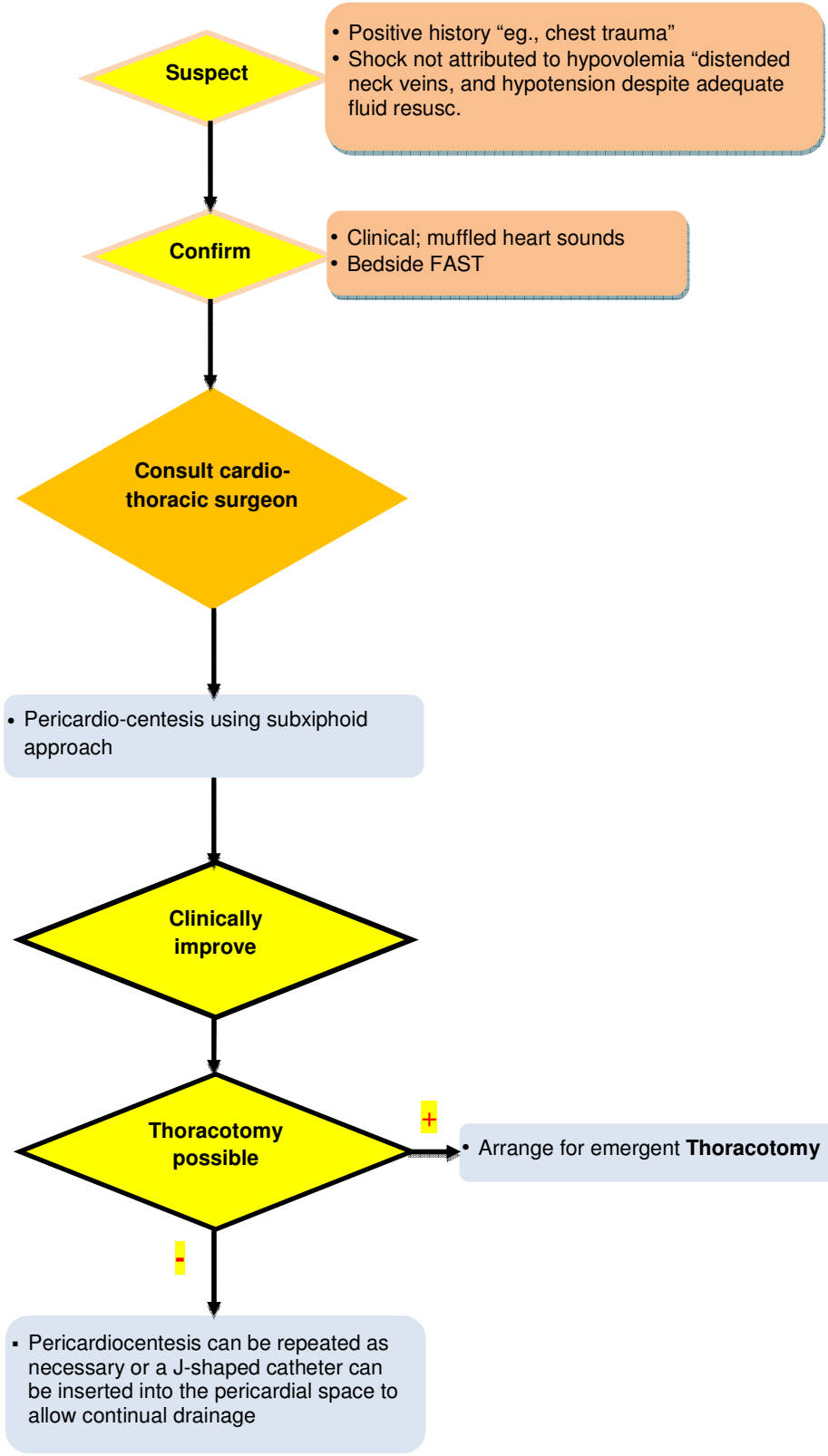
- Positive history “eg., trauma”
- Shock that does not improve despite adequate fluid resusc.
- Bradycardia± respiratory insufficiency in high cervical lesion

- Radiology to detect level of lesion *“see TSCI P83 “*

- Maintaining MAPs of at least **85** - **90** mmHg
  - Use IV fluids, transfusion, and pressors as needed
  - Excess fluids ---further cord swelling & increase damage
  - Fluid, UOP, and electrolyte must be carefully monitored
- **Bradycardia**
  - This usually occurs in severe, high cervical (C1 through C5) lesions in the 1st 2 ws after TSCI
  - ttt as protocol
- **Ventilate** as indicated


Tension pnemothorax
Suspect
• Positive history “eg., chest trauma, CVC insertion”
• Acute respiratory distress & circulatory collapse
• Ventilator troubleshooting
There is not time for CXR
Confirm
• Diminished or absent breath sounds on the affected hemothorax
• Hyper-resonance on percussion
• Ventilator data; high PIP &Pplat
There is no time
• pt deteriorates rapidly
+
▪ Decompressthe chest as a temporizing measure, using a long, large (eg, 12 or 14 gauge) angiocatheter or needle inserted above rib at 2nd intercostal space in mid-clavicular line or the fifth intercostal space in mid-axillary line
▪ Ideal length unclear, but a 4.5 cm needle is a reasonable
Consult cardiothoracic surgeon
▪ Place thoracostomy tube (≥36 French) in lateral chest

Pericardial tamponade



Tachy-arrhythmias

Vagal maneuvers
- **Carotid sinus massage**:5 s unilateral massage of carotid
- **Valsalva maneuver**:Ask pt to try to move the plunger of a 50 ml syringe by pushing air from his own breath

- **ABCDE** approach assessment
- **O2&IV** line
- **Monitor** ECG, bl pr, oximetry, 12 lead ECG
- ttt **reversiblecauses** "eg., electrolytes disorders"

Extreme tachycardia ">**150**b /min. considered by itself as adverse sign.

For sinus tachycardia----- treat he underlying cause & do not attempt

Assess evidence of adverse signs

- Shock
- Syncope
- HF
- Myocardial ischemia

+

Synchronized shocks
- "Up to **3** attempts"
- Consider sedation
- Anticoagulate with AF

Amiodrone300 mg over 10-20 min. and repeat **shock** followed by **Amiodrone900** mg over 24hrs

-

Check QRS

Narrow QRS

Check regularity

Regular

Vagal maneuver

- **Adenosine 6** mg ---- **12** mg ---- **12** mg bolus
- If adenosine CI --- consider **Verapamil2.5** - **5** mg IV over 2 min.

Sinus rhythm restored

-

- Consider A-flutter
  - Rate control

Seek expert

+

- Consider Re-entry PSVT
- **Adenosine** if recur
- Consider ant-arrhythmic prophylaxis

- **Control rate with**
  - **B-blockers**, or **Diltiazem**
  - **Digoxin**, or **amiodrone** if HF
- Do not attempt cardioversion unless anticoagulant is not administered for 3 weeks or TTE to exclude atrial thrombus

Irregular

Consider AF

<48 hrs

-

+

- **Rhythm control**
  - Chemical "**amiodrone**"
  - Electrical cardioversion
- Anticoagulate 'therapeutic **LMWH** or **UFH** to reach 1.5-2 appt, then oral anticoag. For at least 4 ws'

Broad QRS

Check regularity

Regular

- If VT "of uncertain rhythm" ---- **Amiodrone300** mg over 20-60 min. followed by **Amiodrone** 900 mg over 24 hrs
- If known to be SVT with BB --- **Adenosine** as narrow complex tachycardia

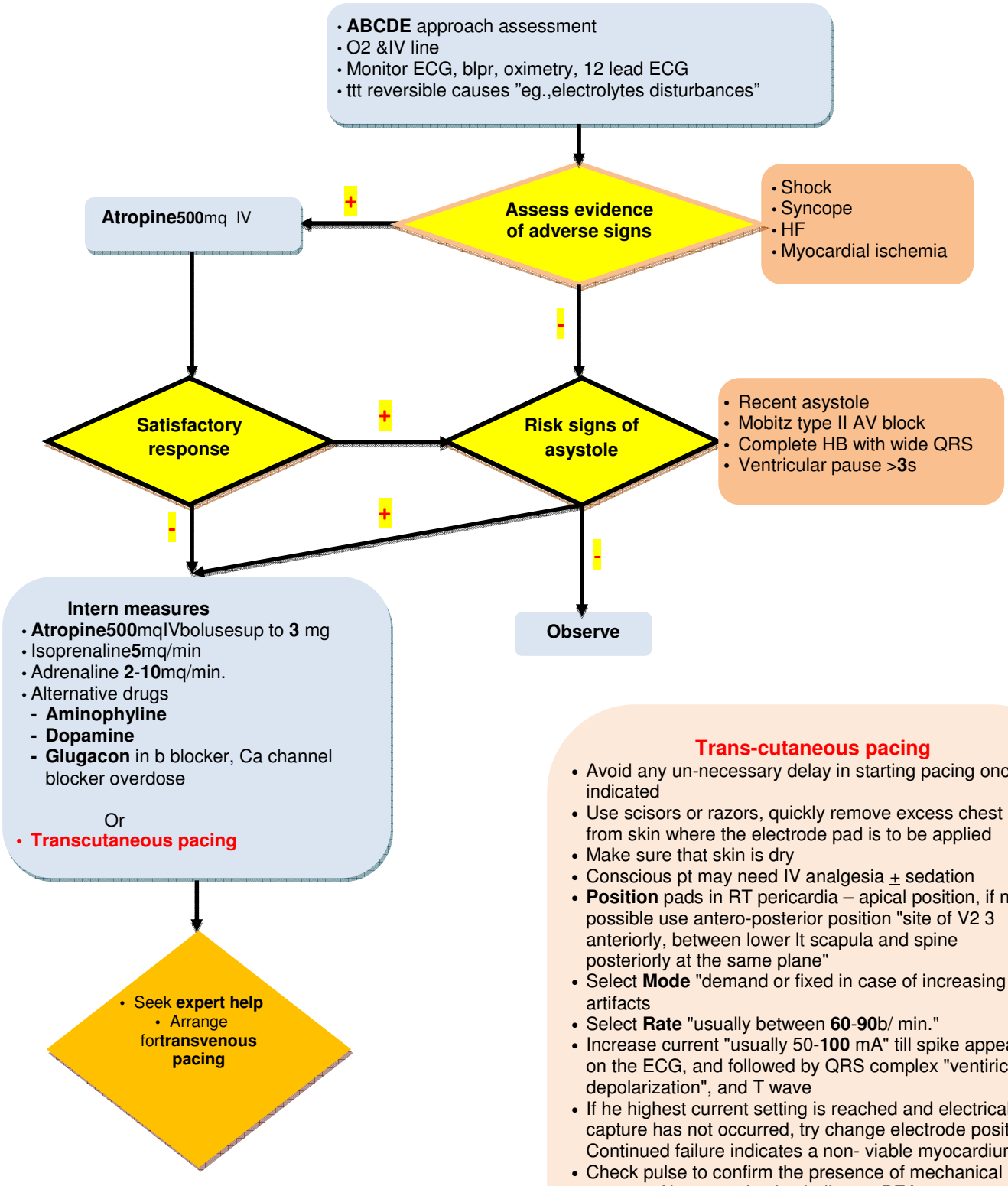
Irregular

Seek expert

Consider
- **AF with BBB** ---treat as narrow complex
- **Pre-excited AF** ----- consider **Amiodrone**
- **Polymorphic VT "Torsae de points"**---- **Mgsulphate** 2gm over **2** min.

- **Broad complex tachycardia&AF** -- start with **120**- **150** J biphasic or **200** J monophasic. If 1 st shock does not terminate arrhythmia, give up to 2 more shocks of increasing energy up to the maximum setting of defib.
- For **Atrialflutter** and **Regularnarrowcomplextachycardia** --- Start with **70**-**120** J biphasic or **100** J monophasic. If 1st shock does not terminate arrhythmia, give up to 2 more shocks of increasing energy up to maximum setting of the defib.

Brady-arrhythmias



Trans-cutaneous pacing

- Avoid any un-necessary delay in starting pacing once indicated
- Use scisors or razors, quickly remove excess chest hair from skin where the electrode pad is to be applied
- Make sure that skin is dry
- Conscious pt may need IV analgesia ± sedation
- **Position** pads in RT pericardia – apical position, if not possible use antero-posterior position "site of V2 3 anteriorly, between lower lt scapula and spine posteriorly at the same plane"
- Select **Mode** "demand or fixed in case of increasing artifacts
- Select **Rate** "usually between **60**-**90**b/ min."
- Increase current "usually 50-**100** mA" till spike appears on the ECG, and followed by QRS complex "ventiricular depolarization", and T wave
- If he highest current setting is reached and electrical capture has not occurred, try change electrode position. Continued failure indicates a non- viable myocardium
- Check pulse to confirm the presence of mechanical capture. Absence of pulse indicates PEA
- When produces COP, seek expert help to arrange for trans-venous pacing
- There is no hazard from trans-cutaneous pacing to others in contact with pt, however, it is best to shut off pacing during CPR

Hypertensive emergencies

- Are acute, life-threatening (usually associated with marked increases in BP, **"≥180/120** mmHg"
- **There are 2 major clinical syndromes induced by the severe hypertension:**
  Malignant hypertension
- Marked hypertension with retinal hges, exudates, or papilledema. There may also be renal involvement, called **Malignant nephrosclerosis**
- Although papilledema had been thought to represent a more severe lesion, it does not appear to connote a worse prognosis than hges and exudates alone (**Accelerated hypertension**)
- ttt the same whether or not papilledema is present
  Hypertensive encephalopathy
- Refers to presence of signs of cerebral edema caused by breakthrough hyperperfusion from severe and sudden rises in blpr
- Malignant hypertension is usually associated with a diastolic pr> 120 mmHg
- In comparison, hypertensive encephalopathy can be seen at diastolic pr as low as 100 mmHg in previously normotensive pts with acute hypertension due to preeclampsia or acute glomerulonephritis

Suspect

- **History** : Long-standing uncontrolled hypertension, many of whom discontinued anti-hypertensives
- **Neurologic symptoms** : Encephalopathy, Insidious onset of headache, N&V, then non-localizing neurologic symptoms as restlessness, confusion, and, if not treated, seizures & coma

Confirm

Fundus examination
- Retinal hge, exudates and papilledema

MRI
- Edema of white matter of parieto-occipital regions (**reversible posterior leuko-encephalopathy S)***or*
- Primarily pontine abnormalities (**Hypertensive brainstem encephalopathy)**
- These neurologic symptoms differ from the abrupt onset of focal neurologic symptoms of stroke or hge (not treated aggressively)

Renal biobsy
- Fibrinoid necrosis in arterioles and capillaries, producing histologic changes " indistinguishable from any of the forms of hemolytic-uremic S

ttt *"see drugs summaryP167"*

- The goal is to rapidly lower diastolic pr to about **100 - 105** mmHg within **2-6**hrs, with maximum initial fall in blpr not exceeding **25** % of the presenting value
- Once blpr is controlled, pt should be switched to oral therapy, with diastolic pr being gradually reduced to **85 - 90** mmHg over **2-3**ms
- Initial reduction to a diastolic pr of approximately **100** mmHg is often associated with a modest worsening of renal function; this change, however, is typically transient as the vascular dis. tends to resolve and renal perfusion improves over **1-3**ms
- Anti-hypertensives should not be withheld unless there is excessive blpr reduction
- A change in medication, however, is indicated if decline in renal function is temporally related to ACE inhibitor or angiotensin II receptor blocker
- **Consider blpr control in special situations**

Special situations

Chronic hypertension without neurological symptoms
- Rapid correction is not necessary in asymptomatic pt
- Slow , gradual reduction indicated in pt with known cerebrovascular dis. or long-standing uncontrolled hypertension

Angina pectoris or AMI
- Use IV vasodilators, **Nitroprusside** ,**Nitroglycerin**, **Labetalol**
- **Hydralazine**is CI

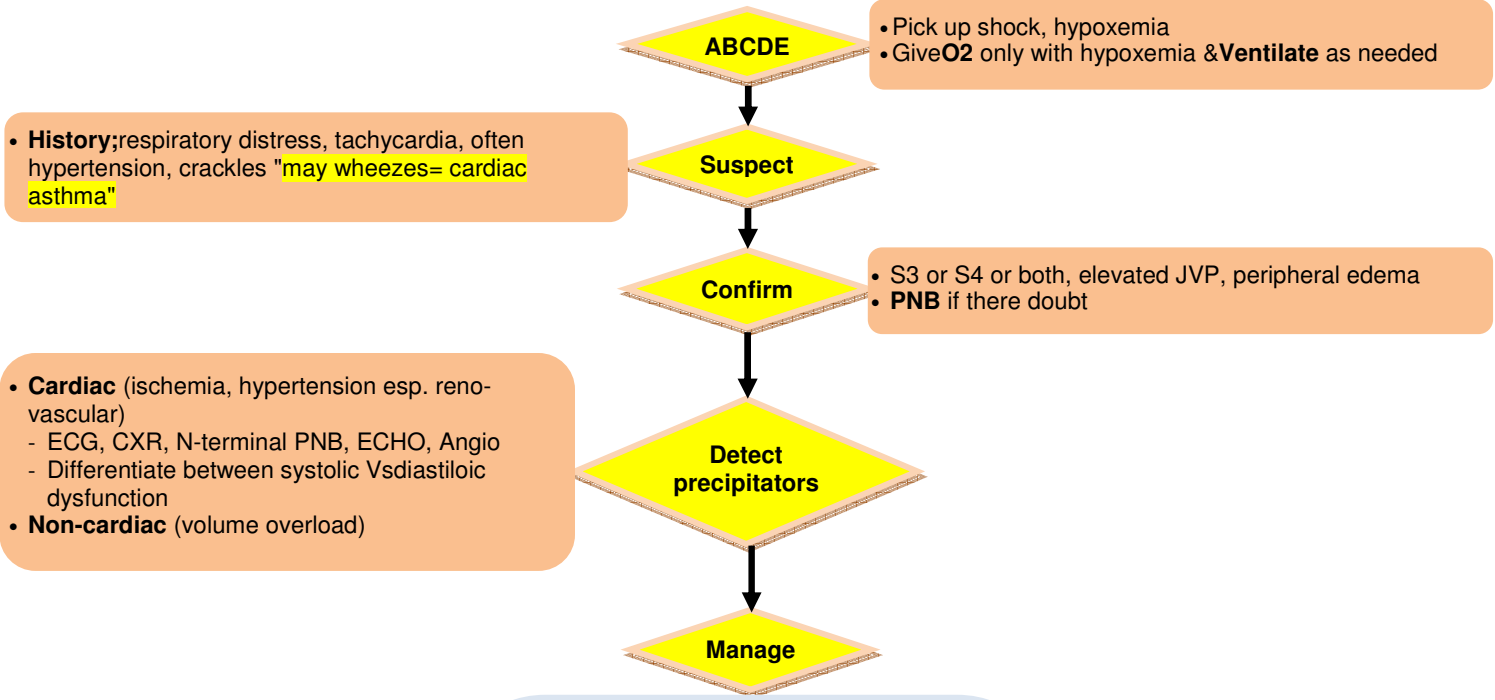
Aortic dissection
- The initial aim is to decrease S blpr of **100 - 120** mmHg if tolerated
- Combine **Nitroprusside** and IV **Bblocker** "**Nitroprussideshould not be given without a B blocker**"

Pregnancy
- IV **Hydralazine** is of choice. **Nicardipine** or **Labetalol** are alternatives
- **Nitroprusside** , **ACEinhibitors** and **angiotensinIIreceptorblockers** are CI

Ischemic stroke or subarachnoid or intra-cerebral hge
- Antihypertensive should be given for pts with blpr ≥**130/80** mmHg after they being stabilized following a stroke
- Combine**Diuretics&ACEinhibitor**
- Monitor for hypotensive symptoms due to failure of cerebral auto-regulation
  Withdrawal of antihypertensives
- Re-administer drug and, if necessary, **Phentolamine**, **Nitroprusside**, or **Labetalol**

Acute pulmonary edema
- **Nitroprusside** or **Nitroglycerin** with a loop **Diuretic** is of choice
- **Hydralazine**) or **Bblocker**) CI

Acute decompensated heart failure"ADHF"



Restrictfluids
- (<**2** L/d) in pts with moderate hyponatremia (<**130**meq/L) & volume overload

Diuresis
- For both systolic & diastolic HF
- Even without volume overload
- Exception ----- hypotension, cardiogenic shock
- **Furosemide** ------ **40** mg IV -------- increase dose to **80** mg "Pts with chronic diuretics"
- If inadequate response -------- salt water restriction & double dose
- If increases in serum creatinine appear to reflect intravascular volume depletion ----reduce or temporary DC diuretic or vasodilator therapy& consider inotropic therapy
- Pts with moderate to severe renal dysfunction and evidence of intravascular fluid overload ---- Continue to be treated with diuretics
- In the presence of severe fluid overload. If substantial congestion persists, diuresis cannot be achieved without an unacceptable degree of azotemia---- consider ultrafiltration or dialysis

Morphinesulfate
- For both systolic & diastolic HF"In selected pts

Vasodilators
- For both systolic & diastolic HF
- In the absence of symptomatic hypotension,----- Consider IV **nitroglycerine**, **Nitroprusside**+ diuretic
- Frequent blpr monitoring
- Dosage of vasodilator agents should be decreased if symptomatic hypotension develops
- Once symptomatic hypotension is resolved ----- Consider reintroduction in increasing doses

ACEinhibitors&Bblockers
- Should be avoidedIn systolic dysfunction,
- ACE inhibitors and B blockers "pts taking chronic medication & with no hypotension" may be useful in acute as well as chronic HF in pts with primarily diastolic dysfunction

Inotropicagents
- Not indicated in pts with diastolic dysfunction with preserved systolic function
- Manage*Cardiogenic shock"p 108 "*

General approach to patient with chest pain

ABCDE

- O2, IV access, cardiac monitor, and vital signs
- A 12-lead ECG and a blood sample for cardiac enzyme should be obtained if possible
- Evaluate the need for emergent care “MI, pneumothorax, aortic dissection, esophageal rupture, or acute abdomen”

History

Quality of pain
- Myocardial ischemia ---- squeezing, tightness, pr, constriction, strangling, burning, heart burn, fullness in the chest, a band-like sensation, knot in the center of the chest, lump in the throat, ache, heavy weight on chest (elephant sitting on chest), like a bra too tight, and toothache (when there is radiation to lower jaw
- Myocarditis can be pleuritic (can also mimic that of myocardial ischemia)

Region or location of pain
- Ischemic pain is a diffuse discomfort that may be difficult to localize
- Pain that localizes to a small area on the chest is more likely of chest wall or pleural origin

Radiation
- Myocardial ischemia --- neck, throat, lower jaw, teeth, upper extremity, or shoulder
- A wide extension of radiation increases the probability that it is due to MI
- Pain radiating to left arm is classically associated with coronary ischemia, radiation to right arm may be a particularly useful finding & Radiation to both arms is a strong predictor of AMI
- Acute cholecystitis can present with right shoulder pain, although concomitant right upper quadrant or epigastric pain is more typical than discomfort
- Pain that radiates between scapulae may be due to aortic dissection
- The pain of pericarditis typically radiates to one or both trapezius ridges
- Cervical radiculopathy may present with chest, upper back or upper extremity

Time course
- Pain of pneumothorax & aortic dissection or PE have abrupt onset “greatest at the start’
- Onset of ischemic pain is more often gradual with an increasing intensity over time
- A crescendo pattern of pain can also be caused by esophageal disease "Functional" or non-traumatic musculo-skeletal chest pain might have a much more vague onset

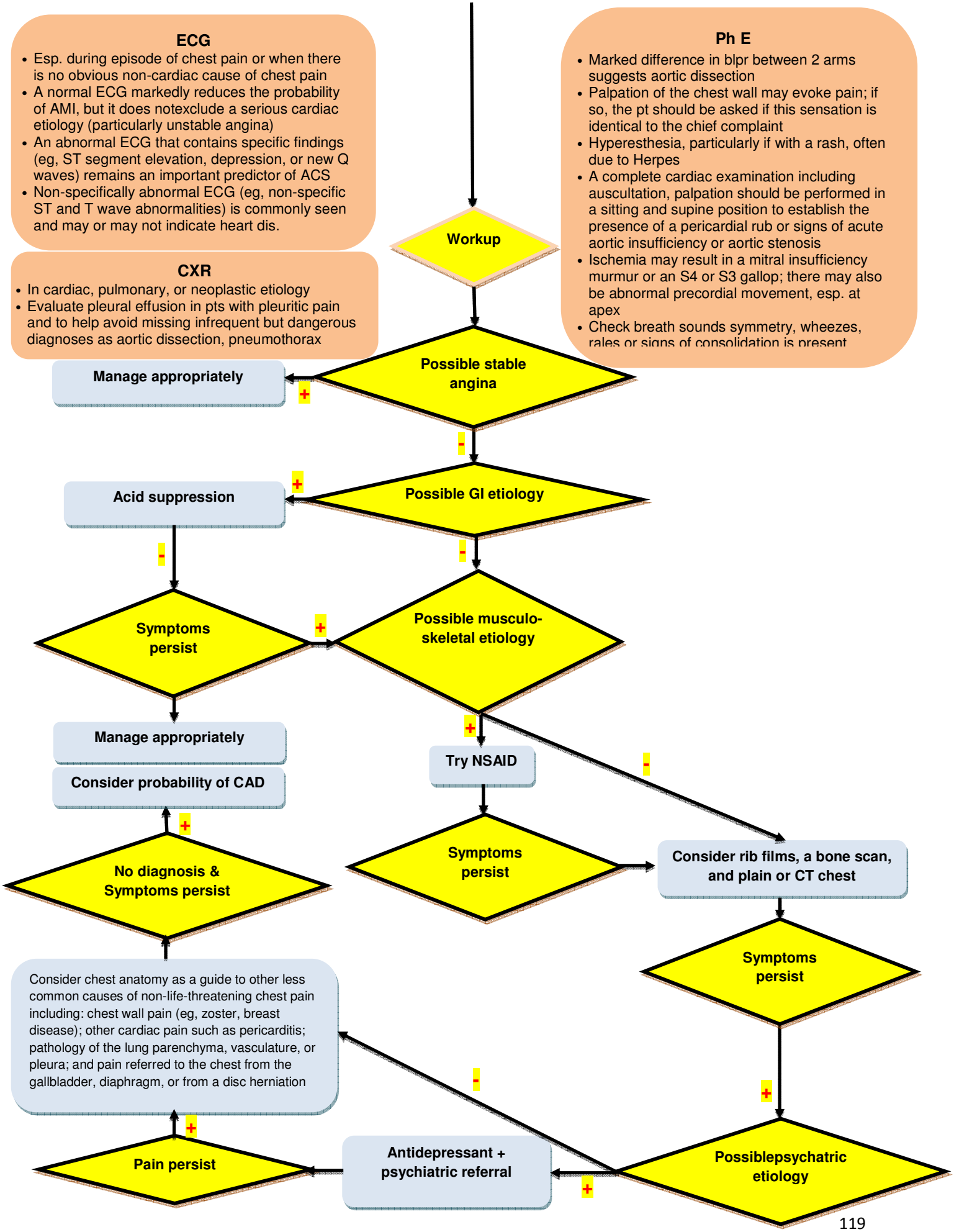
Duration
- Discomfort that lasts only for seconds or pain that is constant over ws is not ischemic
- Pain from myocardial ischemia generally lasts for a few min; it may be more prolonged in MI
- Myocardial ischemia may demonstrate a circadian pattern (more in morning).

Severity
- Not a useful predictor of the presence of coronary heart dis.

Provocation
- Discomfort that reliably occurs with eating is suggestive of upper GI dis.
- Postprandial chest pain may be due to GI or cardiac dis.
- Chest discomfort provoked by exertion is classic angina symptom “esophageal pain the same’
- Other factors that may provoke ischemic pain “cold, emotional stress, meals, or intercourse”
- Pain made worse by swallowing is likely of esophageal origin
- Body position or movement, as well as deep breathing, may exacerbate musculoskeletal pain
- Truly pleuritic chest pain is worsened by respiration and may be exacerbated when lying down
- Causes of pleuritic chest pain include PE, pneumothorax, pneumonia, and pleuro-pericarditis

Palliation
- Pain that is reliably and repeatedly palliated by antacids or food is likely gastro-esophageal
- Pain responds to SL Nitroglycerine is frequently have a cardiac etiology or esophageal spasm
- Relief of pain following "GI cocktail" does not reliably distinguish GI from ischemic chest pain
- Pain that abates with cessation of activity strongly suggests an ischemic origin
- Pain of pericarditis typically improves with sitting up and leaning forward

Associated symptoms
- Associated symptoms " Belching, a bad taste in the mouth, and difficult or painful swallowing "may not reliably distinguish between a cardiac and GI chest pain ‘up to 35 %’
- Vomiting may occur with myocardial ischemia, and GI problems as peptic ulcer, cholecystitis, and pancreatitis (DKA, which can be precipitated by AMI, is another cause of vomiting
- Diaphoresis is more frequently associated with MI than esophageal disease
- Exertional dyspnea may predate, is common when chest pain is due to myocardial ischemia
- Differential diagnosis of chest pain and cough includes infection, as ,CHF, PE, and neoplasm
- Cough, hoarseness, or wheezing may also be the result of gastro-esophageal reflux dis.
- Pts with ischemia can feel palpitations resulting from ventricular ectopy. AF is associated with chronic CHD” new onset isolated AF is uncommon in pts with AMI but common with PE”
- Pt with myocardial ischemia may describe presyncope “syncope with chest pain suggest aortic dissection, a hemodynamically significant PE, a ruptured abdominal aortic aneurysm, or critical aortic stenosis (particularly if the pt has a history of exertional dyspnea)


ECG
• Esp. during episode of chest pain or when there is no obvious non-cardiac cause of chest pain
• A normal ECG markedly reduces the probability of AMI, but it does notexclude a serious cardiac etiology (particularly unstable angina)
• An abnormal ECG that contains specific findings (eg, ST segment elevation, depression, or new Q waves) remains an important predictor of ACS
• Non-specifically abnormal ECG (eg, non-specific ST and T wave abnormalities) is commonly seen and may or may not indicate heart dis.
CXR
• In cardiac, pulmonary, or neoplastic etiology
• Evaluate pleural effusion in pts with pleuritic pain and to help avoid missing infrequent but dangerous diagnoses as aortic dissection, pneumothorax
Ph E
• Marked difference in blpr between 2 arms suggests aortic dissection
• Palpation of the chest wall may evoke pain; if so, the pt should be asked if this sensation is identical to the chief complaint
• Hyperesthesia, particularly if with a rash, often due to Herpes
• A complete cardiac examination including auscultation, palpation should be performed in a sitting and supine position to establish the presence of a pericardial rub or signs of acute aortic insufficiency or aortic stenosis
• Ischemia may result in a mitral insufficiency murmur or an S4 or S3 gallop; there may also be abnormal precordial movement, esp. at apex
• Check breath sounds symmetry, wheezes, rales or signs of consolidation is present
Workup
Possible stable angina
+
Manage appropriately
-
Possible GI etiology
+
Acid suppression
-
Symptoms persist
+
Possible musculo-skeletal etiology
-
+
Try NSAID
-
Symptoms persist
Consider rib films, a bone scan, and plain or CT chest
Symptoms persist
+
Possiblepsychatric etiology
+
Antidepressant + psychiatric referral
-
Pain persist
+
Consider chest anatomy as a guide to other less common causes of non-life-threatening chest pain including: chest wall pain (eg, zoster, breast disease); other cardiac pain such as pericarditis; pathology of the lung parenchyma, vasculature, or pleura; and pain referred to the chest from the gallbladder, diaphragm, or from a disc herniation
No diagnosis & Symptoms persist
+
Consider probability of CAD
Manage appropriately

Acute coronary syndrome "ACS"

Suspect

History:
- IHD

Symptoms
- Heaviness or tightness or indigestion-like discomfort in chest or upper abdomen, often radiates into throat, one or both arms (more commonly left), back or epigastrium & Some pts experience discomfort predominantly in one or more of these other areas rather than in the chest" & Sometimes it may be accompanied by belching "may be misinterpreted as indigestion"
- Usually sustained for at least **20**-**30** min, often longer in STEMI & NON-STEMI

Signs
- Non-specific" sweating, pallor & tachycardia"

Confirm

ECG& Cardiac enzymes
- Normal ECG **+** normal enzs. **(unstable angina)**
- Non- specific changes "T wave inversion" + normal enzs **(unstable angina)**
- ST segment depression **+** normal enzs**(non STE ACS)**
- ST segment**+** elevated enzymes**(STE ACS)**

❖ **Cardiac troponins (troponin T and troponin I)** are very sensitive and specific markers of cardiac injury& Elevated troponin level **6**-**8** hr after onset of pain indicates a higher risk of further coronary events in unstable angina &A combination of ST segment depression and raised troponin identifies a particularly high-risk group for subsequent MI & sudden death

❖ **CKmb** relatively low until **4**-**6** hs after symptom onset **"** a negative test in this period does not exclude infarction (some pts do not show a biomarker" elevation for 12 hrs)

❖ **Tropinins** onset 3-12 hrs peak 18-24 duration 36-48hrs& CKmb onset 3-12 hr peak 18-24 duration 10 ds

❖ In pts without a clear diagnosis but at risk for ACS, ECGs should be repeated at frequent intervals until the pt's chest pain resolves or a definitive diagnosis is made

Echo
- Has no role when diagnosis already apparent
- Assist in identifying or ruling out non-cardiac causes of chest pain & Can visualize RWMAs within seconds of a coronary artery occlusion & detect location & extent of an infarct & Diagnose RV infarction, acute VSD, MR

Immediate Manage

- **O2** given if arterial oxygen saturation <**90** %
- **IVaccess** and blood work obtained
- Sitting up **position**
- **Aspirin162** - **325** mg given
- **Nitrates**
  - SL NG **0.4 mg** / **5 min.** for 3 doses, after which an assessment of blpr and pain relief should guide the need for IV nitroglycerine
  - Nitrates are CI if **sildenafil** (**Viagra**), **vardenafil** (**Levitra**), or **tadalafil** (**Cialis** have been used in the last 24 hrs (or perhaps as long as 36 hrs with tadalafil)
  - Be cautious before giving nitrates in inferior MI with possible involvement of RV
- **Morphine** given (unless CI)
  - Administer IV morphine sulfate **2** - **4** mg, with increments of **2** - **8** mg, repeated / 5-15 min., to relief of chest pain and anxiety
- Anti-emetic, esp. if nausea is present

Detect site

- **Anterior or anteroseptal infarction**-------- **V1** - **4**
- **Inferior infarction** ----------**II**, **III**, and **aVF**
- **Lateral infarction** ---------- **V5**-**V6** and/or **I** and **aVL**
- **Posterior MI** -------- **V1**- **3**" ST segment depression & dominant R wave"
  - Confirmed by repeating ECG with posterior leads (**V8**, **V9** and **V10**) are placed in a horizontal line around the chest, continuing from **V6** (mid-axillary line) and **V7** (posterior axillary line). **V9** is placed to the left of the spine, V8 half way between **V7** and **V9** and **V10** to the right of the spine

Diagnose Rt infarct

- Suspected in posterior & inferior infarction
- Fluid-responsive hypotension and signs of high CVP without pulmonary congestion
- Right-sided precordial leads----- RV infarction
- 2-D**ECHO**

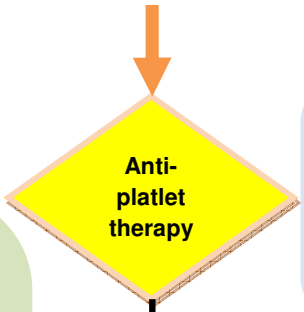
Re-perfusion therapy

- Maximum benefit if delivered within 1 h of onset
- 12 hrs after onset of chest pain of AMI, risks of reperfusion outweigh any small residual benefit
- Should not await the availability of results of cardiac biomarkers (**Level I**)

PCI
- Recommended method for reperfusion therapy in STEMI is **1ry** PCI, provided this can be achieved within **90** min. of 1st medical contact
- **May be 'rescue PCI'"** in case of failed thrombolytics & re-occlusion after thrombolytics

Thrombolytics
- 2nd choice "unavailable PCI or delayed > 90 min.
- The delay from pt arrival to administration of thrombolytics should be < 30 min.

- **Fluid resusc.:**Saline infusion ---- target "PCWP 15 mmHg". If not available, infuse 1-2 L saline ---- target "blpr and UOP and signs of pulmonary congestion"
- If volume loading does not improve hemodynamics or not tolerated---- inotropes
- **Avoid** "Nitrate"
- Afterload reducing agents or an intra-aortic balloon pump in LV dysfunction
- **Coronary reperfusion**
  - Early reperfusion is recommended "Successhigher with 1ry PCI"



Aspirin
- Administer **162** - **325** mg "chew and swallow as soon as possible to any pt with STEMI"

Clopidogrel
- In pts treated with primary PCI (**Level I**)
- **300** -**600** mg loading dose followed by **75** mg / d
- Given when aspirin is rarely CI

Thrombolytic "Streptokinase"

CI

Absolute
- Lack of verbal informed consent
- Active internal bleeding
- Recent TBI, major trauma or surgery within 3 ws
- Cerebro-vascular accident within 6 ms
- Refractory hypertension (>**200**/**100** mmHg)

Relative" should be considered in view of the potential clinical benefit and risk to each pt".
- Known bleeding diathesis
- Current use of anticoagulants (warfarin)
- Active peptic ulceration or other GI bleeding within 6 ms
- Prolonged CRP (> 10 min.) and/or traumatic resusc.
- Pregnancy. 1 w postpartum
- Diabetic proliferative retinopathy
- Non-compressible vascular puncture/injury
- Previous allergic reaction
- Advanced liver disease
- Active peptic ulcer disease

Follow up
- ECG at 1 and 4 hrs post STK/TNK
- Cardiac enzs 6, 12 and 24 hrs post infusion
- IF ST-elevation persists 1 hr post-TNK, contact cardiology regarding "rescue angioplasty"

B- blockers
- Administere to all pts without CI, irrespective of concomitant fibrinolytic therapy or performance of primary PCI
- **Metoprolol** can be given in **5** mg increments by slow IV administration (**5** mg over 1-2 min.), repeated / 5 min. for a total initial dose of **15** mg. Pts who tolerate this regimen should then receive oral therapy beginning 15 min. after the last IV dose (**25** - **50** mg / 6 hs for 48 hrs) followed by a maintenance dose of **100** mg twice daily
- IV **Atenolol** can be given in a **5** mg dose followed by another 5 mg 5 min. later. Pts who tolerate this regimen should then receive oral therapy beginning 1-2 hrs after last IV dose (**50** - **100** mg/day)
- **Esmolol** (**50** mcg/kg / min increase to **200** - **300** mcg/kg / min maximum) can be used if an ultra-short acting B blocker is required

Glycoprotein IIb/IIIa inhibitors
- Administer **Abciximab** as early as possible prior to PCI, with or without stent, in pts with STEMI (**Level IIa**)

IV nitroglycerin
- For ttt of persistent pain, CHF, or hypertension, IV nitroglycerincan be given, provided there are no CI
- Goal of therapy is a 10 % reduction in S blpr or a 30 % reduction in hypertensive pts
- Stop hypotension resulting from NG prevents administration of B- blockers, which are of greater proven benefit in STEMI

Electrolyte repletion
- Maintain serum **K** above **4**.0 meq/L in pts with an acute MI, and they recommend maintaining a serum **Mg** concentration >**2**.0 meq/L (2.4 mg/dL or 1 mmol/L)

Heparin *"see drug summaryP168"*
- Use UFH with STEMI undergoing percutaneous or surgical revascularization, and to pts undergoing thrombolysis with selective fibrinolytic agents(**LevelI**)
- LMWH might be considered (**Level IIb**) an alternative to UFH in pts receiving thrombolysis provided they are < 75 years of age and have no renal dysfunction

Aortic dissection

- The primary event is a tear in the aortic intima. Degeneration of the aortic media, or cystic medial necrosis, is felt to be a prerequisite for the development of non-traumatic aortic dissection
- Bl. passes into the aortic media through the tear, separating the intima from the surrounding media and/or adventitia, and creating a false lumen. It is uncertain whether the initiating event is a primary rupture of the intima with 2ndry dissection of the media, or hge within the media and subsequent rupture of the overlying intima
- Propagation of the dissection can occur both distal and proximal to the initial tear, involving branch vessels and the aortic valve and entering the pericardial space
- Such propagation is responsible for many of the associated clinical manifestations, including ischemia (coronary, cerebral, spinal, or visceral), aortic regurgitation, and cardiac tamponade

Suspect

- Severe, sharp or "tearing" posterior chest or back **Pain** (in dissection distal to Lt subclavian) or anterior chest pain (in ascending aortic dissection). Radiate anywhere in the thorax or abdomen. May be associated with syncope, a cerebro-vascular accident, MI, HF

Consult cardio-thoracic surgeon

Confirm

Involvement of ascending aorta — In addition to pain (chest more often than back or abdominal pain)

- Acute myocardial ischemia or MI, Cardiac tamponade, hemothorax, acute aortic insufficiency"diastolic murmer", considerable variation (>**20** mmHg) in S bl pr between the arms, neurologic deficits, including stroke or decreased conscious-ness, horner syndrome

Involvement of the descending aorta

- In addition to pain, a dissection that involves the descending aorta can lead to splanchnic ischemia, renal insufficiency, lower extremity ischemia, or focal neurologic deficits due to spinal artery involvement and spinal cord ischemia
- Multi-plane**TEE** at the bedside for pts who present with acute chest pain and/or are clinically unstable
- Hemodynamically unstable pts with a very strong suspicion of dissection can be emergently brought to the operating room and undergo TEE after induction of anesthesia as the chest is being prepared
- **MRI** is preferred in pts with chronic chest pain and in those who are hemodynamically stable, or are seen for follow-up of a chronic dissection
- **CTscan** with contrast is reserved for situations in which both TEE and MRI are unavailable or CI. As such, it is often indicated as an initial screening study in pts with suspected aortic dissection, especially in the emergency department setting where TEE and MRI are less available, especially after hrs. If CT is equivocal, or further delineation of the dissection is needed, TEE or MRI are indicated
- **Aortography** is used when ascending aortic dissection is strongly suspected, but non-invasivetests are unavailable or inconclusive
- Coronary **angiography** is generally safe in stable pts, although the delay to surgical invention for ascending dissections should be minimized. Generally attempted in all pts with a prior history or angina or MI, pts older than 60 years of age, and pts with multiple risk factors for coronary dis.

Manage

Surgical management

- Acute dissections involving the ascending aorta are considered surgical emergencies
- Dissections of descending aorta are treated medically unless the pt demonstrates progressive dissection or continued hge into pleural or retro-peritoneal space

Medical management

- Pain control with**Morphine**
- Pts who are hemodynamically unstable or with airway compromise should be intubated
- **bl pr control**
  - Target -----S bl pr **100** - **120** mmHg or the lowest tolerated level, decreaseLV contractionvelocity
  - Initial ttt---- IV **B- blocker** " reduce HR **≤60** b/min
    - **Propranolol** (**1**-**10** mg load, then**3** mg/h)
    - **Labetalol**(**20** mg initially, followed by **20** - **80** mg / 10 min. to a total dose of **300** mg) or as an infusion (**0.5** - : **2** mg/min)
    - **Esmolol:** preferred in acute setting, esp. pts intolerant of B- blockers"eg.,, asthma, HF"
  - **Verapamil** or **Diltiazem** are alternatives in pts who cannot tolerate B- blockers
  - **Hydralazine**, should be avoided
  - If, after B- blockade, the S bl pr remains above **100** mmHg with good mentation and renal function, IV **Na nitroprusside** should be added
    - The initial dose is **0.25 - 0.5** mcg/kg / min.
    - Should not be used without first controlling the HR with B- blockade since vasodilation alone induces reflex activation of the sympathetic nervous system leading to enhanced ventricular contraction and increased aortic wall shear stress
    - Pts should be continuously monitored, preferably using an intra-arterial cannula from the arm with the highest auscultatory pr.
  - The pt can be switched to oral B- blocker therapy after HR control has been achieved
- **Hypotensive pts**
  - Exclude hemopericardium with tamponade, or HF before volume is administered
  - Inotropic agents should be avoided
  - Tamponade --- percutaneous pericardio-centesis can accelerate bleeding and shock

Respiratory disorders

General approach to a patient withpproach to respiratory distress

ABCD E

- O2, IV line, Cardiac monitor, oximetry, vital signs, Airway equipment is brought to the bedside
- **Detect life threatening conditions**(pneumothorax, tamponade airway obstruction)
- Ventilate as indicated

Worku p

PhE

RR
- Pts may have a fast, normal, or slow RR "eg.,pts with a PE may have a RR in the normal range"

Pulse oximetry
- Inaccurate in hypothermia, shock, CO poisoning

Abnormal breath sounds
- Inspiratory stridor ----supra-gloticobstruction (eg, FB, epiglottitis, angioedema)
- Expiratory or mixed stridor ----infra-gloticobstruction (eg, croup, bacterial tracheitis, FB)
- Wheezing ----- obstruction below the level of the trachea and is found with asthma, anaphylaxis, a FB in a mainstem bronchus, ADHF, tumor
- Crackles (rales) ----alveolar fluid as pneumonia or ADHF**.** Absence does not rule out diagnosis
- Diminished breath sounds ----conditions prevent air from entering the lungs (severe COPD, severe asthma, pneumothorax, tension pneumothorax)

Cardiovascular signs
- Heart murmurs ----- acute decompensated or diseased or compromised cardiac valves
- S3 heart sound ------ LV systolic dysfunction, especially in the setting of ADHF
- S4 heart sound ----- LV dysfunction and may be severe hypertension, aortic stenosis, hypertrophic cardiomyopathy, IHD, or acute MR
- Muffled or distant heart sounds ----- cardiac tamponade, but must be interpreted in the context of the overall clinical setting
- Elevated JVP ----- ADHF or cardiac tamponade. It can be assessed by observing jugular venous distension or examining hepato-jugular reflux
- Pulsusparadoxus ---compromisedright heart function (severe asthma, PE, or tamponade)

Skin
- Discoloration ---- hypoxia or poor perfusion, signs of an allergic reaction, and evidence of trauma

Extremities
- Peripheral edema --- not occur with acute left HF, but if present suggests ADHF

History
- **General history;** events leading to episode
- **Past history;** new or recurring problem. Ask about preexisting medical conditions, as asthma
- **Prior intubation**; Have a higher risk for severe dis. and the need for subsequent intubation
- **Time course**; developed suddenly or gradually
- **Severity;**Pts with acute exacerbations of asthma, COPD and ADHF tend to rate their distress as moderately severe, while those with normal pregnancy, neuromuscular disorders or PE tend to rate their distress as only moderate
- **Chest pain**; sizable minority with ACS or PE complain of dyspnea alone
- **Trauma**
- **Fever** : hypersensitivity oraspiration pneumonitis
- **Paroxysmal nocturnal dyspnea (PND):** "Pts with COPD may present with PND "
- **Hemoptysis;** PE, TB, and malignancy, when tumors erode into a vascular structure
- **Cough and sputum**; Purulent sputum suggests pneumonia and white or pink frothy sputum suggests ADHF, frankly bloody sputum suggests inf. (eg, tuberculosis) or pulmonary hge (eg, PE or malignancy). & A non-productive cough is a non-specific symptom, and may be associated with asthma, HF, respiratory inf., or PE
- **Medications &Tobaco**
- **Psychiatric conditions**; may be in a sizable minority < 40 years with no medical conditions

CXR
- Diagnose acute heart failure&pleural effusions & pneumonia&pneumothorax&COPD and asthma

ABGs
- The role in diagnosis and ttt is limited by using transcutaneous pulse oximetry and venous bl. Gas

CO2 "End-tidal CO2 " monitoring
- Provides dynamic monitoring of ventilatory status

D-Dimer
- Ruled out Pts at low risk for PE if -ve
- It is NOT appropriate to screen high risk pts. Pts with malignancy or recent surgery and elderly pts are more likely to be false high

Negative inspiratory pressure (NIF) & FVC
- Assess dyspneicpts with possible neuromuscular disease or musculoskeletal diseases
- If the **NIF** is <**30** cm H2O or the **FVC** is <**20** mL/kg, the pt should be admitted to ICU in anticipation of MV

Cardiacbiomarkers
- Serial measurements to rule out an ACS
- Limited specificity(may be elevated in PE, sepsis, pericarditis, myocarditis, warfarin use, renal failure

Brain natriuretic peptide "BNP"
- Helpful when diagnosis of ADHF is in question
- Not helpful if used indiscriminately in acute dyspnea
- A BNP of <**100**pg/mL has a negative predictive value of > 90 % for ADHF
- A BNP >**500**pg/mL strongly suggests ADHF, with a positive predictive value > 90 %.
- 100 - 500 pg/mL cannot differentiate between ADHF and other causes of elevated BNP
- False +ve----PE, renal and liver failure, critical illness, corpulmonale, pulmonary hypertension

Multi-detector CT (MDCT)Scan and VQ scan
- Diagnose PE, pneumonia, and pulmonary edema
- **VQscanning**used in pts unsuitable

Hypoxemia

Suspect
- Cyanosed pt
- SPO2 <90%

Confirm

ABGs
- **Absolute hypoxemia**; PaO2 <**60** mmHg or
- **Corrected hypoxemia**; PaO2 <**5 * FiO2**

A-a gradient

High

Hypoxemia "V/Q mismatching"

CXR

Workup

History & PhE & CT Chest &Consultation as indicated

Manage

- ttt **underlying cause**
- **O2 therapy**
- **Ventilate** as indicated
- Consider the following strategies to improve hypoxemia on a ventilator
  - PEEP
  - High I: E up to inverse ratio
  - Increase FiO2--- Last step to avoid O2 toxicity
  - Permissive hypoxemia "**88**% SPO2' in severe cases of ARDS

Infiltrate

Focal

Consider
- Pnemonia
- Atelectasis

Bilateral

Consider
- Cardiogenic pulmonary edema
- ARDS
- Pnemonia

Normal

Consider
- PE

Normal

Alveolar hypoventilation "hypercapnea"

Acute respiratory acidosis algorithm *"p34"*

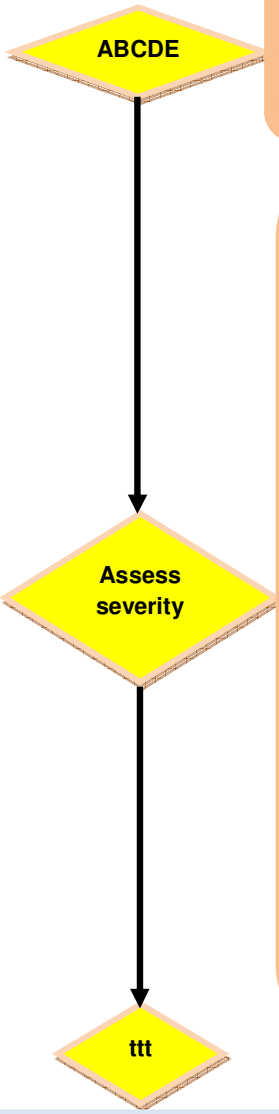
A-a gradient

PaO2 (partial pressure of O2 in the artery), obtained from the ABGs **PAO2** (partial pressure of O2 in the alveoli), obtained from **Alveolar gas equation**

PAO2 =PiO2 (FiO2 * (760-47)–PaCO2/R" 0.8 ")

- **R'respiratoryquotation** = O2 consumption/ CO2 production = **0.8**
- In healthy subjects breathing room air at sea level = **10** mmHg
- It increases **5-7** mm Hg / 10% increase in FiO2
- Mean airway pr should be added to the barometric pressure during PPV

Acute exacerbation of asthma



- Give **O2** ----maintain SaO2 92 % (>95 % in pregnancy)
- Establish IV **access**; may give bolus of NS for prolonged episode to replace insensible losses
- **Ventilate**As indicated

Clinical danger signs

- Use of accessory muscles of respiration; brief, fragmented speech; inability to lie supine; profound diaphoresis; agitation; severe symptoms that fail to improve with initial ttt
- Life-threatening airway obstruction can still occur when these signs are not present
- Inability to maintain respiratory effort, cyanosis, and depressed mental status portend imminent respiratory arrest

Spirometry

- PEFR <**40** % predicted (or <200 L/min. in most adults) indicates severe obstruction
- Pts in severe distress are often unable to perform peak flow tests

ABG

- Severe hypoxemia (eg, SpO2 **95** % despite high flow O2 ttt by non-rebreather mask) portends imminent respiratory arrest
- Continuously monitor pulse oximetry
- Hypercapnia (a sign of impending RF) usually does not occur unless a PEFR <**25** % of normal (generally <100 - 150 L / min.) is present

CXR

- Generally unhelpful
- Obtain if complications suspected (eg, pneumonia, pneumothorax), diagnosis is in doubt, or pt is high-risk (eg, IV drug abuser, immunosuppressed, COPD, CHF)

haled B- agonist

- Give nebulized **Albuterol2.5** mg /20 min. for 3 doses
- Alternatively, administer **10-15** mg by continuous nebulization over 1 hr

Ipratropium bromide

- Give **500** mcg by nebulization / 20 min. for 3 doses, or as needed for up to 3 hrs

Mgsulfate

- Give **2** g IV over 20 min. for life-threatening exacerbation s((ie, impending intubation for RF) or those whose exacerbation remains severe after 1 hr of intensive conventional therapy)

Epinephrine

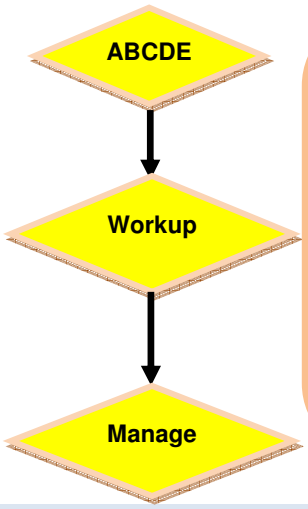
- Give **0.2 - 0.5** mL of 1:1000 solution by SC injection for severe asthma unresponsive to standard therapies

Corticosteroids

Corticosteroids

- Start early systemic gluco-corticoids for all pts who have a moderate (PEFR<70 % of baseline) or severe exa-cerbation (PIF<**40** % of baseline), or in whom inhaled short-acting B- agonists do not fully correct decrement
- IV glucocorticoids should be given to pts who present with impending or actual respiratory arrest, or pts who are intolerant of oral glucocorticoids (seroids may be given IM or orally if IV access is unavailable)
- Administer **60** - **125** mg of IV **Methylprednisolone** / 6 - 8 hrs) in life-threatening asthma (smaller doses may be equally effective "eg, **40** - **60** mg / 12 hrs"). or **Prednisone40**-**60** mg po; alternatives include: **Dexamethasone6**-**10** mg IV or **Hydrocortisone150**-**200** mg IV
- Transit to oral glucocorticoids when the pt can tolerate and absorb oral medication
- Tapering oral glucocorticoids is not necessary if duration of glucocorticoid ttt is <3 ws

Acute COPD exacerbation



Underlying Cause

- 50 - 60 % of exacerbations are due to respiratory inf. (mostly bacterial and viral)
- Others (myocardial ischemia, HF, aspiration, or PE)

- History, PhE, CXR, and routine lab.
- ABG to assess the severity, establish a baseline
- **Sputum gram stain & culture**
  - Advantageous if pts whose exacerbations are due to bacterial inf.
  - Risk factors for Pseudomonas "recent hospitalization (≥ 2 d' duration during the past 90 ds), frequent administration of abs (≥ 4 courses within the past year), severe COPD (FEV1 <**50** % of predicted), isolation of P. aeruginosa during a previous exacerbation, and colonization during a stable period
- Sputum purulence
  - Important not absolute, indicator of bacterial inf.

Glucocoticoids

- Administer systemic Glucocorticoids (**Level 1A**)
  - Inhaled glucocorticoids should not be a substitute
- Oral glucocorticoids are rapidly absorbed =efficacy is comparable to that with IV therapy
- IV glucocorticoids should be given to pts who present with a severe exacerbation, who respond poorly to oral glucocorticoids, who are vomiting, or who may have impaired absorption due to splanchnic hypoperfusion (shock)
- Administer IV -- **Methylprednisolone** (**60**-**125** mg, 2-4 times daily) Oral ---**Prednisone** (**40**-**60** mg orally/d "Lower doses (eg, equivalent of **30** - **40** mg of Prednisone") may be equally effective to higher doses and safe
- Should be treated for 7 - 10 ds
- Taper over about 7 ds as a trial to see if continued gluco-corticoid therapy is required
- Tapering solely because of concerns about adrenal suppression is not necessary if duration of therapy is < 3 ws

tttUnderlying cause

O2 therapy

- Adequate oxygenation must be assured, even if it leads to acute hypercapnia
- Target"PaO2 60-70 mmHg, SPO2 90-94% (**Level 2C**)
- Inability to correct hypoxemia with a relatively low FiO2 should prompt consideration of PE, ARDS, pulmonary edema, or severe pneumonia as the cause of RF

B- Adrenergic agonists

Anti-cholinergics

- **Ipratropium500** mcg by nebulizer / 4 hrs as needed

Glucocoticoids

Ab therapy

- Is not initiated in pts whose exacerbation is mild" not requiring ventilatory assistance"
- Duration is usually **3** - **7** ds, depending upon the response to therapy
- Started on parenteral abs, switched to oral regimen when able to take medications orally

Antiviral therapy

- Pts whose exacerbation was triggered by influenza virus and who are diagnosed within 48 hrsof the onset of influenza symptoms should be treated with **oseltamivir** (75 mg orally twice daily)
- **Zanamivir** is also effective in the ttt of influenza but is CI in this pt population due to the risk of airway reactivity

Mucoactive agents, mechanical techniques to augment sputum clearance, and methylxanthines

- Have not been shown to confer benefit for patients with a COPD exacerbation

B adrenergic agonists

- Administer inhaled short-acting B- adrenergic agonist and an inhaled short-acting Anti-cholinergic agent, rather than either medication alone (**Level 1B**)
- **Albuterol2.5** mg (diluted to a total of 3 mL) by nebulizer / 1 - 4 hrs as needed
- Continuous nebulized B-agonist has no advantage
- SC injection if inhaled administration is not possible
- Parenteral use results in greater inotropic and chrono-tropic effects (may cause arrhythmias or ischemia in susceptibles)

Acute respiratory distress syndrome "ARDS"

Berlin definition of ARDS

- **Timing**: Within 1 w of a known clinical insult or new or worsening respiratory symptoms
- Chest **Imaging**; Bilateral opacities – not fully explained by effusion, lobar/lung collapse, or nodules
- Orgin of **Edema**: Respiratory failure not fully explained by cardiac failure or fluid overload. Need objective assessment "ECCHO"to exclude hydrostatic edema if no risk factor present
- **Oxygenation**:
  Mild ---**200**- **300**mmHg with PEEP ≥5 cmH2O
  Moderate ----**100**- **200**mmHg with PEEP≥ 5 cmH2O
  Severe ----<**100**mmHg with PEEP ≥5 cmH2O

Precipitators

Sepsis - Aspiration - Infectious pneumonia - Severe trauma - Surface burns - Multiple blood transfusions -Leukoagglutin reactions - Pancreatitis - Drug overdose - Near drowning - Smoke inhalation - Cardiopulmonary bypass - Pulmonary contusion - Multiple fractures - Following upper airway obstruction -Following bone marrow transplantation - Drug reaction - Venous air embolism - Amniotic fluid embolism - Neurogenic pulmonary edema - Acute eosinophilic pneumonia
* Bronchiolitis obliterans organizing pneumonia (BOOP)
* Miliary tuberculosis*

Suspect

- **Precipitators** "e.g. sepsis"
- Typical onset--- **48**-**72** hrs
- Tachypnea, refractory hypoxemia

Confirm

- **Ph E** ---- tachycardia, cyanosis, tachypnea, and either normal auscultation, or diffuse rales
- **ABGs** ---- respiratory alkalosis, hypoxemia, elevated alveolar-arterial O2 gradient
- **Lab**. ---- Related to the underlying risk factors "e.g. "leukocytosis, DIC, and lactic acidosis"
- **CXR** ---- Typically has bilateral alveolar infiltrates
- **CT** ------ Reveals patchy abnormalities that are denser in dependent lung zones

Exclude

- **Cardiogenic pulmonary edema ---- PNB"100** pg/mL favors ALI/ARDS"**, TTE** "Normal EF"
- **Pnemonia** ---- Pneumonia is characterized by cough, fever, and dyspnea. Aerobic bacteria, Legionella pneumophila, Pneumocystis jirovecii (previously called Pneumocystis carinii), respiratory viruses, or viral inclusion bodies can frequently be identified in sputum or BAL specimens
- **Diffuse alveolar hge** may be associated with a large, otherwise unexplained drop in Hgb and Ht. While hemoptysis may be minimal or absent, bronchoscopy often reveals frothy bloody secretions throughout the airways and invariably detects an increasing amount of RBCs in serial BAL specimens
- **Cancer** ---- Can disseminate through lungs so rapidly that the ensuing RF may be mistaken for ARDS. This is most often due to lymphoma or acute leukemia, but lymphangitic spread of solid tumors occasionally behaves this way. Cytological preparation of bronchoscopic specimens eg, brushings, lavage) may reveal malignant cells
- **MiliaryTB---**Considered when ARDS develops in pts who are hospitalized because of a systemic febrile illness. The diagnosis is made by recovery of acid-fast bacilli from BAL fluid

Manage

Supportive

- **GIT**, **DVTprophylaxis**, **Nutritionsupport**, **sedation**, **NMblockade**, **prevention**, **managementofVAP**, **glycemiccontrol**, **hemodynamicmonitoring** "Swan ganz catheter not routinely done"
- Conservative strategy of **Fluidmanagement** is warranted, as long as hypotension and organ hypoperfusion can be avoided. It is reasonable to target a CVP of <4 mmHg
- **PRBCs** can be withheld until the Hb<**7** g/dL, unless there are alternative reasons for transfusion
- No benefit from **Inotropes** in normal cardiac function
- Decrease O2 consumption "antipyretics, sedation, NMB

Novel

Corticosteroids

- Have no role in prevention and late phase of ARDS
- There is silver lining in early ARDS using these agents. There is distinct advantage in improving organ function score, lung injury score, and oxygenation which result in reduction in duration of MV requirement

Fat embolism syndrome"FES"

Suspect

- **Presence of predisposing factors**
  - Trauma (long bone fractures "closed > open " is the most common cause, pelvic fractures, soft tissue injuries, burns, liposuction)
  - Non- trauma (pancreatitis, diabetes, osteomyelitis)
- Presence of respiratory insufficiency, neurologic impairment, and a petechial rash
- Onset **24** - **72**hr after the initial insult (rarely, as early as **12**hr or as late as **2**ws after the event)

Confirm

- No **laboratory** test is sufficiently sensitive or specific to be diagnostically useful
- **CXR**are normal in the majority

Prevent

- Early immobilization of fractures
- Operative correction rather than conservative management of fractures (ie, traction alone)
- **Corticosteroid**
  - Controversial
  - Give prophylactic steroid therapy only to those pts at high risk for FES, eg, those with long bone or pelvic fractures, especially closed ones
  - **Methylprednisolone1.5** mg/kg IV can be administered / 8 hrs for 6 doses
- Limit elevations in intra-osseous pr. during orthopedic procedures in order to reduce intra-vasation of intra-medullary fat and other debris
- Use of cementless fixation of hip prostheses and un-reamed intra-medullary femoral shaft stabilization

ttt

- **Supportive** care is the mainstay of therapy for clinically apparent FES
- Do not start **Steroid** therapy once FES is established

Air embolism

- Air embolism is an uncommon, but potentially catastrophic, event that occurs as a consequence of entry of air into vasculature. It can be venous or arterial
- It requires direct communication between a source of air and vasculature, and a pr gradient favoring passage of air
- Minor cases of air embolism are commonand cause minimal or no

Pathophysiology

- If the capacity of the lung to filter microbubbles of air from the venous circulation can be exceeded. When this capacity is exceeded, air may pass through the pulmonary capillaries, enter arterial circulation, and cause arterial air embolization with organ ischemia. It is estimated that **300**-**500** mL of gas introduced at a rate of **100** mL/sec is a fatal dose for humans. This flow rate can be attained through a 14-gauge catheter with a pr gradient of only 5 cm H2O
- Arterial air embolism may be due to either direct introduction of air into arterial system or paradoxical embolization through a septal defect, patent foramen ovale, or pulmonary arterial-venous malformation. In pts with a left-to-right shunt, venous air embolization into the pulmonary circulation can raise right heart prs and reverse the direction of the shunt, allowing paradoxical embolism to occur. Air in the arterial circulation can occlude the microcirculation and cause ischemic damage to end organs, as brain, spinal cord, heart, and skin. The organs may also be damaged secondarily by the release of inflammatory mediators and O2 free radicals. In addition, there may be **Hemodynamic complications** — Large bubbles obstruct pulmonary outflow tract (air lock) ---diminishes bl flow from right heart, increaseCVP, decrease pulmonary arterial pr, and decrease systemic arterial pr. Smaller bubbles lodge within pulmonary arterioles or pulmonary microcirculation, directly impeding blflow and inducing vasoconstriction---- increase pulmonary vascular resistance, increase pulmonary arterial pr, and increase right ventricular pr. There may be an initial brief increase in the COPand systemic arterial pr due to tachycardia followed by a decrease in COPand systemic arterial pr, and myocardial ischemia due to hypoxia, right ventricular overload, and/or air emboli to the coronary arterial circulation, and **Pulmonary complications** — Bubbles in pulmonary microcirculation --- local endothelial damage and accumulation of neutrophils, platelets, fibrin, and lipid droplets at the gas-fluid interface. Additional endothelial damage ---- non-cardiogenic pulmonary edema, bronchoconstriction, hypoxemia (alveolar flooding and ventilation-perfusion mismatching), increased physiologic dead space (with a rise in PaCO2 if ventilation is held constant), decreased lung compliance (2ndry to pulmonary edema), and increased airway resistance

Suspect

Sudden onset of cardiopulmonary or neurologic decompensation in a **predisposed pt**

DD

- Acute pulmonary decompensation (PE, pneumothorax, bronchospasm, pulmonary edema)
- Acute cardiovasculardecompensation (hypovolemia, cardiogenic shock, MI, septic shock)
- Acute neurological decompensation (cerebral hypoperfusion, stroke,SAH, hypoxic brain injury, TBI, and metabolic disorders)

Confirm

- Diagnosis of exclusion, requiring appropriate clinical setting and exclusion of other life-threatening processes
- The following tests provide supportive evidence
  - **ECG**---- sinus tachycardia, right heart strain , non-specific ST-segment and T-wavechanges, and acute ischemia or infarction
  - **CXR**---- usually normal
  - **ABGs**--hypoxemia"may severe", hypercapnea
  - **Lab** – thrombocytopenia, high CK
  - **Echo** ---- detect emboli in right ventricle
  - **V/Qscan**&**Angio** ---resolve within 24 hrs
  - **CTscan** --- detect large emboli

Prevent

- **MV**----- minimize airway pr
- **CVC** ----ttt hypovolemia prior to placement, occlude hub of catheter during insertion, keepall connections to a central line closed and locked when not in use, and place pt supine and do Valsalva or exhale during CVC removal

ttt

- **Supportive----MV&**High flow supplemental **O2 & Vasopressor &**
- **Prevent further emboli; Venous----** place pt into left lateral decubitus position, Trendelenburg position, or left lateral decubitus head down position &**Arterial** ---place pt supine
- **Restoration of circulation** ---- If above positioning fails ---- chest compressions "force air out of pulmonary outflow tract and into smaller pulmonary vessels, improving forward blood flow"
- **Removal of embolized air** — from right ventricle via an already in place CVC or per-cutaneously introduced needle, of limited benefit because the volume recovered **≤20** mL
- Pts with evidence of cardio-pulmonary compromise orneurologic deficits ---- **HBO**

Predisposed pt

Surgical procedures

Surgical incision is superior to heart at a distance that is greater than CVP

- Neurosurgery (craniotomy, shunt)
- Otolaryngological procedures
- Orthopedic surgery (arthroscopy)
- Ob-Gyn procedures(hysteroscopy/laparoscopy, CS)
- Cardiothoracic surgery (cardiopulmonarybypass, lung resection, transplantation)

IV catheterization

- CVC& Hemodialysis
- CABG/angioplasty
- Pacemaker &Radiofrequency ablation

Radiologic procedures

- IV contrast injection &Arthrography

Trauma

- Head and neck injuries
- Penetrating and blunt chest trauma
- Blunt abdominal trauma

PPV

Decompression sickness

Massive hemoptysis

Generally used to describe the expectoration of a large amount of blood and/or a rapid rate of bleeding, although the precise thresholds that constitute massive hemoptysis are controversial.massive hemoptysis defined as **≥500** mL of expectorated blood over a 24 hr period or bleeding at a rate ≥ **100** mL/hr

Identify bleeding site

- May be difficult
- Occasionally, on the bleeding side there is a history of lung disease or there may be a gurgling sound that can be auscultated, or an abnormal sensation
- Many signs are misleading because they occur away from the bleeding site “eg, upper lobe hge may manifest as wheezing, rhonchi, or air space disease in the lower lobe, due to accumulation of blood in the lower lobe with gravitational pooling

Initial management

- Placepts immediately with presumed bleeding lung in the dependent position
- Establish a **patent airway** using
  Single lumen ETT
  - Insert into mainstem bronchus of the non-bleeding lung.The right upper lobe bronchus may become blocked
  Double-lumen ETT
  - MV permits ventilation of both lungs & preventing aspiration from one lung to the other
  - Recommended only for pts who are exsanguinating and/or asphyxiating from their bleeding, when no other approaches are possible, due to its practical limitations (difficult to insert& maintain, pts need to be paralyzed, and easily obstructed ”narrow lumen”)
- **Ventilate** as indicated
- Optimize cardiovascular function
- Volume replacement:Crystalloid first, blood products are an appropriate alternative in coagulopathic, anemic, ptsand/or bleeding rapidly
  - Arrhythmias are best managed with restoring adequate gas exchange

Cardio-thoracic consultation

Bleeding control

- Rapidly reverse known or suspected coagulation abnormalities
- Flexible bronchoscopy ----- assess & control bleeding & advised overarterio-graphic embolization & surgery (**level 2C**)
- Rigid bronchoscopy ----- accepted alternative if flexible bronchoscopy is either inadequate or insufficient “It has greater suctioning capacity that may provide superior visualization.
  - Bronchoscopic strategies to control pulmonary hge include balloon tamponade, iced saline lavage, administration of a topical vasoconstrictor or a topical coagulant, laser therapy, and electro-cautery
- If bronchoscopy fails to identify the cause & bleeding has ceased ----- do CT chest with high-resolution cuts
- For pts with massive hemoptysis who continue to bleed despite a flexible bronchoscopic&stable enough to leave the ICU ----- doArterio-graphic embolization rather than surgery during active bleeding episodes (**Grade 2C**)
- Pts who continue to bleed despite both a flexible bronchoscopic intervention and arterio-graphic embolization ---- do another attempt of rigidbronchoscopic bleeding control ----
  - If bleeding cannot be controlled via rigid bronchoscopy, surgery may be the best

Aspiration pnemonia

- Aspiration pneumonia refers to the pulmonary consequences resulting from abnormal entry of fluid, particulate exogenous substances, or endogenous secretions into lower airways
- There are usually 2 requirements to produce aspiration pneumonia:
  - Compromise in usual defenses that protect lower airways including glottic closure, cough reflex, and other clearing mechanisms
  - An inoculum deleterious to the lower airways by a direct toxic effect, stimulation of an inflammatory process from a large enough bacterial inoculum, or obstruction due to a sufficient volume of material or particulate matter
- Most pneumonia arises following the aspiration of less virulent micro-organisms which are common constituents of the normal oral or nasopharyngeal flora in a susceptible host prone to aspiration, primarily anaerobes.The 3 syndromes that are most frequently seen clinically and best studied are **chemical pneumonitis, bacterial infection,** and **airway obstruction**. Although there may occasionally be overlap and inability to classify individual cases, this classification is essential to an understanding of aspiration pneumonia
- The term chemical pneumonitis refers to the aspiration of substances that are toxic to the lower airways, independent of bacterial infection. The prototype and best studied clinical example is chemical pneumonitis associated with gastric acid aspiration

Suspect → **Respiratory distress in a predisposed pt**

Confirm

Chemical pneumonitis "inoculum = **acids**"
- Abrupt onset of dyspnea
- low-grade fever
- Diffuse crackles on lung auscultation
- Severe hypoxemia
- CXR; infiltrates typically appear within 2 hrsinvolve dependent segments "in upright position are lower lobes&in recumbent position are the superior segments of lower lobes and posterior segments of upper lobes
- Bronchoscopy---- erythema of bronchi

Bacterial inf." inoculum = **Oropharyngeal bacteria"**
- Insidious onset, some have abrupt onset suggestive of a pyogenic pneumonia, but without chills
- Cough, fever, purulent sputum
- Complications " lung abscess, and empyema"
- CXR ; infiltrate involving dependent segment or lobe ± cavitation
- Putrid diagnostic of anaerobic inf

Mechanical Obstruction
- Acute dyspnea, cyanosis ± apnea&Pulmonary edema "inoculum = inert fluids"
- Ranging from acute apnea and rapid death to irritative chronic cough ± recurrent inf. Depends on level of obstruction "inoculum +particulate matter"

Predisposed pt

- Reduced consciousness, compromising cough reflex and glottic closure
- Dysphagia from neurologic deficits
- upper GITdisorders including esophageal dis., surgery involving the upper airways or esophagus, and gastric reflux
- Mechanical disruption of glottic closure or cardiac sphincter due to tracheostomy, intub., bronchoscopy
- Miscellaneous --- protracted vomiting, large volume tube feedings, feeding gastrostomy

ttt

- **Ventilate**as indicated
- Pts with an observed aspiration --- immediate tracheal suction to clear fluids and particulate matter ----- will not protect the lungs from chemical injury
- Witnessed aspiration ---- Empiric antimicrobial ,if no infiltrates develop after 48 -72 hrs--- stop ab
- When anerobes are suspected----- ***clindamycin*** (**600** mg IV /8 hrs followed by **300** mg orally 4 times daily or **450** mg orally /8 hrs) as first-line therapy. Alternative are ---- ***amoxicillin-clavulanate*** (**875** mg /12 hrs); or ***metronidazole*** (**500** mg orally or IV /8 hrs) + amoxicillin (**500** mg orally /8 hrs) or ***penicillin*** G (**1**-**2** million units IV /4-6 hrs)
- For nosocomial pneumonia ---- ***carbapenem***or ***piperacillin-tazobactam***
- The duration of ab---- is arbitrary and not well studied. The usual duration of therapy for cases that are not complicated by cavitation or empyema is **7**- **10** ds


Neurologic disorders

General approach to a patient with disturbed conscious level

History: From witnesses, friends or family members, and old hospital chart
- What was the time course of the loss of consciousness?
- Did focal signs or symptoms precede coma?
- What recent illness has the pt had?
- What prescription or non-prescription drugs are used?

Seeup-todate for details

Ph E
- **General**: Vital signs, breathing pattern, and skin lesion
- **Neurologic** : GCS, motor exam, posture, pupils, cranial nerves, eye movement, reflexes

Seeuptodate for details

Lab
- CBC Glucose S electrolyes BUN, creatinine
- PT, PTT ABG LFTs
- Drug screen
- Adrenal and thyroid function tests
- Blood cultures
- Blood smear: screen for TTP or DIC
- CO-Hgb if CO poisoning is suggested
- Serum drug concentrations for specific drugs

Neuro-imaging
- Prioritize emergent if focal neurologic signs, papilledema, or fever
- CT is inferior to MRI for detecting abnormalities in pts with HZ encephalitis, early ischemic strokes (esp., brainstem), multiple small hge or white matter tract disruption associated with traumatic diffuse axonal injury, anoxic-ischemic damage from cardiac arrest
- MRI takes longer time than CT, requires the pt to be farther from monitoring, and may be problematic for unstable pt
- CT is the test of choice for initial evaluation
- Follow-up MRI is recommended when CT and other testing do not explain, or incompletely explain, the clinical picture

LP
- Prioritize emergent after CT scan if fever, elevated WBC, meningismus; otherwise do according to level of suspicion or if cause remains obscure
- Evaluation of CSF is a necessary inurgent evaluation of a pt with suspectedCNSinf. In a pt with altered level of consciousness, neuro-imaging to exclude an intracranial mass lesion is required prior to LP "avoid herniation"
- Obtain Coagulation test beforehand
- Empiric antimicrobial ttt is recommended when diagnosis of bacterial meningitis or herpes encephalitis is strongly suspected
- Obtain bl cultures prior to antibiotic
- CSF exclude subarachnoid hge when CT is normal and diagnosis remains suspect, and may help in diagnosis inf., as demyelinating, inflammatory, and neoplastic conditions

EEG
- If the pt has clinical finding suggestive of non-convulsive seizures, or if the cause of coma remains obscure after other testing
- Non-epileptiform EEG findings can help
- Can help in determining prognosis in cardiac arrest "somatosensory evoked potential more prognostically definitive"
- Continuous EEG may help in showing effects

ABCDE

- **Intubate** if GCS 8 **Stabilize C- spine** Supplement **O2 &IV access**
- **Blpr**supportas needed
  - **Hpotension**(Fluids & Vasopressors)
  - **Hypertension** (Labetelol 5 - 20 mg)
- **Glucose 50 %** IV **50** mL (after blood drawn, before results back) &**Thiamine 100** mg IV
- Treat definite seizures with **Phenytoin** orequivalent

Workup

Manage

- **For possible infection:**
  - **Ceftriaxone** 2 g IV / 12 hrs and **Vancomycin** 2 g/d IV in 4 divided doses
  - **Acyclovir** (10 mg/kg IV every/ 8 hrs)
- **For possible ingestion**
  - **Naloxone**
  - **Flumazenil**
- **Gastric lavage/activated charcoal**
- **For possible increased ICP:**
  - **Mannitol** (**1** g/kg IV)
  - Hyperventilation
- **For possible non-convulsive status:**
  - **Phenytoin** or equivalent (**15**-**20** mg/kg phenytoin equivalent IV)

New onset seizures

Epilepsy is defined as recurrent unprovoked seizures

History

Medication history
- Review medications that may cause seizure
- Partial-onset are less likely to be drug-induced than generalized tonic-clonic seizures

Past medical history
- Review risk factors for epileptic seizures as TBI, stroke, inf., and alcohol or drug abuse

Family history
- A +ve family history is highly suggestive that pt has epilepsy. In particular, absence seizures and myoclonic seizures may be inherited
- Occasionally, a family member does not have seizures but has an abnormal EEG

- **ABCDE** approach
- O2 &IV line
- Monitor ECG, blpr, oximetry, 12 lead ECG

Workup

PH E
- *See approach to comatosedpt*

Lab.
- Determine a serum **Glucose** and sodium level on pts with a 1st time seizure with no comorbidities who have returned to their baseline (**Level II**)
- Pregnancy test in childbearing age woman (**Level II**)

LP
- After CT scan, either in ED or after admission, on immune-compromised pts (**Level II**)

Neuro-imaging
- When feasible

EEG
- Consider an emergent EEG in pts suspected of being in non-convulsive status epilepticus or in subtle convulsive status epilepticus, pts who have received a long-acting paralytic, or pts who are in a drug-induced coma (**Level III**)

- To diagnose **Non-convulsive status epilepticus** (ie, complex partial status and absence status) and subtle convulsive status epilepticus (often the terminal stage of convulsive status), emergency physicians need to maintain a high index of suspicion

Causes of provoked epileptic seizures
- Alcohol & drug withdrawal
- Drug intoxication
- Non-ketotic
- Hyperglycemia
- Porphyria
- Uremia
- Hyperthyroidism
- Dialysis disequilibrium S
- Hypoxia
- Hyponatremia,
- Hypernatremia
- Hypomagnesium
- Hypocalcemia
- Hypoglycemia

ttt

- Pts with a normal neurologic examination can be discharged with outpatient follow-up (**Level III**)
- Pts with normal neurologic exam., no comorbidities, and no known structural brain dis. do not need to startan anti-epileptics(**Level III**)
- Administer IV or oral loading dose of **Phenyttoin** or IV & restart daily oral maintenance dosing (**Level III**)
- Administer 1 of the following agents IV: “high-dose **Phenytoin**,” **Phenobarbital**, **Midazolam** infusion, or **Propofol** infusion should be administered to a pt in status epilepticus who continues to seize after having received benzodiazepine and phenytoin (**Level III**)

Status epilepticus "SE"

- ***SE*** should be defined as 5 min or more of continuous clinical and/or electrographic seizure activity or recurrent seizure activity without recovery between seizures
- SE should be classified as either **convulsive SE** (convulsions that are associated with rhythmic jerking of the extremities) or **non-convulsive SE** (seizure activity seen on EEG without the clinical findings associated with convulsive SE)
- **Refractory SE** should be defined as SE that does not respond to the standard treatment regimens, such as an initial benzodiazepine followed by another antiepileptic drug (AED)

Manage initially

- As most seizures terminate spontaneously within 3 min., **the following measures should only be instituted for seizures lasting > 7 - 10 min.,** unless pt is known to have longer seizures with self-termination (this information may be obtained from relatives or friends or from pt's epilepsy card or diary)

- Protect pt from damage during the seizures
  - Make environment safe "use padded bed rails'
  - **Do not restrain pt**
  - Once the flurry of seizures has ceased, place pt in a semi-prone **Position** with head down till full consciousness is restored"
  - Note the **time**
- **ABCD**
  - During inter-ictal period insert airway &**O2**
  - Do not attempt to insert anything in pt's mouth during a seizure, even if tongue is injured
- Set up an **IV line** (at least 2) as soon as possible to gain access to the circulation
- Estimate bl. **Glucose** rapidly using a bl. glucose test
  - If the pt is hypoglycaemic rapidly infuse a 50% solution of glucose to give **1**-**2**mg / kg BW
- Consider **Thiamine** IV
- Draw venous bl for measurement of glucose, urea, Na, K, Ca, liver function and anticonvulsant drug levels, CBC and clotting studies
- Correct **Metabolic abnormalities** if present
- **Monitor** temp., ECG, monitor respiration and blpr
- Gain **information**

Neurological Consultation

- **Neurologic exam**

- **Search for**: trauma ,inf., stroke, and drug ingestion
- **Consider**: EEG,LP, and imaging

Initial therapy

In the 1st IV

- Give**Diazepam0.1** mg/Kg or
- Give**Midazolam0.05** mg/Kg then Wait 1 min. for response
- Give additional Diazepam or midazolam prn

In the 2nd IV

- **Phenytoin20** mg/Kg at 25-50 mg/min "dilute in NS"

2nd line therapy "if seizures persists 20 min."

- **Phenytoin10** mg/Kg

No response

If seizures persist 20 min. Later

- Give**Phenobarbitone 20** mg/kg at **100** mg/min."if unavailable, start GA"

If seizures persists20 min. Later

- Administer **General anesthesia**
  - **Propofol** (**1–2** mg/kg bolus, then 2–10 mg/kg/hr) titrated to effect
  - **Midazolam** (**0.1–0.2** mg/kg bolus, then **0.05–0.5** mg/kg/hr) titrated to effect
  - **ThiopentalNa** (**3–5** mg/kg bolus, then **3–5** mg/kg/hr) titrated to effect; after 2–3 ds infusion rate needs reduction as fat stores are saturated
  - Anesthetic continued for 12–24 hrs after the last clinical seizure, then dose tapered

Further manage

- If phenytoin or phenobarbital has been used in emergency ttt, maintenance doses can be continued orally or IV guided by serum level monitoring
- Other maintenance AEDs can be started also, with oral loading doses
- Care needs to be taken with NG feeds, which can interfere with the absorption of some AED

Subarachnoid hemorrhage "SAH"

Rupture of an aneurysm releases bl. directly into CSF under arterial pressure. The blood spreads quickly within the CSF, rapidly increasing ICP. The bleeding usually lasts only a few seconds, but re-bleeding is common and occurs more often within the first d

Suspect

- Sudden, severe **Headache** (97 %) classically described as the "worst headache of my life"
- Disturbed conscious level

Confirm

Clinical
- Association --- brief loss of consciousness, seizure, N&V, and meningismus
- History of physical exertion

Non-contrastCT±LP
- A -ve head CT and LP effectively eliminate diagnosis if performed within a few ds
- LP is mandatory if there is a strong suspicion despite normal head CT
- Elevated opening pr & RBC count "does not diminish from CSF tube 1 to tube 4" &exclude SAH only if final tube is normal
- Unexplained xanthochromic supernatant "pink or yellow tint "in CSF -- highly suggest SA----- Indicates that bl has been in the CSF for at least 2 hrs& can last for 2 ws or more
- **BrainMRI**
  - Limited data suggest that MRI may be as sensitive as CT for acute detection of SAH
- **Cerebralangiography** if diagnosis in doubt

Detect cause

- **Digitalsubtractionangiography** (**DSA**) have highest resolution to detect intracranial aneurysms and define their anatomic features
  - Some pts with an initially -ve angiogram have bl in the cisterns around the midbrain, which reflects a peri-mesencephalic (non-aneurysmal) pattern of hge "10 % of cases", Repeat angiography is not necessary
- **CTangiography** (**CTA**) and **Magneticresonanceangiography** (**MRA**) non-invasive tests -- screen and plan pre-surgical
  - Both identify aneurysms 3 - 5 mm or larger with a high degree of sensitivity, not achieve resolution of conventional angiography
  - A major advantage of CTA over conventional angiography --- speed and ease by which it can be obtained
  - CTA is particularly useful in acute setting in a rapidly declining pt who needs emergent craniotomy for hematoma evacuation. Furthermore, CTA offers a more practical approach to acute diagnosis than MRA

Consult Neuro-surgeon

Re-bleeding
- Risk is highest in 1st 24 hrs,esp. within 6 hrs
- Diagnosis ---- acute deterioration of neurologic status **+** new hge on head CT scan
- LP is harder to evaluate because xanthochromia can persist for 2 ws or more
- Only aneurysm ttt is effective for prevention

Vasospasm, delayed cerebral ischemia
- Typically no earlier than d 3, & peak ds 7 – 8
- Diagnosis ---- decline in neurologic status, including onset of focal neurologic abnormalities + TCD sonography " precede clinical sequelae of vasospasm "

Hydrocephalus (acute and chronic)
IncreasedICP &**Seizures** &**Hyponatremia**
Cardiacabnormalities
ECG abnormalities
- ST segment depression, QT prolongation, deep symmetric T wave inversions, and prominent U waves. Life-threatening rhythm disturbances such as torsades de pointes, AF

Hypothalamic dysfunction & pituitary insufficiency

Detect comp-lications

General measures
- Stool softeners, bedrest, and analgesia
- DVT prophylaxis -- pneumatic compression stockings start prior to aneurysm ttt**&**AddSC UFH **5000** U / 8hrs once aneurysm is treated
- ttt metabolic, cardiovascular instability

Ventriculostomy
- In pts with high ICP with acute hydrocephalus

Bl pr control
- Decrease risk of re-bleeding, but may be offset by an increased risk of infarction
- Avoid vasodilators. **Labetalol** is preferred
- **Nimodipine** (**60** mg by mouth or NGT/ 4 hrs) improve neurologic outcome. start within 4 ds
- Prophylactic **AED** --- consider in unsecured aneurysms and large conc. of bl. at cortex
  - Continuation not necessary in pts without acute seizures after securing aneurysm
  - Continue for 6 months in pts experienced an acute seizure (within 7 ds) following SAH
- **Surgicalclipping** and **Endovascularcoiling**--- effective in preventing re-bleeding with acceptable complications rates. The choice depends on experienced clinicians, anatomic characteristics of the aneurysm, and the neurologic and clinical status of the pt
- **Anti-fibrinolyticagents** (eg, **Tranexamicacid**,) not shown to be overall effective
- Avoid Hypovolemia
- **Hyperdynamic therapy**-- prevent symptomatic vasospasm & ameliorate vasospasm with secured aneurysm
  - Employs modest hemodilution, induced hypertension with pressors & hypervolemia
  - If vasospasm persists despite **---** percutaneous intra-arterial angioplasty or administration of vasodilators" limited data "
- **Statintherapy** (**Pravastatin 40** mg /d or **Simvastatin 80** mg /d) --- reduce incidence of vasospasm, delayed ischemic deficits and

ttt

CT
- Clot is demonstrated in 92 % if performed within 24 hr of the bleed
- Performed with thin cuts through the base of the brain to increase the sensitivity
- Sensitivity is highest in 1ST 6 - 12 hrs after SAH (nearly 100 %) and then progressively declines over time to about 58 % at d 5

Cardiacabnormalities
- Myocardial injury --- elevations of CK-MB or serum troponin I (>0.1 µg/L)
- RWMAs, can occur not always reversible
- Some develop a pattern of transient apical LV dysfunction mimics MI (in absence of significant coronary artery disease)"**Takotsubocardiomyopathy**

Brain abscess

Suspect

- Headache is the most common complain
- Fever is not a reliable indicator
- Focal deficit ± Seizures + Signs of increased ICP

Confirm& DD

CT with contrast

- Less sensitive than MRI
- The lesion has different appearances according to its age

MRI

- More sensitive than CT in detecting cerberitis, and satellite lesions
- More accurate in detecting the extend of cerebral necrosis, ring enhancement, and cerebral edema
- Better visualization of brain stem

LP

- If focal symptoms (eg, unilateral headache) or signs (eg, unilateral cranial nerve deficits, hemiparesis) or papilledema ---- Do not perform LP before ruling out mass lesion
- The specimen obtained from stereotactic CT-guided aspiration or surgery should be sent for Gram's stain, aerobic, anaerobic, mycobacterial, and fungal cultures, special stains, and histopathology
- Pts at risk for parasitic abscess should have serology of bl and/or CSF sent for diagnostic testing

DD; Epidural and subdural empyema,septic dural sinus thrombosis, mycoticcerebral aneurysms, septic cerebral emboli with associated infarction, metastatic or1ry brain tumors, acute focal necrotizing encephalitis, and pyogenicmeningitis

ttt

Empirictherapy

- Oral, otogenic, or sinus source --- **Metronidazole**(**15**mg/kg IV, then **7.5** mg/kg IV/ 8 hrs; not to exceed 4 g /d) **+ either penicillinG** (**20-2**4 million U / d IV in 6 divided doses) for suspected oral focus, or **ceftriaxone** (**2**g IV/12 hrs) **OR cefotaxime** (**2**g IV/4-6 hrs) for suspected sinus or otogenicfocus
- Hematogenous spread --- **Vancomycin** (**30** mg/kg IVdaily in 2 divided doses adjusted for renal function) for empiric coverage of MRSA. If susceptibility testing reveals methicillin-sensitive S. aureus, replace vancomycin with **nafcillin** (2g IV/4hrs) **or oxacillin** (**2**g IV /4 hrs)
- PO neurosurgery --- **Vancomycin+ ceftazidime** (**2**g IV/8hrs), **cefepime** (**2**g IV/8 hrs) or **meropenem** (**1**g IV/8 hrs)for empiric coverage of MRSA. If susceptibility test reveals methicillin-sensitive S. aureus, replace as above
- Penetrating trauma ---- **Vancomycin+ ceftriaxone** (**2** g IV / 12 hrs) **or cefotaxime** (**2** g IV / 4 - 6 hrs)for empiric coverage of MRSA. If susceptibility test reveals methicillin-sensitive S. aureus, replace as above
- Vancomycin has poor penetration into CSF. **Rifampin 600** mg orally once daily or **300 - 450** mg twice daily can be added (it achieves bactericidal concentrations in CSF regardless of meningeal inflammation “limited data “)
- Vancomycin alternatives ---- **linezolid** (**600** mg IV or orally twice daily), **trimethoprim-sulfamethoxazole** (**5** mg/kg
- Subsequent ttt **:**De-escalate as C/S
- Duration of therapy; 4 - 8 ws.til there is good clinical response and resolution of CT or MRI findings. Contrast enhancement at site of abscess may persist for several ms” alone is not an indication for continued ab or exploration”

Aspiration

- Preferable to excision
- Burr hole &needle aspiration CT or US guided
- Culture aspirate for aerobes and anaerobes, fungi, and Mycobacterium TB
- If fails to change in size or expands -- re-aspirate
- Delayed or not required in: Early cerebritis without cerebral necrosis &abscesses located in vital regions of brain or those inaccessible
- Repeat scan ---in 48 hrs at 1 w, and any change in clinical status requires immediate imaging

Surgical excision

- Inducesmore deficits & infrequently performed
- Indicated in: Traumatic, multi-loculatedcases
- Indications for excision after initial aspiration and drainage: No clinical improvement within 1 W, depressed sensorium, signs of increased ICP, progressive increase in ring diameter of abscess
- Pts with mult-loculated lesions are more likely to recur and may require a 2nd procedure
- The course of IV ab can be shortened to 4 s following excision compared with drainage
- Excised lesions are less likely to relapse than lesions that have only been drained

Glucocorticoids

- substantial mass effect demonstrated on image
- **Dexamethasone 10** mg IV followed by 4 mg / 6 hrs; DC as soon as possible
- Disadvantage: Reduce contrast enhancement on CT,slow capsule formation, Increase ventricular ruptureris k, decrease penetration intoabs

Brain abscess is a focal collection within thebrain parenchyma, which can arise as acomplication of a variety of inf., trauma, or surgery

- The direct spread of organisms from a contiguous site usually causes a single brain abscess. 1ry inf. that can directly spread to the cerebral cortex include subacute and chronic otitis media and mastoiditis (spread to the inferior temporal lobe and cerebellum), frontal or ethmoid sinusitis (spread to the frontal lobes), and dental inf. (usually spreads to the frontal lobes)
- Brain abscesses associated with bacteremia usually result in multiple abscesses that are most commonly located in the distribution of the middle cerebral artery
- A wide variety of organisms may cause brain abscess. The pathogens involved differ depending upon the site of the 1ry inf.

Viral encephalitis

- Viral infections of the CNS result in the clinical syndromes of aseptic meningitis or encephalitis
- The true incidence of these infections is difficult to determine because the diagnosis may not be considered, many cases are unreported, or a specific viral etiology is never confirmed

Meningitis versus encephalitis

- The presence or absence of normal brain function is the important distinguishing feature between encephalitis and meningitis
- Pts with meningitis may be uncomfortable, lethargic, or distracted by headache, but their cerebral function remains normal
- In encephalitis, however, abnormalities in brain function are expected, including altered mental status, motor or sensory deficits, altered behavior and personality changes, and speech or movement disorders
- Seizures and postictal states can be seen with meningitis alone and should not be construed as definitive evidence of encephalitis
- Other neurologic manifestations of encephalitis may include hemiparesis, flaccid paralysis, and parasthesias
- However, the distinction between the 2 entities is frequently blurred since some pts may have both a parenchymal and meningeal process with clinical features of both
- The pt is usually labeled as having meningitis or encephalitis based upon which features predominate in the illness although meningo-encephalitis is also a common term that recognizes the overlap
- The importance of distinguishing between encephalitis and aseptic meningitis relates to the fact that the likely cause of each syndrome is different

Suspect

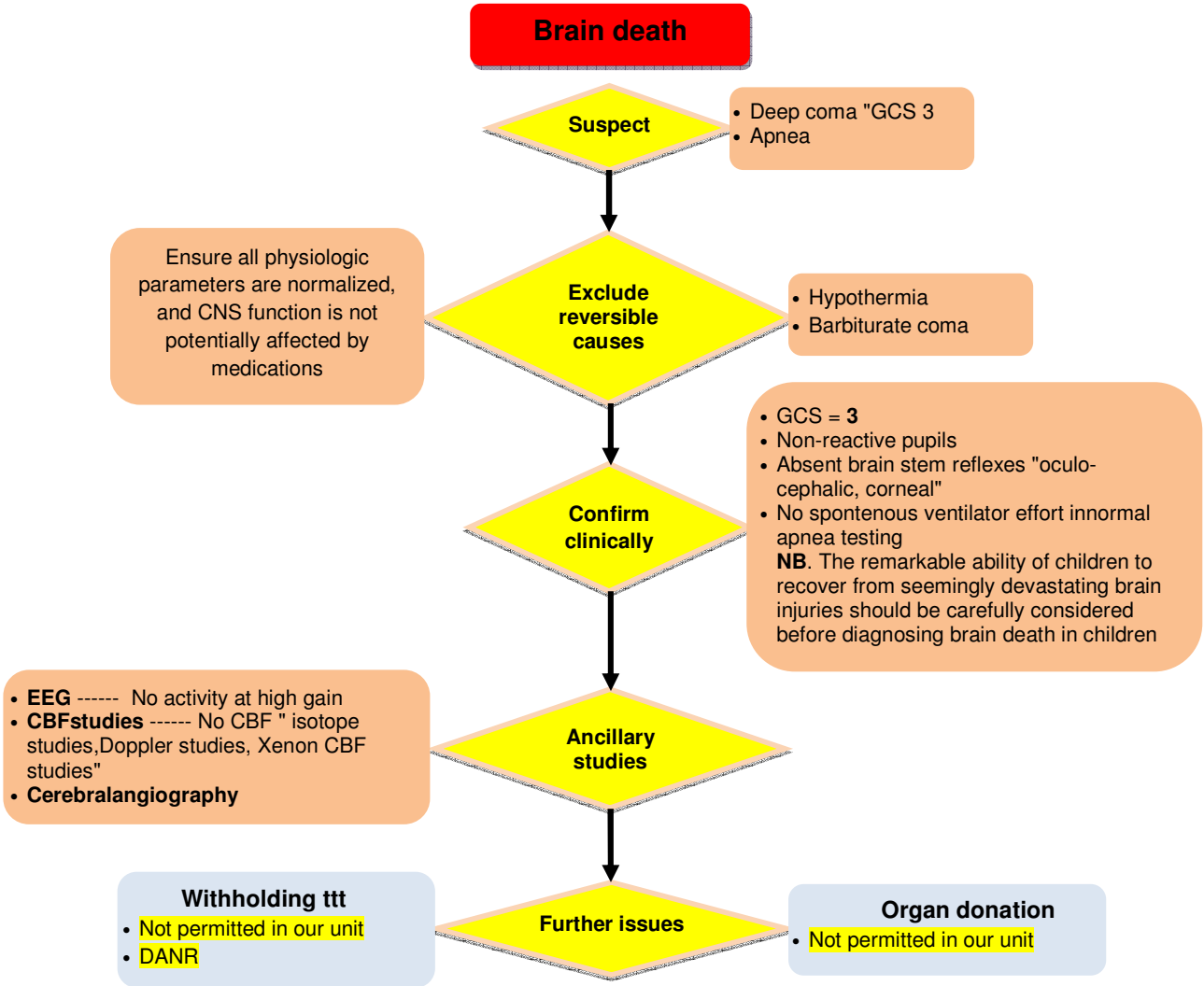
- Altered mental status
- sexual, travel, and exposure **History**

Confirm

- Altered mental status ranging from subtle deficits to complete unresponsiveness
- Symptoms and signs of meningeal irritation (photophobia and nuchal rigidity) are usually absent with a pure encephalitis but often accompany a meningo-encephalitis
- Seizures are common with encephalitis, and focal neurologic abnormalities can occur, including hemiparesis, cranial nerve palsies, and exaggerated deep tendon and/or pathologic reflexes
- Pts may appear confused, agitated, or obtunded
- **Suggestiveclues**
  - Parotitis strongly suggests diagnosis of mumps encephalitis
  - Flaccid paralysis suggests the possibility of West Nile virus inf.
  - Grouped vesicles in a dermatomal pattern suggest varicella-zoster virus
- Obtain **Neuro-imaging** to assess possibility of a localized process, as abscess, and to look for focality, which may suggest a specific etiology of encephalitis (eg, temporal lobe involvement and HSV-1)
- **CSF examination**: Reveals lymphocytic predominance "suggestive of a viral etiology"; red blood cells in the absence of a traumatic tap "suggestive of HSV or other necrotizing viral encephalitides".elevatedptn concentration, but usually **≤150** mg/dL. Usually normal glucose concentration (>50 % of blood value), but moderately reduced values are occasionally seen with HSV, mumps, or some entero-viruses
- **Diagnostic CSF PCR for HSV-1&IgMantibodyonCSF&serumforWestNilevirus**. Testing for other viral pathogens will depend on travel or exposure history to insects and animals. Serologic testing for West Nile virus, mumps, and Epstein-Barr virus can also be considered in the appropriate clinical setting

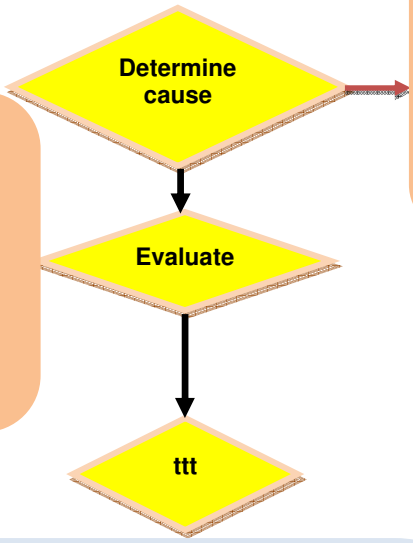
Empiric therapy

- The most important viral etiology to rule out in a pt with encephalitis is HSV, since this clinical entity is usually fatal if untreated
- HSV should be considered particularly if there is temporal lobe focality suggested by symptoms, signs or imaging studies
- Diagnosis is most readily made by detecting HSV DNA by polymerase chain reaction on CSF
- While awaiting confirmation, empiric therapy with **acyclovir** should be initiated **10** mg/kg /8hrs IV


Brain death
Suspect
• Deep coma "GCS 3
• Apnea
Exclude reversible causes
Ensure all physiologic parameters are normalized, and CNS function is not potentially affected by medications
• Hypothermia
• Barbiturate coma
Confirm clinically
• GCS = 3
• Non-reactive pupils
• Absent brain stem reflexes "oculo-cephalic, corneal"
• No spontenous ventilator effort innormal apnea testing
NB. The remarkable ability of children to recover from seemingly devastating brain injuries should be carefully considered before diagnosing brain death in children
Ancillary studies
• EEG ------ No activity at high gain
• CBFstudies ------ No CBF " isotope studies,Doppler studies, Xenon CBF studies"
• Cerebralangiography
Further issues
Withholding ttt
• Not permitted in our unit
• DANR
Organ donation
• Not permitted in our unit

GIT & Hepatic disorders

Acute liver failure "ALF"

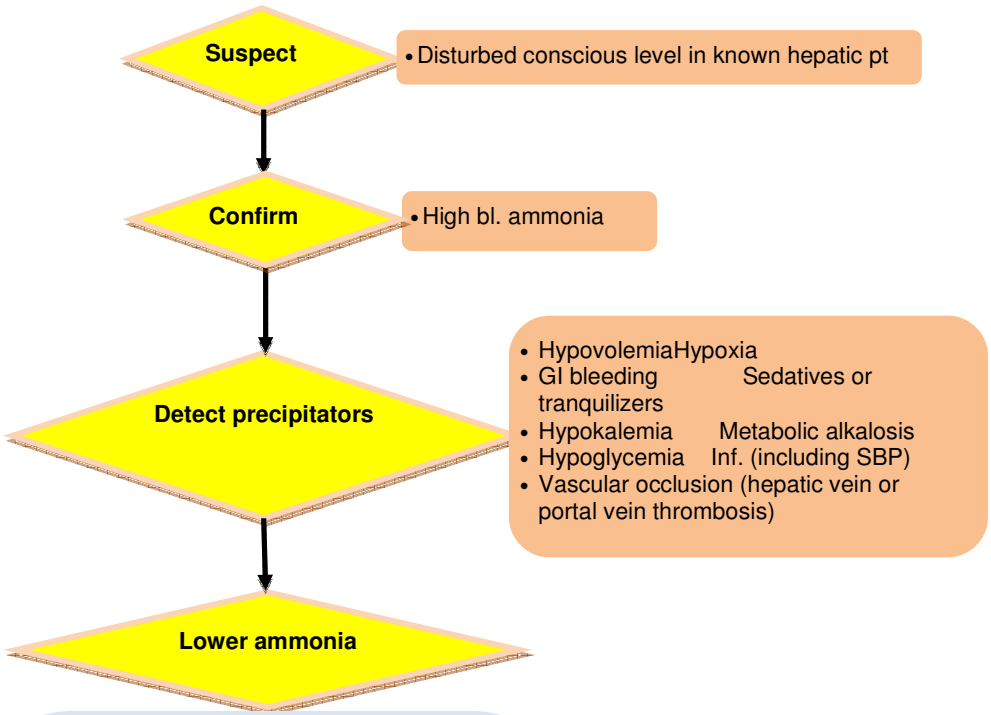


A	Acetaminophen, hepatitis A, autoimmune hepatitis
B	Hepatitis B
C	Cryptogenic, hepatitis C
D	Hepatitis D, drugs
E	Esoteric causes - Wilson's disease, Budd-Chiari syndrome
F	Fatty Infiltration - acute fatty liver of pregnancy, Reye's syndrome

- Hepatic encephalopathy
- Cerebral edema in **75**-**80** % of grade IV encephalopathy
- Acute renal failure complicates ALF in approximately **30** - **50** %
- Increased risk of inf. and sepsis
- Pulmonary edema & pulmonary inf. in 30 %
- Electrolyte disturbances (Hypokalemia, hyponatremia, and hypophosphatemia)
- Coagulopathy & Malnutrition

- **Othotopiclivertransplantation** "only therapy proven to improve pt outcome"
  - Should be transferred as early as possible to a transplant center (**Level 1C**)
  - The decision to transplant depends upon the probability of spontaneous hepatic recovery, which cannot be predicted by any single factor alone " transportation may be hazardous if complications, such as severe coagulopathy or increase ICP develop
  - The most important variables for predicting the outcome in ALF are PT, pt's age degree of encephalopathy, and cause of ALF
- **Lactulose** For hepatic encephalopathy
  - Intubation is mandatory (**Level 2C**)
- Use invasive ICP monitoring to guide therapy
  - An epidural ICP monitor be placed in pts with grade IV encephalopathy or in those in whom grade III encephalopathy is progressing rapidly (**Level 2C**)
  - Maintain ICP <**20** mmHg & CPP >**50** mmHg
  - Interventions to achieve these are:
    - Minimize sensory stimulation
    - Keep thept from becoming agitated
    - The use of NGT should be minimized
    - Endo-tracheal suction should be minimized
    - Fluid status should be closely monitored
    - The head of bed should be elevated to 30$^{O.}$ Pts should remain supine if the CPP <**50** mmHg with bed elevation"
- For persistent intra-cranial hypertemsion
  - Pts with ICP >**20** mmHg should be hyper-ventilated to keep PCO2 <**25** mmHg
  - If no response or relapse is noted, **Mannitol** (**0.5 - 1** g/kg) as an IV bolus and then on an as-needed basis to maintain plasma osmolality between **310**&**325**mosmol/kg
  - Fluid should be removed via **UF** with a goal to remove 3 -5 times the fluid volume of the infused mannitol in pts with compromised renal function and oliguria
  - If no response or relapse is noted after mannitol, **Pentobarbital** coma should be induced using a bolus of **3 - 5** mg/kg IV
  - **Dexamethasone** not effective in ttt of cerebral edema & should not be given

Hepatic encephalopathy



Whether or not the values are frankly elevated

- Correct hypokalemia
- **Dietary ptn restriction is not recommeded**
- **Lactulose** is recommended (**Level 2C**)
  - **45** - **90** g/d should be titrated to achieve 2 - 3 soft stools / d with a pH < 6
  - Lactulose enemas can be given if the pt cannot take lactulose orally
- For those who have not improved within 48 hrs, second-line therapy with non-absorbed abs is recommended (**Level 2C**)
  - Added to rather than substituted for lactulose
  - **500** mg **Neomycin** /8hrs or **1**gm twice daily
  - With regard to the nephrotoxicity, only a few cases have been described and thus the risk (although unknown) is probably very low
  - In deep hepatic encephalopathy ------ give lactulose by enema for several ds, reserving neomycin for those who do not respond

Chronic therapy

- In pts with recurrent encephalopathy caused by cirrhosis and portal hypertension /or systemic shunts ---- administer lactulose (**Level2B**)
- Ptn restriction is not needed unless encephalopathy is resistant to lactulose
- In ptswhose symptoms worsen with ptn intake ---- substitute ptnfish, milk, or meat with vegetable ptn may improve N2 balance
- Add **BCAA** to a low ptn diet. BCAA supplementation is indicated only in severely ptn-intolerant pts

Acute GI bleeding

- Overt bleeding: - blood in the NGT
  - Haematemesis or malaena

  Plus either:
  - Mean arterial pr>**20** mmHg decline from baseline
  - Requirement of at least **2**Ubl.transfusion, ↓ Hb≥ **2** g/100ml in 24 hr

❖ Bl. in the NG tube is frequently due to local erosion and by itself **doesnot**constitute clinically significant GI bleeding

Documented GI bleeding

- ABC / Resusc. including
  - 2 wide bore **IVaccess**
  - **Fluid**resusc.
  - **Bloodcomponenttherapy**

Gastro-entologist consultation

Workup

Simultaneously with resusc.
- **Lab**. ---- CBC, Coagulation profile
- **NGT** insertion
- **Endoscopy**

Manage

- Correct coagulopathy / cease heparin
- **Endoscopy± sclerotherapy**
- Consider **Labelledred cell scan**, **Angiography** (+/- embolization) or **Colonoscopy** if the bleeding source not identified &Hb loss continues
- **Drug therapy for clinically significant GI bleeding**

Drug therapy for clinically significant GI bleeding

Pantoprazole
- **40** mg/day r bd IV acutely
- Once daily for maintainance

Octreotide
- **50**uq IV , then **50**uq/hr
- For variceal bleeding
- As effective as sclera-therapy

Ranitidine
- **50** mg IV/ 8 hrs
- For peptic ulceration"first line"
- Does not prevent re-bleeding
- Reduce dose in renal failure

Tranexamic acid

- Standard dose1 mg IV /24 hrs "adjust for renal function"
- Inhibitor of fibrinolysis
- Indications
  - Major uncontrolled hge

Vitamin K

- Standard dose**10** mg IV
- Indications

  Correction of prolonged INR in:
  - Warafarin overdose "use 2 mg if anticoagulant is planned"
  - Vitamin K deficiency


Neuro-muscular disorders

Acute muscle weakness

- **Intubate & Ventilate as indicated**
  - Avoid **Succinyl** if neuro-muscular process present for 3 ds
  - Stabilize circulation if there is autonomic instability

ABCD

PhE → **Association &Time course**

Unilateral

–

Consider
- Cortical
- Sub-cortical "brain stem"
  - Cranial nerve affection

Confirm

- **Head non-contrast CT**
  - Suspected cortical & sub-cortical lesions
- **Head contrast CT**
  - Intra-cranial tumour or inf. is suspected
- **MRI**
  - Suspected brain stem, Spinal cord or nerve root lesions
- **CT with myelo-graphy**
  - If MRI not available or CI if myleopathy is suspected

Consult

- **Neurologist**
- **Nephrologist**
  - Plasmaphersis decision
- **Spinesurgeon** if epidural compression syndrome suspected
  - Initiate ttt with steroids

+

Consider
- **Large cerebral lesion**
  - Diminished mental status
- **Brain stem lesion**
  - Cranial nerve affection
- **Poly-neuropathy**
  - Sensory involvement
  - Lower motor neurone signs
  - Legs more than arms
  - Distal more than proximal
- **Myelopathy**
  - Mid, upper cervical "all 4 limbs "spastic tetra-paresis, upper motor neurone signs, sensory level, bladder dysfunction"
  - Lower cervical"legs, hands"upper motor neurone signs"
  - Thoracic "legs, hands'upper motor neurone signs"
  - Lumbar "legs"spasticparaparesis, upper motor neurone signs"
- **Myopathy**
  - All limbs
  - No sensory involvement
  - Proximal more than distal
- **Neuro-muscular weakness**
  - Involvement of eyelids, jaw, tongue, occulomotor, pharynx; fatiguable weakness
  - Most common causes **botulism** "intestinal involvement, or **Myastheniagravis**"

Confirm

- If **myelopathy** is suspected "epidural compression S is not suspected"
  - MRI at the suspected
- If **poly-neurapthy** is suspected
  - Lumbar puncture if guilianbarre
  - Heavy metal screen if pure motor neuropathy
- If **NM- junctiondisorder** is suspected
  - Acetyl-choline receptor antibody test if myasthenia
  - Botulism test if botulism
- If **myopathy** is suspected
  - Electrolytes, C. phophokinase
  - EMG, nerve conduction velocity, muscle biobsy, thyroid function

Review causes & Determine if Pre-existing ,undiagnosed or new onset or critical illness related
-

- **Paresis**
  - Partial or incomplete paralysis
- **Plegia, or paralysis**
  - Total loss of contractility
- **Quadri-plegia, or tetra-plegia**
  - Involves all 4 limbs"implies pathology of cervical cord
- **Parplegia**
  - Both lower limbs "thoracic, lubar disc dis."
- **Hemiplegia**
  - Upper, lower limb of the same side
- **Monoplegia**
  - Only one limb
- **Myleopathy**
  - Lesion of the spinal cord
- **Myopathy**
  - Lesion of the muscle
- **Spinalnerve**
  - A nerve existing the anterior horn cell of the spinal cord
- **Poly-neuropathies**
  - Are "downstream" to spinal nerves in peripheral nervous system
- **Bulbar**
  - Refer to the tongue, jaw,face , and larynx
  - Involves dysarthria, dysphagia, and difficult secretion handling

Localization	Pre-existing	Undiagnosed & New onset	Critical illness related
Spinal cord	Trauma InF. Transverse myelitis	Acute ischemia Epidural abscess Acute Transverse myelitis	Not described
Anterior horn cell	Amyotropic lateral scelrosis Poliomylitis"Westnile virus"	Amyotropic lateral scelrosis Poliomylitis"Westnile virus"	Hopkins syndrome
Peripheral nerve	GBS Chronic inflammatory demylenatingpolymilitis	Incidental GBS Toxic, compressive, vasculitis, porphyria	Critical illness poly-neuropathy
NM-junction	Myasthenia Lambert-eaton s Botulism	Unmasked MG Toxic	Prolonged neuro-muscular blockade
Muscle	Muscle dystrophy Polymisitis Metabolic	Rhabdomyolysis Toxic Polymisitis Hypokalemic Hypophosphatemic	Critical illness myopathy

	Polyneuropathy	Myopathy	NM-junction disorders
Distribution	More distal	More proximal	Diffuse"ocular,bulbar, respiratory"
Relexes	Decreased	Normal, decreased	Normal
Sensory involvement	+	-	-
Atrophy	±	±	-
Fatigue	±	±	+
Serum CPK	Normal	Normal to increased	Normal

Association	Examples
Arches, tenderness	Myopathy
Aphasia	Cortical stroke
Diplopia	NM-junction disorders
Bulbar symptoms	NM-junction disorders
Vomiting, diarrhea, diuretics	Electrolytes induced myopathy
Rash	Dermato-myositis
Heavy metal exposure	Poly- neuropathy
Tick exposure	Tick paralysis
Bladder symptoms	Myelopathy
Recent viral illness	Poly- neuropathy, Myelopathy
Sensory deficit at or below v level point	Myelopathy
Thyroid abnormalities	Thyrotoxic weakness

Time course	Examples
Sudden	Stroke or vascular related
Acute/suacute	Polyneuropathy, myelopathy, NM – junction disorders
Insidious	Amyotropic lateral sclerosis, muscle dystrophies, myopathy,
Episodic	NM-junction disorders, hypo, hyperkalemic periodic paralysis

Clinical test	Upper motor neurone	Lower motor neurone
Reflexes	Hyper-reflexia	Hypo-reflexia
Muscle tone	Increased/spastic	Decreased/flacid
Fasciculations	None	Present
Atrophy	None	Severe
Babiniski	Present	Absent

Critical illness poly-neuropathy

- Sepsis, trauma, and burn are predisposing factor
- Electrophysiology reveals axonal motor and sensory polyneuropathy
- Weakness is more distally ,and in lower limbs
- Cranial nerves spared "may there is facial weakness"
- Sensory impairment in only %
- Hyo or areflexia is common

Critical illness myopathy

- In pts treated with neuro-muscular blockers, and steroids
- Pts with asthma,pneumonia, ARDS, renal failure are more predisposed
- Symmetric diffuse weakness, muscle wasting, hyporeflexia, challenge to wean
- Diagnosis: elevated CPK, EMG study
- Strict bl sugar control is beneficial

Prolonged NM blockade

- In pts treated with high doses of NDMR even after discontinuation
- Lasts from hrs to ws
- Renal, hepatic failures, acidosis, and hypermagnesmia are more prone
- Ph E:generalized weakness, normal sensation, normal or reduced reflexes, bilateral ptosis, and facial and jaw muscle weakness
- Diagnosis: Electro-physiology

Drugs associated with weakness

- **Peripheralnerve** "amiodrone, metronidazole
- **NM-junction** "NDMR, aminoglycosides, B blockers, Ca channel blockers, phenytoin
- **Muscle** "steroids, amiodrone, cholesterol lowering drugs

Causes of weakness grouped by anatomic subunits

Central "Upper motor neurone"

Cerebrum

- Stroke
- Space occupying/structural lesions
  - Left dominant cerebral hemisphere lesion
  - Right dominant cerebral hemisphere lesion

Subcortex/brainstem

- Stroke
- Space occupying/structural lesions
  - Lacunar syndrome
  - Midbrain/brainstem syndromes
- Spinal cord
  - Acute transverse myelitis
  - Spinal cord infarct
  - Spinal epidural, subdural he
  - Central intevertebral disc herniation
  - Tumours"primary, metastatic"
  - Multiple sclerosis

Peripheral "lower motor neurone"

- **Anterior horn**
  - Amyotropic lateral scelerosis *
  - Poliomyelitis *
- **Spinal nerve root**
  - Inter-vertebral disc herniation *
- **Ply-neuropathy**
  - Guillianbarre syndrome * #
  - Porphyria *
  - Lead, heavy metal poisoning *
  - Alchohol, drug induced
  - Diabetic
- **NM-junction disorders**
  - Myasthenia gravis *
  - Eaton-lambert myasthenic syndrome *
  - Botulism *
  - Tick paralysis *
  - Organophosphorus poisoning *
- **Myopathies**
  - Inflammatory"polimyosistis" *
  - Electrolyte induced *
  - Alchohol, drug induced
  - Muscle dystrophy *
  - Endocrinal related *

○ **Neuro-muscular cause of RF*

○ *#Frequently associated with autonomic dysfunction*

Botulism

- Potentially life-threatening neuro-paralytic disorder caused by the toxin of Clostridium botulinum that prevents release of acetylcholine from nerve endings at NM- junction

Causes

- Contaminated canned foods, wound inf., esp. in parenteral drug abuser

presentation

- Symptoms onset usually 18-36 hrs after exposure"range:6 hrs - 8 ds"
- Cranial nerve involvement "ocular, bulbar finding"
- Autonomic involvement "parasympathetic blockade as constipation and urinary retention"
- NM-junction involvement "bilateral weakness more proximally and more in upper extremity
- Muscle involvement may be severe "unable to mobilize in bed, or use hands to lift the head
- Deep tendon reflexes are normal 'but may be hypoactive or absent"

Diagnosis

- Normal neuro-imaging, and CSF
- In foodborne botulism ---- stool, serum, and implicated food should be tested for botulism neurotoxin
- In wound botulism --- obtain a swab of the wound exudates "for anerobic culture" and serum toxin assay

ttt

- Supportive in form of MV
- **Ant- toxin**"neutralize toxin molecule that are not bound to nerve endings"
- Early use prevent disease progression and decrease duration of ventilator support'
- Only one vial per pt, with no additional doses

Acute transverse myelitis

- Acute inflammation of the spinal cord

Pesentation

- Symmetric numbness, weakness, with bladder symptoms
- Significant pain may be absent
- Can be patchy, functional loss is often incomplete

Ph E

- Paraplegia "symmetric', weakness more profound in lower exteremities

Diagnosis

- **MRI** is the best diagnostic modality
- There may be a lag of 5 ds between onset and appearance of lesion
- **CSF analysis**
  - Pleocytosis, normal glucose, normal or slightly elevated ptn

Prognosis

- Varies, with most recovery achieved within 3 months of diagnosis with a direct relationship between initial deficit severity and long term outcome

Myasthenic crisis

ABCD

Intubate & Ventilate as indicated

Suspect

Generalized weakness can mask the usual signs of respiratory distress, as accessory muscle use. In addition, weak respiratory muscles may fatigue, producing precipitous respiratory collapse

Known myasthenia

- Pt presented by neuromuscular RF, severe bulbar weakness that produces dysphagia and aspiration, increasing generalized weakness as a warning
- May respiratory insufficiency out of proportion to their limb or bulbar weakness" RF may be the only presentation"
- Precipitating factors "infection, surgery, or tapering of immuno-suppression, or drug induced"

Exclude cholinergic crisis

Acetyl-choline receptor antibody

- Paradoxical weakening with anti-cholinesterase medications
- Rarely if ever seen with dose limitation of pyridostigmine to **<120** mg / 3 hrs
- So rare that it should not be the presumed cause of increasing weakness unless doses taken are known to significantly exceed this range. Otherwise, even in presence of cholinergic side effects, it should be assumed that pt's underlying myasthenia is worsening and appropriate ttt should be initiated

Any factors that may have precipitated or exacerbated the pt's weakness should be sought and corrected esp., inf.

Consult

Nephrologist

Neurologist

Rapid therapy

Plasmapheresis

- Start working over several ds, benefits last a few ws

Anticholinesterase

- Can be restarted at a low dose after the pt has shown a response to rapid therapy

High-dose Steroids

- **Prednisone60-80**mg /d
- Because therapeutic effect of plasmapheresis or IVIG is short-lived.
- Onset of benefit begins within 2-3ws& peaks after a mean of 5.5 ms
- Transient worsening of weakness and myasthenic symptoms that is serious in up to 50 % and associated with RFrequiring MV in up to 10 %
  - Transient worsening usually occurs 5-10 ds after the initiation of corticosteroids & lasts about 5-6 ds
  - Quick onset of action of rapid therapies helps to prevent worsening

Consider **Imuno-suppresants** "azathioprine, mycophenolatemofetil, or cyclosporine if steroids are CI or previously ineffective

Guillainbarre syndrome

- Acute immune-mediated polyneuropathies
- Heterogenous syndrome with several variant

Variants

Acute infective demylenating polyneuropathy

- Acute motor axonal neuropathy
- Involvement of predominately motor nerves, and electrophysiologic pattern suggesting axonal damage

Acute axonal motor sensory neuropathy

- Has sensory symptoms and more prolonged course

Miller fisher syndrome

- External ophthalmoplegia, ataxioa, muscle weakness, areflexia
- >90% of pts reach the nadir of their function within 2-4 s, followed by return of function occurring slowly over a course of ws to ms
- A chronic" >2 ms" slowly progressing or relapsing inflammatory demylenating polyneuropathy generally is considered a distinct entity from acute GBS
- Clinical course is shorter in children and recovery is more complete

ABCD

- Intubate & Ventilate as indicated
  - **Avoid Succinylcholine**
- Close monitoring
- Hemodynamic support

Suspect

- Associated with recent immunization
- Suspect acute inflammatory demylenating polyneuropathy if
  - Onset 2-4 ws after having febrile respiratory or GI inf.
  - Classic presentations:parsthesia in fingers and toes followed by lower extremity symmetric weakness that may ascend over hrs to ds to involve arms, and in severe cases respiratory muscles
- Facial nerve palsy in 70% of cases
- Autonomic dysfunction
  - Arrhythmias ", bradycardia, tacharrythmias"
  - Orthostatic hypotension & Paralytic ileus
  - Transient or persistent hypertension
  - Bladder dysfunction & Abnormal sweating

Confirm

Ph E

- Bilateral symmetric weakness
- Diminished or absent reflexes "10% normal "
- Minimal sensation loss despite parasthesia

CSF

- Normal pr
- **Albumino-cytologicdissociation**
  - Few cells "<10 typically mononucellar"
  - In 50% after 1 wk, 75% in the 3 rdwk "repeated puncture is needed"
  - Initial pleocytosis less than 100 lymphocytes may occur
  - Elevated ptn "more than 45 mg/dl"
    - Maximal protein values may be not seen for 4-5 ws

Electro-physiology

- Normal study after several ds of symptoms esp, with severe weakness render diagnosis is unlikely
- In motor, sensory variant, study is normal

Consult

Neurologist& Nephrologist

Manage

IV immune-globulin "IVIG" & Plasma exchange

- They are the main modalities of disease modifying therapy
- Equivalent and improve outcome. Shortens time to walking independently by 40 - 50 %
- For non-ambulatory adult within 4ws of neuropathic symptom onset, --- tttwith plasma exchange or IVIG (Grade 1A). For ambulatory adult who are not yet recovering within 4ws of neuropathic symptom onset ---ttt with plasma exchange or IVIG (Grade 2B)
- The choice between plasma exchange and IVIG is dependent on local availability and on pt preference, risk factors, and CI. When both therapies are equally available and there are no CI for either --- ttt with IVIG (Grade 2B)
- **IVIG ----- 2**gm /Kg, given as **1** g/Kg for 2 ds or **400**mglKg for 5 ds as a single course. Preferred in children"easy use, safe", however with the same results. Avoided in renal failure

- No need for **glucocorticoids** (Grade 1A)

Supportive

- Nutritional support
- DVT prophylaxis
- Frequent careful appropriate positioning & supportive mattress
- Repeated assurance is better than sedation
  - **Sedation&NMblockers** should be avoided "obscure course of illness"
- Laxative
- Analgesia
  - Avoid **opiods** "acute nocipetive pain
  - **Carbamezapime** "may aggrevatehyponatermia due SIADH"
  - **Amitriptyline** or ant-epileptic in chronic non-nociceptive neuropathic pain "may aggravates constipation"
- Prevent corneal ulceration

Endocrinal disorders

Diabetic keto-acidosis "DKA"

Euglycemic DKA, in which the serum glucose is normal or near normal but the pt requires insulin therapy for clearance of ketoacidosis, has been described, particularly in presence of poor oral intake or pregnancy

Suspect

- Positive history; can affect all age groups, but common in old age in type II diabetes
- Presence of precipitator; mostly, inf."esp,. pneumonia, UTI", discounted or irregular insulin therapy, MI, Cerebro-vascular accident, and pancreatitis

False -ve test for ketone bodies

- Nitroprusside reacts with acetoacetate and acetone (produced by the decarboxylation of acetoacetic acid), but not with B-hydroxybutyrate.
  - B-hydroxybutyrate is the predominant ketone, particularly in severe DKA
  - It is therefore possible, although unusual, to have a negative serum nitroprusside reaction in the presence of severe ketosis
- An indirect method to circumvent the masking of ketoacidosis is to add a few drops of hydrogen peroxide to a urine specimen
  - Will non-enzymatically convert B-hydroxybutyrate to acetoacetate, which will then be detectable by nitroprusside
- An alternative is to directly measure B-hydroxybutyrate in bl.; monitors are available to measure beta-hydroxybutyrate at the bedside, but this assay may not be available

Confirm

Clinical

- Neurologic symptoms, including lethargy, focal signs, and obtundation which can progress to coma in later stages
- Hypeventilation "**Kussmaulrespirations"**abdominal pain, fever is rare
- Symptoms & signs of volume depletion

Lb.

- Blood glucose level "**500-800** mg/dl"
- Plasma osmolarity
- ABGs"arterial pH <7.30 ,can be <than 6.90"
- Serum electrolytes"variable Na, hypokalemia, hypophosphatemia"
- Serum Anion gap">**20**meq/L
- Urinalysis, BUN, and serum creatinine, and urine ketones by dipstick
- Differential CBC "leucocytosis"
- Measure **A1C** " determine whether the acute episode is the culmination of an evolutionary process in previously undiagnosed or poorly controlled diabetes or a truly acute episode in an otherwise well-controlled pt"
- Differential CBC, culture of urine, sputum, and blood, serum lipase & amylase, ECG , and

False +ve test for ketones

- Sulfhydryl drugs, such as captopril, penicillamine, and mesna, interact with the nitroprusside reagent and can lead to a false positive ketone test
- Thus, a positive nitroprusside test cannot be reliably interpreted in pts treated with these drugs and directly measure B-hydroxybutyrate is
- If it is not available, diagnosis of DKA should be made on the basis of clinical presentation and an otherwise unexplained high anion gap metabolic acidosis in association with hyperglycemia

Manage

- The aim of therapy is to replete the ECF without inducing cerebral edema due to too rapid reduction in the plasma osmolality
- The average fluid loss is **3** - **6** L

- **ABC**
- Obtain large bore IV (16 G) line; monitor using a cardiac monitor, capnography, and oximetry
- Monitor serum glucose /hr, and basic electrolytes, plasma osmolality, and venous pH / 2-4 hrs until the pt is stable
- Determine and ttt any underlying cause of (eg, pneumonia or UTI, myocardial ischemia)

- The final serum Na concentration will reflect the balance between dilution of Na due to osmotic water movement out of the cells, and concentration of Na due to glucosuria-induced osmotic diuresis resulting in water loss in excess of Na
- Most pts are mildly hyponatremic
- Hyponatermia may devolpe due to hyperlipidedmia"pseudo-hponatermia"

Fluid repletion

Cardiogenic shock

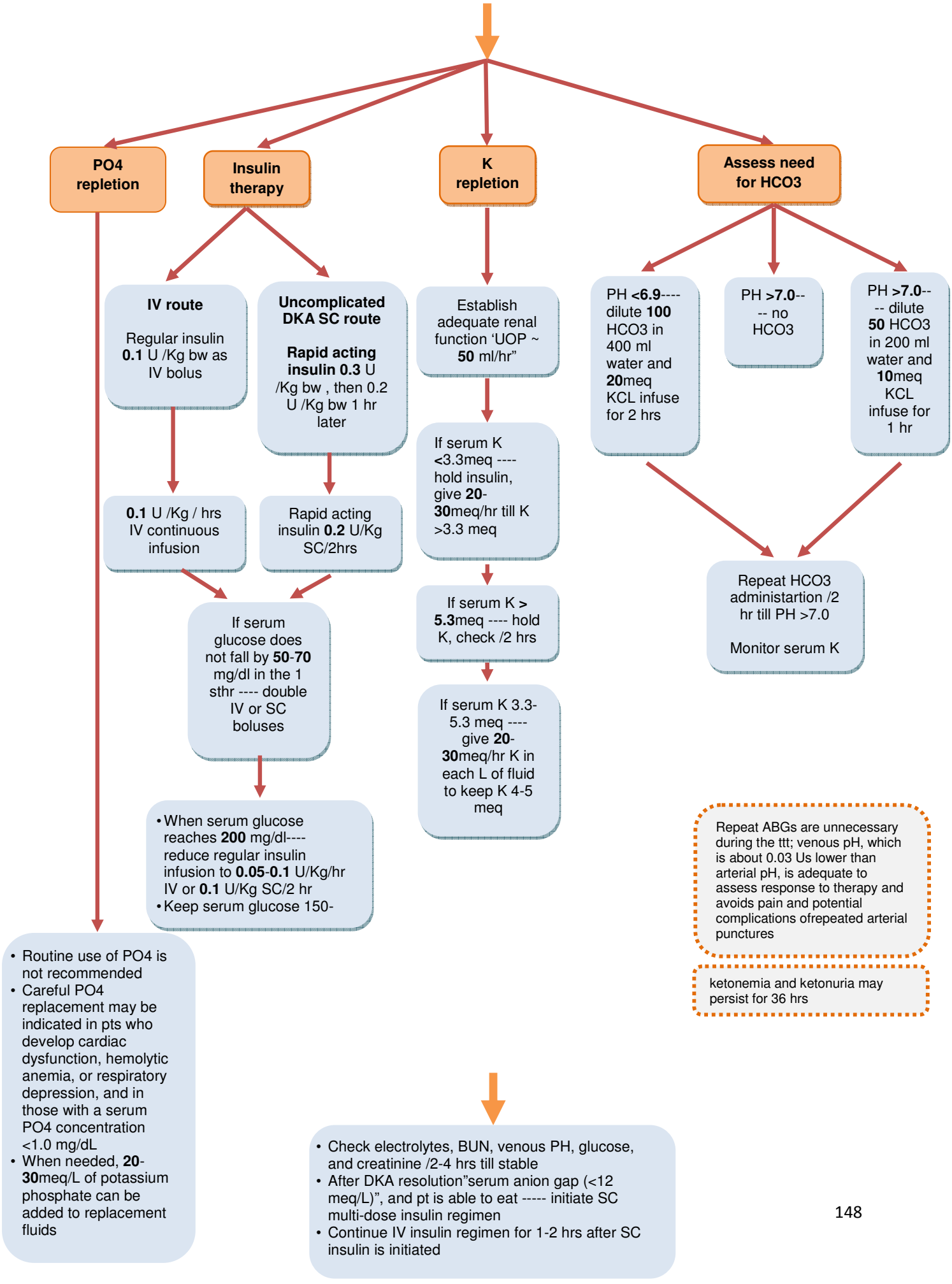
- Hemodynamic monitoring &Pressors

Severe hypovolemia

- Hemodynamic monitoring &Pressors

Mild dehydration

- Evaluate serum Na
- Normal & high Na ---- **0.45**% NaCL"**250**-**500** ml/hr according to hydration state
- Low Na **----- 0.9**% NaCL "**250**-**500** ml/hr according to hydration state ------- Change to 5% glucose and **0.45**% NaCL"**150**-**250** ml/hr when serum glucose reaches **200** mg/dL


PO4 repletion
Insulin therapy
K repletion
Assess need for HCO3
IV route
Regular insulin 0.1 U /Kg bw as IV bolus
Uncomplicated DKA SC route
Rapid acting insulin 0.3 U /Kg bw , then 0.2 U /Kg bw 1 hr later
Establish adequate renal function 'UOP ~ 50 ml/hr"
PH <6.9---- dilute 100 HCO3 in 400 ml water and 20meq KCL infuse for 2 hrs
PH >7.0---- no HCO3
PH >7.0---- dilute 50 HCO3 in 200 ml water and 10meq KCL infuse for 1 hr
0.1 U /Kg / hrs IV continuous infusion
Rapid acting insulin 0.2 U/Kg SC/2hrs
If serum K <3.3meq ---- hold insulin, give 20-30meq/hr till K >3.3 meq
If serum K > 5.3meq ---- hold K, check /2 hrs
If serum K 3.3-5.3 meq ---- give 20-30meq/hr K in each L of fluid to keep K 4-5 meq
Repeat HCO3 administartion /2 hr till PH >7.0
Monitor serum K
If serum glucose does not fall by 50-70 mg/dl in the 1 sthr ---- double IV or SC boluses
• When serum glucose reaches 200 mg/dl---- reduce regular insulin infusion to 0.05-0.1 U/Kg/hr IV or 0.1 U/Kg SC/2 hr
• Keep serum glucose 150-
• Routine use of PO4 is not recommended
• Careful PO4 replacement may be indicated in pts who develop cardiac dysfunction, hemolytic anemia, or respiratory depression, and in those with a serum PO4 concentration <1.0 mg/dL
• When needed, 20-30meq/L of potassium phosphate can be added to replacement fluids
Repeat ABGs are unnecessary during the ttt; venous pH, which is about 0.03 Us lower than arterial pH, is adequate to assess response to therapy and avoids pain and potential complications ofrepeated arterial punctures
ketonemia and ketonuria may persist for 36 hrs
• Check electrolytes, BUN, venous PH, glucose, and creatinine /2-4 hrs till stable
• After DKA resolution"serum anion gap (<12 meq/L)", and pt is able to eat ----- initiate SC multi-dose insulin regimen
• Continue IV insulin regimen for 1-2 hrs after SC insulin is initiated

Hyperosmolar non-ketotoc coma "HONKC"

- Characterised by severe hyperglycaemia with marked serum hyperosmolarity, without evidence of significant ketosis
- Hyperglycaemia causes an osmotic diuresis, with hyperosmolarity leading to osmotic shift of water into the intravascular compartment, resulting in severe intracellular dehydration
- Ketosis does not occur " basal insulin secretion sufficient to prevent ketogenesis, but insufficient to reduce bl glucose"

The presence of stupor or coma in diabetic pts with an effective plasma osmolality lower than 320 mosmol/kg demands immediate consideration of other causes of the mental status change

Suspect

- Positive history; can affect all age groups, but common in old age in type II diabetes
- Presence of precipitator; mostly, inf. "esp,. pneumonia, UTI", discounted or irregular insulin therapy, MI, CV accident, and pancreatitis
- Disturbed conscious level

Confirm

Clinical

- Symptoms develop more insidiously with polyuria, polydipsia, and weight loss often persisting for several ds before admission
- Most pts present with severe dehydration and focal neurologic deficits± seizures
- Coma is present in < 10% of cases

Lab.

- Blood glucose level >"**1000** mg/Dl"
- Plasma osmolarity "reach **380**"
- ABGs"arterial pH < 7.30 ,HCO3 <20"
- Serum electrolytes"variable Na, hypokalemia, hypophosphatemia"
- Serum Anion gap">**20**meq/L
- Urinalysis, BUN, and serum creatinine, and urine ketones by dipstick
- Differential CBC "leucocytosis"
- Measure **A1C** " determine whether the acute episode is the culmination of an evolutionary process in previously undiagnosed or poorly controlled diabetes or a truly acute episode in an otherwise well-controlled pt"
- Differential CBC, culture of urine, sputum, and blood, serum lipase & amylase, ECG , and CXR, abd CT for pancreatitis as needed

ttt

- Aim --- replete the ECF volume without inducing cerebral edema due to too rapid reduction in the plasma osmolality
- The average fluid loss is 3 - 6 L

- **ABC**
- Obtain large bore IV (16 G) line; monitor using a cardiac monitor, capnography, and oximetry
- Monitor serum glucose hourly, and basic electrolytes, plasma osmolality, and venous pH / 2-4 hrs until the pt is stable
- Determine and ttt any underlying cause of (eg, pneumonia or UTI, myocardial ischemia)

- The final serum Na concentration will reflect the balance between dilution of Na due to osmotic water movement out of the cells, and concentration of Na due to glucosuria-induced osmotic diuresis resulting in water loss in excess of Na
- Most pts are mildly hyponatremic
- Hyponatermia may devolpe due to hyperlipidedmia"pseudo-hponatermia"

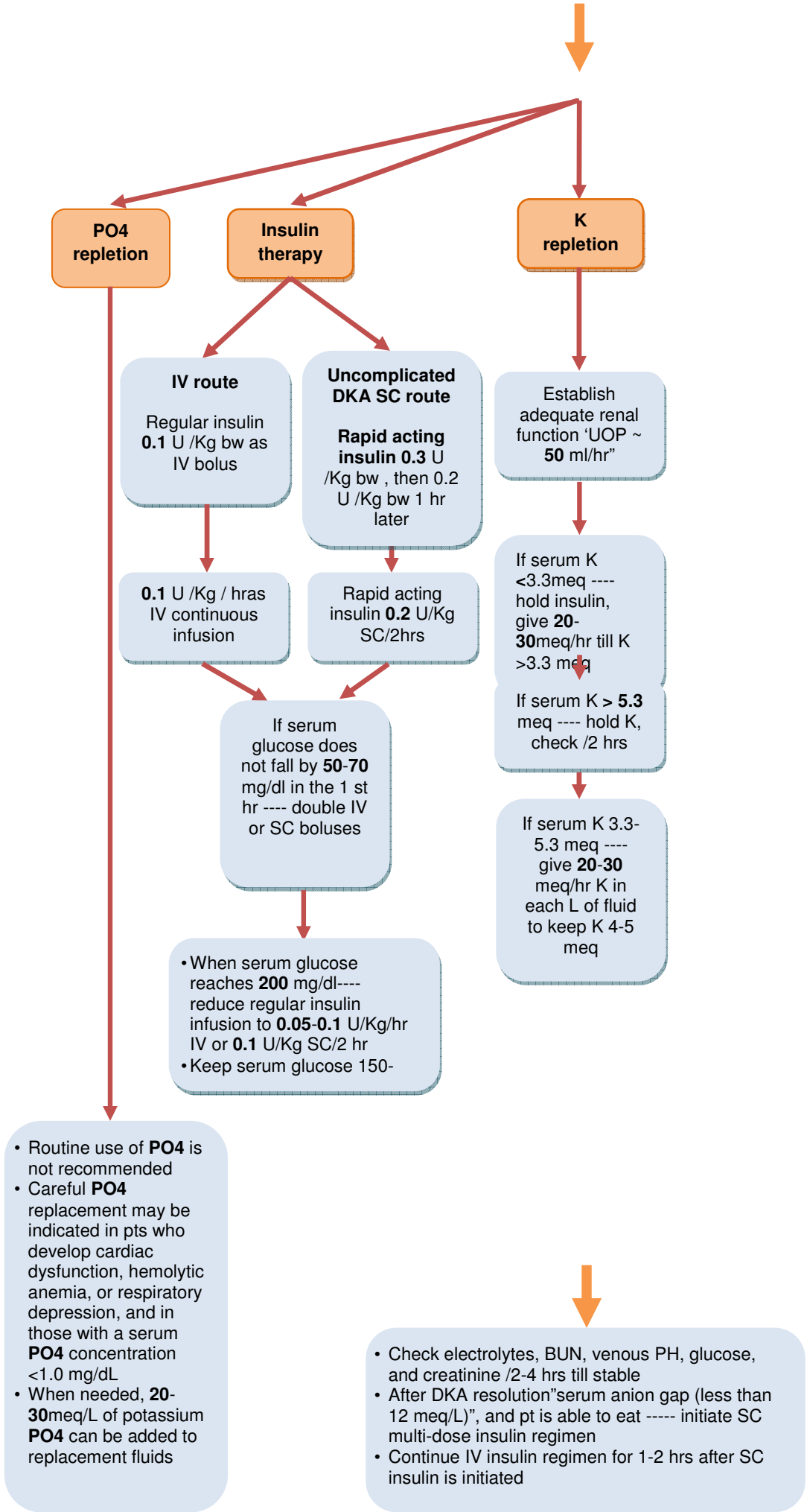
Fluid repletion

Cardiogenic shock
- Hemodynamic monitoring &Pressors

Severe hypovolemia
- Hemodynamic monitoring &Pressors

Mild dehydration

- Evaluate serum Na
- Normal & high Na ---- **0.45**% NaCL"**250**-**500** ml/hr according to hydration state
- Low Na **-----** **0.9**% NaCL "**250**-**500** ml/hr according to hydration state ------- Change to 5% glucose and **0.45**% NaCL"**150**-**250** ml/hr when serum glucose reaches **200** mg/dL


PO4 repletion
Insulin therapy
K repletion
IV route
Regular insulin 0.1 U /Kg bw as IV bolus
Uncomplicated DKA SC route
Rapid acting insulin 0.3 U /Kg bw , then 0.2 U /Kg bw 1 hr later
Establish adequate renal function 'UOP ~ 50 ml/hr"
0.1 U /Kg / hras IV continuous infusion
Rapid acting insulin 0.2 U/Kg SC/2hrs
If serum K <3.3meq ---- hold insulin, give 20-30meq/hr till K >3.3 meq
If serum glucose does not fall by 50-70 mg/dl in the 1 st hr ---- double IV or SC boluses
If serum K > 5.3 meq ---- hold K, check /2 hrs
If serum K 3.3-5.3 meq ---- give 20-30 meq/hr K in each L of fluid to keep K 4-5 meq
• When serum glucose reaches 200 mg/dl---- reduce regular insulin infusion to 0.05-0.1 U/Kg/hr IV or 0.1 U/Kg SC/2 hr
• Keep serum glucose 150-
• Routine use of PO4 is not recommended
• Careful PO4 replacement may be indicated in pts who develop cardiac dysfunction, hemolytic anemia, or respiratory depression, and in those with a serum PO4 concentration <1.0 mg/dL
• When needed, 20-30meq/L of potassium PO4 can be added to replacement fluids
• Check electrolytes, BUN, venous PH, glucose, and creatinine /2-4 hrs till stable
• After DKA resolution"serum anion gap (less than 12 meq/L)", and pt is able to eat ----- initiate SC multi-dose insulin regimen
• Continue IV insulin regimen for 1-2 hrs after SC insulin is initiated

Myxedema coma

History
- long-standing hypothyroidism or be precipitated by inf., MI, cold exposure, or administration of sedatives, esp. opiates

Clinical
- Hypothermia, Hyponatremia, and Hypoglycemia
- Cardiovascular abnormalities" Diastolic hypertension, Overt CHF is quite rare in the absence of preexisting cardiac disease, and May pericardial effusion"

Lab.
- Before giving thyroid H --- measure serum TSH, free T4, and cortisol to rule outassociated adrenal insufficiency or hypopituitarism
- **Cortrosyn** should be given before bl.sample is obtained to permit evaluation for coexisting adrenal insufficiency
- Most have primary hypothyroidism, with high serum TSH and low free T4 values
  - A normal or low serum TSH value in a pt with a low free T4 value indicates that hypothyroidism is secondary to hypothalamic or pituitary dysfunction

Suspect
- **Decreasedmentalstatus**
- Inappropriate **Bradycardia**

Confirm

DD
- Other causes of decreased conscious level

ttt

ttt should be instituted without waiting for lab.

Steroids
- Give in stress doses until the possibility of coexisting adrenal insufficiency has been excluded

Supportive measures
- **MV** if necessary
- Judicious administration of IV **Fluids** including electrolytes and glucose
  - Avoid dilute fluids in hyponatremicpts
- Hypotension, if present and not caused by volume depletion, will be corrected by thyroid hormone therapy over a period of hrsto ds
  - Severe hypotension that does not respond to fluids , treated with a **Vasopressor** drug until the T4 has had time to act
- Passive **Rewarming** with a heating blanket is preferred for correction of hypothermia
  - Active rewarming carries a risk of vasodilatation and worsen hypotension
- Empiric administration of **abs** for inf. should be considered til appropriate cultures are proven -ve

Thyroid hormone
- **T4** is given in a loading dose of **200** - **300** mcg followed by **50** mcg daily
- **T3** is given simultaneously in a dose of **5** - **20** mcg, followed by **2.5** - **10** mcg / 8 hrs, depending upon pt's age and coexistent cardiac risk factors
- T3 is continued til there is clinical improvement and pt is stable

Thyroid storm

History:
- Long-standing untreated hyperthyroidism & surgery, trauma, inf., or acute iodine load

Clinical
- Tachycardia "may exceed 140 b/min, along with CHF
- Hyperpyrexia is common
- Agitation, delirium, psychosis, stupor, or coma are common "considered essential by many for diagnosis"
- May severe N&V, or diarrhea, and hepatic failure with jaundice
- ABGs ---- Hypercapnea

Suspect
- Un-explained **tachycardia** "may exceed 140 b/min
- Un-explained **hypercapnea**

Confirm

DD
- Other causes of tachycardia, hypercapnea

ttt

B blockers
- Used with caution if pt has CI to B-blockade
- **Propranolol**; frequently selected for initial therapy " can be given IV"
  - IV dose ---**1** mg/min. until several Mgs have been administered or adequate B-blockade has been achieved
  - Concurrently, propranolol is given orally or via NGT at a dose of **60 - 80** mg / 4 hr
- An alternative regimen is to utilize the short-acting B - blocker **Esmolol**
  - Loading **250 - 500** µg/kg, followed by **50 - 100** µg/kg / min.

Thionamides
- Block de novo thyroid hormone synthesis within 1 - 2 hrs
- They have no effect on release of preformed hormone from the gland
- **Methimazole** has a longer duration of action than **PTU** (more effective unless PTU is given at regular frequent intervals)
- Administer **30** mg of **methimazole** / 6 hrs, or **200** mg **PTU** / 4 hr, orally or via NGT
- Both drugs can also be suspended in liquid for rectal administration, PTU can be prepared for IV administration by dissolving the tablets in NS made alkaline (pH 9.25) with Na hydroxide, and methimazole can be dissolved in pH-neutral isotonic saline and prepared for IV administration by filtering through a 0.22 µm filter

Glucocorticoids &Lithium &Plasmapheresis

Glucocorticoids
- Reduce T4-to-T3 conversion, and may have a direct effect on the underlying autoimmune process if thyroid storm is due to Graves' dis.
- **Hydrocortisone100** mg IV / 8 hr
- not used in pts with severe, but not life-threatening, hyperthyroidism

Lithium
- Has been given to acutely block the release of thyroid hormone. its renal and neurologic toxicity limit its utility

Plasmapheresis
- Has been tried when traditional therapy has not been successful

Supportive
- Many require a substantial amount of **Fluid**& others may require **Diuresis** (CHF)
  - Digoxin requirements may be quite high
- Inf. needs to be identified & treated
- Hyperpyrexia should be aggressively corrected
  - **Acetaminophen** is preferable to aspirin

Iodinated radio-contrast agents
- **Iopanoicacid** and other iodinated radio-contrast agents have little efficacy in thyroid storm
- Potent inhibitors of T4-to-T3 conversion, and they have been extremely useful in treating severe hyperthyroidism or in preparing hyperthyroid pts for urgent surgery
- A dose of **0.5 - 1** g given once daily
- Given at least 1 hr after thionamide

Glucocorticoids withdrwal

Presumed to have HPA suppression

- Pt who received a glucocorticoid dose comparable to >**20** mg **Prednisone** /d > 3 w
- Pt who received an evening/bed time dose of prednisone for > a few ws
- Pt who has a Cushingoid appearance

- No need test to evaluate HPA function
- Treat like any pt with secondary adrenal insufficiency

Less likely to have a suppressed HPA

- Pt who has received any dose of glucocorticoid for < 3 w
- Pts treated with alternate-day glucocorticoid therapy

- May have glucocorticoids as is appropriate for the underlying disease

Intermediate or uncertain risk of HPA suppression

- Pt take**10**-**20** mg **Prednisone** / d for > 3 w
- Any pt who has taken **<10** mg of **Prednisone** or its equivalent / d, providing that it is not taken as a single bedtime dose for > a few ws

- If withdrawal from glucocorticoids is otherwise indicated, gradual reduction in dose is appropriate
- Do not need to be tested for HPA functional reserve unless abrupt discontinuation is being considered or pt is facing an acute stress (surgery)
  - Give stress doses or, if time permits, test for responsiveness of the adrenal with an ACTH (**Cosyntropin**) stimulation test

Tapering regimen

- Short-term glucocorticoid therapy (up to 3 w), even if at a fairly high dose, can bestopped and need no tapering
- Most pts on **5** mg/d daily dose of do not have to be tapered

The dose is tapered by:

- **10** mg/d / 1 – 2 w at an initial dose **>60** mg prednisone or equivalent / d
- **5** mg/d / 1 -2 w at 60-20 mg/day prednisone
- **2.5** mg/d / 1 -2 w at 19-10 mg/day prednisone
- **1** mg/d / 1 -2 w at 5-9 mg/day prednisone
- **0.5** mg/d / 1 -2 weeks at prednisone doses **<5** mg/day
- This can be achieved by alternating daily doses, eg, **5** mg on day 1 and 4 mg on d 2

Drug	Equival-ent	Glucoco-rticoids	Mineralcorti-coids
Hydrocortisone	100	1	1
Prednisone	25	4	0.3
Methyprednisolone	20	4	0
Dexamethasone	4	30	0
Cortisone acetate	125	0.8	0.8
Fludrocortisone	1	10	250

Estimation of HPA suppression

- Identifying the degree of HPA suppression is not simple clinically (unusual to perform any testing of HPA function prior to beginning the glucocorticoid withdrawal process)
- In certain settings (eg, the pt for whom elective surgery is planned) such testing may be warranted
- Testing for HPA-axis function is appropriate when pt are using **≤ 5** mg/d prednisone and there is difficulty reducing dose further because of non-disease related symptoms
- Response to giving synthetic (ACTH [cosyntropin]) is the preferred method to assess adrenocortical function
- Test results should be available within hr to d thereafter

<u>The standard high-dose ACTH test and the criteria of a normal adrenal response are summarized as follows:</u>

- Standard high-dose ACTH stimulation test A baseline venous blood sample is taken prior to ACTH injection
- Synthetic ACTH (cosyntropin**250** µg [85 nmol, or 40 IU]) is injected IV or IM
- Venous blood is obtained 30 and 60 min. after the injection and serum cortisol concentrations are measured on these and the baseline sample
- If ACTH is given IV a serum cortisol value of **>20** µg/dL (550 nmol/L) at any time during the test, including before injection, is indicative of a normal adrenal response
- After IM injection, a serum cortisol value of **16** µg/dL (440 nmol/L) or more at any time ---- normal adrenal function
- If ACTH stimulation testing indicates normal adrenal responsiveness but a pt continues to have non-disease related symptoms with further attempts to reduce glucocorticoid dosing ---- **CRH stimulation test "** can assess both the ACTH and cortisol responses and may be used instead of the cosyntropin test if there is concern about pituitary function"

Hematologic disorders

Anemia

Anemia defibed as Hgb <13 in males, and <12mg/dl in females. Severe anemia; Hgb<8 mgdl

Bl transfusions are associated with increased inf. rates, increased risk of organ failure, increased length of stay in the ICU, and increased mortality

Suspect

- Pallor
- History of trauma, or major surgery

CBC

- Detect **Hgb** level
- Detect **Platelet** count
- Detect **Pancytopenia**

Workup to detect cause

Obvious cause

Consider

- Bl loss: trauma " 4, and bl in the floor",surgery; bl in the surgical drains, and NGT, coagulopathy
- Nutritional deficiencies
- Erythropoietindeficiency: renal failure, inf.

Non-obvious cause

Consider

- Review numbers of phlebotomies
- Occult bl in the stool
- Abdominal US to detect source of bleeding in trauma "exclude retro-pertonealhge", and PO abdominal surgery
- CXR to exclude hemothorax in trauma
- Diagnose hemolysis ---- reticulocytic count, fragmented RBCs

Manage

- Assess hemodynamic stability and signs of adequate organ perfusion
- Control surgical bleeding
- RBCs transfusion trigger ----- "*see next p*"
- If pt is anemic with chronic renal failure ----- consider erythropoietin therapy and iron as alternatives to increase bl cell mass
- When pt is able, add iron supplementation in conjunction with bowel regimen
  - Minimize bl loss by decreasing phlebotomy frequency
  - Minimize frequency of labs for subacutepts
  - Serial labs are unnecessary for monitoring solid organ injury
- Optimize nutrition
- Monitor continuously for signs of hypoperfusion with anemia
  - Follow neurologic and mental status, vital signs, end organ perfusion
  - Invasive monitoring indicated by physiology and comorbidities, not by anemia

Causes of anemia in ICU

- Hemodilution
- Nutritional deficiencies
  - Folic acid - Iron - Vit B
- Coagulopathy
  - Thrombocytopenia - Sepsis
  - Liver dis. - Splenomegaly
- Hemolysis
  - Toxins - Drugs "*see table*"
- Bl loss
  - Phelpotomies - Trauma
  - Surgery - Stress ulcer
- Erythropoietindeficiency
  - Renal failure- Chronic dis.-Inf.

Drugs causing hemolysis of anemia in ICU

Mechanism of hemolysis	Common medications
Immune	Cephalosporins/cephamycins Cefotetan Ceftriaxone
	β lactams Penicillin derivatives Piperacillin
	Nonsteroidal anti-inflammator Diclofenac Ibuprofen
	Antineoplastics Fludarabine
	Others Methyldopa Quinine/quinidine
Nonimmune	Nitrofurantoin
	Phenazopyridine
	Primaquine
	Sulfa drugs

PRBCs transfusion

BlLoss

40 % loss (>2000 ml):

- Rapid fluid replacement, including RBCs

30 - 40 % loss (1500 - 2000 mL):

- Rapid fluid replacement, probably RBCs transfusion

Hgb Concentration

- Transfusion thresholds in
  - Active IHDor acute ischemic syndromes ----- Hgb<**10** g/dL, hematocrit <**30**
  - Stable critical care pts ---Hgb<**7** g/dL, hematocrit <**21** unless they exhibit: Impaired O2 delivery, shock, ongoing bl loss
  - Pts with hematocrit **21**-**30** ------ mandate an attending discussion

Blood lost acutely (surgery, trauma or bleeding)

\+

Transfusion needed peri-opertively

\+

Pts with asymptomatic anemia &Hgb< 7 g/dL may need transfusion if:

- A scheduled surgery is expected to produce significant blood loss
- Risks with general anesthesia are high

- DC anti-platelet drugs, reverse anticoagulation
- Considering various strategies for auto-geneic transfusion
- Use pharmacological agents to raise Hgb and reduce surgical bleeding

-

There is chronic anemia

\+

- CI if specific replacement therapy is possible (*e.g.*, iron, vitamin B12, folic acid). Indicated only in case of emergency surgery, acute bl. loss, or trauma
- Anemia 2ndry to aplasia or bone marrow suppression
  - Asymptomatic pts and:
    - No high risk factors -- a Hb of **6**-**7** g/dL may be sufficient
    - Evidence of cardiovascular, pulmonary or cerebrovascular dis. -- transfuse ifHb of <**7** g/dL

-

There is Specific indications ?

Specific indications

Uremic bleeding:

- Hgb<**10** g/dL

Sickle cell disease

- Pts with a history of or at high risk for stroke, other severe complications, on chronic transfusion or require acute RBC exchange ---- transfuse to reduce Hb S to <**30** - **50** %.

Burn pts

- Not critically ill and without cardiopulmonary problems ----if Hb≤**8** g/dL
- Critically ill pts and/or those with cardiopulmonary problems ---- if Hgb≤**10** g/dL

- Monitor transfuse pt carefully for adverse reaction
- Wait **3**hrs after transfusion before redrawing labs unless pt clinically unstable or ongoing bl loss

\+

Inadequate response

- 1 U of whole bl or PRBCs increases Ht by **3%**, or the Hb by **1 g/dL**, in a 70-kg non-bleeding adult

\+

Consider

- High bl. volume from fluid infusion
- Incomplete transfusion
- Ongoing bleeding
- Hemolysis
- RBC units near outdate

Thrombocytopenia

Normal platelet count in adults **150,000-450,000**/microL, 2.5 % of the normal population will have a platelet count **<150,000**/microL. A fall in platelet count to this level may herald severe clinical problems, and requires active follow-up, although thrombocytopenia is not usually detected clinically until count has fallen to levels significantly **<100,000**/microL

Suspect

- Bleeding diathesis
- Petichae, echomisis, purpura
- Massive bl transfusion
- Dis. that cause thrombocytopenia as sepsis

Platelet count

<150,000/microL

Rule out pseudo-thrombocytopenia

- If anticoagulation of bl sample is inadequate, the resulting thrombin-induced platelet clumps can be counted as leukocytes by automated cell counters
- Platelet rosetting around neutrophils, monocytes, and rarely, around circulating lymphoma cells . This in vitro phenomenon, induced at room temp. in EDTA-anticoagulated blood has been called "**plateletsatellitism**".
- Administration of murine monoclonal antibody abciximab, which is directed against the GP IIb/IIIa receptor

Review causes

Causes

- **Dilutional thrombocytopenia**; massive bl transfusion
- **Distributional thrombocytopenia**;Splenomegally
- **Decreased production**
  - After viral inf.
  - HIV
  - After Chemo, and radiotherapy
  - Alcohol toxicity
  - Vit B12, folic acid deficiency
  - Cong, acquired bone marrow aplasia "fanconi S"
- **Increased destruction**
  - ITP &SLE
  - Drugs "heparin, vancomycin, pipracilin"
  - Alloimmune destruction "post-transfusion, post-transplant'
  - TTP-HU&DIC
  - Following certain inf. "CMV"
  - HIV
  - Anti-phospholipid S
  - HELLP S
  - Physical destruction during CPB

Workup

PhE

- Fundus & skin for evidence of bleeding, Examination for lymphadenopathy, hepato-splenomegaly, Examination of the stool for occult blood

CBC and peripheral smear;

- Estimation of platelet numbers, morphology, presence or absence of platelet clumping, as well as evaluation of associated white and red blood cell changes cannot be over-emphasized. It is important to use CBC and bl smear to help rule in or out TTP and acute leukemia
- Findings that suggest reduced production of platelets as the cause include presence of small platelets, malignant cells, a leuko-erythroblastic bl picture, leukopenia, anemia, and/or large (macrocytic) red cells
- Large platelets (mega-thrombocytes) on peripheral smear without significant bleeding suggest presence of young, hemostatically active platelets in response to peripheral destruction. However, very large platelets, approaching size of red cells, suggest congenital thrombocytopenia
- Other findings that suggest increased peripheral destruction of platelets as the cause of thrombocytopenia include presence of fragmented red cells, hemolytic anemia, increased serum concentration of LDH
- A presumptive diagnosis of ITP is made when the history (eg, lack of ingestion of a drug, beverage, or food that can cause thrombo-cytopenia), PhE, CBC, and examination of peripheral blsmear do not suggest other etiologies for the isolated thrombocytopenia

Bone marrow aspiration & biopsy

- Is indicated in virtually all pts with unexplained thrombocytopenia severe enough to constitute a risk for major bleeding. The only valid exclusion to this requirement is the pt <60 years of age with a presumptive diagnosis of ITP

Considerhematologicconsultation

ttt

- Stop offending drug, manage **HITS***"p158"*
- Thrombocytopenia as part of a multi-system disorder (eg, DIC)---**ttt cause**
- If ttt of underling cause not possible ---- maintain **safe platelet count***"see next p"*
- If cause (of any degree of severity) is not clear after retesting and initial evaluation---**Consult hematologist**
- **Safety of ttt with antiplatelet agents and anticoagulants** — Risk of bleeding is increased, but it may not be excessive at platelet counts >**50,000**/microL
- **Emergency management of critical bleeding** — Immediate transfusion of platelets, regardless of the etiology of the thrombocytopenia. Adjunctive ttt with high doses of glucocorticoids, as 1000 mg of **methylprednisolone**, and **IVIG** (0.4 - 1 gm) is also appropriate
- **Invasive procedures** *"see next p"*
- **Transfusion trigger** *"see next p"*

Gestational thrombocytopenia

- Mild asymptomatic thrombocytopenia discovered near term, present in about 5 % of pregnant women. The etiology is unknown. It is clinically unimportant, unless count is **<100,000**/microL and there is concern regarding the safety of epidural anesthesia.By definition, this disorder does not cause severe or symptomatic thrombocytopenia, resolvesspontaneously following delivery, and is not associated with thrombocytopenia inthe infant

Platelets transfusion

- "**Safe**" **plateletcount**is an imprecise concept without evidence-based varying from disorder to disorder and pt to pt with the same disorder. Considered if all other aspects of hemostasis are normal
- Platelet >**50**,000/microL ---- not recognized unless a platelet count is performed
- Platelet **30**,000 -**50**,000/microL---rare purpura, even with significant trauma
- Platelet counts of **10**,000 - **30**,000/microL ----- may be asymptomatic, even with usual trauma of everyday activities. However these pts may be at risk for excessive bleeding following more extensive trauma
- Spontaneous bleeding typically do not occur unless platelet counts are <**10**,000/microL. Such ts commonly have some spontaneous bruises and some petechiae, but even they may be entirely asymptomatic on occasion
- Platelet <**5000**/microL (ie,<2% of normal) ----cause critical spontaneous bleeding
- Pts who have thrombocytopenia primarily caused by increased peripheral platelet destruction (eg, ITP) seem to have less risk for bleeding with severe thrombo-cytopenia than pts for whom marrow failure is the principal cause of the thrombocytopenia (eg, aplastic anemia)

Abnormal platelets count

\- May be indicated despite an apparently adequate platelet count if there is known platelet dysfunction and micro-vascular bleeding

\+

ITP & TTP & HIT diagnosed

\+ • Ineffective and rarely indicated "urgent situation" when is due to increased destruction (e.g., ITP)

\-

Prophylaxis indicated

\+ **Surgery**
- In surgical pts with thrombo-cytopenia due to decreased production, prophylactic transfusion rarely indicated >**100** x 1,000,000,000/l. Usually indicated when count <**50** x 1,000,000,000/. (**50** -**100** x1,000,000,000/l) therapy based on the risk of bleeding

Invasive procedures
- >**40**,000 - **50**,000/microL ---safe for interventional procedures as LP
- **80**,000 - **100**,000/microL ---- safe for central neuro-axial blockade
- **30**,000 - **50**,000/microL --- safe for childbirth and tooth extractions
- **Severe thrombocytopenia**--- safe for bone marrow aspiration/biopsy

\-

Surgical pts with micro-vascular bleeding

\- Do not transfuse

\+

- Surgical and obstetric pts with micro-vascular bleeding --- transfuseif count <**50** x 1,000,000,000/l & do not if >**100** x 1,000,000,000/l. &With intermediate platelet counts (**50**-**100** x 1,000,000,000/l), the determe based on pat's risk for more significant bleeding

Chronic ITP
- Although platelet survival in the circulation of pts with ITP and other disorders with accelerated platelet destruction may not be normal, platelet transfusion, supplemented as necessary with high-dose glucocorticoids or IVIG, nearly always provides prompt satisfactory hemostasis, if only for a short duration
- 1 W of prednisone **1** mg/kg/d can be used, abruptly discontinued afterwards. For pts unresponsive to prednisone ---IVIG **0.4 - 1** gm/kg will increase the platelet count to a safe level in 1-3 ds in most pts

Adjust dose

- Therapeutic dose is **1** platelet concentrate / **10** kg bw
- Should be transfused immediately before or during invasive procedure for maximal effectiveness

Inadequate response

- Transfusion of **1** U / **10** kg bw will typically increase count **10**,000/ml
- Obtain a post-transfusion count 10 min. - 1 hr after transfusion

\+

- Pts repeatedly transfused over a prolonged period may become allo-immunized and refractory to platelet transfusion
- Confirmed by demonstration of antibodies to platelets (e.g., to human leukocyte antigens [HLA])
- In such pts, HLA -matched or cross-matched platelets may be required

Heparin induced thrombocytopenia syndrome "HITS"

Type II (HIT-II)

- Immune-mediated disorder characterized by formation of antibodies against heparin-platelet factor 4 complex. Has also been called **heparin-associated immune thrombocytopenia**, **white clot,** and **syndromeheparin-associated thrombocytopenia and thrombosis**,
- **White clot syndrome**refers to platelet-rich arterial thrombosis (rather than fibrin-rich venous thrombosis), which occurs with high frequency in pts who develop this disorder

Type I heparin-induced thrombocytopenia)

- Typically characterized by a lesser fall in platelet count that occurs within the first 2 ds after heparin initiation and often returns to normal with continued heparin administration
- The mechanism is non-immune and appears to be due to direct heparin effect on platelet activation

Pretest probability

Thrombocytopenia

- Count fall >**50** % & nadir >**20**,**000**: **2** points
- Count fall **30** - **50** % & nadir **10**-**19**,**000**: **1** points
- Count fall <**30** % or nadir <**10**,**000**: **0** points

Timing of count fall

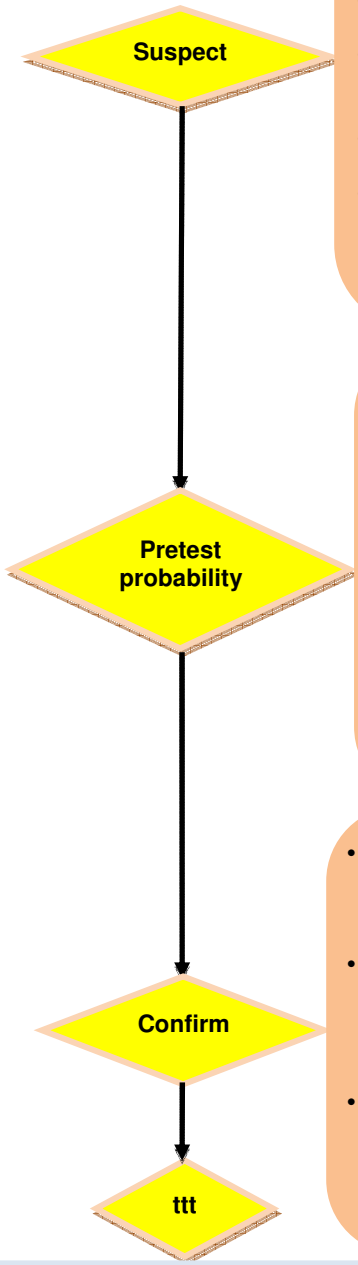
- Clear onset between ds **5** and **10** or platelet count fall at ≤**1** d if prior heparin exposure within the last **30** ds: **2** points
- Consistent with fall at **5** - **10** ds but unclear (eg, missing platelet counts), onset after d **10**, or fall ≤**1** d with prior heparin exposure within **30**-**100** ds: **1** point
- Count fall at <**4** ds without recent exposure: **0** points

Thrombosis & other sequelae

- New thrombosis, skin necrosis, or acute systemic reaction after IV UFH bolus: **2** points
- Progressive or recurrent thrombosis, non-necrotizing skin lesions, or suspected thrombosis which has not been proven: **1** point
- None: **0** points

Other causes for thrombocytopenia

- None apparent: **2** points
- Possible: **1** point
- Definite: **0** points



*Any of the following raise possibility of HIT in a pt begun on heparin therapy within the preceding **5**-**10** ds, or in a pt receiving prolonged ttt of LMWH*

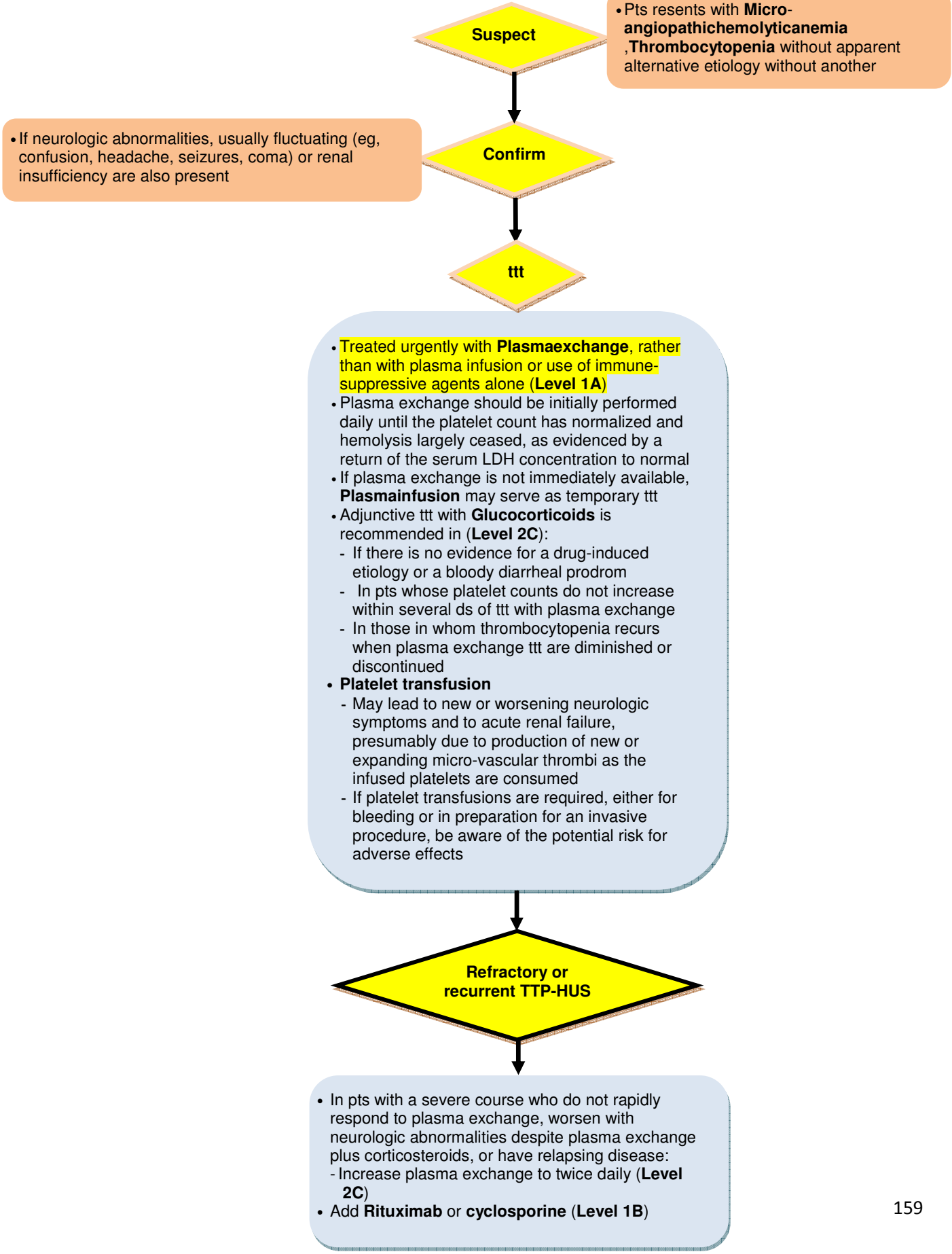
- Onset of otherwise unexplained thrombocytopenia
- Venous or arterial thrombosis associated with thrombocytopenia
- Platelet count fall ≥**50**% from a prior value, even if there is no absolute thrombocytopenia
- Necrotic skin lesions at heparin injection sites
- Acute systemic (anaphylactoid) reactions (eg, fever/chills, tachycardia, hypertension, dyspnea, cardiac arrest) occur after IV heparin bolus injection

- **Low probability** (**0**-**3** points) — Causes for thrombocytopenia other than heparin should be re-evaluated. Lab. testing for HIT is not generally appropriate
- **Intermediate & high probability** (**4**-**8** points) — Pts should have all forms of heparin and LMWH immediately stopped and lab. testing for HIT urgently obtained
- Because results of functional assays for HIT may be delayed for as long as 1 w, they are useful only for final confirmation of diagnosis

- Diagnosis established in a pt with intermediate or high pretest probability and +ve solid phase **ELISA** test for HIT
- The most specific diagnostic tests include serotonin release assays, heparin-induced platelet aggregation assays, and solid phase immunoassays
- Diagnosis is initially made on clinical grounds "assays with the highest sensitivity & specificity may not be readily available & have a slow turnaround time

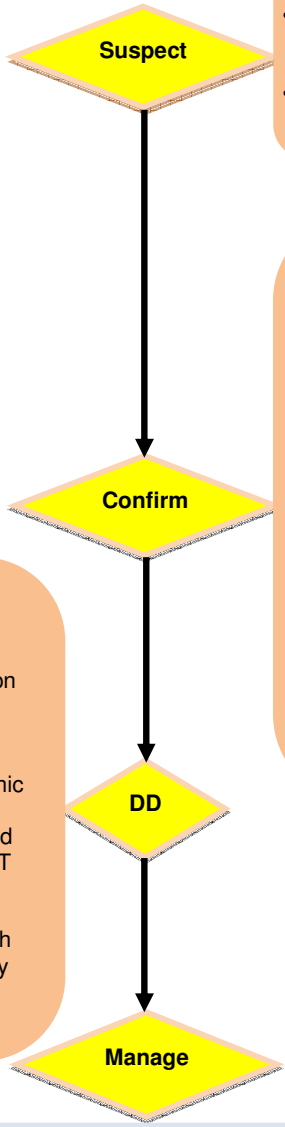
- For intermediate or high pretest probability, and normal renal & hepatic functions --- use non-heparin anti-coagulant (eg**Fondaparinux**) (**Level 1B**). For abnormal renal & normal hepatic function --- use **Argatroban** "standard doses" &**Lepirudin**"reduced doses" **Level 2C**)
- Anticoagulate for at least 2-3 months in absence of a thrombotic event and 3-6 months if occured (**Level 2C**). Warfarin can be started once pt has been stabilized with a non-heparin anticoagulant and the platelet count has recovered to ≥**150**,000/microl. Start with low initial doses of warfarin, rather than high "loading" doses. The non-heparin anticoagulant should be continued for at least 5 ds along with warfarin, until platelet count has stabilized and the INR has reached the intended target range

Thrombotic thrombocytopenic purpra hemolytic uremic syndrome "TTPHUS"



Disseminated intravascular coagulation "DIC"

DIC, also called **Consumptioncoagulopathy** and **Defibrinationsyndrome** is a systemic process producing both



- History (sepsis, trauma, malignancy)
- Petechiae and ecchymoses, bl.oozing from wound sites, IV lines, catheters, and mucosal surfaces
- Moderate to severe thrombocytopenia (<**100**,**000**/microL)

Acute

- **Clinical** (Bleeding, hepatic dysfunction "jaundice", acute renal failure, pulmonary hge, CNS dysfunction "coma, delirium, and transient focal neurologic symptoms. Micro-thrombi, hge"
- **Lab**. Increased thrombin generation (eg, decreased fibrinogen) and increased fibrinolysis (eg, elevated FDPs and D-dimer); degree of abnormality may correlate with extensiveness of organ involvement

Chronic

- The above laboratory studies are variable in chronic DIC because a slower rate of consumption of coagulation factors may be balanced by enhanced synthesis of these ptns. In such pts, the diagnosis may be largely based upon evidence of micro-angiopathy on peripheral blood smear and increased f FDPs and particularly, D-dimer

Severe liver disease

- It may be difficult to distinguish the coagulopathy associated with primary severe liver disease from liver dysfunction often seen in patients with acute DIC

TTP-HUS

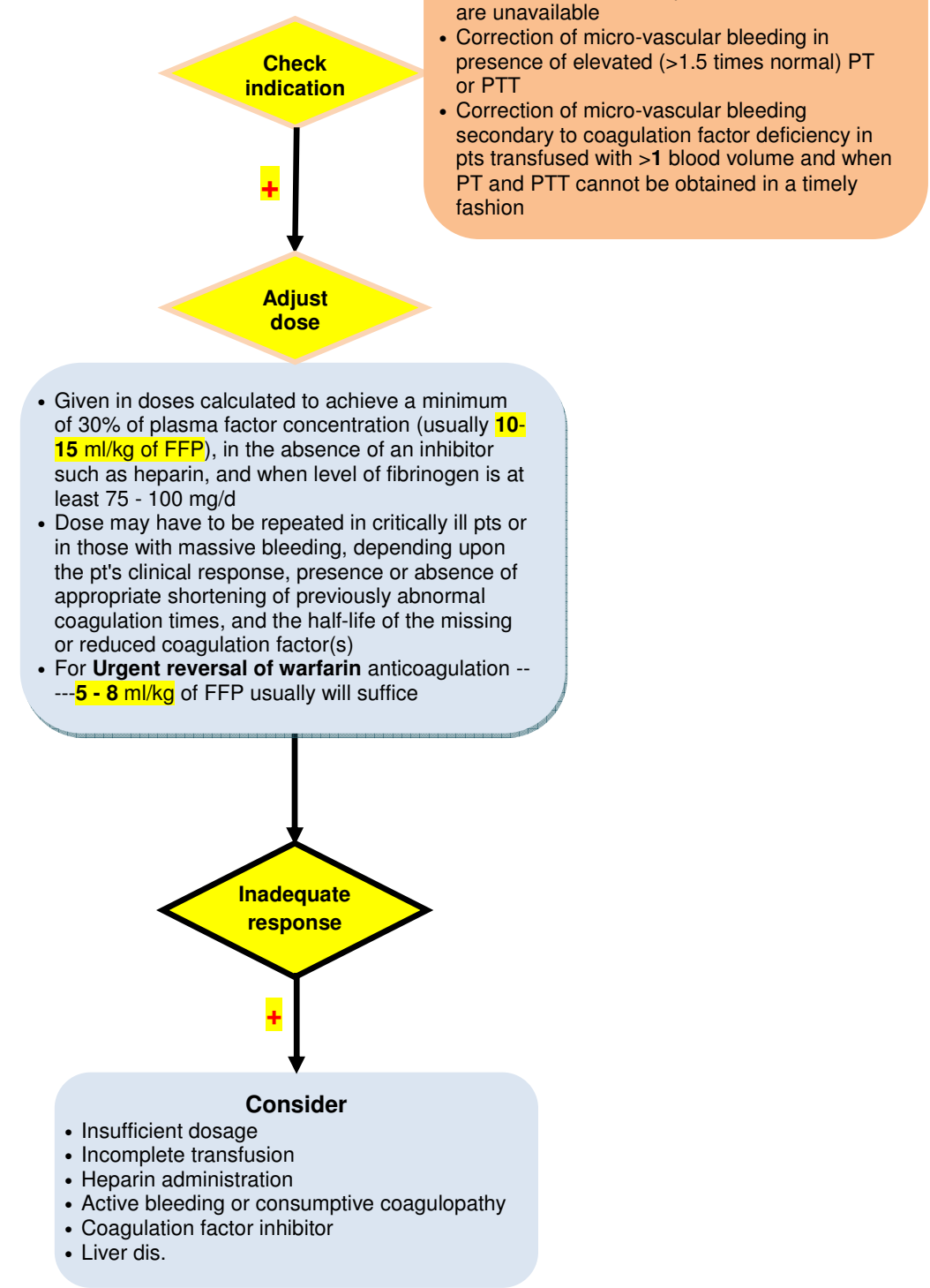
- Pts with TTP-HUS present with thrombocytopenia and a micro-angiopathic blood smear, but usually have normal levels of the coagulation components and little or no prolongation of the PT or aPTT

Heparin-induced thrombocytopenia

- Diagnosis of HIT is initially made on clinical grounds, because the assays with the highest sensitivity and specificity may not be readily available and have a slow turnaround time

- **ttt underlying dis**. (eg, sepsis)
- **Hemodynamic support**
- There is no evidence to support **transfusions** of platelets and coagulation factors in pts who are not bleeding or who are not at high risk of bleeding. Use of one or more of the following supportive modalities for symptomatic pt (**Grade 2C**):
  - **Platelets**&**Coagulationfactors** is justified in pts who have serious bleeding, are at high risk for bleeding (eg, post- surgery), or require invasive procedures. Pts with marked or moderate thrombocytopenia (<**50**,**000**/microL) and serious bleeding should be given platelet
  - Actively bleeding pts with a significantly elevated INR and/or a fibrinogen concentration <**50** mg/dL, should receive **FFB** or **Cryoprecipitate** to keep fibrinogen level >**100** mg/dL
- **Heparin**: generally limited to subset of pts with chronic, compensated DIC who have predominantly thrombotic manifestations
  - Be sure that the pt's anti-thrombin (AT) level is near normal (ie, 80-100 %)

Fresh frozen plasma "FFP" transfusion



Cryoprecepitate transfusion

- Cellular blood component
- Compatibility test unnecessary
- Rh type considered
- It is preferable to use ABO-compatible cryoprecipitate with the recipient's red cells
- Thawed cryoprecipitate should be kept at room temp. and transfused as soon as possible after thawing or within 6 hrs if it is a closed single U or has been pooled prior to freezing
- Should be transfused within 4 hrs if it is an open system or units have been pooled after thawing
- **Cryoprecipitated antihemophilic factor** (**CAF** or "**cryo**") is a concentrate of Factor VIII, von Willebrand's factor, fibrinogen, and Factor XIII
- Each U contains a minimum of **80** U of Factor VIII and typically **250** mg fibrinogen
- If the label indicates "**CryoprecipitatedAHF**, **Pooled**," several U of cryoprecipitate have been pooled into 1 bag, and volume of the pool is indicated on the label
- Each U of FFP contains 2 - 4 mg of fibrinogen /ml. Therefore, it should be noted that each U of FFP delivers equivalent amount of fibrinogen as 2 U of cryoprecipitate

Check indication

- Prophylaxis in non-bleeding peri-operative or peri-partum pats with congenital fibrinogen deficiencies or von Willebrand's disease unresponsive to **DDAVP** ---- Consult hematologist
- Bleeding pts with von Willebrand's dis.
- Correction of micro-vascular bleeding in massively transfused pts with fibrinogen concentrations <**80**-**100** mg/dL (or when cannot be measured in a timely fashion)
- Factor VIII deficiency when Factor VIII concentrate is not available
- Hypo-fibrinogenemia
- Factor XIII deficiency
- Uremic bleeding; decrease bleeding due to uremic platelet dysfunction

+

Adjust dose

- **1** U cryoprecipitate / **10** kg BW raises plasma fibrinogen concentration by **50** mg/dL in absence of continued consumption or massive bleeding
- For **Factor VIII** replacement ---- dose can be calculated assuming **80** U / bag. Current methods of manufacturing typically yield higher Factor VIII content
- For **Fibrinogen** replacement, the dose can be calculated assuming **250** mg / bag
- For **Other indications**, given as **1** U / 10 kg, or a pool of **10** U for an adult

Inadequate response

- Clinical response is usually the best
- Factor VIII activity, fibrinogen, or von Willebrand's factor activity should be measured pre-transfusion, 1 hr after transfusion
- **1** U / 10 kg BW raises plasma fibrinogen by ~ **50** mg/dL in absence of continued consumption or massive bleeding

+

Consider

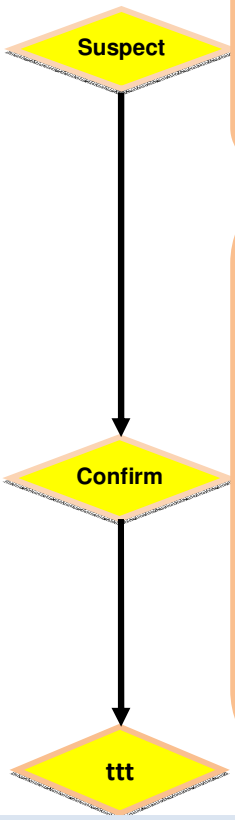
- Inadequate dosage
- Incomplete transfusion
- Presence of a Factor VIII inhibitor
- Bleeding
- IV coagulation

DDVAP

- Standard dose: **0.3 - 0.4** uq /Kg over 30 min.
- Increases in facror VII:C, VIII : Vwf and platelet adhesion
- Indications
  - Hemophilia A
  - Type I von Willebrand disease
  - Uremic platelet dysfunction

Deep venous thrombosis "DVT"

- **Deep vein thrombosis (DVT)** and **acute pulmonary embolism (PE)** are 2 manifestations of the same disorder, **venousthromboembolism** (**VTE**). DVT of the lower extremity is subdivided into 2 categories:
- **Distal** (calf) vein thrombosis, in which thrombi remain confined to the deep calf veins
- **Proximal** vein thrombosis, in which thrombosis involves the popliteal, femoral, or iliac veins. Proximal vein thrombosis is of greater importance clinically, since it is more commonly associated with the development of PE

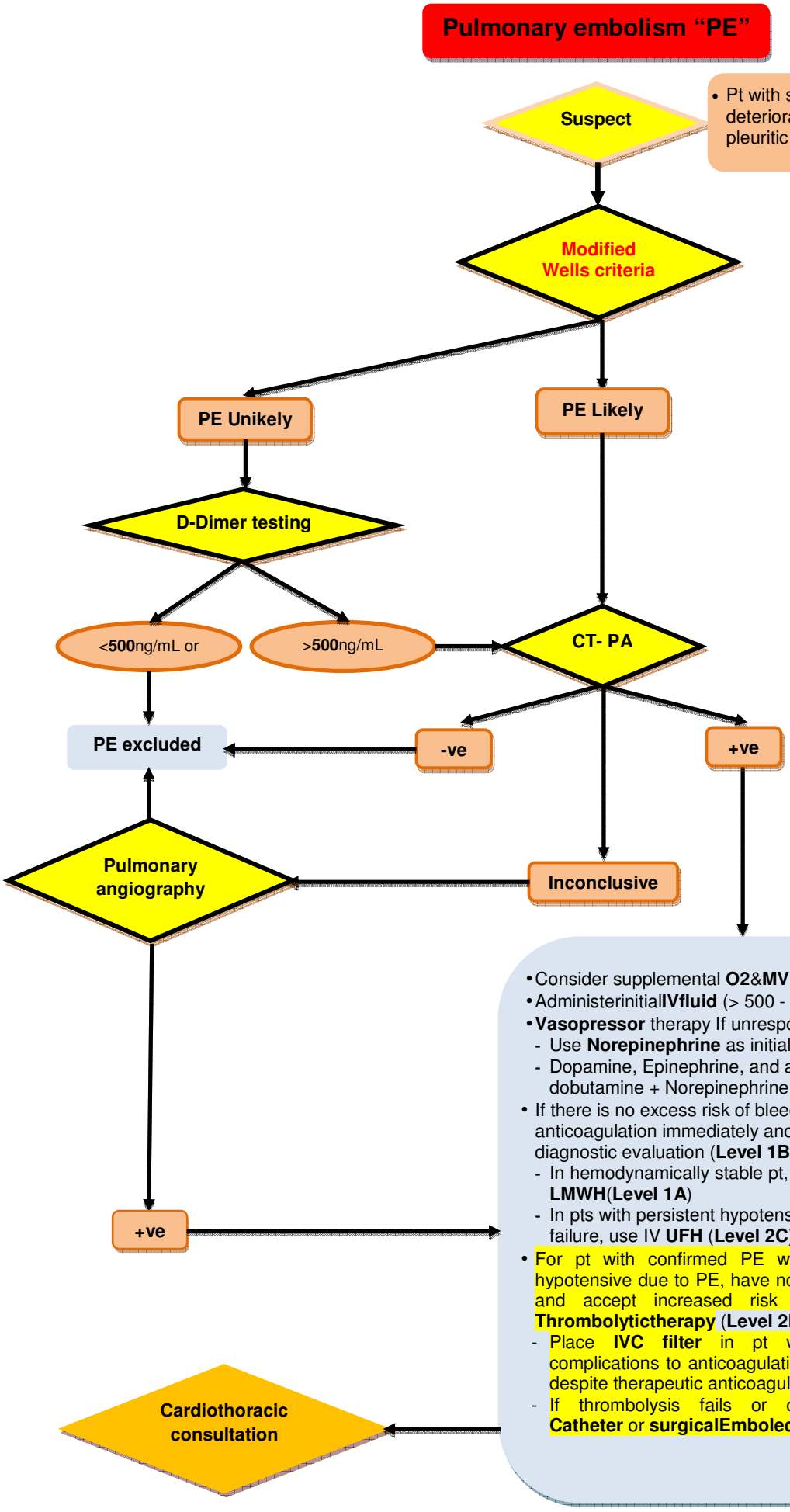


- Presence of risk factors "prolonged immobilization, bed rest, obesity, recent surgery, prior episode of VTE, malignancy, oral contraceptive use, pregnancy or post-partum status, stroke, and lowerextremity trauma
- Swelling, pain, and erythema of the involved extremity

- **PhE**------palpable cord (reflecting a thrombosed vein), calf or thigh pain, unilateral edema or swelling with a difference in calf diameters, warmth, tenderness, erythema, and/or superficial venous dilation.pain and tenderness in the thigh along the course of the major veins ("**Painful deep vein syndrome**"). Tenderness on deep palpation of the calf muscles is suggestive, but not diagnostic. **Homan's sign** is also unreliable
- **Compression Ultrasonography** ----- if negative, do serial US or IPG
- **Duplex** or **color-flowUS** may be utilized, although there is no proven superiority over compression US alone
- **Venography** when noninvasive testing is not clinically feasible or the results are equivocal
- **MRI** appears accurate for lower extremity DVT (**level II**), it is more expensive than US or IPG and is generally not indicated as the initial diagnostic test

- Initial ttt should be started acutely with **UFH**, **LMWH**, or **Fondaparinux**
- When UFH is used, the dose should be sufficient to prolong aPTT to **1.5** - **2.5** times the mean of the control value, or the upper limit of the normal aPTT range
- **Warfarin** should be initiated simultaneously with the heparin, at an initial oral dose of approximately **5** mg/d. In elderly pts and in those at high risk of bleeding or who are undernourished, debilitated, or have HF or liver disease, the starting dose should be reduced. The heparin product can be discontinued on d 5 or 6 if the INR has been therapeutic for 2 consecutive ds
- Oral anticoagulation with warfarin should prolong the INR to a target of **2.5** (range: **2.0**-**3.0**). If oral anticoagulants are CI or inconvenient, long-term therapy can be undertaken with either adjusted-dose UFH, LMWH, or Fondaparinux
- Pts with hemodynamically unstable PE or massive iliofemoral thrombosis (ie, phlegmasiaceruleadolens), and who are also at low risk to bleed, are the most appropriate candidates for use of thrombolytic agents, surgical **Thrombectomy**, or percutaneous mechanical thrombectomy
- Pts with a first DVT due to a reversible or time-limited risk factor (eg, trauma, surgery) and those with a first unprovoked or provoked episode of distal DVT should be treated for at least 3 months
- **Inferior vena caval filter** placement is recommended when there is a CI to, or a complication of, anticoagulant therapy in an individual with, or at high risk for, proximal vein thrombosis or PE

Pulmonary embolism "PE"



- Pt with sudden onset of **Dyspnea**, deterioration of existing dyspnea, or onset of pleuritic chest pain without apparent cause

Modified Wells criteria

Criterion	Score*
Clinical signs or symptoms of DVT	3
Alternative diagnosis less likely than PE	3
Heart rate 100 beats per minute	1.5
Immobilization (>3 days) or surgery in last 4 weeks	1.5
Previous history of DVT or PE	1.5
Hemoptysis	1
Active cancer within the last 6 months	1

DVT = deep venous thrombosis; PE = pulmonary embolism. Source: Lim W, Korenstein D. Physicians' Information and Education Resource: Pulmonary embolism. Available to ACP members at http://pier.acponline.org/physicians/diseases/d239/d239.html.

*Modified Wells criteria: <2 points = low risk for PE; 2-6 points = moderate risk for PE; >6 points = high risk for PE. Simplified Wells criteria: ≤4 points = PE unlikely; >4 points = PE likely.

- Consider supplemental **O2**&**MV** if indicated
- Administer initial **IVfluid** (> 500 - 1000 mL)
- **Vasopressor** therapy If unresponsive to IV fluids
  - Use **Norepinephrine** as initial agent (**Level 2C**)
  - Dopamine, Epinephrine, and a combination of dobutamine + Norepinephrine may be effective
- If there is no excess risk of bleeding, initiate empiric anticoagulation immediately and continue during the diagnostic evaluation (**Level 1B**)
  - In hemodynamically stable pt, use SC **LMWH**(**Level 1A**)
  - In pts with persistent hypotension or severe renal failure, use IV **UFH** (**Level 2C**)
- For pt with confirmed PE who are persistently hypotensive due to PE, have no CI to thrombolysis, and accept increased risk of bleeding, start **Thrombolytictherapy** (**Level 2B**)
  - Place **IVC filter** in pt who have CI, or complications to anticoagulation), or recurrent PE despite therapeutic anticoagulation (**Level 2C**).
  - If thrombolysis fails or contraindicated, do **Catheter** or **surgicalEmbolectomy** (**Level 2C**)

Critical care drug summary

Vasopressors & Inotropes

Drug	Receptor affinity	Dose	Adverse effects	Special consideration
Nor-epinephrine	Alfa1 > B1	**0.05 – 1.25**uq/Kg/min.	Tachycardia& peripheral GI ischemia	
Epinephrine	B1 > Alfa1 Low dose ---alfa Large dose ----B	**0.05 – 1** uq/Kg/min.	Tachycardia& peripheral GI ischemia	
Dopamine	DA BI Alfa1	**<5**uq/Kg/min. **5-10**uq/Kg/min. **>10**uq/Kg/min.	Tachycardia Arrythmias	Renal protective doses should not be used
Vasopressin	VI	**0.01 – 0.04** U/min.	Cardiac&mesentric ischemia, skin lesion	Doses > 0.04 U/min. causes cardiac ischemia
Dobutamine	BI&B2	**5-20**uq/Kg/min.	Arrythmias Hypotension	

Relative vasopressor activity

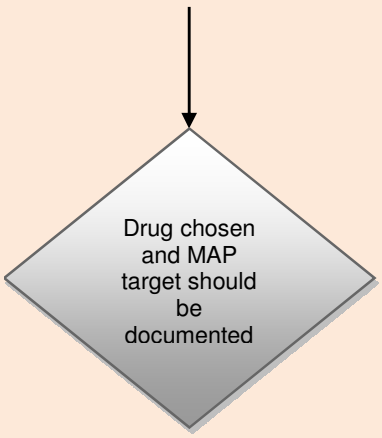
Alfa activity

Norepinephrine = epinephrine > Dopamine

Strongest → Weakest

Dopamine > epinephrine> Norepinephrine

B activity



Anti-arrythmic drugs

Drug	Indication	Dose	Special consideration
Adenosine	• Paroxysmal SVT with re-entrant circuits includes AV node" AVNRT, AVRT"	**6** mg IV bolus, if unsuccessful give up to 2 doses of **12** mg after 1-2 min. interval	• Has extremely short half life "10-15s" • Give as a rapid bolus into a rapid running IV infusion, or followed by rapid flush • Warn pt of undesirable side effects as nusea, flushing, and chest discomfort • CI ---- asthma
Amiodrone	• To control hemo-dynamically stable mono-morphic VT & poly-morphic VT and wide complex tachycardia of uncertain origin • To control rapid ventricular rate caused by accessory pathway conduction in pre-excited atrial arrhythmias "AF",or achieve electrical cardioversion • After successful electrical cardioverion to achieve chemical cardioversion, or to increase the likehood of further electrical cardioversion success	**300** mg IV over 10-60 min., according to the hemodynamic stability , followed by **900** mg infusion over 24 hrs	• It is preferable to other anti- arrythmics for atrial, and ventricular arrythmias, if cardiac functions are impaired • Major side effects as hypotension and bradycardia can be minimized by slowing the infusion rate. • Give via CV catheter whenever, possible, otherwise given via a large peripheral line in emergency "it causes thrombo-phelbitis"
B-blockers	• Narrow complex regular tachycardia uncontrolled by vagal maneuvers, or adenosine in pts with preserved cardiac function • Control rate in AF, flutter when ventricular function is maintained	• **Propanolol** "B1,2 " • **100**uq/Kg IV slowly in 3 equal doses at 2-3 min. interval • **Esmolol**"selective B1" • **500**uqlKg IV over 1 min., followed by titrated doses of **50**-**200**uq/Kg /min.	• Side effects include;bradycardia, bronchospasm, AV conduction delay, and hypotension • CI ---- hypotension, severe CHF, 2nd, 3rd HB, and lung diseases associated with bronchospasm
Verapamil	• As B blockers	• **2.5-5** mg IV over 2 min. • In absence of a therapeutic response or undesirable side effects, give repeated doses of **5**-**10** mg over 15-20 min. to a maximum of **20** mg	• If given in VT ---- cause cardiovascular collapse • Reduce contractility ,and COP in pt with severe LV dysfunction
Mg	• Ply-morphic VT "torsaes de pointes" • Digoxin toxicity	• **2**gm peripherally over 2 min. • May repeat once if necessary	

Emergent Anti-hypertensive drugs

Drugs	Dose	Onset	Duration	Adverse effects
Esmolol	Bolus **500**uq Continuous **25-300**uq/Kg/min. Titrate by **50**uq/Kg /min./4 min	**60** s	**10-20** min.	Bradycardia
Labetalol	Bolus **10-20** mg Double dose at 10 min. interval till **80** mg Continuous **2-10** mg/min. Titrate by **1** mg /min.	**2-5** min.	**2-6**hr	Bradycardia
Verapamil	**0.075 -0.15** mg/Kg	**3-5** min.	**0.5-6**hr	Bradycardia
Hydralazine	Bolus **2.5 -5** mg Continuous **10-200**uq/min.	**5-15** min.	**3-10**hr **10-20** min.	Lupus Tachycardia
Nitroglycerine	**10-200**uq/min Titrate increase **5-10**uq / min. /5-10 min.0.25-0.5	**2-5** min.	**10-20** min.	Tachycardia
Na nitroprusside	**0.5-10**uq/Kg/min. Titrate **0.25-0.5**uq/Kg/min./2-3 min.	**3**s	**1-2**min.	Tachyphylaxis Muscle twich

Oral anticoagulant

Mechanical prosthetic heart valves
- The recommended target INRs are given in the table
- If embolic event occurs during anticoagulation within target, elevate INR target or add anti-platelet drugs (2C)

Bio-prosthetic heart valve
- In the mitral position should receive 3 ms of anticoagulation with warfarin with an INR target of **2.5** (1B)
- History of systemic embolism ---at least 3 ms anticoagulation with warfarin with an INR target of **2.5** (1C)
- Left atrial thrombus at surgery should receive warfarin until the clot has resolved with an INR target of **2.5** (1C)
- Other pro-hrombotic risk factors, as AF and low ventricular EF, should receive warfarin with INR target of **2.5** (1C)

MI and cardiomyopathy
- MI ----- INR target for anticoagulation is **2.5** (2A)
- Dilated cardiomyopathy --- INRtarget of **2.5** (2C)

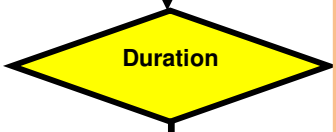
VTE
- 1st episodes of VTE ---- INR target of **2.5** (1A)
- Warfarin + parenteral anticoagulation (1A) which continue for at least 5 ds and INR **>2** for at least 24 hrs (1C)
- Recurrent VTE whilst anti-coagulated and within the therapeutic range --- INR target of**3.5** (2C)

Anti-phospholipid S
- INR target should be **2.5**

AF
- INR target should be **2.5**(1A)

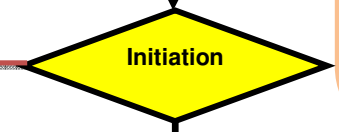
Cardio-version
- Anticoagulated with warfarin for at least 3 ws prior to and 4 ws post cardioversion with a target INR of **2.5** (2C). Target INR of **3.0** can be used prior to the procedure

Mitral stenosis or regurgitation
- Who have AF (1A) or a history of systemic embolism (1A) or left atrial thrombus (1A) or an enlarged leftatrium (2C) should receive warfarin with an INR target of **2.5**



Duration of initial anticoagulation
- Proximal VTE--- at least **3**ms(1A)
- Isolated calf veinDVT is employed,ttt of such clots can be restricted to **6**ws (1A)

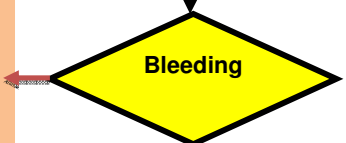
Continued anticoagulation beyond initial period of 3 ms
- Not recommended inpts with VTE provoked by surgery (1B). pts with VTE provoked by non-surgical transienttrigger factors (1B) ,and with VTE confined to the calf (i.e. not extendinginto popliteal vein) (1A)
- Pts with unprovoked proximal VTE----- long-term anticoagulation, takeintoaccount information that help predict risk ofrecurrence and risk of bleeding in individual pt(2B)



Rapid induction regimens for pts with acute Thrombosis
- No evidence to suggest a **10** mg loadingdose is superior to a **5** mg loading dose. However in theelderly lower initiation doses or age-adjusted doses maybe more appropriate as they lead to fewer high INRs (2B)

Induction of anticoagulation in outpatients with AF
- For outpatients who do not require rapid anticoagulationa slow-loading regimen is safe and achieves therapeuticanti-coagulation in majority of pts within 3-4 ws (2C)

- Pre-operative carries a lower risk of bleeding thanPO bridging. PO bridging should not bestarted until at least 48 hrs after high bleeding risk surgery(1C)
- Pts with VTE > 3 ms earlier can be given prophylactic LMWH (or a suitable alternative) rather than bridging therapy (2C)
- Pts with low risk AF (no prior stroke or TIA) do notrequire bridging therapy (2C)
- Pts with a bileaflet aortic MHV with no other riskfactors do not require bridging (2C)
- Pts with a VTE within the previous 3 ms,pts with AF and previous stoke or TIA or multipleother risk factors, and pts with a mitral MHVshould be considered for bridging therapy (2C)



Major bleeding
- Stocka 4-factor prothrombin complex concentrate"PCC"(1C)
- Emergency reversal ---**25–50** u/kg 4-factorprothrombin complex concentrate and 5 mg IVvitamin K (1B)
- FFB produces suboptimal anticoagulationreversal and should only be used if prothrombincomplex concentrate is not available (1C)
- Recombinant factor VIIa not used for emergent reversal (1B)

Non major bleeding
- Pts with an INR >**5.0**& not bleeding ---- 1-2 doses of warfarin withheld & reduce theirmaintenance dose (1B)
- Investigate for causes of elevated INR (1C)
- NR >**8. 0**---- 1–5 mg of oral vitamin K (1B)

Emergency surgery for pts on warfarin

- For surgery that requires reversal of warfarin and that can be delayed for 6–12 h, the INR can be corrected by giving IV vitamin K. For surgery that requires reversal of warfarin and which cannot be delayed, for vitamin K to have time to take effect the INR can be corrected by giving PCC and IV vitamin K. PCC should not be used to enable elective or non-urgent surgery (2C)

TBI

- All pts on with TBI should havetheir INR measured as soon as possible (1C)
- A lower threshold for performing a head CT scan should be used for pts on warfarin (2C)
- Pts on warfarin presenting with a strong suspicionof intra-cerebral bleed should have their anticoagulationreversed before the results of any investigations (2C)

sub-therapeutic anticoagulation

Sub-therapeutic anticoagulation in the 1st m after acuteVTE

- Bridging therapy be considered if theINR ɔ-therapeutic within the 1st m of acute VTE

Pt on anti-platlets

Pts on antiplatelet therapy who develop anindication for warfarin

- Pts receiving an anti-platelet agent as 1ryprophylaxis for cardiovascular dis. on developing an indication forwarfarin should stop their antiplatelet agent (1B)
- Pts with peripheral artery dis. or previousischaemic stroke on antiplatelet therapy should stopthis agent if warfarin is commenced (1B)
- Pts on aspirin or clopidogrel as 2ndry prophylaxiswith stable IHD (often defined as>12 ms following acute MI)should stop their antiplatelet agent while being treatedwith warfarin (2B)
- Pts on a single antiplatelet agent <12 msfollowing an ACS, who require to start warfarin therapyshould continue aspirin therapy until 12 ms postACS, unless they are regarded as having a high bleedingrisk (2B)
- Pts on aspirin and clopidogrel, following an ACS orstent placement, who develop an indication for warfarinshould be carefully assessed for bleeding risk anddiscussed with their cardiologist, with a view to introducingwarfarin and minimizing the duration of tripletherapy (2C) . When combined warfarin and single antiplatelet agent are indicated, consideration should be given to use ofaspirin given the higher bleeding risk associated withclopidogrel (2C)

Pts on warfarin who develop an indication for antiplatelet agents

- Pts requiring a coronary artery stent, should beconsidered for bare metal stent (rather than drugelutingstent) which would only necessitate triple therapy for 4 ws, followed by aspirin and warfarin to 12 ms (2B)
- Pts who do not undergo PCI should be considered for 4 ws triple therapy, after which clopidogrel should be stopped, and aspirin continued for a further 11 ms (2C)

Recommended target INRs for mechanical heart valves

(Level 2B) [adapted from Vahanian et al (2007), copyright (2007),with premission from Oxford University Press].

Prosthesis Thrombogenicity*	INR target No patient risk factors	INR target Patient-related risk factors†
Low	2·5	3·0
Medium	3·0	3·5
High	3·5	3·5‡

*Prosthesis thrombogenicity: Low: Carbomedics (aortic position), Medtronic Hall, St Jude Medical (without silzone), Medium: Bjork-Shiley, other bileaflet valves; High: Starr-Edwards, Omniscience, Lillehei-Kaster.

†Patient-related risk factors for thrombosis: Mitral, tricuspid or pulmonary position; Previous arterial thromboembolism; Atrial fibrillation; Left atrium diameter >50 mm; Mitral stenosis of any degree; Left ventricular ejection fraction <35%; Left atrial dense spontaneous echo contrast.

‡Was 4·0 in Vahanian *et al* (2007).

UFH

CI
- Hypersensitivity to heparin
- Increased risk for hemorrhagic complications
- Pts who are actively bleeding
- Thrombocytopenia
- <72 hrsPO
- Recent hemorrhagic stroke

- Use heparin 1000 units/mL vial for bolus
- Use heparin 25,000 units/500 mL D5W premixed bags

Check CI

Prescribe

- The prescriber initiate heparin protocol by writing an order in the order form
- The order must specify the intended dosing regimen and if an initial bolus is desired
  - **Low** Intensity Regimen – acute MI treated with alteplase, tenecteplase orabciximab/eptifibatide/tirofiban*(note: no bolus is recommended if <6 hrs from arterial sheath removal)*
  - **Medium** Intensity Regimen – non-ST segment myocardial infarction, mechanical valve
  - **High** Intensity Regimen – established VTE, ventricular/atrial thrombus

Review initial order

Regimen	Bolus Dose (units/kg)	Maximum Bolus (units)	Initial Infusion (units/kg/hr)	Maximum Initial Infusion Rate (units/hr)
Low	60	4000	12	1000
Medium	70	7000	15	1400
High	80	10,000	18	2000

Make calculations using actual body weight

Order labs

- Stat baseline aPTT and INR/P
- Hematocrit and platelet count every other day until D 14, beginning the day of initiation
- Stat aPTT 6 hrs after initiation of heparin
- Repeat stat aPTT 6 hrs after each adjustment

Titration

- Stat aPTT 6 hrs after initiation and 6 hrs after any dose change. Adjust heparin infusion as indicated in the dosing adjustment table until aPTT is therapeutic. Use supplemental bolus if ordered
- Once 3 consecutive aPTTs (drawn / 6 hrs) are therapeutic, order routine aPTT only / 24 hrs. If dose adjustment again becomes necessary, recheck aPTT in 6 hrs and repeat the process

Monitor

- /8 hrs: Inspect line/surgical/wound sites for bleeding & check pt forematomas, bruising, and respiratory symptoms
- Provider should be notified if:
  - Baseline aPTT>**34**s or baseline INR >**1.2**
  - Hgb decreases by >**2** g/dL from baseline or platelets <**100**,000/microliteror platelets decrease by >1/3 of baseline value
  - aPTT is <38 s or >84 s if pt is on low or medium intensity regimen
  - aPTT is >45 s or >85 s if pt is on higintensity regimen
  - Pt has any deterioration in neurologic status

Titration

Low and Medium Intensity (Arterial Thrombosis) Heparin Anticoagulation Dose Adjustments

aPTT (seconds)	Bolus/Hold	Infusion
<34	Give supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 ml /hr
34 37	Give ½ supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 mL/hr
38-44	0	↑ 50 units/hr = ↑ 1 mL/hr
45-54	0	NO CHANGE
55-64	0	↓ 50 units/hr = ↓ 1 mL/hr
65 84	0	↓ 100 units/hr = ↓ 2 mL/hr
85-100	Hold infusion 1 hour & inform MD	↓ 150 units/hr – ↓ 3 mL/hr
101-125	Hold infusion 1 hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr
>125	Hold infusion 1 ½ hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr

High Intensity (Venous Thromboembolism) Heparin Anticoagulation Dose Adjustments

aPTT (seconds)	Bolus/Hold	Infusion
<34	Give supplemental bolus if ordered & inform MD	↑ 100 units/hr – ↑ 2 mL/hr
34-44	Give ½ supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 ml /hr
45 54	0	↑ 50 units/hr = ↑ 1 mL/hr
55-70	0	NO CHANGE
71-85	0	↓ 100 units/hr = ↓ 2 mL/hr
86-100	Hold infusion 1 hour & inform MD	↓ 150 units/hr = ↓ 3 ml /hr
101-125	Hold infusion 1 hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr
>125	Hold infusion 1 ½ hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr

- If 2 consecutive aPTTs are >125 s, pt should not be maintained on the heparin protocol ---Consult Hematologist
- If 2 consecutive aPTTs are sub-therapeutic ---Consult hematologist

Oral anticoagulant

Indications &Target INR

Mechanical prosthetic heart valves
- The recommended target INRs are given in the table
- If embolic event occurs during anticoagulation within target, elevate INR target or add anti-platelet drugs (2C)

Bio-prosthetic heart valve
- In the mitral position should receive 3 ms of anticoagulation with warfarin with an INR target of **2.5** (1B)
- History of systemic embolism ---at least 3 ms anticoagulation with warfarin with an INR target of **2.5** (1C)
- Left atrial thrombus at surgery should receive warfarin until the clot has resolved with an INR target of **2.5** (1C)
- Other pro-hrombotic risk factors, as AF and low ventricular EF, should receive warfarin with INR target of **2.5** (1C)

MI and cardiomyopathy
- MI ----- INR target for anticoagulation is **2.5** (2A)
- Dilated cardiomyopathy --- INRtarget of **2.5** (2C)

VTE
- 1st episodes of VTE ---- INR target of **2.5** (1A)
- Warfarin + parenteral anticoagulation (1A) which continue for at least 5 ds and INR **>2** for at least 24 hrs (1C)
- Recurrent VTE whilst anti-coagulated and within the therapeutic range --- INR target of**3.5** (2C)

Anti-phospholipid S
- INR target should be **2.5**

AF
- INR target should be **2.5**(1A)

Cardio-version
- Anticoagulated with warfarin for at least 3 ws prior to and 4 ws post cardioversion with a target INR of **2.5** (2C). Target INR of **3.0** can be used prior to the procedure

Mitral stenosis or regurgitation
- Who have AF (1A) or a history of systemic embolism (1A) or left atrial thrombus (1A) or an enlarged leftatrium (2C) should receive warfarin with an INR target of **2.5**

Duration

Duration of initial anticoagulation
- Proximal VTE--- at least **3**ms(1A)
- Isolated calf veinDVT is employed,ttt of such clots can be restricted to **6**ws (1A)

Continued anticoagulation beyond initial period of 3 ms
- Not recommended inpts with VTE provoked by surgery (1B). pts with VTE provoked by non-surgical transienttrigger factors (1B) ,and with VTE confined to the calf (i.e. not extendinginto popliteal vein) (1A)
- Pts with unprovoked proximal VTE----- long-term anticoagulation, takeintoaccount information that help predict risk ofrecurrence and risk of bleeding in individual pt(2B)

Initiation

Rapid induction regimens for pts with acute Thrombosis
- No evidence to suggest a **10** mg loadingdose is superior to a **5** mg loading dose. However in theelderly lower initiation doses or age-adjusted doses maybe more appropriate as they lead to fewer high INRs (2B)

Induction of anticoagulation in outpatients with AF
- For outpatients who do not require rapid anticoagulationa slow-loading regimen is safe and achieves therapeuticanti-coagulation in majority of pts within 3-4 ws (2C)

Pre-operative

- Pre-operative carries a lower risk of bleeding thanPO bridging. PO bridging should not bestarted until at least 48 hrs after high bleeding risk surgery(1C)
- Pts with VTE > 3 ms earlier can be given prophylactic LMWH (or a suitable alternative) rather than bridging therapy (2C)
- Pts with low risk AF (no prior stroke or TIA) do notrequire bridging therapy (2C)
- Pts with a bileaflet aortic MHV with no other riskfactors do not require bridging (2C)
- Pts with a VTE within the previous 3 ms,pts with AF and previous stoke or TIA or multipleother risk factors, and pts with a mitral MHVshould be considered for bridging therapy (2C)

Bleeding

Major bleeding
- Stocka 4-factor prothrombin complex concentrate"PCC"(1C)
- Emergency reversal ---**25–50** u/kg 4-factorprothrombin complex concentrate and 5 mg IVvitamin K (1B)
- FFB produces suboptimal anticoagulationreversal and should only be used if prothrombincomplex concentrate is not available (1C)
- Recombinant factor VIIa not used for emergent reversal (1B)

Non major bleeding
- Pts with an INR >**5.0**& not bleeding ---- 1-2 doses of warfarin withheld & reduce theirmaintenance dose (1B)
- Investigate for causes of elevated INR (1C)
- NR >**8. 0**---- 1–5 mg of oral vitamin K (1B)

Emergency surgery for pts on warfarin

- For surgery that requires reversal of warfarin and that can be delayed for 6–12 h, the INR can be corrected by giving IV vitamin K. For surgery that requires reversal of warfarin and which cannot be delayed, for vitamin K to have time to take effect the INR can be corrected by giving PCC and IV vitamin K. PCC should not be used to enable elective or non-urgent surgery (2C)

TBI

- All pts on with TBI should havetheir INR measured as soon as possible (1C)
- A lower threshold for performing a head CT scan should be used for pts on warfarin (2C)
- Pts on warfarin presenting with a strong suspicionof intra-cerebral bleed should have their anticoagulationreversed before the results of any investigations (2C)

Sub-therapeutic anticoagulation

Sub-therapeutic anticoagulation in the 1st m after acuteVTE

- Bridging therapy be considered if theINR ɔ-therapeutic within the 1st m of acute VTE

Pts on antiplatelet therapy who develop anindication for warfarin

- Pts receiving an anti-platelet agent as 1ryprophylaxis for cardiovascular dis. on developing an indication forwarfarin should stop their antiplatelet agent (1B)
- Pts with peripheral artery dis. or previousischaemic stroke on antiplatelet therapy should stopthis agent if warfarin is commenced (1B)
- Pts on aspirin or clopidogrel as 2ndry prophylaxiswith stable IHD (often defined as>12 ms following acute MI)should stop their antiplatelet agent while being treatedwith warfarin (2B)
- Pts on a single antiplatelet agent <12 msfollowing an ACS, who require to start warfarin therapyshould continue aspirin therapy until 12 ms postACS, unless they are regarded as having a high bleedingrisk (2B)
- Pts on aspirin and clopidogrel, following an ACS orstent placement, who develop an indication for warfarinshould be carefully assessed for bleeding risk anddiscussed with their cardiologist, with a view to introducingwarfarin and minimizing the duration of tripletherapy (2C) . When combined warfarin and single antiplatelet agent are indicated, consideration should be given to use ofaspirin given the higher bleeding risk associated withclopidogrel (2C)

Pts on warfarin who develop an indication for antiplatelet agents

- Pts requiring a coronary artery stent, should beconsidered for bare metal stent (rather than drugelutingstent) which would only necessitate triple therapy for 4 ws, followed by aspirin and warfarin to 12 ms (2B)
- Pts who do not undergo PCI should be considered for 4 ws triple therapy, after which clopidogrel should be stopped, and aspirin continued for a further 11 ms (2C)

Pt on anti-platlets

Recommended target INRs for mechanical heart valves

(Level 2B) [adapted from Vahanian et al (2007), copyright (2007),with premission from Oxford University Press].

Prosthesis Thrombogenicity*	INR target No patient risk factors	INR target Patient-related risk factors†
Low	2·5	3·0
Medium	3·0	3·5
High	3·5	3·5‡

*Prosthesis thrombogenicity: Low: Carbomedics (aortic position), Medtronic Hall, St Jude Medical (without silzone), Medium: Bjork-Shiley, other bileaflet valves; High: Starr-Edwards, Omniscience, Lillehei-Kaster.

†Patient-related risk factors for thrombosis: Mitral, tricuspid or pulmonary position; Previous arterial thromboembolism; Atrial fibrillation; Left atrium diameter >50 mm; Mitral stenosis of any degree; Left ventricular ejection fraction <35%; Left atrial dense spontaneous echo contrast.

‡Was 4·0 in Vahanian *et al* (2007).

UFH

CI
- Hypersensitivity to heparin
- Increased risk for hemorrhagic complications
- Pts who are actively bleeding
- Thrombocytopenia
- <72 hrsPO
- Recent hemorrhagic stroke

- Use heparin 1000 units/mL vial for bolus
- Use heparin 25,000 units/500 mL D5W premixed bags

Check CI

Prescribe

- The prescriber initiate heparin protocol by writing an order in the order form
- The order must specify the intended dosing regimen and if an initial bolus is desired
  - **Low** Intensity Regimen – acute MI treated with alteplase, tenecteplase orabciximab/eptifibatide/tirofiban *(note: no bolus is recommended if <6 hrs from arterial sheath removal)*
  - **Medium** Intensity Regimen – non-ST segment myocardial infarction, mechanical valve
  - **High** Intensity Regimen – established VTE, ventricular/atrial thrombus

Review initial order

Regimen	Bolus Dose (units/kg)	Maximum Bolus (units)	Initial Infusion (units/kg/hr)	Maximum Initial Infusion Rate (units/hr)
Low	60	4000	12	1000
Medium	70	7000	15	1400
High	80	10,000	18	2000

Make calculations using actual body weight

Order labs

- Stat baseline aPTT and INR/P
- Hematocrit and platelet count every other day until D 14, beginning the day of initiation
- Stat aPTT 6 hrs after initiation of heparin
- Repeat stat aPTT 6 hrs after each adjustment

Titration

- Stat aPTT 6 hrs after initiation and 6 hrs after any dose change. Adjust heparin infusion as indicated in the dosing adjustment table until aPTT is therapeutic. Use supplemental bolus if ordered
- Once 3 consecutive aPTTs (drawn / 6 hrs) are therapeutic, order routine aPTT only / 24 hrs. If dose adjustment again becomes necessary, recheck aPTT in 6 hrs and repeat the process

Monitor

- /8 hrs: Inspect line/surgical/wound sites for bleeding & check pt foreматomas, bruising, and respiratory symptoms
- Provider should be notified if:
  - Baseline aPTT>**34**s or baseline INR >**1.2**
  - Hgb decreases by >**2** g/dL from baseline or platelets <**100**,000/microliteror platelets decrease by >1/3 of baseline value
  - aPTT is <38 s or >84 s if pt is on low or medium intensity regimen
  - aPTT is >45 s or >85 s if pt is on higintensity regimen
  - Pt has any deterioration in neurologic status

Titration

Low and Medium Intensity (Arterial Thrombosis) Heparin Anticoagulation Dose Adjustments

aPTT (seconds)	Bolus/Hold	Infusion
<34	Give supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 mL/hr
34-37	Give ½ supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 mL/hr
38-44	0	↑ 50 units/hr = ↑ 1 mL/hr
45-54	0	NO CHANGE
55-64	0	↓ 50 units/hr = ↓ 1 mL/hr
65-84	0	↓ 100 units/hr = ↓ 2 mL/hr
85-100	Hold infusion 1 hour & inform MD	↓ 150 units/hr = ↓ 3 mL/hr
101-125	Hold infusion 1 hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr
>125	Hold infusion 1 ½ hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr

High Intensity (Venous Thromboembolism) Heparin Anticoagulation Dose Adjustments

aPTT (seconds)	Bolus/Hold	Infusion
<34	Give supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 mL/hr
34-44	Give ½ supplemental bolus if ordered & inform MD	↑ 100 units/hr = ↑ 2 mL/hr
45-54	0	↑ 50 units/hr = ↑ 1 mL/hr
55-70	0	NO CHANGE
71-85	0	↓ 100 units/hr = ↓ 2 mL/hr
86-100	Hold infusion 1 hour & inform MD	↓ 150 units/hr = ↓ 3 mL/hr
101-125	Hold infusion 1 hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr
>125	Hold infusion 1 ½ hour & inform MD	↓ 200 units/hr = ↓ 4 mL/hr

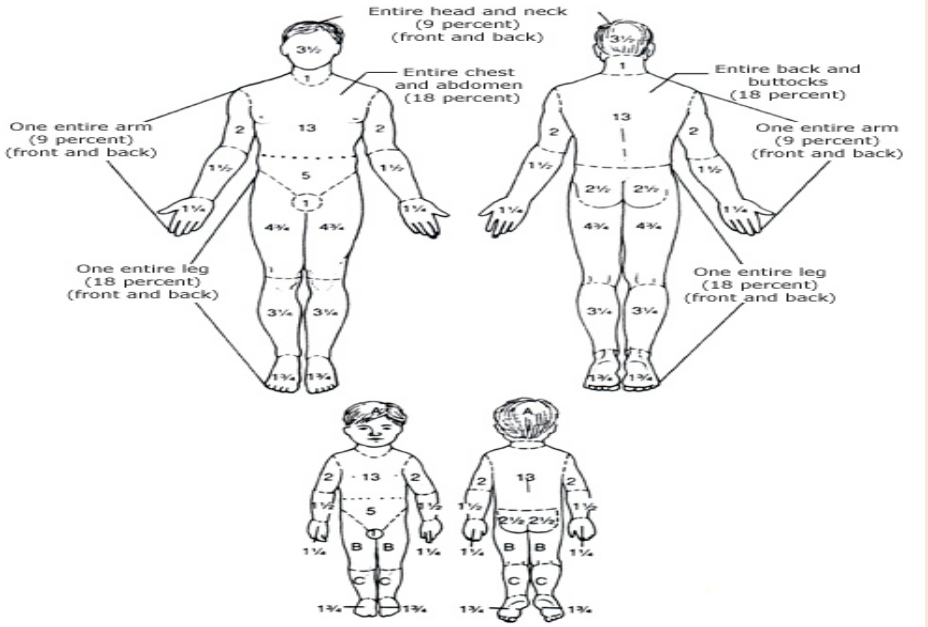
- If 2 consecutive aPTTs are >125 s, pt should not be maintained on the heparin protocol ---Consult Hematologist
- If 2 consecutive aPTTs are sub-therapeutic ---Consult hematologist

Toxins Antidotes

Poison/syndrome	Antidote(s)	Adult dose
Acetaminophen	N-acetylcysteine (Mucomyst 20%)	Initial oral dose: 140 mg/kg, then 70 mg/kg q 4h x 17 doses
Anticholinergic agents	Physostigmine (Antilirium)	Initial dose: 0.5-2.0 mg slow IV over 3-5 min
Benzodiazepines	Flumazenil (Romazicon)	Initial dose: 0.1-0.2 mg IV over 30-60 sec, repeat 0.1-0.2 mg IV ever prn up to 1.0 mg
Beta-blockers	1) Glucagon	1) Initial dose: 5-10 mg IV bolus, then 2-10 mg/hr IV infusion
	2) Calcium	2 Calcium chloride 10%: 1 gm (10 cc) IV; repeat as necessary
	3) Insulin + dextrose	3) Insulin load: 0.5 units/kg IV bolus, then 0.5-1.0 U/kg/h IV Dextrose 10% IV infusion (with KCl) - titrate to euglycemia
Calcium-channel blockers	1) Calcium	1) Calcium chloride 10%: 1-4 gm (10-40 cc) IV; repeat as necessary
	2) Glucagon	2) Initial dose: 5-10 mg IV bolus, then 2-10 mg/hr IV infusion
	3) Insulin + dextrose	3) Insulin load: 0.5 units/kg IV bolus, then 0.5-1.0 U/kg/h IV
Carbon monoxide	Oxygen ± hyperbaric chamber	100% oxygen by ventilator or NRB; high-flow oxygen by tight-fitting f
Crotalid snakebite	Wyeth polyvalent crotalidae antivenin (equine)	Mild: 3-5 vials; moderate: 6-10 vials; severe: 10-20 vials Mix reconstituted antivenin in 1000 ml NS over 4-6 hours
Cyanide	1) Amyl nitrate pearls	1) 1 ampule by inhalation for 15 sec every 3 min until IV access
	2) Sodium nitrite (3% solution)	2) 10 ml (300 mg) IV over 3 min
	3) Sodium thiosulfate (25%)	3) 50 ml (12.5 g) IV over 10 min
Digitalis	Digoxin immune Fab (Digibind)	1) (# mg ingested x 0.8) ÷ 0.6 = #vials needed 2) (Dig concentration [in ng/ml] x 5.6 x kg [weight]) ÷ 600 = #vials 3) Empiric dose: 10 vials (acute poisoning); 1-3 vials (chronic) 4) Reconstitute Digibind in NS and administer IV over 5-30 min
Ethylene glycol **Methanol**	1) Ethanol 10% in D5W ± hemodialysis	1) Initial load: 10 ml/kg IV of 10% ethanol over 30 min, then 1.5 ml/k infusion (titrate drip to serum ethanol 100 mg/dL); double to triple inf during hemodialysis
	2) Fomepizole [4-MP] (Antizol) ± hemodialysis	2) Initial load: 15 mg/kg IV over 30 min, then 10 mg/kg every 12 hou 30 min (re-bolus during HD)
Heparin	Protamine sulfate	1 mg neutralizes 90-115 U heparin; Initial dose: 1 mg/min to total do mg in 2 h
Hydrofluoric Acid	Calcium gluconate	1) Topical Cagluconate gel 3 percent applied for 1-2 days; or2) Intra SQ Cagluconate injection 5 percent at burn site (0.5 mL/cm2 burn a Regional intravenous (Bier block): 10 ml 10 percent in 40 mL NS inje locally in venous system x 20-30 min4) IntraarterialCagluc 10 percer ml in 40 ml NS over 4 hrs; repeat as necessary until pain relief
Iron	Deferoxamine (Desferol)	15 mg/kg/h IV infusion until urine color clears or patient clinically wel exceed 6 gm/24 h)
Isoniazid	Pyridoxine (Vitamin B6)	Initial dose: 1 gm pyridoxine for every gm INH ingested or empiric 5 over 10 min if amount ingested unknown
Lead	2,3-dimercaptosuccinic acid [DMSA] (Succimer); 100 mg cpsl	30 mg/kg po in three divided doses x 5 days, then 20 mg/kg in twice doses x 14 days; repeat therapy prn after 2 week rebound
Mercury **Arsenic** **Gold**	British antilewisite, dimercaprol (BAL); in peanut oil	Initial dose: 4-6 mg/kg IM every 4-6 h x 2 days
Methemoglobinemia	Methylene blue (1 percent solution)	Initial dose: 1-2 mg/kg (0.1-0.2 mL/kg) IV over 5 min; repeat prn
Opiates	Naloxone (Narcan) Nalmefene, naltrexone	Initial dose: 0.1-2.0 mg IV push (opioid dependent patients should re mg IV every 30-60 sec until clinical response); synthetic opiates may to 10 mg for initial reversal dose
Organophosphates **Carbamates** **Nerve agents**	1) Atropine	1) Initial dose: 0.5-2.0 mg IV; repeat q 3-5 min until sweat and secre clear
	2) Pralidoxime [2-PAM] (Protopam)	2) Initial dose: 1 gm IV over 15 min, then IV infusion of 3-4 mg/kg/h f hrs or until clinical toxicity resolves
Sulfonylurea	Octreotide (Sandostatin) + dextrose	Initial dose: 50-100 mcg SQ or IV, then 50 mcg q 12 h until euglycer maintained without supplemental dextrose
Tricyclic antidepressants	Sodium bicarbonate ($NaHCO_3$)	Initial dose: 1-2 ampules (50-100 mEq) IV push, then IV infusion to r blood pH 7.45-7.55 and PCO_2 30 mmHg (Usual drip: 3 amps NaHC D5W infused at 200-250 mL/h)

Appedix

TBSA of burned patient



Percentage based on age

	Birth- 1 yr	1-4 yr	5-9 yr	10-14 yr	15 yr	Adult
A. Head	19	17	13	11	9	7
B. Thigh	5.5	6.5	8	8.5	9	9.5
C. Leg	**5**	5	5.5	6	6.5	7

Equations

Creatinine clearance

Cockcroft-Gault GFR = (140-age) * (Wt in kg) * (0.85 if female) / (72 * Cr)

Use MD-calculator

Ideal body weight

1- Determine pt height

2- ***Use MD-calculator***

Corrected Ca for hypocalcemia

0.8 * (4 – serum albumin) + Serum Ca*r*

Fractional excretion of Na (FENa)

Plasma creatinine * Urinary Na / Plasma Na * Urinarycreatinine

- **<1** ------ Prerenal
- **>1** ------ Renal
- **>4** ------ Postrenal

Mean arterial blood pressure

1/3 Systolicpr + 2/3 Diastolic pr

Appache score II

A. Total Acute Physiology Score (*use MD calculator*)

B. Age points (years) (***use MD calculator***)

C. Chronic Health Points (see below)

Total APACHE II Score (add together the points from A+B+C)

Chronic Health Points: If the pt has a history of severe organ system insufficiency or is immunocompromised as defined below, assign points as follows:

immunocompromised state must have been evident **prior** to this hospital admission and conform to the following criteria:

- **Liver** – biopsy proven cirrhosis and documented portal hypertension; episodes of past upper GI bleeding attributed to portal hypertension; or prior episodes of hepatic failure/encephalopathy/coma.
- **Cardiovascular** – New York Heart Association Class IV.
- **Respiratory** – Chronic restrictive, obstructive, or vascular disease resulting in severe exercise restriction (i.e., unable to climb stairs or perform household duties; or documented chronic hypoxia, hypercapnia, secondary polycythemia, severe pulmonary hypertension (>40 mmHg), or respirator dependency.
- **Renal** – receiving chronic dialysis.
- **Immunocompromised** – the patient has received therapy that suppresses resistance to infection (e.g., immunosuppression, chemotherapy, radiation, long term or recent high dose steroids, or has a disease that is sufficiently advanced to suppress resistance to infection, e.g., leukemia, lymphoma, AIDS).

Interpretation of Score:

Score	DeathRate (%)
0-4	4
5-9	8
10-14	15
15-19	25
20-24	40
25-29	55
30-34	75
>34	85

Glasgow coma scale"GCS" & Full outline of unresponsiveness score

Table 1: Definition of the FOUR score and the Glascow Coma Score

FOUR score	Glascow Coma Scale
Eye response	Eye response
4 = eyelids open or opened, tracking, or blinking to command	4 = eyes open spontaneously
3 = eyelids open but not tracking	3 = eye opening to verbal command
2 = eyelids closed but open to loud voice	2 = eye opening to pain
1 = eyelids closed but open to pain	1 = no eye opening
0 = eyelids remain closed with pain	Motor response
Motor response	6 = obeys commands
4 = thumbs-up, fist, or peace sign	5 = localising pain
3 = localising to pain	4 = withdrawal from pain
2 = flexion response to pain	3 = flexion response to pain
1 = extension response to pain	2 = extension response to pain
0 = no response to pain or generalised myoclonus status	1 = no motor response
Brainstem reflexes	Verbal response
4 = pupil and corneal reflexes present	5 = oriented
3 = one pupil wide and fixed	4 = confused
2 = pupil or corneal reflexes absent	3 = inappropriate words
1 = pupil and corneal reflexes absent	2 = incomprehensible sounds
0 = absent pupil, corneal, and cough reflex	1 = no verbal response
Respiration	
4 = not intubated, regular breathing pattern	
3 = not intubated, Cheyne-Stokes breathing pattern	
2 = not intubated, irregular breathing	
1 = breathes above ventilator rate	
0 = breathes at ventilator rate or apnoea	

FOUR score = Full Outline of UnResponsiveness.

Date…/…./20…

Admission sheet

ICU doctor:

Pt N

Sex:Male Female

Age: …Y

IBW…..

Priority: I II III

Referral site: ED OR Ward

Provisional diagnosis: ……

Apache: ……

Past history

Medical: D HTN IHD COPD Asthma Stroke Hepatic…… Renal ……Other ……..……

Drugs: ……..……

Operations: ……

Procedures:……

Allergy: ……

Recent History prior to admission

……

……

……

……

……

……

Presentation at admission

Conscious level*and/or***GCS**……**Pupil** ……..**Lateralization**+ -**Bl sugar** ……mg/dl

Airway Patent At risk Obtunded ……

Blpr…../…..**HR**…..b/min.**Rhythm** ……**JVP** ……**LL** …… **Temp**. ……^{0}

RR ……b/min.**Breathing pattern** ……..……**Chestexam**……..……

Abdominalexam: ……

CT……

CXR……**ECG**……
.**Invasive devices:**ETT Tracheostomy NG tube Ur. Catheter CVC Periph. Line ICD Others ……..

Screen:GIT prophylaxis+ -DVT prophylaxis+ -Antibiotic+ -Risk of malnut.+ -Malnut. **+ -**

Nutrition plan…… Target Calories…… Ptn…… CHO …….Fat …..

Referral plan

……

……

……

……

……

……

……

……

……

Consultant ……

ICU plan

……

……

……

……

……

……

……

……

……

Consultant ……

Cardiac arrest sheet

Sign.: ……

Pt Name:……..…	**Date**…./..../20…	**Time**.…am pm.	**Bed**…...	**Witnessed+ -**

Nurse:……..	**Attendant doctors**:……	**Rhythm**……

Intervention:

Possible causes:……

EtCO2 :…… **CV ABGs: PH**….. **PaCO2**…… **HCO3**……. **Time of resuscitation:** …….**Min.**

Drugs given …… **DC given**……

Pre-arrest signs:

	Sign	Time	Intervention
Consc. level	…… …… …… …… ……	…….	…… …… …… …… ……
BIPr	……...……..…… …..……..…….. ……...……...…… ……...…… ……..…….. ……..……..……	…….	……..……... …… ……..……... …… …… ……
Seizures	……..	…….	……..……...
HR	……..	…….	……
Rhythm	…….....	…….	……
RR	……..….....	…….	……
Spo2	…….....	…….	…….....

Comments:

……
……

Recommendations:

……
……

Consultation form

Date…/..../20…

Pt

Age: …..Y

Admission date…/…../20…

Department

Provisional diagnosis……

Level of consultant: Resident Assist. LecturerSenior consult.

Urgency of consultation Urgent Within 2hrs Within 24 hrs.

Brief History

……
……
……

Indications of consultation

……
……

Sign: ……..……..

Delivery time ……..…

Sign.: ……..……..

Consultation notes

……
……
……
……
……
……
……
……

Conclusion: ……

Drugs prescribed:

……
……
……
……
……
……
……
……

Investigations ordered:

……
……
……
……
……
……
……
……

Sign.: ……..……..

Initiation of ventilation sheet

Pt Name:……..… | **IBW** ..... | **Date:** ……/……../20..… | **Time:**…..... am pm

Indication of ventilation: Apnea RF Impending RF Refractory hypoxemiaOthers……..…….

Most probable pathology: ……

Patient candidate for NIV+ -NIV tried + -NIV succeeded + -

ETT needed + - Adverse events during intubation……..

Adverse events post-intubation&vent.……

Ventilatory objectives

……

Ventilatory workup "radiology-consultation-analgesia-etc,."

……

……

ABG	SaO2			
	PaO2			
	P/F			
	HCO3			
	PaCO2			
	PH			
Monitored data	WOB			
	Dyn. Compliance			
	Stat Compliance			
	RR			
	MAP			
	Pplat			
	PIP			
	VTE			
Ventilator Setting	Triggering			
	PEEP			
	Flow cycling of PS			
	PS			
	Rise time			
	P Max			
	Peak flow			
	Pi			
	Ti			
	I : E			
	R R			
	VT			
	FiO2			
	Mode			
	Time	**Before**	**Just after**	**Within 30 min.**

Date…./…./20…

Ventilator flow sheet

Sign.: ……..

Pt Name……..…

Day NO

ABG	BE					
	SaO2					
	PaO2					
	HCO3					
	PaCO2					
	PH					
Monitored data	RSBI					
	P/F					
	RR					
	MAP					
	Pplat					
	PIP					
	VTE					
Ventilator settings	PEEP					
	PS					
	Flow					
	I:E					
	VT					
	RR					
	FiO2					
	Mode					
	Action	……	……	……	……	……
	Time					
	Event	……	……	……	……	……

Daily screening for weaning

1. Weaning / Daily Screening (If All Y, Go To 2)			
Resolved Underline Cause	Y N	Can initiate respiratory effort	Y N
P/F ratio >200	Y N	FiO2 ≤ 40 %	Y N
PEEP ≤ 5	Y N	Temp. ≤ 38 C°	Y N
Hb > 8	Y N	No need for heavy sedation	Y N
Minimal presors & inotropes	Y N		
2. Calculate RSBI (if Y, Go To 3)			
RSBI ≤ 100	Y N		
RR < 35	Y N	HR & Bl pr.stability "< 20% of baseline"	Y N
SaO2> 90% ± FiO2 10 % higher	Y N	Normal WOB	Y N
T V > 5 ml / kg during CPAP	Y N	I C P < 20 mmHg	Y N
SBT ≥ 30-120 Min *(.....min)*	Y N	SBT No*".....*"	1 2 3
4. Readiness for Extubation (If All Y, Extubate)			
No excessive secretion	Y N	Effective Cough	Y N
GCS ≥ 8	Y N	+ Ve Cuff Leak	Y N

5. Pt. is candidate for Weaning / Extubation or Not / Plan

.....

.....

.....

If Extubated Postextubation Events:

.....

.....

Patient progression sheet

Date: ...../...../20.....

Sign.: .....

PtName:.....

Day ( )

Major progress in different body systems

Cerebro-vascular:.....

.....

.....

.....

Cardio-vascular:

.....

.....

.....

.....

Respiratory:.....

.....

.....

.....

Renal&Electrolytes & Acid base: .....

.....

Additional notes

.....

.....

.....

.....

.....

.....

Change in referral plan

.....

.....

.....

.....

.....

.....

.....

.....

.....

Change in ICU plan

.....

.....

.....

.....

.....

.....

.....

.....

.....

Radiology.....

Procedures.....

Physiotherapy

plan.....

Pt Name:……..

Secondary trauma survey

Date……/……../20…..

Part	Lesion	Yes	No	Description	Plan	Sign	Investigations
Head							**Blood** Hb…….. Glucose……..…… Creatinine……. Cross match…….. Pregnancy test……. **X-ray** C-spine …… …… CXR …… …… Pelvis …… …… **Ultrasound**"FAST" …… …… ……..…… …… …… **Dpl** …… **CT head** ……..… ……..… …….. …… …….. **Angiography** Aorta …… Peripheral ……..….. **CT chest** …… …… …… **Cystogram** …… **Urethrogram** ……..…… **ECG** ……
Scalp	Laceration						
Scull	Vault fr						
	Basal fr suspected						
Face	Laceration						
	Fracture						
	Midface/maxilla instability						
Eyes	Orbit.globe or eye lid injury						
	Decreased visual acuity						
Ears	Hemotympanuim						
	CSF leak						
Nose	Bleeding						
Mouth	Tooth fr						
Neck	C-spine injury suspected						
	Soft tissue injury "larynx"						
	Laceration through platysma						
Chest	Fr ribs						
	Flail segment						
	Open pnemothorax						
	Surgical emphysema						
	Pnemothorax						
	Hemothorax						
	Pulmonary contusion						
Abdomen	Skin contusion/abrasion						
	Distension						
	Guarding						
	Tenderness						
PP	Lax anal tone						
	Blood						
	High prostate						
PV	Injury						
Peri-neum	Blood at meatus						
	Hematuria						
Ortho-pedic Injury	Spinal						
	Shoulder girdle						
	Upper limb						
	Wrist/Hand						
	Pelvis						
	Lower limb						
	Ankle/Foot						

Sign:……

Pt

Order form

	.../.../20…	.../.../20…	.../.../20…	.../.../20…	.../.../20…	.../.../20…	.../.../20…
Positioning							
Supine							
Special consideration							
O2 therapy							
Form							
Target							
DC							
Pressor&Ino-trope							
Drug 1							
Start rate							
Target MAP or…							
Drug 2							
Start rate							
Target MAP or…							
Drug 3							
Start rate							
Target MAP or…							
Intense insulin therapy							
Target Bl sugar							
Therap.Heparin							
Target a PTT							
Pnematic cuff compression							
Start							
DC							
EN							
Cont. gastric							
Intermit. gastric							
Post-pyloric							
Oral							
DC							
Lab./ CXR							
Type / Time	/	/	/	/	/	/	/
Type / Time	/	/	/	/	/	/	/
Type / Time	/	/	/	/	/	/	/
Type / Time	/	/	/	/	/	/	/
Stop sedation							
At 6 am							
Start Sedation							
Light/Time							
Heavy/Time							
Morph. /Fent. Inf.	/	/	/	/	/	/	/
Other orders							
……..…….. /Time	…….	…….	…….	…….	…….	…….	…….

Pt Name:……

Peri-operative sheet

Date: …/……/20…….

Pre-operative

Anesrhetist……..

Medical history

……
……
……

Presentation

……
……
……

Investigations

……
……
……

Anesth.Plan /Preparation

……
……
……
……
……
……
……
……

Date: …/……/20…….

Intra&Post-operative

Anesrhetist……..

Anesthesia given

……

Fluids/blood component given

……
……

Uneventful events……
……
……
……
……
……
……
……
……

Recovery……
……
……
……
……

ICU presentation

……
……
……
……

ICU doctor…..……..