EDITION 1.01

The RESUSCITATION CRISIS MANUAL

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CRISIS PROTOCOLS

SECTION **1** | SECTION **2**

CRISIS PROCEDURES

Before using this manual, please visit

resuscrisismanual.com/how-to-use

The Resuscitation Crisis Manual (The RCM) is designed for use as a cognitive aid in the Emergency and ICU departments, although it can be useful in any procedural area. It is not a substitute for experience, clinical acumen or simulation training, but it can provide immediate accessible guidance in common emergencies.

It is well documented that in potentially stressful, time dependent clinical scenarios, even senior clinicians can forget important steps in treatment pathways. Written in the same aviation checklist format as The Anesthetic Crisis Manual, The RCM is the next in the crisis manual series.

Embedding cognitive aids into resuscitation management may contribute to a more systematic and effective approach to emergency medicine. As more studies support their use, we expect The RCM will prove to be a valuable resource for all clinicians, particularly for those working in resuscitation and intensive care medicine.

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Dedicated to the memory of Dr John Hinds

Resuscitation expert | Motorcycle enthusiast | Life saver

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CRISIS PROTOCOLS | SECTION 1

CARDIOVASCULAR

RESPIRATORY
AIRWAY

ANAPHYLACTIC SHOCK

Salim Rezaie | Anand Swaminathan

1. Remove any triggers and call for assistance.
2. Consider early intubation for airway compromise or impending obstruction as edema can progress rapidly.
3. Give high-flow oxygen.
4. Place two large bore IVs and infuse at least 20 mL/kg crystalloid.
5. Give epinephrine.
6. If symptoms persist, start an IV epinephrine infusion and consider arterial line placement.
7. If still no response, consider alternative agents listed opposite.
8. Start adjunctive therapy once patient is hemodynamically stable.

ANAPHYLACTIC SHOCK

Salim Rezaie | Anand Swaminathan

Intubation

Be prepared to perform emergent cricothyrotomy before attempting intubation (double setup). Consider awake intubation if time permits.

Route	Dose
Nebulized	5 mg standard epinephrine (5 mLs of 1 mg/mL) if patient has stridor
IM bolus	0.3-0.5 mg (use 1 mg/mL concentration)
IV bolus	5-20 mcg (use 10 mcg/mL concentration)
IV infusion	1-20 mcg/min

Alternatives if poor response to conventional therapy

- epinephrine 100 mcg IV bolus
- norepinephrine infusion 0.1 mcg/kg/min
- vasopressin 0.01-0.04 units/min or 2 U bolus (0.03 U/kg)
- glucagon 1 mg IV over 5 min (for beta blocker reversal)
  start infusion 5-15 mcg/min if needed (common side effect of nausea/vomiting)
- methylene blue 1.5-2 mg/kg IV bolus
  add continuous infusion of 0.5 mg/kg/hr in refractory cases

Adjunctive therapy when hemodynamically stable

- PO prednisone 1 mg/kg (maximum of 50 mg) *or*
  IV hydrocortisone 2-4 mg/kg (maximum 200 mg) *or*
  IV dexamethasone 0.2-0.4 mg/kg (maximum 10 mg) *or*
  IV methylprednisolone 125 mg
- diphenhydramine 1-2 mg/kg (up to 50 mg)
- H2 blocker: cimetidine 300 mg or ranitidine 50 mg PO or IV

AORTIC DISSECTION

Peter Weimersheimer

1. Establish large bore IV access and titrate FiO_2 to $SpO_2 \geq 90\%$.

2. Place right radial arterial line and use left arm for NIBP.

3. Use fentanyl and esmolol as first line therapy to control pain, keep HR < 60 and reduce SBP < 120 mmHg.

4. If additional control is needed, use the agents listed (in order of preference) in the table opposite.

5. Prepare for rapid CT angiogram but use bedside TEE or TTE as alternatives if unstable or while waiting for CT.

6. Notify blood bank, request blood products and activate massive transfusion protocol if indicated 06.

7. Seek early consultation with CT surgeon for decision on imaging, blood pressure, and heart rate control.

8. Notify theatre early if surgery is indicated.

9. If hypotensive, consider tamponade, myocardial infarction, aortic valve incompetence or aortic rupture.

10. Prepare for transfer to ICU or the OR.

AORTIC DISSECTION

Peter Weimersheimer

Principles are to control BP, stop bleeding and replace volume.

Morbidity and mortality is directly proportional to the delay in diagnosis and management. Early placement of arterial line for monitoring and sampling helps guide treatment.

> Dissection progression and mortality is proportional to flow velocity and pressure differentials (dP/dT) so decrease HR, inotropy and SBP.

Aim to keep HR < 60, SBP < 120 mmHg (or < 100 in the young).

Use fentanyl and esmolol for pain and BP control. Fentanyl 25-50 mcg IV boluses can also decrease sympathetic surge during intubation.

Agents for controlling BP are listed in order of preference in the table below.

	Bolus Dose	Starting Infusion	Titration Dose	Max Dose
Esmolol	500 mcg/kg SIVP	50 mcg/kg/min	↑ by 50 mcg/kg/min q5-10min, rebolus with each increase	300 mcg/kg/min
Nicardipine	none	drop to 5 mg/hr	↑ by 2.5 mg/hr q5min, when desired BP obtained, drop to 3 mg/hr	15 mg/hr
Clevidipine	none	1-2 mg/hr	variable	32 mg/hr
Nitroprusside	none	0.1 mcg/kg/min	↑ by 0.5 mcg/kg/min	10 mcg/kg/min

If the above are unavailable, consider metoprolol or diltiazem for HR and labetalol for additional BP control.

Rapid imaging options
CTA chest/abdomen ± neck or lower extremities
TEE/TTE if unstable or awaiting CT scanner.

CTA (CT with arterial contrast) is generally the most available imaging modality with similar accuracy to TEE and MRA. Bedside TTE, aortic, or carotid imaging are available rapid adjuncts to make the diagnosis and begin focused management but are not sufficiently sensitive to exclude dissection.

Hypotension may be due to myocardial infarction, aortic incompetence, tamponade or aortic rupture. Use ECG and bedside echo to look for treatable causes.

Thoracic aortic dissection is a rare but lethal disease (3.5/100000). Only 85% of patients present with a sudden onset of severe pain. **Beware patients who have sudden symptoms involving disparate anatomical systems, or chest pain *plus* other symptomatology** and have a low threshold for investigating further.

CARDIOGENIC SHOCK

Scott Weingart

1. Give supplemental oxygen to achieve $SpO_2 \geq 90\%$.
2. Intubate if SpO_2 not maintained or altered mental state.
3. Get a 12-lead ECG and screen for STEMI equivalents using right side and posterior leads as appropriate [39].
4. Give aspirin and start heparin if ECG shows ischemia.
5. Request blood chemistry, CBC, coagulation panel, type and screen, troponin, lactate, ABG and CXR.
6. Review the differential diagnosis.
7. Perform focused echocardiography and RUSH exam for hemodynamic status and possible causes [09].
8. Monitor cardiac output if equipment available.
9. Start peripheral norepinephrine to obtain MAP ≥ 65 mmHg.
10. If poor cardiac function after MAP corrected, begin inopressor or inotrope.
11. Insert CVC, arterial line and urinary catheter for infusions and monitoring.
12. Request interventional cardiology review for diagnostic catheterization and placement of mechanical support device.
13. Consider ECMO team consultation [40].

CARDIOGENIC SHOCK

Scott Weingart

Drug Doses	
Norepinephrine	start at 5 mcg/min and titrate to 1 mcg/kg/min
Epinephrine (inotropic)	0.01-0.08 mcg/kg/min
Dobutamine	2-20 mcg/kg/min
Levosimendan	0.05-0.2 mcg/kg/min (no loading dose)

Methods of measuring cardiac output

TTE - Transthoracic Echocardiography
PiCCO - Pulse Contour Cardiac Output
LiDCO - Lithium Dilution Cardiac Output
NICOM - Non Invaive Cardiac Output Monitoring
FloTrac - Arterial Pulse Waveform Analysis

Differential Diagnosis of Cardiogenic Shock	
Myocardial infarction	Papillary muscle rupture
Valvular dysfunction	Ventricular wall disruption
Cardiomyopathy (including peripartum and Takotsubo)	Dysrhythmia
Myocarditis	Toxicologic
Pericarditis	Metabolic disturbance
Cardiac tamponade	Thyrotoxic crisis
Pulmonary embolus (PE)	Pneumothorax

Cardiogenic shock masqueraders include sepsis and aspirin toxicity.

The internal jugular vein is preferred for the CVC and the femoral artery for the arterial line. Aim to leave the right femoral and radial arteries available for interventionists. Use ultrasound to guarantee location in the common (rather than superficial) femoral artery.

Echocardiography and RUSH

These allow visualisation of the myocardium and valvular structures, as well as realtime hemodynamic assessment.

HYPERTENSIVE EMERGENCY GENERAL

Joseph Colla

Do not aggressively treat unless there is end organ dysfunction.

1. Obtain IV access and place patient on cardiac monitor.

2. Request CBC, blood chemistry, cardiac enzymes, ECG, CXR and urinalysis.

3. Consider early insertion of an arterial line.

4. If altered mental state, consider endotracheal intubation.

5. Perform a systematic review of possible causes including pregnancy and toxidrome of catecholamine excess.

6. Initiate disease specific, parenteral antihypertensive therapy.

7. Avoid using beta blockers in bradycardia, SCAPE, toxidrome of catecholamine excess and cocaine/amphetamine toxicity.

8. If headache, seizure, or other neurologic symptoms, request immediate head CT to exclude intracranial event.

9. Consider performing RUSH exam 09 to complete hemodynamic assessment.

HYPERTENSIVE EMERGENCY GENERAL

Joseph Colla

Causes of Hypertensive Emergency

Acute on chronic hypertension	Renal artery stenosis
Pre-eclampsia	Thyroid disease
Drugs (cocaine, amphetamines, sympathomimetics)	Intracranial mass
Drug withdrawal	Stroke/Bleed/Head injury
Pheochromocytoma	Epilepsy/Post-ictal

Hypertensive emergency requires immediate, disease specific lowering of the blood pressure. Lowering of the BP is crucial to preventing cerebral ischemia secondary to autoregulation. Optimal control of hypertensive emergencies balances the benefits of treating BP against the risk of decreasing target organ perfusion.

It is end organ dysfunction that determines aggressiveness of treatment.

Disease Specific Treatment

Intracerebral hemorrhage 23	Aortic dissection 02
Intracranial hemorrhage 23	Hypertensive encephalopathy 05
Ischemic stroke 19	Pre-eclampsia 05
SCAPE 10	Cocaine/amphetamine toxicity 05

Until additional evidence accumulates, using beta blockers in sympathomimetic excess or toxicity is not recommended.

HYPERTENSIVE EMERGENCY SPECIFIC

Joseph Colla

Pre-eclampsia

1. Start treatment if SBP >160 mmHg or DBP >110 mmHg.
2. In severe hypertension, first give magnesium sulfate 4-6 g IV as a loading dose, then 1-2 g every hour for seizure prophylaxis.
3. Do not reduce MAP more than 25% in the first two hours. Aim to keep SBP 140-160 mmHg and DBP 90-110 mmHg to avoid hypoperfusion.
4. Treat with a nicardipine infusion but if unavailable use labetalol or hydralazine.
5. NTG can be used as a last resort but exclusively for pulmonary edema.
6. Immediately consult with OB/GYN.
7. Avoid ACE inhibiters, angiotensin II and direct renin blockers.

Cocaine/Amphetamine Toxicity

1. Use diazepam, phentolamine, and NTG/SNP as preferred drugs.
2. Treat agitation, hypertension and tachycardia initially with benzodiazepines.
3. Give antihypertensive medication if evidence of end-organ damage.
4. Use phentolamine as first line therapy.
5. Avoid beta blockers until alpha blockade is established.
6. Use NTG if associated coronary vasoconstriction is suspected.

Hypertension and tachycardia from cocaine toxicity rarely require specific treatment. Alpha-adrenergic antagonists are the preferred agents for cocaine-associated acute coronary syndromes.

HYPERTENSIVE EMERGENCY SPECIFIC

Joseph Colla

Hypertensive Encephalopathy

1. Suspect if DBP > 110 mmHg and patient has severe headache, restlessness, agitation, seizure and no evidence of stroke or ICH on CT scan.

2. Be vigilant as it usually develops insidiously.
   - Early: headache, nausea and vomiting
   - Later: non-focal neurologic signs (restlessness, anxiety and confusion)

3. If evidence of papilledema, retinal hemorrhage or exudate on fundoscopic exam, treat as hypertensive encephalopathy regardless.

4. Lower MAP 10-20% in the first hour and not more than 25% total in the ED (guidelines recommend 25% over 24hr) using clevidipine or nicardipine.

5. Avoid centrally acting antihypertensives (clonidine, methyldopa or reserpine) to prevent CNS depression and clouding of mental state.

If not treated properly, symptoms can proceed to coma or death.
Consider MRI screening for Posterior Reversible Encephalopathy Syndrome (PRES).

Drug Doses	
Clevidipine	start 1-2 mg/hr, double rate q90s until close to goal, then increase q5-10min up to max 32 mg/hr (usually goal achieved by 21 mg/hr)
Esmolol	loading dose of 500 mcg/kg over 1 minute, infusion 25-50 mcg/kg/min, titrate every 10 min along with re-administration of loading dose
Hydralazine	start with 5 mg IV over 1-2 min, repeat dose of 5 mg in 20 min if goal not achieved up to max 30 mg (reduction should occur within 10-20 min of dose)
Labetalol	give 20 mg IV, then 40-80 mg q10min up to 300 mg max
Magnesium Sulphate	4-6 g loading dose then 1-2 g/hr
Nicardipine	start infusion at 5 mg/hr, increase by 2.5 mg/hr q5min (max 15 mg/hr) drop to 3 mg/hr when desired BP obtained
Nitroglycerin (NTG)	infuse at 5-400 mcg/min
Nitroprusside (SNP)	infuse at 0.25-10 mcg/kg/min (slowly titrate and consider arterial line)
Phentolamine	5 mg every 5-10 min as needed

MASSIVE HEMORRHAGE

Joe Nemeth | Nisreen Hamza-Maghraby

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CRISIS PROTOCOLS
SECTION ONE | SECTION TWO
CRISIS PROCEDURES

Control bleeding in ED/ICU/OR/Interventional Suite.

1. Assess SpO_2 and ventilation with view to early intubation.
2. Review clinical condition and consider activation of massive transfusion protocol (MTP).
3. Insert large caliber CVC or two wide bore peripheral IV lines.
4. Reverse any anticoagulation therapy.
5. Request blood chemistry, CBC, ABG, type and screen, and coagulation panel including TEG/TEM (if available).
6. Give antifibrinolytic agent if bleeding started < 3hr prior and there are no contraindications.
7. Monitor hemodynamics and aim for MAP ≥ 65 mmHg but if suspected traumatic brain injury increase MAP ≥ 80 mmHg.
8. Strictly limit use of all non-blood product fluids.
9. Monitor progress with CBC, coagulation panel, fibrinogen, ABG and iCa.
9. Actively warm patient, IV fluids and room.
10. Make early contact with surgical or medical proceduralists depending on the etiology and site of bleeding.

MASSIVE HEMORRHAGE

Joe Nemeth | Nisreen Hamza-Maghraby

Massive hemorrhage = significant bleeding leading to death within minutes if there is failure to gain definitive control.

Indications to initiate MTP

Trauma

- Assessment of Blood Consumption (ABC) score of 3 or 4.

For each parameter below 0 = no; 1 = yes	
ED SBP < 90 mmHg ED HR > 120 bpm Penetrating mechanism Positive fluid on FAST exam	Score of 3 - massive transfusion required in 45% of cases. Score of 4 - massive transfusion required in 100% of cases.

- Critical Administration Threshold:
  Initiate MTP if giving 3rd unit of RBCs in the first hour.
- Significant hemorrhage with either shock or abnormal coagulation as determined by a senior clinician.

Non-Trauma

- Significant hemorrhage with either shock or abnormal coagulation as determined by a senior clinician.

Early endotracheal intubation and mechanical ventilation is strongly recommended.
Use vitamin K, prothrombin complex or protamine if indicated to reverse any anticoagulation.

The intra-osseous route is only suitable for initial resuscitation until large bore IV access is established.

Blood chemistry should include SMA-10, ABG, iCa and lactate.
TEG/TEM are excellent methods of quickly assessing and monitoring coagulation status.
Monitor every 4-6 units of RBCs.

For antifibrinolytic therapy in adults, administer tranexamic acid 1g IV bolus and consider a second 1g bolus or sustained drip.

To avoid dilution minimize the use of crystalloid and colloid therapy.

MASSIVE PULMONARY EMBOLISM

James Horowitz | Oren Friedman

1. Establish IV access, arterial line and titrate FiO_2 to SpO_2 > 90%.

2. Bolus unfractionated heparin and start weight based protocol.

3. Treat associated hypotension by titrating norepinephrine to a MAP > 65 mmHg and avoid large fluid boluses.

4. If intubation is required beware of hemodynamic collapse 45.

5. Use bedside echo to assess RV function and look for Clot-in-Transit.

6. Start thrombolytic therapy if bleeding risk is *low*.

7. Consider surgical embolectomy or endovascular alternatives if thrombolysis bleeding risk is *high*.

8. Contact ECMO team early if bridging to these procedures (or to recovery) is required.

9. If a PE is suspected in cardiac arrest, give 50 mg alteplase or full dose tenecteplase as IV push, followed by 15 min of CPR and repeat once if indicated.

10. If known massive PE prior to cardiac arrest, immediate transition to ECMO is indicated.

MASSIVE PULMONARY EMBOLISM

James Horowitz | Oren Friedman

Use the hospital's protocol for heparin bolus and infusion. Unfractionated heparin may be preferable to LMWH if lysis is likely.

For a norepinephrine infusion, use hospital protocol or start with 4mg in 50mL at 5 mL/hr (scale up for 100mL or 250mL dilutions).

Doses of medications for stabilization

- norepineprhine 1-30 mcg/min (may increase to 1 mcg/kg/min with caution)
- inotropic epinephrine 0.01-0.08 mcg/kg/min
- dobutamine 1-20 mcg/kg/min
- nitric oxide 10-40 ppm

Focused echocardiographic assessment in PE

- assess RV to LV ratio in apical 4 chamber view
- assess RV function
- assess interventricular septal bowing in parasternal short axis view
- look for clot-in-transit

Always review contraindications before commencing thrombolytics.

Thrombolysis

The standard PE dose is alteplase 100 mg IV over 2 hr (tenecteplase as a weight based dose is a less widely used alternative). Consider 10-20 mg IV push if the patient is very unstable, followed by an infusion of the remaining 80-90 mg over 2 hr.

All invasive procedures (including IVs, arterial lines, catheters) should be completed before infusion when possible and peripheral IVs tested to confirm integrity.

Stop unfractionated heparin (UFH) during the infusion and restart UFH when PTT falls to 1.5x normal. Restart without a bolus at the previous therapeutic infusion rate.

Consider stopping thrombolytic at 50 mg if bleeding risk is moderately elevated and the patient is responding.

Contraindications to Thrombolysis	
Absolute	**Relative**
prior intracranial hemorrhage	> 75 years
ischemic stroke < 3 months old	current anticoagulation
aortic dissection	pregnancy
active major bleeding	traumatic CPR
intracranial malignancy	internal bleed within 4 weeks prior
intracranial AVM	severe hypertension (greater than 180/110)
recent neurosurgery	dementia
recent traumatic head injury	major surgery within 3 weeks prior
	ischemic stroke > 3 months old

SEPTIC SHOCK

Steve Mathieu

1. Confirm the diagnostic triad of infection, hypotension and tissue hypoperfusion.

2. Give supplemental oxygen to achieve $SpO_2 \geq 90\%$.

3. Request laboratory screen including blood cultures and give broad-spectrum antibiotics immediately.

4. Give 500-1000 mL bolus of crystalloid, then titrate boluses up to 20-30 mLs/kg against monitored parameters and echo.

5. Consider arterial line and CVC placement.

6. Monitor hourly urine output and use serial lactate to assess response to therapy.

7. Initiate vasopressor therapy if MAP remains < 65mmHg.

8. Perform focused echocardiography and ultrasound exam for hemodynamic status and possible sources of infection.

9. Consider blood transfusion if Hb falls to 7 g/dL or less.

10. Initiate appropriate diagnostic imaging and contact relevant specialty teams for ongoing care and source control.

SEPTIC SHOCK

Steve Mathieu

The diagnostic triad is based on suspected or documented evidence of infection, hypotension (MAP ≤ 65 mmHg after appropriate initial fluid load) and tissue hypoperfusion (lactate > 2 mmol/L).

Septic shock is an emergency requiring prompt clinical diagnosis and treatment. Priorities are volume, pressors and locating the source. Frequent reassessment is essential.

Antimicrobial therapy
Blood and body fluid cultures should be obtained immediately and broad-spectrum antibiotics to cover any possible source should be started promptly.

Vasopressor therapy
Initiate peripherally via large bore cannula avoiding the hand and wrist. Central venous access should be secured at the earliest opportunity and vasopressor infusion transferred. Follow departmental guidelines.

Norepinephrine is a good initial vasopressor choice, as is epinephrine (especially if patient is demonstrating insufficient cardiac output). Add vasopressin if norepinephrine dose gets high (> 20mcg/min).

Consider steroids if despite multiple vasopressors hypotension persists.

Aim for a higher target MAP if patient is normally hypertensive.

The Surviving Sepsis Campaign (SSC) still recommends CVP and $ScvO_2$ targeting. Although this can be considered, there is no evidence that it improves mortality.

Inotropic therapy
Dobutamine can be considered if the patient has evidence of significant myocardial dysfunction and ongoing signs of hypoperfusion, despite achieving adequate intravascular volume and MAP. Targeting supranormal cardiac indices and BP with dobutamine should be avoided. Epinephrine at inotropic doses (0.01-0.08 mcg/kg/min) may also be considered and may be the preferred agent.

Fluids
Choice of fluid is influenced by efficacy, safety, availability, cost, and desire to minimize interstitial edema. The recommended choice of isotonic crystalloid is either a balanced solution (e.g. Ringers lactate) or 0.9% sodium chloride. There are concerns about hyperchloremia and increased risk of kidney injury with the latter, but this remains unproven. There is no clear benefit (or harm) from albumin. Starches are associated with increased risk of kidney injury and should be avoided.

Blood transfusion
Transfuse if Hb less than 7 g/dL. Consider leukocyte-reduced blood if local policies allow.

UNDIFFERENTIATED HYPOTENSION

+ Rapid Ultrasound for Shock and Hypotension (RUSH)

Mike Stone | Heidi Kimberly

1. Assess airway, breathing, circulation and conscious state.
2. Secure airway and start CPR if necessary.
3. Establish cardiac monitoring.
4. Give supplemental O_2 and titrate FiO_2 to SpO_2 > 90%.
5. Consider getting help and delegate according to skill level.
6. Prioritize IV access and start empiric fluid resuscitation.
7. Obtain ECG, blood chemistry and ABG.
8. Perform point-of-care ultrasound using the HI-MAP approach.
9. Use results to implement appropriate treatment pathway.

UNDIFFERENTIATED HYPOTENSION

+ Rapid Ultrasound for Shock and Hypotension (RUSH)

Mike Stone | Heidi Kimberly

Target Organ	Hypovolemic	Cardiogenic	Obstructive	Distributive
Heart	Hyperdynamic	Low LVEF	Pericardial effusion RV strain	Hyperdynamic (early sepsis) Low LVEF (late sepsis)
IVC	Small Increased respiratory variation	Distended Decreased respiratory variation	Distended Decreased respiratory variation	Small/normal
Morison's	Free fluid possible (ruptured ectopic, intra-abdominal bleeding)	Small amount ascites possible	Normal	Evaluate gallbladder/ kidneys for sepsis with unknown source
Aorta	AAA Aortic dissection	Normal	Normal	Normal
Pulmonary	A-lines	B-lines Pleural effusion	Pneumothorax (absent lung sliding)	A lines B lines (ARDS) Pleural fluid (empyema) US findings of PNA

Heart - A hyperdynamic left ventricular ejection fraction (LVEF) in the patient with atraumatic hypotension is highly suggestive of septic shock, although LV systolic failure may also be encountered in late stages of sepsis.

Although cardiogenic shock is often due to LV systolic failure, consider valvular etiologies such as acute aortic or mitral regurgitation. Evaluate with color Doppler and/or obtain consultative echocardiography if significant suspicion exists.

IVC - An IVC that collapses with respiration argues strongly against the presence of obstructive or cardiogenic shock. Complete collapse of the IVC is suggestive of hypovolemia in this patient population.

Morison's - Consider a full evaluation of the right upper quadrant, left upper quadrant and pelvis in patients with hypotension and suspected intra-abdominal source (young female with concern for ectopic pregnancy, patients with abdominal distention or tenderness on exam).

Aorta - Assess the aortic arch using the supra-sternal notch view in patients with suspected Type A dissection, particularly if pericardial effusion or aortic root dilation noted on evaluation of the heart.

Pulmonary - Absent lung sliding is suggestive (though non-diagnostic) of pneumothorax.
B-lines may confirm the presence of acute decompensated heart failure.

Supplementary Views - DVT: consider compression ultrasound of the common femoral, femoral, and popliteal veins if suspicious for massive pulmonary embolism (RV dilation, hypoxia, appropriate clinical scenario) as presence of DVT may confirm diagnosis.

- Ectopic: if pregnancy test positive, careful scanning trans-vaginally may show ectopic pregnancy.

SYMPATHETIC CRASHING ACUTE PULMONARY EDEMA SCAPE

Michelle Welsford

10A
CRISIS PROTOCOLS
SECTION ONE | SECTION TWO
CRISIS PROCEDURES

For the hypertensive patient in severe acute pulmonary edema.

1. Establish IV access and consider arterial line placement.

2. Start NIPPV using 100% O_2 and settings listed opposite.

3. Give NTG 200-400 mcg IV bolus over 1 min and repeat in 2 min if BP remains high.

4. Start 100 mcg/min NTG infusion and titrate up to 400 mcg/min.

5. If hypotension develops, reduce or turn off NTG and consider a 250 mL fluid bolus.

6. Avoid giving diuretics early.

7. Avoid morphine.

8. Perform bedside echo/US assessment of cardiovascular system.

9. Screen for ischemia with serial ECGs and cardiac enzymes.

10. Once stable, transfer to ICU or CCU as appropriate.

SYMPATHETIC CRASHING ACUTE PULMONARY EDEMA SCAPE

Michelle Welsford

Principles are to support ventilation, reduce afterload and treat cause.

Sympathetic crashing acute pulmonary edema (SCAPE) is the extreme end of the spectrum of acute pulmonary edema.

Patients present with sudden respiratory distress and severe hypertension from sympathetic overload. There is severe pulmonary edema but little peripheral edema. The main problem is very high afterload.

Low-dose NTG primarily affects preload so high-dose NTG is required to reduce afterload. Patients are not acutely volume overloaded and don't require diuresis.

Treat as hypertensive emergency with simultaneous NIPPV and IV nitrates and aim to avoid endotracheal intubation. It is only indicated in a very limited number of cases and carries inherent risks and challenges.

NIPPV settings
Patients require primarily CPAP/EPAP. Start at 5 cm H_2O and titrate up to 15 cm H_2O. IPAP is supplementary.

Diuretics should not be given early. Acute diuresis with furosemide may lead to an increase in afterload due to neurohormonal mechanisms (increase in sympathetic and renal angiotensin). Morphine leads to greater intubation rates and doesn't improve the primary problem.

If the patient is not hypertensive then treatment is directed more towards cardiogenic shock or other causes of edema rather than SCAPE.

Bedside echocardiography can determine cardiac function, valvular function and monitor pulmonary edema.

> Acute pulmonary edema has a high mortality, but SCAPE is one of the high yield conditions in Emergency where early recognition and prompt treatment can avoid intubation and ICU admission, with its associated morbidity and mortality.

Consider causes of acute pulmonary edema requiring immediate intervention:

- acute valvular dysfunction
- myocardial infarction
- cardiomyopathy

CRASHING ON VENTILATOR

Sean Scott

1. Call for help, airway cart, ultrasound and scalpel.
2. If patient arrests, start CPR, delegate two staff members to chest compressions and continue to seek underlying cause.
3. Disconnect patient from ventilator and allow exhalation.
4. BMV 100% oxygen with PEEP valve at 10 breaths/min but don't delay if valve not immediately available.
5. Confirm endotracheal tube position with waveform $EtCO_2$.
6. Listen for any air leak from the mouth or neck.
7. Pass a bronchoscope (preferred), tube exchanger, bougie or suction catheter to exclude obstruction.
8. Consider reintubation unless a patent, correctly positioned tube is directly visualized.
9. Use bedside ultrasound to diagnose pneumothorax and if confirmed, treat with finger thoracostomy.
10. If no pneumothorax, review the list of common causes.

CRASHING ON VENTILATOR

Sean Scott

Disconnecting the patient from the ventilator and bagging eliminates the machine as a cause of deterioration.

Allow exhalation to relieve dynamic hyperinflation (auto-PEEP), which obstructs venous return and results in hypotension.

Any air leak suggests tube displacement or cuff issues.

Hand ventilation delivers 100% oxygen and allows assessment of compliance. Uneven rise and fall of the chest suggests pneumothorax, mucus plug or bronchial intubation. Subcutaneous crepitus suggests barotrauma.

Ideally, actions should occur in parallel, with one operator focused on airway issues while a second operator identifies and treats any pneumothorax.

Continuous waveform capnography confirms ventilation but tube position must also be verified by laryngoscopy or bronchoscopy. Consider ultrasound to assess mainstem intubation and palpate cuff balloon to assess cuff integrity.

If flexible bronchoscopy is immediately available, it can simultaneously confirm tube position and exclude obstruction.

If bronchoscopy is not available, replacement of the tube may be necessary to rule out ball-valve obstruction.

Absent lung sliding on ultrasound suggests pneumothorax or mainstem intubation. The former should be immediately treated with finger thoracostomy.

Post intubation hemodynamic collapse
Hemodynamic collapse in the immediate post intubation period is often due to inadequate preload or sedative medications. Treat by administration of intravenous fluid (10 mL/kg) and/or epinephrine in 10-20 mcg boluses [45].

In an arrest or peri-arrest situation make bilateral finger thoracostomies and follow up with formal chest drains when the patient is stabilized. This should be considered even in the absence of ultrasound.

Common causes

- displaced tube
- cuff leak/rupture
- barotrauma
- auto-PEEP
- pneumothorax
- mainstem intubation
- ball valve obstruction
- tube obstruction
- post intubation hemodynamic collapse

CRICOTHYROIDOTOMY

Ernesto Brauer

Stand on same side of patient as operator's dominant hand.

If the cartilage anatomy is palpable

1. Immobilize the thyroid cartilage between the thumb and middle finger of the non-dominant hand.
2. Identify cricothyroid cartilage by palpating with non-dominant index finger down thyroid cartilage.
3. Make at least a 3 cm longitudinal incision from middle of thyroid cartilage to the bottom of cricoid cartilage.
4. Re-palpate cricothyroid membrane with index finger.
5. Use scalpel to make a horizontal incision through full extent of the cricothyroid membrane, first one way until stopped by cartilage and then the other.
6. Remove the scalpel, insert finger and feel cartilage surrounding it and underneath the fingertip.
7. Ride the tip of the bougie along the finger pad and advance until below sternal notch.
8. Place 6.5 endotracheal tube over bougie just until the cuff disappears through incision, or insert 6.0 Portex tracheostomy tube until the hub is reached.
9. Confirm with waveform $EtCO_2$.
10. Secure with large sutures.

CRICOTHYROIDOTOMY

Ernesto Brauer

Stand on same side of patient as operator's dominant hand.

If the cartilage anatomy is not palpable

1. Use at least 8 cm midline vertical neck incision from sternal notch until at least 2/3 up the patient's neck.

2. Spread fat and soft tissue with four fingers of each hand until midline cartilage/trachea can be palpated.

3. Locate thyroid, cricoid and membrane.

4. Continue from step 5 on opposite page.

FAILED INTUBATION

George Kovacs

Continue BMV between attempts, call for help and remember each attempt mandates a different approach.

1. If SpO_2 permits allow a maximum of 3 attempts.
2. For direct or Macintosh videolaryngoscopy optimize technique, ELM, head position and consider using adjuncts.
3. If there is hold up at the glottic inlet when using a bougie, rotate ETT *counterclockwise*.
4. For hyperangulated videolaryngoscopy, pull back if glottis is occupying more than the screen's upper half.
5. Manage hold up *after* the glottic inlet with partial retraction of stylet (~ 3cm) and/or rotation of ETT *clockwise*.
6. Ensure assistant's technique is not impeding progress (MILS, cricoid pressure).

If BMV fails at any point move to failed oxygenation protocol.

7. Aim to achieve rescue oxygenation within 90 seconds.

Rescue attempt 1 single attempt at supraglottic airway (SGA) with $EtCO_2$ monitoring.

Rescue attempt 2 emergency surgical airway (ESA) if SGA not successful within 45 seconds. ESA may be primary rescue approach.

8. Place gastric tube.

FAILED INTUBATION

George Kovacs

Failed intubation is an emergency mandating action, but as long as the patient can be oxygenated there is time. **Failed oxygenation** is a crisis mandating *immediate action* and there is time pressure.

The number of laryngoscopy and intubation attempts is associated with an increase in morbidity and mortality. **Three attempts is a suggested limit.** However, it may be appropriate to abort attempts earlier (<3 attempts) based on predicted likelihood of success or patient condition.

> Placement of a polyvinyl tube does not define success so don't persist. The goal of airway management is to maintain end organ oxygen delivery whether it be by BVM, SGA, ETT or ESA.

The method of intubation should be based on the skill set of the clinician, predicted difficulty, and availability of equipment. Regardless of the plan A device choice, the clinician must be equally skilled with their plan B device option.

Successful rescue of the patient from a *cannot intubate cannot oxygenate (CICO)* scenario requires regular decision-making and procedural practice using predefined equipment.

CricCon2

Simplified Cricothyrotomy Alert Posture ©emcrit

Ready (All Patients)	**Discuss/Feel/See Kit**
Set (Difficult Airway)	**Mark/Kit Bedside**
About to Go (Crashing/Hypoxemic)	**Inject/Prep/Open and Set Kit Scalpel in Hand**

For **all patients** rescue oxygenation equipment should be chosen and within reach, the neck assessed for landmarks and procedure roles assigned (CricCon2 green).

For **high risk patients** equipment should be out of packaging, the neck landmarks marked and the decision to act defined and communicated (CricCon2 yellow).

For the **crashing patient** a single primary supraglottic approach can be attempted with emergency surgical airway (ESA) in assigned clinician's hands (CricCon2 red).

While reversal of neuromuscular blockade with sugammadex is rapid (~4 min to 90% TOF) critically ill patients in a failed oxygenation scenario are not likely to return to their already compromised preintubation state. A partially awake patient may make rescue conditions even more difficult.

MASSIVE HEMOPTYSIS

Calvin Brown

Asphyxia is the greatest threat so prioritize airway.

1. If bleeding side is known, lay patient in lateral decubitus position *with that side down*.

2. Place two large bore peripheral IVs and consider arterial line.

3. Intubate for airway protection, patency, or oxygenation.

4. Attempt lung isolation with mainstem intubation, but if there is a tracheostomy in situ go to 17.

5. Place bronchial blocker for lung isolation as soon as possible.

6. Reverse any coagulopathy.

7. Obtain blood products and transfuse to keep MAP > 65 mmHg.

8. Communicate early with interventional radiology, anesthesiology, pulmonology and cardiothoracic surgery.

9. Transfer patient to the specific interventional suite, or operating room for definitive treatment.

MASSIVE HEMOPTYSIS

Calvin Brown

Intubate with the largest possible endotracheal tube (at least 8.0) to facilitate bronchoscopy.

Direct laryngoscopy (DL) with brisk suction may be required. **Videolaryngoscopy (VL) can be used but may get soiled by blood.** Be prepared for surgical airway [12].

Lung isolation (preferably with bronchial blocker) may be required if bleeding is extensive and positioning doesn't help. Consult anesthesia early if this is a possibility.

Mainstem intubation of the unaffected lung can be a temporizing measure. Turning the tube 90º towards the desired side can increase success.

Diagnostic and therapeutic strategies

If the patient stabilizes, CT angiography of the chest is the diagnostic imaging of choice.

Early bronchoscopy should be arranged and is both diagnostic and therapeutic. Prompt patient transfer is required if bronchoscopy is unavailable.

The top 5 causes of hemoptysis

- bronchiectasis
- active TB
- malignancy
- fungal infection
- immunolgical lung disease

Consider glucocorticoids for diffuse alveolar hemorrhage (DAH) associated with autoimmune disorders.

Bronchial artery embolization is an effective strategy and should be considered for patients with massive hemoptysis who are poor surgical candidates or for whom bronchoscopy has failed.

Surgical exploration is the next step if bronchoscopy and embolization fail.

POSTERIOR EPISTAXIS

Rob Cooney

1. Establish two large bore IVs or place introducer sheath.
2. Consider intubation if the airway is at risk.
3. Have patient blow nose to clear clots from nares.
4. Insert dual-balloon pack or Foley catheter.
5. *Slowly* inflate the posterior balloon and apply traction.
6. Inflate the anterior balloon or pack the anterior nares.
7. Administer sedation and analgesia as needed.
8. Place gauze between exterior nares + catheter, then secure.
9. Discuss case with ENT specialist and prepare for admission.
10. Reverse any coagulopathy and review need for antibiotics.
11. If bleeding continues, consult interventional radiology and oral-maxillofacial surgery.

POSTERIOR EPISTAXIS

Rob Cooney

Signs of posterior bleeding

- heavy bleeding
- bleeding in the posterior pharynx
- bleeding not controlled with an anterior pack

Procedure can be painful so judicious use of sedation and analgesia is recommended. Slow inflation of the balloons can allow hemostasis while minimizing pain.

Familiarity with the devices prior to an emergent event is essential as inflation substance (air/water) and volumes are unique to the devices. A dedicated pre-made ENT kit containing all needed supplies will improve management of these cases.

Treatment of any coagulopathy should be directed by the coagulation panel.

Posterior packing with Foley(s)

Intubation is preferable. For epistaxis due to trauma, use Foleys (12-14F) in preference to commercial devices.

Procedure

- Witness passage into the posterior pharynx by both Foleys with laryngoscope.
- Inflate using smaller volume first (6-8 mL) and apply traction until balloon wedges in posterior choana.
- Inflate to 20 mL, apply traction and secure.
- Reapply the anterior packs bilaterally.

Suggested Contents for ENT Kit

headlight	gloves, gown, mask
nasal speculum	topical anesthetic agent
bayonet forceps	topical vasoconstrictor
ring forceps	gauze, vasoline, nu-gauze
wall suction unit	Foley catheter (12F)
tongue depressor	umbilical clamp
dual balloon pack	

THE CRASHING ASTHMATIC

Lillian Emlet

1. Give continuous nebulized albuterol + ipratropium bromide.
2. Give methylprednisolone 2 mg/kg IV.
3. In addition, consider magnesium sulfate 2 g IV over 20 min.
4. Observe work of breathing and prepare for RSI if diaphoretic or unable to speak in short sentences 43.
5. Start NIPPV and infuse 30 mL/kg of IV fluid.
6. Consider epinephrine 0.3-0.5 mg IM and arterial line.
7. Only intubate if poor response after 5-10 min of NIPPV.
8. For the intubated patient use safe mechanical assist-control, volume cycle ventilation (settings are listed opposite).
9. Continue beta 2 agonists through NIPPV or ventilator circuit.
10. Use IV sedation and analgesia when on ventilator.
11. Consider VV ECMO if there is continued oxygenation or ventilatory failure.

THE CRASHING ASTHMATIC

Lillian Emlet

Aim to delay or avoid intubation if possible.

Maximise medical management and allow NIPPV time to work to avoid intubation in these patients. It is associated with increased morbidity and mortality.

Clinical findings for severe life-threatening asthma exacerbation

HR >120, RR > 30	accessory muscle use
oxygen saturation < 90%	diaphoresis
peak expiratory flow rate < 50%	agitation

Severity of wheezing does not correlate with degree of airway obstruction or severity. Patients are volume depleted so administer IV fluids early. It will also reduce peri-intubation hypotension [45].

NIPPV sedation

If patient's anxiety is the only thing preventing tolerance of NIPPV, consider ketamine or dexmedetomidine sedation with 1:1 nursing observation.

NIPPV settings

IPAP 10 cm H_2O EPAP 5 cm H_2O
(IPAP may be titrated up if required)

RSI medications

ketamine 2 mg/kg IV (preferred), propofol 1.5 mg/kg, or etomidate 0.3 mg/kg
succinylcholine 1.5 mg/kg or rocuronium 1.2 mg/kg

Post-intubation sedation and analgesia

Propofol, ketamine and fentanyl are all appropriate. Inhaled anesthetics can also be administered in operating room or at bedside with AnaConDa device.

Ventilator management

- assist-control volume cycle ventilation
- minimize rate (start at 10)
- increase flow rate (60-80 L/min)
- reasonable Vt (start at 8 mL/kg IBW)
- minimize inspiratory time, extend expiratory time
- raise peak pressure alarm to allow full breath
- permissive hypercapnea goal
- lower respiratory rate if expiratory flow graph shows incomplete exhalation or plateau pressure > 30 cm H_2O

Consider VV ECMO for patients still unable to oxygenate or ventilate.

Cardiac arrest

To diagnose pneumothorax perform ultrasound (US) for lung sliding. If US not available, treat with finger thoracostomies empirically.

TRACHEOSTOMY DISASTERS

Justin Morgenstern

Respiratory distress

1. Apply 100% oxygen to both face and tracheostomy.
2. Remove the inner cannula and any attached devices.
3. Attempt to pass a suction catheter and suction the airway.
4. ***If the suction catheter does not easily pass or $EtCO_2$ waveform is absent, deflate the cuff and remove the tracheostomy tube.***
5. If upper airway patent, use BMV, SGA or intubate to maintain oxygenation.
6. If not, ventilate via the stoma using a pediatric facemask or #2 LMA.
7. Replace the tracheostomy tube or intubate the stoma.
8. Assess patency of large airways with fiberoptic scope.
9. If there is no obstruction search for other causes.

Significant bleeding ▸ tracheo-innominate fistula

1. Call for help and delegate tasks accordingly.
2. With a cuffed tracheostomy tube, overinflate the cuff.
3. If tube is uncuffed, or overinflation unsuccessful, intubate orally and *only then* remove tracheostomy tube.
4. Ensure cuff is distal to bleeding to limit aspiration of blood.
5. Insert finger through stoma into pre-tracheal space to compress artery against posterior aspect of sternum.
6. Order blood products to the bedside.
7. Notify surgical team and prepare for urgent OR transfer.

TRACHEOSTOMY DISASTERS

Justin Morgenstern

If a suction catheter cannot be passed through the tracheostomy tube, assume it is either **obstructed** or **displaced**. Remove it immediately.

If the suction catheter passes easily, suction and search for other causes of respiratory distress, such as pneumothorax, aspiration, or bronchospasm.

If conditions permit, a fiberoptic scope is very useful for assessing tube position and possible obstruction.

A key distinction exists between patients with a patent upper airway and those with a prior laryngectomy or obstructing pathology. Steps 1 to 4 are the same but in those with a patent upper airway the next step is to focus on oxygenating through the orally placed airway. In the laryngectomy patient the focus is on oxygenating through the neck.

If using a BVM or supraglottic device to ventilate orally an assistant must use their hand to seal the tracheostomy stoma.

To ventilate through the stoma use a pediatric facemask or a #2 LMA to create a seal.

When ventilating through the stoma in a patient with a patent upper airway, an assistant must hold the mouth and nose closed or place a BVM with a PEEP valve attached dialled to its maximum setting.

Assume all patients with a tracheostomy have a difficult airway and prepare accordingly.

When emergently reinserting a tracheostomy tube, start with a size smaller than the one removed. A small (6.0) endotracheal tube can also be used. A bougie can be used as a guide but placement of the tube over a video rhinolaryngoscope or bronchoscope is preferred.

Immediately verify the tube is in the trachea using capnography. Reinsertion of a tracheostomy less than 7 days old may be difficult, and with higher rates of complications, is relatively contraindicated if other options are available.

If multiple practitioners are available they should work simultaneously, attempting oral intubation while another works on the neck.

NEUROLOGICAL

METABOLIC
ELECTROLYTES
DRUGS

TRAUMA

OBSTETRICS

ACUTE ISCHEMIC STROKE

Aarti Sarwal

Aim for complete evaluation within 45 minutes of patient arrival.

1. ***Check glucose and ensure > 50 mg/dL (2.8 mmol/L).***
2. Control the airway if necessary.
3. Call a code stroke, stat neurology or telestroke consult.
4. Exclude stroke mimics, check onset time and perform an NIHSS.
5. Request CBC, coagulation panel and blood chemistry but don't wait to give tPA unless an abnormality is suspected.
6. Perform non contrast head CT to rule out ICH.
7. Secure two peripheral IVs, keep BP < 180/110 mmHg at all times and review tPA contraindications before proceeding.
8. If there are no contraindications give tPA ASAP within a 3 hour window from stroke onset.
9. If 3-4.5 hr have elapsed, NIHSS score < 25 and imaging evidence suggests < 1/3 of MCA territory involved, give tPA.
10. Perform CT angiogram and CT perfusion when practical in all patients (including those not receiving tPA).
11. Consider transfer to comprehensive stroke center for endovascular interventions in the following:
    - causative occlusion of the ICA or proximal MCA
    - pre-stroke mRS 0-1 and NIHSS ≥ 6
    - treatment possible within 24 hr of symptom onset
12. After tPA is given keep BP < 180/105 mmHg.

ACUTE ISCHEMIC STROKE

Aarti Sarwal

Stroke mimics

- seizure (17%)
- systemic infection (17%)
- brain tumor (15%)
- toxic/metabolic disorders (13%)
- positional vertigo (6%)
- conversion disorder

Stroke Syndromes

Anterior circulation
One sided face, arm and leg weakness or sensory loss, aphasia, gaze preference, favoring one side of the body during spontaneous movements.

Posterior circulation
Field defects, dysphagia, cranial nerve deficits, central vertigo, dizziness, imbalance, falls. Crossed findings - motor or sensory loss of hemi body opposite side of cranial nerve of face involvement.

Unexplained nausea, vomiting or altered mental status.

Ongoing Management

Control BP using nicardipine or clevidipine as first line therapy but if not available use labetolol as an alternative 05.
Blood glucose should be maintained between 80 and 180 mg/dL (4.4-10 mmol/L).
If intubation is required, consider short-acting induction and muscle relaxant agents 46.
CT angiogram and CT perfusion needs IV access - use a 20G or larger cannula.

tPA Contraindications	
Symptoms suggestive of SAH	Postpartum < 14 days
Active internal bleeding	Intracranial aneurysm, AVM, neoplasm
BP > 185/115 despite treatment	Within last 7 days: arterial puncture at a non-compressible site
CT shows hypodensity > 1/3 cerebral hemisphere	Within last 21 days: GI/GU bleed
Current use of anticoagulants	Within last 3 months: ▸ intracranial or intra-spinal surgery ▸ severe head trauma ▸ intracranial hemorrhage or prior ischemic stroke
INR > 1.7 , Plt < 100K, aPTT > 40s, PT > 15s	
Heparin within 48 hr with abnormal pTT	
LMWH within 24 hr	

AGITATED DELIRIUM

Minh Le Cong | Tim Leeuwenburg

1. Call security, police, or behavioral emergency response team.

2. Perform rapid ABCD assessment for immediately reversible causes including ***hypoxemia*** and ***hypoglycemia***.

3. Attempt oral sedation using diazepam and/or olanzapine.

4. If oral sedation fails, use IV or IM sedation.

5. Titrate drugs aiming for a sleepy but rousable patient.

6. Use procedural sedation monitoring including BP, ECG, SpO_2, RR and nasal $EtCO_2$.

7. Titrate supplemental oxygen to SpO_2 > 90%.

8. Prepare drugs and equipment for rapid sequence intubation if there is potential for loss of airway 43.

9. Once control is established, repeat ABCD, request a full laboratory screen and include BGL.

10. Arrange appropriate placement for ongoing monitoring.

AGITATED DELIRIUM

Minh Le Cong | Tim Leeuwenburg

Causes of Delirium	
Immediately reversible	hypoxia, hypoglycemia, hypotension, drug action or withdrawal (prescribed or recreational)
Others	infection/septicemia, head injury, electrolyte disturbance, hypo/hyperthermia, renal/liver failure, endocrine disorders, serotonin or sympathomimetic syndrome, acute psychiatric conditions, head injury/CVA/intracranial bleed, epilepsy

Investigations

- Immediate point of care glucose.
- CBC, blood chemistry, osmolality, urinalysis
- ABG (carbon monoxide, cyanide, hypercarbia, acidosis)
- Drug levels
- CXR, CT head

General Principles

Use a designated safe area of hospital with exits and duress alarms.
Assess situation and patient for airway, anesthesia and risk to self or others.
Provide a low stimulus and calm environment.

Select one sedative (benzodiazepine) and one antipsychotic agent and titrate to a targeted level of sedation.

Avoid switching agents as effects can be unpredictable. **Avoid multiple doses before sufficient time given for effect,** as this can lead to overdose and respiratory depression. Use longer acting agents whenever possible to avoid the roller coaster effect of agitation/over-sedation.

If using rapid takedown agents, be prepared to manage the airway.
Establish an airway plan and team brief. Have basic, advanced and difficult airway equipment available including suction 43.

Administer medications with patient supine, one staff member to each limb and one to give drugs. Avoid prone restraint.

Doses differ between IM and IV routes of administration.

THERAPY	NO IV ACCESS	IV ACCESS ESTABLISHED
1st line	olanzapine wafer 10-20 mg PO (max 30 mg/24 hr) ± diazepam 10-20 mg PO	IV haloperidol or droperidol 5-10 mg IV (max dose 20 mg/24hr)
2nd line	haloperidol or droperidol 10 mg IM	IV ketamine 1-1.5 mg/kg
3rd Line	ketamine 4 mg/kg IM	consider RSI only if absolutely necessary

INCREASED ICP
+ HERNIATION

Minh Le Cong | Tim Leeuwenburg

1. Optimize oxygenation, ventilation and blood pessure.

2. Use the neuroprotective protocol for intubation [46].

3. Correct any coagulopathy and consider correction of platelet function or reversal of anti-platelet therapy.

4. Confirm there is elevated ICP by urgent CT scan, ocular nerve sheath diameter, clinical examination or ICP monitoring.

5. Notify neurosurgeon for surgical intervention if indicated.

6. Position head of bed to 30 degrees, place head midline, and check cervical collar is not impeding jugular blood flow.

7. Minimize pain and agitation with short acting agents.

8. Maintain $PaCO_2$ at 35-40 mmHg.

9. Treat seizures and administer seizure prophylaxis.

10. If signs of herniation:
    - hyperventilate to $PaCO_2$ of 30 mmHg
    - give osmolar therapy (23.4% hypertonic saline preferred)
    - arrange for immediate operative intervention

INCREASED ICP
+ HERNIATION

Minh Le Cong | Tim Leeuwenburg

Causes of Increased ICP	
Extra axial	bleed, empyema, tumor, pneumocephalus
Focal lesions	neoplasm, hematoma, abscess, 2° focal edema
Diffuse lesions	encephalitis, meningitis, encephalopathy, TBI, SAH
Venus sinus obstruction	central vein thrombosis, depressed fracture
Hydrocephalus	obstructive, communicating
Idiopathic	benign intracranial hypertension

Clinical signs of elevated ICP/herniation

- altered mental state
- pupil abnormalities - unilateral, fixed, dilated pupil or bilateral non-reactive midposition pupils
- cranial nerve palsy
- loss of upward gaze
- stroke syndromes from vessel compression
- cushing's response - hypertension and bradycardia, erratic respiratory pattern

Diagnosis

Urgent CT Scan
Ultrasound of optic nerve sheath diameter:
outer diameter > 6mm in high risk patient - *likely elevated ICP*
outer diameter < 5mm in low risk patient - *likely normal ICP*

Intubation is for airway protection and CO_2 control.

Aim to avoid hypotension, hypoxemia and hypo/hypercapnia.
Ketamine 1-2 mg/kg is a suitable alternative to etomidate in hypotension.
$PaCO_2$ should be maintained at 35-40 mmHg unless herniation.

Osmolar therapy

hypertonic saline: 500 mL 3% or 30-60 mL 23.4%
mannitol: 1 g/kg if not hypotensive and no ESRD
Insert IDC (Foley) and replace urinary losses with normal saline to avoid hypotension.

Seizure treatment

Use IV midazolam 2-4 mg or IV lorazepam 2-4 mg.

Prophylaxis options: levitarecetam 1g BID (preferred), or fosphenytoin 20 mg/kg load, then 100 mg TID.

Surgical intervention may include ventricular drain or more specific procedures such as evacuation of hematomas, resection of tumor or SOL lesions, and craniectomies.

Do not use steroids in traumatic brain injury (TBI) or intracranial hemorrhage (ICH).
Do not adjust ventilation on $EtCO_2$ only - set to $EtCO_2 \leq 35$ and then use $PaCO_2$.

STATUS EPILEPTICUS

Josh Farkas

Resuscitate, terminate seizures and treat the underlying cause.

1. Perform ABC assessment, maintain airway and establish IV access as soon as practicable.

2. Call for skilled assistance and delegate immediately.

3. Give lorazepam 0.1 mg/kg IV or midazolam 10 mg IM (if no IV).

4. Check fingerstick BGL or give empiric glucose 25 g IV.

5. Start a conventional anti-epileptic drug intravenously.

6. If known hyponatremia, treat according to protocol 28.

7. If seizure stops, reassess, consider intubation and prepare using the intubation checklist 43.

8. If seizures continue 10 min after benzodiazepine, induce the patient and secure the airway with endotracheal tube.

9. Start a propofol or high dose midazolam infusion immediately.

10. Request laboratory screen and look for underlying cause.

STATUS EPILEPTICUS

Josh Farkas

Definition
More than 5 min of convulsive seizure or multiple seizures without neurological recovery in between. Rapid control is necessary to limit any seizure induced neuronal injury and prevent respiratory compromise.

Causes
epilepsy, infective, hypoxic, vascular, metabolic, structural, physical (hyperthermia), drug induced or withdrawal effect, drug non-compliance

Laboratory screen
CBC, blood chemistry, BGL, ABG, toxicology screen, anticonvulsant levels, iCa and Mg.

When in doubt, intubate. The primary goals are terminating the seizure and stabilizing the patient. Intubation using anti-epileptic induction agents can achieve both.

Don't delay intubation while awaiting a conventional anti-epileptic drug. The anti-epileptic drug should be given as soon as possible. However, it may take 10-20 min to arrive from pharmacy and has limited efficacy in stopping the seizure.

Hypotension
Propofol is a powerful anti-epileptic agent, but can cause hypotension.
Before intubation, consider starting or preparing a vasopressor infusion (target pre-intubation SBP > 120 mmHg) [45]. For unmanageable hypotension use midazolam.

Conventional Anti-Epileptic Drugs	
Levtiracetam (KEPPRA)	60 mg/kg (max 4500 mg) IV over 10 min Considered safest (including pregnancy)
Valproic acid	40 mg/kg (max 3000 mg) IV over 7 min. Well tolerated with proven efficacy but contraindicated in liver disease and rare disorders causing hyperammonemia.
Anti-Epileptic Induction Regimen for Intubation	
1st Push	propofol 1.5-2 mg/kg
2nd Push	ketamine 2 mg/kg
3rd Push	succinylcholine 1.5 mg/kg or rocuronium 0.6 mg/kg
Anti-Epileptic Infusions	
Propofol infusion	load with 1.5-2 mcg/kg bolus infuse at 80 mcg/kg/min (5 mg/kg/hr) continue infusion 50-110 mcg/kg/min (3-7 mg/kg/hr)
Midazolam infusion	load with 0.2 mg/kg (may re-load to max total 2 mg/kg) infuse at 0.1 mg/kg/hr (range: 0.1-1 mg/kg/hr) May be more suited in hypotension.

Consider daily administration of thiamine.

Be aware of complications: aspiration, respiratory depression, neurogenic pulmonary edema, trauma and electrolyte abnormalities.

INTRACRANIAL HEMORRHAGE

SUB ARACHNOID (SAH) INTRACEREBRAL (ICH)

Jane Sturgess

Evidence of blood on non-contrast head CT

1. Intubate if GCS ≤ 8 or the patient cannot protect airway.
2. Ventilate to maintain $PaCO_2$ 35-40 mmHg.
3. Reverse coagulopathy and consider anti-platelet reversal.
4. Assess the grade of SAH or size of ICH on CT imaging.
5. Consider giving prophylactic anticonvulsants in the immediate post-hemorrhagic phase.
6. Maintain euvolemia while keeping SBP < 140 mmHg and MAP > 80 mmHg.
7. Treat hyperthermia with acetaminophen 650-1000 mg and actively cool to normothermia if necessary.
8. Consider RBCs if cerebral ischemia risk and Hb < 7 g/dl.
9. Screen for neuro-cardiac sequelae with ECG, echo and CXR.
10. Perform CT angiogram for SAH and ICH as soon as possible.
11. For SAH give oral or NGT nimodipine 60 mg q4hr.
12. Transfer to dedicated neurocritical care unit if indicated.

INTRACRANIAL HEMORRHAGE

SUB ARACHNOID (SAH) INTRACEREBRAL (ICH)

Jane Sturgess

Hunt and Hess scale for subarachnoid hemorrhage

Introduced in 1968 to determine the severity of the subarachnoid hemorrhage based on the patient's clinical condition. Mortality increases from a score of 1 through to 5.

1	asymptomatic, mild headache, some neck stiffness
2	moderate to severe headache, neck stiffness, may have a cranial nerve palsy but no other neurological deficit
3	mild neurological deficit, drowsiness or confusion
4	stupor, moderate to severe hemiparesis
5	coma, decerebrate posturing

Sizing of intracerebral hemorrhage from CT imaging

ABC/2
A = longest axis (cm)
B = longest axis perpendicular to A (cm)
C = number of slices with hemorrhage x slice thickness (cm)

- ≥ 75% area of hemorrhage count as 1 slice
- 25-75% area of hemorrhage count as 0.5 slices
- < 25% area of hemorrhage count as 0 slices

Calculator available at **mdcalc.com**

Essential information for referral

History

Timing of headache, collapse, and seizures.
Background including functional status and comorbidities.

Examination

Neurological exam including pupils.
Most recent vitals, GCS, and trends since admission.
Localizing neurological signs (eg. third nerve palsy right side).

Investigations

Ensure the scan images are reviewed and available to surgeons.

Patients with high-grade SAH or ICH may benefit from dedicated neurocritical care.
Consider transfer if indicated.

UNDIFFERENTIATED COMA

Scott Weingart | Eelco Wijdicks

1. Intubate if airway protection is needed.

2. Take fingerstick glucose, request laboratory screen and measure core temperature.

3. Treat BP to keep SBP < 185 mmHg and MAP > 80 mmHg.

4. Perform stroke/coma neuro exam and assess a ***FOUR Score***.

5. Request an immediate non-contrast head CT and additional imaging (± MRI, angiogram).

6. If etiology is still uncertain, consider a full toxicology screen and CSF examination relevant to the differential diagnosis.

7. Obtain neurology consultation if no cause has been discovered or if required for ongoing management.

8. Admit to a high acuity monitored bed.

UNDIFFERENTIATED COMA

Scott Weingart | Eelco Wijdicks

When confronted with a comatose patient, review ABC to assess the need for airway protection and blood pressure control before further neurological assessment.

Initial laboratory screen should include blood chemistry, CBC, coagulation panel, ABG/VBG, type and screen, and pregnancy test.

Coma exam should include NIHSS stroke exam plus careful examination of the eyes for vertical skew, anisocoria and abnormal movements, as well as brainstem reflexes, muscle tone and FOUR score.

The FOUR score (**F**ull **O**utline of **U**n**R**esponsiveness) consists of 0-4 points for each of eye response, motor response, brainstem reflexes and respiratory pattern. See website for full description.

Imaging

- If the patient is posturing or has no motor response *with a unilateral, fixed and dilated pupil,* perform an immediate CT non-contrast scan of the head followed by an MRI if patient is compatible.
- If the patient is posturing or has no motor response *with anisocoria, skew deviation or abnormal brainstem responses,* perform an immediate CT non-contrast scan of the head and CT angiogram of the head and neck vessels to screen for an acute brainstem lesion. Follow with an MRI if patient is compatible.
- If the patient doesn't fit into either of the above, perform an immediate CT non-contrast scan of the head, followed by an MRI if no other cause is found on lab testing and clinical exam.

Etiology of Coma

Diffuse brain dysfunction
hypoxia, hypoglycemia, shock, cyanide, CO

Endogenous CNS toxins
NH_4, uremia, CO_2 narcosis, hyperglycemia

Exogneous CNS toxins
alcohols, poisons, drugs, heavy metals

Endocrine disorders
thyroid, adrenals, pituitary diseases

Abnormal electrolytes
Na, Ca, Mg, PO_4, pH disturbance

Seizures / post-ictal state

Focal lesions
bleed, tumour, infarct, abscess with mass effect

Inflammation/infiltration
meningitis, encephalitis, encephalopathy vasculitis, SAH, carcinoid meningitis, traumatic axonal shear injury

Temperature regulation
hypothermia, heat stroke, NMS, MH

Intracranial hypertension
hypertensive encephalopathy, PRES, pseudotumor cerebri

1° neuronal or glial disorders
Creutzfeldt-Jacob, Marchiafava-Bignami, adrenoleukodystrophy, gliomatosis cerebri, PML

Psychogenic
hysterical, malingering, acute catatonia

ACCIDENTAL HYPOTHERMIA

Doug Brown | Scott Weingart

Vital signs present

1. Immediately place cardiac and core temperature monitors.
2. Consider securing airway to protect or ventilate but assess risk of hemodynamic collapse and review 45 before proceeding.
3. If there is one or more of ventricular arrhythmia, core temp < 28°C, or SBP < 90 mmHg, consider transfer to ECMO centre.
4. Start minimally invasive rewarming and consider bladder lavage.
5. Start IV fluid resuscitation using crystalloid at 38-42°C.

Cardiac arrest

1. Start CPR. Use up to 3 doses of ACLS medications and defibrillation.
2. Exclude normothermic cardiac arrest *leading to* hypothermia.
3. Start minimally invasive rewarming but do not apply heat to head.
4. Transfer to ECMO center (if available) *within 6 hr from start of CPR*.
5. Add bladder lavage ± peritoneal lavage ± chest tube lavage.
6. Re-attempt defibrillation when ≥ 28°C, there is a rhythm change or after every 2°C increase in core temperature.
7. Above 30°C reimplement ACLS medication cycles.
8. If > 32°C and still in cardiac arrest, consider ending resuscitation.

ACCIDENTAL HYPOTHERMIA

Doug Brown | Scott Weingart

Core temperature monitors include esophageal, rectal or bladder.

For bladder lavage use 3-way Foley and 40°C saline, given at 2-4 L/hr by gravity. Confirm volume in = volume out and be aware it will invalidate bladder and rectal temperature measurements.

Titrate fluid resuscitation to clinical volume status and avoid overload.
IV fluids are not a source of significant rewarming.

Use either femoral, shallow internal jugular or subclavian insertion for central venous access.

At all times, to prevent arrhythmias, avoid stimulation of the heart with catheter and guidewire.

Vasopressors are withheld, as relative hypotension may be physiologically appropriate depending on core temperature - consider expert consultation.

Bradycardia is to be expected with hypothermia and usually doesn't require treatment.

For rewarming, consider multiple heat delivery devices, but do not apply heat to the head. Instead, allow warm oxygenated blood to rewarm the brain centrally.

It is critical that lavage procedures do not impede CPR quality.

In hypothermic cardiac arrest, be prepared for prolonged CPR. If the presumed cause of the arrest is hypothermia, a good outcome is possible despite prolonged resuscitation.

HYPERKALEMIA

Doug Brown | Scott Weingart

1. Establish cardiac monitoring and venous access.

2. Identify and remove any source of K^+ intake.

3. Assess clinical stability and start treatment if:
   - patient is unstable
   - ECG shows any signs of hyperkalemia
   - K^+ levels > 6.5 mmol/L

4. Stabilise the myocardiocyte membrane by giving calcium gluconate 10% (15-30 mL) or calcium chloride 10% (5-10 mL).

5. To redistribute plasma potassium:
   - give soluble insulin 10 units + dextrose 50% 50 mL, both as IV push
   - start albuterol 5-10 mg nebulizer
   - hyperventilate if on mechanical ventilation

6. If hypovolemic consider 1 L of isotonic sodium bicarbonate (150 meq of $NaHCO_3$ in 1 L sterile water or D5W).

7. If there is urine production, consider saline with 20-80 mg of furosemide (but maintain euvolemia).

8. If K^+ > 6.5 mmol/L, repeat treatment at 40-60 min and consider emergent hemodialysis.

9. Correct any reversible precipitating factor including potassium sparing agents.

10. Check K^+ q1hr until normalized.

HYPERKALEMIA

Doug Brown | Scott Weingart

Treatment of hyperkalemia should be considered when levels are > 6.5 mmol/L or when there are ECG changes

Always send a second sample to exclude artifact, but if clinically unstable with ECG changes, don't wait for confirmation to start treatment.

ECG

Classic findings include peaked T-wave, loss of P-wave, prolonged PR interval, loss of R amplitude, widened QRS complex, sine wave pattern and asystole.

In the *unstable* patient ECG changes that arouse suspicion and may warrant immediate therapy include sinus bradycardia, bradycardia associated with a wide QRS, regular wide QRS complex tachycardia with rate < 120.

Hyperkalemia can present with any rhythm disturbance and even with ST segment alterations but a normal ECG doesn't exclude hyperkalemia diagnosis.

Stabilizing membrane

Calcium is used to stabilize the myocardial cellular membrane. It does not decrease serum concentration of K^+. Calcium chloride is more potent but also more irritating to the veins.

Redistribution

Plasma potassium moves to the intracellular space.

Insulin + glucose using only 1 amp of D50W has been associated with hypoglycemia, especially in patients with renal failure.

Beta 2 agonists are useful but should not be used as a single treatment as 12-42% of population don't respond. Inhaled administration is as effective and associated with less side effects than intravenous route. Insulin + glucose associated with beta 2 agonists have a synergic effect on lowering serum potassium.

Bicarbonate is only useful for its dilution effects or if there is associated metabolic acidosis. Not effective with normal pH. Administration by IV push not recommended.

Increased elimination

Use lactated Ringers, isotonic bicarbonate and/or diuretics but maintain euvolemia. Normal saline is not recommended as it will increase potassium.

Always check for inadvertent ongoing potassium administration, potassium sparing agents and precipitating factors.

Ion exchange resins are not recommended for acute treatment. Their peak effect varies from hours to weeks and more recently both their safety and efficacy has been questioned.

LOCAL ANESTHETIC SYSTEMIC TOXICITY LAST

Kariem El-Boghdadly

1. If symptoms or signs of toxicity appear during procedure *stop giving local anesthetic.*
2. Call for help and specifically request the LAST kit.
3. Maintain airway, give 100% O_2 and consider intubation.
4. Confirm intravenous access is established.
5. If cardiac arrest, start CPR but use < 1 mcg/kg epinephrine.
6. Treat seizures with benzodiazepines as first line therapy.
7. ***Administer 20% intravenous lipid emulsion.***
8. Prepare for prolonged CPR and alert the nearest ECMO facility.

LOCAL ANESTHETIC SYSTEMIC TOXICITY
LAST

Kariem El-Boghdadly

Symptoms
circumoral and/or tongue numbness, metallic taste, light-headedness, dizziness, visual/auditory disturbance, drowsiness, disoriented

Signs
muscle twitching, convulsions, unconsciousness, coma, respiratory depression, dysrhythmias, cardiovascular depression, collapse

Cardiopulmonary resuscitation
Follow ACLS algorithm but use reduced dose epinephrine (< 1 mcg/kg) and give intralipid early. *Avoid beta-blockers, Ca^{2+} channel blockers, lidocaine and vasopressin.*

Drugs for seizure termination

Drug	IV Dose	70 kg Adult	Notes
Midazolam	0.05 mg/kg	4 mg	first line, titrate dosage
Lorazepam	0.05 mg/kg	4 mg	
Thiopentone	1 mg/kg	70 mg	cautious dosing
Propofol	0.5-1 mg/kg	35-70 mg	avoid if cardiovascular instability

20% Intravenous Lipid Emulsion Therapy	
Immediate dosing	bolus 1.5 mL/kg over 1 min (100 mL for 70 kg adult) infusion of 0.25 mL/kg/min (1000 mL/hr for 70 kg adult)
Persistent instability for > 5 min	repeat two more bolus doses every 5 min double infusion rate to 0.50 mL/kg/min (2000 mL/hr for 70 kg adult) continue infusion for 10 min after return of cardiovascular stability maximum cumulative dose of 10-12 mL/kg (700-840 mL for 70 kg adult)

Reduce the risk of LAST by using correct local anesthetic dosing, aspirating before injecting, fractionating doses and using ultrasound.

Always be prepared for LAST when blocking patients. Consider stocking a LAST kit anywhere nerve blocks are performed *and know the location*. The kit should include 20% lipid emulsion and a LAST checklist.

Neurological signs, which can be non-specific, depressive or excitatory, often but not always precede cardiovascular signs.

SEVERE HYPONATREMIA

Christina Lu | Sean Smith

1. Treat if Na ≤120 mmol/L, or ≤ 125 mmol/L with symptoms.
2. Request initial laboratory screen and include urine studies.
3. Strictly monitor fluid balance (I/O). Consider urinary catheter.
4. Fluid restrict until full evaluation complete if asymptomatic.
5. Consider head CT imaging.
6. If the patient has seizures or altered mental state (AMS) give 100 mL of 3% NaCl over 10 min and repeat if needed.
   Aim to increase Na by 3 mmol/L and alleviate symptoms but if symptoms persist, increase to a max of 6 mmol/L.
7. If 3% NaCl is unavailable use 50 mL of 7-8.4% $NaHCO_3$.
8. Consider DDAVP to prevent overcorrection.
9. Do not correct Na > 6 mmol/L in the first 24 hr.
10. Monitor during the acute phase using serial labs q1-2hr.
11. If rise > 6 mmol/L/24hr give DDAVP and D5W.
12. Consult nephrology.

SEVERE HYPONATREMIA

Christina Lu | Sean Smith

Initial laboratory screen

Blood - chemistry, hepatic panel, osmolality, uric acid, TSH, cortisol.

Urine - urinalysis, osmolality, creatinine, electrolytes, urea, uric acid.

Osmolality and Na of both plasma and urine confirm diagnosis.

Classification of hyponatremia is based on plasma tonicity and ECF volume.

True hyponatremia is hypotonic hyponatremia (serum osmolality < 275 mOsm/kg).

Isotonic hyponatremia is known as pseudohyponatremia and is caused by an 'apparent' decrease in Na due to lipids or proteins. Hypertonic hyponatremia is usually due to significantly elevated glucose.

True hyponatremia can be classified on fluid volume status.

- Hypovolemic (dehydrated): renal loss (diuretics, mineralocorticoid deficiency, renal tubular acidosis, salt wasting nephropathy), body fluid losses (vomiting, diarrhea, sweating), third spacing.
- Euvolemia: SIADH, psychogenic polydipsia, beer potomania, low solute intake, hypothyroidism, adrenal insufficiency.
- Hypervolemic (fluid overloaded): congestive heart failure, hepatic failure, chronic renal failure.

Drug Doses	
3% NaCl	100 mL IVPB over 10 min x 1 peripherally or centrally in patients with AMS or seizure. For neurologically stable patients, consider 3% NaCl over 20 min. Repeat after 60 min if response inadequate.
DDAVP	2 mcg IV and then q6-8hr for 24-48 hr for patients with euvolemia/ hypovolemia or those who have overcorrected.
D5W	give over 15 min. Amount required is calculated based on total water needed to decrease Na back to 6 mmol/L > baseline. FW deficit in liters = 0.6 (Wt kg) x $\left[\left(\frac{\text{current Na}}{\text{desired Na}}\right) - 1\right]$

DDAVP can be given empirically to *avoid* overcorrection.
DDAVP and D5W are given to *correct* overshoot.

Rule of sixes

Maximum correction of 6 mmol/L in 24 hr for safety, but in severe cases correct by 6 mmol/L in the first 6 hr and then stop.

Beware of potassium supplementation as it will increase Na.
Rapid correction can lead to **Osmotic Demyelination Syndrome**.

THYROID STORM

Tim Warhurst | Jennifer Ng

1. Identify and aggressively treat the precipitating event.
2. Treat extreme agitation with dexmedetomidine (preferred first line therapy) or benzodiazepines.
3. Start fluid and electrolyte replacement.
4. Control hyperthermia with acetaminophen and active cooling.

To reduce effects of thyroid hormone, follow the steps below *strictly* in the order written to prevent deterioration.

5. Inhibit peripheral effects with beta-blocker.
6. Inhibit new synthesis with thionamides (PTU or methimazole).
7. Inhibit peripheral conversion with dexamethasone.
8. Inhibit release using iodine but wait *at least 1 hour* after first thionamide dose.
9. Inhibit enterohepatic circulation with cholestyramine 4 g q12hr.
10. For severe refractory storm consider plasma exchange.

THYROID STORM

Tim Warhurst | Jennifer Ng

Drug Doses	
Beta-blockers	
Propranolol	1 mg IV q10-15min until HR < 100 60-120 mg PO q4-6hr once enteral route working
Esmolol	500 mcg/kg IV push followed by infusion of 50-300 mcg/kg/min
Thionamides	
Propothiouracil (PTU)	load with 500-100 mg PO then 250 mg PO q4hr
Methimazole	20-25 mg PO q4hr
Dexamethasone	2-4 mg IV q6hr

Both thionamides can be given rectally. IV methimazole is available in Europe. There is a boxed warning for PTU - severe life threatening hepatotoxicity, issued by FDA in 2010. Consider changing to methimazole once thyroid storm controlled.

Inorganic Iodine

Wait at least an hour after thionamide given to prevent exacerbation.

- Saturated solution potassium iodide (SSKI): 5 drops PO/PR q6hr (20 drops/mL)
- Lugol's solution: 8 drops PO/PR q8hr (20 drops/mL)
- Sodium iodide: 500 mg IV q12hr

If there is a true allergy or contra-indication to iodine therapy (e.g. amiodarone induced thyrotoxicosis), substitute lithium carbonate 300 mg PO q6hr.

For iatrogenic or intentional (exogenous source) thyroid storm, cease exogenous thyroid hormone, control increased adrenergic tone with beta blockade and inhibit peripheral conversion with dexamethasone.

Heart failure

Results from excessive tachycardia and can precipitate pulmonary edema.

Use titratable beta blocker such as esmolol to decrease heart rate and allow diastolic relaxation, and consider non-invasive positive pressure ventilation (NIPPV) if required.

Aggressively treat hyperthermia with ice packs, cool IV fluids and cooling blankets.

TOXIC BRADYCARDIA

Chris Nickson

1. Ensure a secure airway and titrate FiO_2 to SpO_2 94-98%.

2. Get ECG, establish IV access, and insert an arterial line.

3. Consult a toxicologist early for support in management.

5. Treat bradycardia and hypotension.
   - fluid bolus (1-2 L IV crystalloid)
   - atropine 1-3 mg IV
   - 10% calcium chloride 20 mL IV, if calcium channel blockade
   - inotrope and/or vasopressor support

6. Give specific antidotes early.
   - digoxin-specific Fab fragments for digoxin toxicity
   - high dose insulin euglycemic therapy for calcium channel blocker and beta blocker toxicity. ***Perform q30min fingerstick BGL.***
   - organophosphate toxicity (see website for treatment)

7. Guide therapy with echo and hemodynamic monitoring.

8. Consider intralipid, methylene blue and circulatory assist devices in refractory cases.

9. Notify ECMO team for severe toxicity or cardiac arrest.

TOXIC BRADYCARDIA

Chris Nickson

Toxicological causes of bradycardia are highly lethal without aggressive therapy.
Suspect a toxicological cause of bradycardia if:

- patient had access to, or was administered a relevant agent
- patient is at risk of suicide
- no other cause is likely

In undifferentiated cases, signs and symptoms of toxicity may indicate the cause.

Signs and symptoms of toxicity	
Digoxin	increased automaticity (PVCs, AF, Aflutter, SVT) in combination with AV nodal blockade, gastrointestinal symptoms (nausea, vomiting, abdominal pain), altered mental state, elevated digoxin level
Calcium channel blocker	hyperglycemia (correlates with severity), may be normal HR (some classes of CCB), mental state usually good
Beta-blocker	hypoglycemia, potential bronchospasm, mental state may be depressed
Local Anesthetic	perioral numbness, dizziness, visual disturbance, light headedness, confusion, muscle twitching, seizures

Treat propanolol overdoses as sodium channel blocker toxicity.
Anticipate QT prolongation ± torsades de pointes from sotalol overdoses.

Hypotension (SBP < 90 mmHg) and bradycardia refractory to IV fluid therapy heralds the onset of serious cardiotoxicity. Atropine can be used as temporizing therapy, as can calcium in calcium channel blockade.

Echocardiography and hemodynamic monitoring can be useful for the selection and titration of therapies, including inotropes and vasopressors. In overdose, calcium channel blockers may lose selectivity and have unanticipated hemodynamic effects. Epinephrine is useful to improve inotropy and chronotropy. Norepinephrine is useful for vasopressor effects.

Vasopressin and methylene blue have been used to treat refractory vasoplegia due to calcium channel blockade.

BURNS

Dennis Djogovic

1. Assess airway and if signs of smoke inhalation, airway burn or edema, consider early intubation 43 .

2. Titrate high flow O_2 or increasing FiO_2 to $SpO_2 > 90\%$.

3. Establish two secure, reliable 14-18g IVs.

4. Send venous or arterial co-oximetry for CO and MetHb.

5. Cautiously expose the patient to properly assess and remove jewelry and burnt or wet clothe*s unless stuck on*.

6. Be prepared to treat symptomatic respiratory restriction or loss of extremity pulses with escharotomy.

7. Cover wounds with clean dressings or sterile sheets and aggressively manage body temperature loss.

8. Fluid replacement formulae vary but the resuscitation aim is to achieve good urine output.

9. Early hemodynamic instability may not be due to the burn. Use trauma principles and investigate for medical problems.

10. Tetanus prophylaxis is indicated but antibiotics are not.

11. Transfer the large, unstable, or critical location burn to an appropriate health center.

BURNS

Dennis Djogovic

Airways will become more challenging as fluid resuscitation proceeds, as will vascular access. Intervene early if the burn is significant in size or there is hemdynamic instability and assume it will be difficult.

Escharotomy is meant to relieve local compartment syndrome. Chest and limbs are the most common early sites at risk. Ideally these procedures should be done in the OR with sterile field and cautery. Bilateral incisions at anterior axillary lines with a connecting incision across the lower chest should be sufficient to allow for chest expansion.

Burn patients are not often unstable in the first few hours. If there is early instability, consider other surgical or medical comorbidities:

- traumatic injuries of all types during burn exposure
- pre-existing or concurrent medical emergencies
- cyanide exposure - treat using the Cyanokit and give 5 g IV (2 vials) over 15 min
- carbon monoxide exposure - treat with 100% O_2
- met Hb - treat with 2 mg/kg methylene blue IV

Most burn units will take off any dressings and replace with their own. Moist dressings can contribute to significant body temperature loss and should be avoided. Keep wounds clean and covered.

Traditional teaching uses the **Parkland formula** (4 mL/kg/% TBSA burned; the first half spread over 8 hr, the second half over the next 16 hr). However, over-resuscitation can be as detrimental as under-resuscitation, so current practice focuses on hourly fluid titration to attain urine output of 0.5 mL/kg/hr.
The **Rule of Tens** (burn size to nearest 10% x 10 = mLs/hr for 40-80 kg patient, increasing by 100 mL/hr for each 10 kg over 80 kg) is used by the US military.

Common indications for transfer to a burn center include:

- burns > 20% TBSA
- pediatric burns > 10% TBSA
- burns involving face, hands, feet, perineum, joints
- burns associated with electrical/chemical injury

MASS CASUALTY INCIDENT MCI

Howard Mell

1. Assemble all necessary resources as soon as possible.
2. Clear the department of all non-critical patients.
3. Contact the on-scene EMS personnel ASAP to receive estimates of number and type of patients.
4. Request additional staff (on-call and support personnel) and contact other agencies or institutions for assistance.
5. Appoint a nurse/physician team free from clinical duties to act as command and control for the response.
6. Triage all patients and divide department into 3 areas:
   - MCI patients requiring surgery to stabilize
   - MCI patients not requiring surgery
   - patients who are critically ill or injured outside of the MCI
7. Determine if decontamination of patients is needed.
8. Stockpile medications and IV fluids outside of pharmacy control machines to allow rapid access.
9. Consider using paper records and *downtime* procedures.
10. Communicate with the media early to control the message.

MASS CASUALTY INCIDENT MCI

Howard Mell

Managing MCI effectively requires knowledge of the institution's available resources, how they are accessed, and having them assembled before being needed to prevent any delay in treatment.

To clear department of non critical patients:

- Consider observation placement before workup complete for those who clearly require further care (severe abdominal pain, chest pain, neurological deficits).
- Return patients who are not likely to require admission to the waiting room (isolated orthopedic injuries, non-toxic rashes).

Additional security personnel may be required to control department access as volunteers, family and news media can hinder department operations.

Use surgical subspecialists as primary surgeons for initial damage control. OB/GYN, ENT, and urology are all surgically trained and can perform basic procedures if needed (controlling hemorrhage, line placement).

When contacting on-scene EMS personnel, limit it to a description of likely injuries and a patient number estimate to save EMS time - they will spread the load appropriately.
If EMS requests ED personnel to the scene, make the necessary arrangements if resources are available.

Some MCIs may have minimal EMS transport. Police cars or private vehicles may also be used, impacting on the patient 'balance' and ED preparation.

Incoming patients will be assigned a color by START triage but this is insufficient for department use.

A principle of START triage that should be employed in the ED is not to attempt CPR or heroic, labor intensive resuscitation unless no other patients require care.

When assessing the need for decontamination, be aware explosions may result from a chemical process.

Other institutions can help with patient care. They include nearby hospitals, community resources (Red Cross) and local emergency management agencies.

Make contact with the news media early to guide the message and prevent misinformation, and return operations to normal as soon as possible, as emergency mode is more prone to error with potential for patient harm.

MULTI-TRAUMA

Amit Maini | Francis O'Keeffe

1. Control life threatening external bleeding.
2. Control internal bleeding with pelvic binding and straightening of long-bone fractures.
3. Give high flow O_2 via non-rebreather mask and nasal cannula each ≥ 15 L/min.
4. Insert two large IVs and request CBC, blood chemistry, lactate, coagulation panel and blood type.
5. Limit crystalloid to < 1000 mL.
6. Perform a brief neurological examination.
7. If airway or ventilation is compromised, consider intubation and review appropriate checklist beforehand 43.
8. Fully expose patient for assessment, include log roll with MIS, and then cover completely to avoid hypothermia.
9. Give IV analgesia early in the resuscitation.
10. Use eFAST and/or CXR to identify and treat obstructive shock.
11. Transfuse in hemorrhagic shock using a 1:1:1 ratio aiming for SBP 80-90 mmHg (or MAP > 80 mmHg in head injury).
12. If blood is required, give TXA 1g unless contraindicated.
14. Once stable refer to relevant team or tertiary institution.

MULTI-TRAUMA

Amit Maini | Francis O'Keeffe

Pre-hospital notification applying major trauma call-out criteria will allow effective team assembly, early delegation of tasks and critical management planning.
The team leader will prioritize and determine which tasks can be done in parallel.

Prompt airway control is crucial in the multi-trauma patient.

Early intubation should occur in the following circumstances:

- Direct threats: airway burns, laryngeal trauma, bleeding facial fractures
- Indirect threats: altered mental state, severe agitation, uncontrolled pain, hypoventilation (chest trauma/spinal injury)

Delegate IV cannulation to suitably experienced personnel. A minimum 18g cannula is required and can be readily converted to a rapid infusion catheter (RIC).

Obstructive shock may rapidly lead to cardiac arrest. In the peri-arrest or arrested patient, urgent eFAST and/or CXR help identify tension pneumothorax or pericardial effusion. Treat with rapid bilateral finger thoracostomies and resuscitative thoracotomy respectively.

Hemorrhagic shock remains the most common cause of death in the multi-trauma patient. Blood loss is frequently underestimated. Fluids should be administered utilising a rapid infuser and combined blood warmer.

Treatment should focus on damage control principles.

Hemorrhage control
Control of external exsanguination via direct pressure, utilising tourniquets and/or pressure dressings. This allows temporary control of most external bleeding.

Permissive hypotension
Targeted lower BP by restricting fluid to avoid disruption of an unstable clot, dilutional coagulopathy and acidosis.

Hemostatic resuscitation
Early administration of warmed blood products in a balanced ratio to replace coagulation factors, platelets and RBCs.

To identify the need for laparotomy in trauma, FAST has high sensitivity, specificity and negative predictive value if done by experienced operator.

Once stabilized the patient may be transferred to CT for focused imaging. The liberal use of whole-body CT scanning of stable blunt trauma patients is controversial due to excessive radiation exposure but has become common practice at many trauma centers.

RESUSCITATIVE HYSTEROTOMY RH

Peri-Mortem Cesarean Delivery

Sara Gray

Initiate maternal resuscitation

1. Start CPR, *note the time* and follow standard ACLS.
2. Call for help - Code OB or maternal cardiac arrest team.
3. Displace obvious gravid uterus toward left lateral position.
4. Prepare emergency cesarean kit - if no kit use thoracotomy tray.
5. Prepare for neonatal resuscitation - warmer, blankets, BVM, airway tray, IV access, epinephrine.

Make the decision

6. Check fundus is above umbilicus or GA > 24 weeks.
7. If no ROSC by 4 min strongly consider RH.
8. Start RH earlier if maternal injury is clearly non-survivable.

Do the procedure

1. Continue ACLS and do not waste time transporting to OR.
2. Speed is more important than sterility but breathe slowly to reduce tremor.
3. Use #10 scalpel and make midline vertical incision from top of fundus to symphysis pubis.
4. Blunt dissect to peritoneum, use Kelly to pick it up and scissors to open.
5. Use #10 scalpel and make 5 cm vertical incision of uterus.
6. Use fingers to lift uterine wall away from fetus, then extend uterine incision with scissors.
7. Reach in, grasp and deliver head. The body will follow using external uterine pressure if needed.
8. Clamp cord in two places and cut between clamps then hand baby to neonatal team.
9. Deliver placenta then pack or close uterus and overlying layers.

RESUSCITATIVE HYSTEROTOMY RH

Peri-Mortem Cesarean Delivery

Sara Gray

Maternal resuscitation

Lateral uterine displacement in the supine position when GA > 20 weeks is recommended.
Lift the uterus cephalad and pull to patient's left. Tilting is no longer recommended.

Ensure IV/IO access above the diaphragm. Consider hypovolemia and treat with IV fluid bolus.

Ventilate with 100% O_2, anticipate a difficult intubation with high aspiration risk and use the most experienced operator.

If patient is on IV/IO magnesium, stop it and give IV/IO calcium - 30 mL of 10% calcium gluconate or 10 mL of 10% calcium chloride.

Call for help

Ideally the Code OB team should include a leader to coordinate three teams for:
1. Maternal ACLS 2. Resuscitative hysterotomy 3. Neonatal resuscitation

Resuscitative hysterotomy physiology

Emptying the uterus improves maternal survival via aorto-caval decompression, reduced uterine O_2 consumption, and improved pulmonary mechanics.

Earlier is better than later. The literature supports performing RH within 10 min of maternal arrest, but maternal survivors are reported out to 15 min, and fetal survivors to 30 min.

Gestational age considerations

Visibly pregnant patients likely have aorto-caval compression and impaired venous return. Emptying the uterus may improve maternal hemodynamics regardless of fetal viability.

Some centres may choose to perform RH only for viable fetuses (GA > 24 wks).

Equipment

Minimum: #10 scalpel, scissors, sponges/gauze packs, 2 Kelly clamps
Have emergency C-section kits (or a thoracotomy tray) stocked in high risk areas.

Search for treatable causes of maternal arrest - CAUSE HOPE

Cardiac (MI, dissection, cardiomyopathy)	**H**eme (DIC, bleeding)
Anesthetic complications	**O**ther (standard ACLS differential)
Uterine atony	**P**lacenta previa/abruption
Sepsis	**E**clampsia/Preeclampsia/HTN
Embolism	

Adapted from the 2015 AHA ACLS guidelines.

Joint session training with obstetric, pediatric, nursing and midwifery colleagues is essential for effective team performance.

POST PARTUM HEMORRHAGE PPH

Casey Parker | Penny Wilson

35A
CRISIS PROTOCOLS
SECTION ONE | SECTION TWO
CRISIS PROCEDURES

TEAM 1 Obstetrics ▸ manual hemostasis

To control ongoing blood loss

1. Deliver the placenta.
2. Examine for vaginal trauma and clamp bleeding lacerations.
3. Massage the uterus continuously.
4. Catheterize to empty the bladder.
5. Perform and maintain bimanual uterine compression.

TEAM 2 Nursing/Midwifery ▸ oxytocics

To increase uterine tone give in order listed (as needed)

1. Oxytocin 10 units IM.
2. Oxytocin 60 units in 1L crystalloid at 250 mL/hr IV.
3. Misoprostol 1000 mcg per rectum.
4. Ergometrine 250 mcg IV or methylergonovine 200 mcg IM.
5. Prostaglandin F2α 250 mcg IM.

TEAM 3 Anesthesia/Critical Care ▸ resuscitation

To replace volume loss

1. Insert two large bore IVs.
2. Take blood for massive transfusion panel.
3. Establish monitoring and consider arterial line.
4. Target MAP > 65, transfuse RBCs/plasma/PLT/cryo if indicated.
5. Consider tranexamic acid 1000 mg IV.

If bleeding persists activate MTP and transfer to the OR for definitive surgical management.

POST PARTUM HEMORRHAGE PPH

Casey Parker | Penny Wilson

Forming teams is the ideal if enough staff present, otherwise prioritize:

- manual hemostasis
- delivery of oxytocic agents
- IV access and transfusion
- definitive/surgical control

Review the 4 Ts of postpartum bleeding

TONE - the majority are primarily due to poor uterine contraction
TISSUE - retained placental tissue bleeds and impairs tone
TRAUMA - lacerations to the perineum, cervix or uterine rupture
THROMBUS - DIC, coagulopathy can occur early or be iatrogenic

If the placenta is still in situ, remove it with steady cord traction whilst bracing the uterus with the other hand above the pubic symphysis to avoid uterine inversion. If placenta cannot be removed then proceed to theatre as soon as possible.

Bimanual compression compresses the uterus between one hand in the vagina and one on the abdomen. The aim is to tamponade the bleeding uterus. **This will buy time until help arrives.** Emptying the bladder (via Foley insertion) also aids uterine contraction.

Oxytocic	Dose	Comment
Oxytocin	10 units IM bolus 60 units in 1L crystalloid 250 mL/hr IV	can cause hypotension / tachycardia when used as a bolus
Ergometrine **Methylergonovine**	250 mcg IV 200 mcg IM	is contraindicated in patients with pre-eclampsia, hypertension or ischemic heart disease vomiting is common
Misoprostol	1000 mcg per rectum	can also be given sublingually
PGF2a (Carboprost)	250 mcg IM	can be used directly intramyometrial can cause bronchospasm

Laboratory screen CBC, cross-match, coagulation panel, iCa and VBG.
Use TEG if available and beware hyperfibrinolysis. Early use of fibrinogen/cryoprecipitate may be of benefit and avoid excessive crystalloid fluids as coagulopathy may worsen. Only use if blood not immediately available and shock is present.

If an epidural is in situ check dermatome level and consider use of vasopressors for neurogenic component of shock.

Indications to activate massive transfusion protocol

- persistent bleeding despite initial oxytocic agents and manual maneuvers
- total estimated blood loss > 1500 mL
- persistent signs of shock inconsistent with external hemorrhage
- genital tract trauma or retained placenta requiring operative care

Consider placement of REBOA catheter

PRECIPITOUS DELIVERY
Newborn Resuscitation

Natalie May | Hazel Talbot

1. ***Start the clock.*** Aim to complete steps 1-5 in first 60 sec.
2. Dry, warm and cover the baby to conserve heat.
3. Assess the colour, tone, breathing and heart rate.
4. Call for assistance and delegate tasks if the baby is floppy, pale or deteriorating.
5. Open the airway in neutral head position and give *5 inflation breaths* of 3 sec duration at 30 cm H_2O (use 25 cm H_2O if preterm).

60s

6. Reassess heart rate and chest movement and if no improvement, reposition and repeat breaths.
7. Visualise the pharynx, gently suction under direct vision and intubate if necessary.
8. Following adequate inflation breaths, if heart rate remains < 100/min, start *standard positive pressure ventilation*.
9. If the heart rate falls below 60/min commence chest compressions at 120/min and use PPV with 3:1 ratio.
10. Reassess every 30 sec and if no response give epinephrine 10-30 mcg/kg and glucose 10% 2 mL/kg.
11. If hypovolemic or anemic, 10 mL/kg of isotonic crystalloid or O neg/CMV neg blood can be given and repeated if needed.
12. Admit to Neonatal ICU if successful resuscitation or prepare for family and team debrief.

PRECIPITOUS DELIVERY
Newborn Resuscitation

Natalie May | Hazel Talbot

Low birth weight pre-term infants require additional measures to prevent heat loss; consider plastic food wrapping below the neck, a thermal mattress, a resuscitaire, or fresh dry towels to preserve warmth.

Inflation breaths are delivered more slowly than ventilation breaths and aim to assist initial inflation of the baby's fluid-filled lungs. If there is not adequate breathing at birth, these breaths are usually sufficient to stimulate an increase in heart rate.

Ventilation breaths are indicated if heart rate increases in response to inflation breaths. Continue to ventilate with normal ventilation breaths at a rate of 30-40/min until there is adequate spontaneous ventilation.

If there is no chest movement, reposition and try airway maneuvers to ventilate more effectively including:

- positioning of head (neutral)
- jaw thrust (assistance may be required)
- oropharyngeal airway (Guedel)
- laryngoscopy, suction ± intubation

Routine suctioning is not indicated but gentle oropharyngeal suction may still be required. Nasopharyngeal suctioning has been associated with bradycardia. Endotracheal suctioning is still indicated in non-vigorous babies when meconium is present.

Avoid prolonged intubation attempts. Bag-valve-mask ventilation may be preferable or a supra-glottic airway can be used as an alternative. Monitor with end-tidal CO_2.

Color is not a reliable indicator of SpO_2 in the newborn; cyanosis is common and usually corrects but pallor may indicate acidosis or anemia. Apply SpO_2 monitoring - soon after birth it should be 60% increasing to > 90% at 10 min.

Room air is appropriate for resuscitation but if bradycardia (<60) persists after 90 sec, introduce blended oxygen at 21-30% and use oximetry for guidance. Hyperoxemia should be avoided and has not been shown to confer a survival benefit.

Assess HR by auscultation or ECG. If heart rate persistently < 60/min after adequate ventilation with supplementary oxygen for 30 sec, compressions are indicated. Centre compressions over the lower third of the sternum and aim to depress 1/3 of the anterior-posterior diameter of the chest (two-thumb encircling hands technique).

Ventilation chest compression ratio should be 1:3 with a pause for ventilation. Once intubated, the pause is no longer necessary. **Most babies requiring resuscitation will respond to airway opening and inflation breaths alone.** A small proportion will require further ventilation and cardiovascular support. Drugs are rarely indicated.

Epinephrine dose is 10 mcg/kg but can be increased to 30 mcg/kg. Bicarbonate is not recommended.

Post resuscitation care should include therapeutic hypothermia if evidence of evolving encephalopathy.

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CRISIS PROCEDURES

BALLOON TAMPONADE

Upper GI Bleed

David Menzies | Jess Mason

This guidance is compatible with both Blakemore and Minnesota tubes.

1. Intubate with head of bed elevated to 45 degrees.
2. Optimize coagulation and give terlipressin. Somatostatin, octreotide, or vasopressin can be used as alternatives.
3. Check the balloons on the occlusion tube for leaks.
4. Pass the lubricated tamponade balloon through the mouth to the stomach.
5. Inflate the gastric balloon with 50 mL of air (or contrast) to confirm the position using X-ray.
6. Fully inflate the gastric balloon using 250 mL for the Blakemore tube, or 500 mL for the Minnesota tube.
7. Apply 1 kg of traction using 1 L bag of saline and rolled gauze over a fulcrum.
8. For the Blakemore, insert an orogastric (OG) tube to just above the gastric balloon.
9. Irrigate and suction esophagus to assess ongoing bleeding.
10. Tamponade persistent bleeding by inflating the esophageal balloon while also removing the OG (if using a Blakemore).
11. Mark the tube at the teeth to monitor for any tube migration.
12. Repeat a CXR to review tube position.

BALLOON TAMPONADE
Upper GI Bleed

David Menzies | Jess Mason

Balloon tamponade is a bridge to banding or TIPSS.

Departments should have a kit for upper GI bleeding management.
See **resuscrisismanual.com** for supplies to assemble an appropriate kit.

Key differences between tubes

BLAKEMORE	MINNESOTA
gastric balloon holds 250 mL	gastric balloon holds 500 mL
no suction holes for the esophagus (OG tube is needed)	suction holes for the esophagus

Before commencing, check for leaks by inflating balloons in a basin of water.

Traction setup

1 kg of traction is provided from a 1 L bag of saline.

- Use rolled gauze to tie a slipknot around the tube at the base of the ports.
- Pull the other end of the gauze through the hole in the bag of saline.
- Hang the rolled gauze over an IV pole.
- If using the Hollister ETAD device a second plastic attachment can be added to secure the tube while maintaining traction (optional).

The gastric balloon tamponades the portal veins in the cardia of the stomach. This will often stop both gastric and esophageal varices. If bleeding persists after inflation of the gastric balloon, the esophageal balloon should be inflated but this is rarely required.

If the esophageal balloon is needed, the Blakemore tube requires removal of the OG. Attach the insufflating manometer to the 3-way stopcock on the esophageal balloon port and inflate the balloon to 30-45 mmHg.

Be aware the esophageal balloon should never be inflated alone and gastric balloon inflation in the esophagus can cause esophageal rupture.

ECG PCI REFERRAL
Indications

Madeleine Alexeeva

ECGs Prompting Activation of PCI Team

Classic Criteria

ST elevation in 2 contiguous leads at J point
Men < 40 years of age: 2.5 mm in V2-V3 and 1 mm in all other leads
Men > 40 years of age: 2 mm in V2-V3 and 1 mm in all other leads
Women: 1.5 mm in V2-V3 and 1 mm in all other leads

Presumed new Left Bundle Branch Block with one or more of

Unstable patient or
concordant ST changes or
discordant ST changes: Ratio of Deviation/(R or S) > 0.25

New Right Bundle with LAFB

LAFB: small Q waves with tall R waves in I and aVL,
small R waves, with deep S waves in II, III, aVF

Inferior Wall MI

ANY elevation in two contiguous leads (II, III, aVF)
with ANY amount of ST segment depression in aVL

Right Ventricular Infarction

Suspect in inferior wall MI with V1 elevation unless concomitant posterior MI
V3R and V4R elevation ≥ 0.5 mm increases specificity

Posterior MI

Precordial ST depression ≥ 1 mm maximal in leads V1-V4
Elevations ≥ 0.5 mm in V8 and V9 add specificity

High Lateral MI

Any degree of ST elevation in aVL with ST depression in lead III
(with or without II and aVF)

De Winter ST/T Complex

ST depression > 1mm upsloping at the J point in V1-V6
Tall T waves and normal QRS duration

ECG PCI REFERRAL
Indications

Madeleine Alexeeva

ECGs Prompting Activation of PCI Team

STEMI with Q Waves

May still be an acute MI especially with R wave

Diffuse ST Depressions with aVR Elevation

Activate if pain unrelieved and ST depression persists after treatment
PCI consult in all cases

Differentiating STEMI and Mimics

Left Ventricular Aneurysm

At least one lead with T wave amplitude/QRS ratio > 0.36 favors aneurysm

LVH

ST elevation discordant to deep S wave in V1-3 and
ST depression discordant to high voltage R wave in V5-6 favors LVH
Consider same criteria as LBBB

Anterior Early Repolarization

Download subtleSTEMI app or use calculator on **hqmeded-ecg.blogspot.com**

ECGs Prompting Consultation of PCI Team

Transient STEMI

Patients are at high risk for re-occlusion

Hyperacute T Waves

Get serial ECGs - will evolve to STEMI pattern
but consider hyperkalemia

Unrelieved Pain with NSTEMI

Patients should go to PCI

Wellens Phenomenon

In a chest-pain free patient who previously had anginal signs:
Biphasic T waves (up then down) or deep inverted T waves

ECMO REFERRAL

Jay Menaker | Terence Lonergan

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Acute Severe Respiratory Failure ▸ Veno-Venous ECMO

1. Confirm the patient is < 65 years old with a reversible cause of respiratory failure and without severe comorbidities.
2. Check ventilatory support has been maximized including all adjunctive therapies.
3. Confirm time on the ventilator has been < 7 days.
4. If there is failure to respond and the above criteria fulfilled, implement the ECMO referral process.

Circulatory Failure of Cardiac Origin ▸ Veno-Arterial ECMO

1. Confirm the patient is < 65 years old with a reversible cause of cardiac failure and without severe comorbidities.
2. Check the diagnosis is primary cardiac failure due to either cardiogenic shock or pulmonary embolus.
3. Check all conventional support modalities are maximized.
4. If shock persists despite maximal therapy and the above criteria fullfilled, implement the ECMO referral process.

ECMO REFERRAL

Jay Menaker | Terence Lonergan

Severe respiratory failure

Optimise conventional therapy and use ARDSnet ventilation strategy.
Consider prone positioning and/or neuromuscular blockade.

Assess the response

Does PaO_2 remain < 150 on FiO_2 0.6 or greater?
Is CO_2 retention severe (pH < 7.2) despite high plateau pressure (>30 cm H_2O)?
Murray Score 2.5 or greater?

To calculate the Murray Score add the point value below for each parameter and divide by 4.

Parameters / Points	0	1	2	3	4
PaO_2/FiO_2 on 100% O_2	300	225-299	175-224	100-174	< 100
CXR quadrants involved	normal	1	2	3	4
PEEP	5 or less	6-8	9-11	12-14	15 or >
Lung compliance mL/cmH_20	> 80	60-79	40-59	20-39	< 20

Acute cardiogenic or obstructive shock

Check adequate intravascular volume and correct inotrope and pressor doses. Consider intra-aortic balloon, percutaneous mechanical support devices and revascularization if indicated and available.

Refer patient in cardiogenic shock with one of the following indications:

Hemodynamic

- rapid deterioration with hemodynamic instability requiring repeated vasopressor boluses to maintain MBP > 50 *and* LVEF < 35% or LVEF 35-55% with mitral regurgitation or aortic stenosis
- cardiac index < 2.0 *and* norepinephrine > 0.1 mcg/kg/min, dobutamine > 5 mcg/kg/min and/or epinephrine > 0.02 mcg/kg/min
- SBP < 100 mmHg *and* norepinephrine > 0.2 mcg/kg/min, dobutamine 5 mcg/kg/min and/or epinephrine > 0.02 mcg/kg/min and LVEF < 35% or LVEF 35-55% with mitral regurgitation or aortic stenosis

Metabolic

- lactate - 2 consecutive values > 3.0 (with at least 30 min between measurements) with non-decreasing trend on steady dosing inotropes and/or vasopressors
- $ScvO_2$ - 2 consecutive values < 50% (with at least 30 min between measurements) with non-increasing trend on steady dosing inotropes and/or vasopressors

The patient's recoverability, appropriateness for ventricular assist device and possible cardiac transplantation must be considered. There should be no contraindications to anticoagulation.

If there is any doubt, consultation with the ECMO team may be helpful in the decision making process. Earlier referral often leads to increased survival.

ESSENTIAL PROCEDURES

Lateral Canthotomy Pericardiocentesis Penile Aspiration

Anne Messman | Abdallah Ajani

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CRISIS PROTOCOLS
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Lateral Canthotomy

1. Anesthetize the lateral canthus using 1-2% lidocaine with epinephrine.
2. Use a hemostat to compress the lateral canthus in the direction in which the incision will be made.
3. Use scissors to incise the canthus.
4. Incise inferior crus of the lateral canthal tendon from the orbital rim.
5. Remeasure pressure and if it's still elevated incise superior crus.

Considerations

Although Ophthalmology may be consulted prior to the procedure, the emergency physician must be prepared to perform this procedure if assistance is not available in a timely manner.

These incisions generally heal well and should not deter the emergency physician from performing this potentially vision-saving procedure.

Pericardiocentesis

1. Establish IV access, place patient on cardiac monitor and if possible, confirm pericardial effusion/ tamponade with ultrasound. Prepare 3-5" 18 gauge spinal needle with 60mL syringe. Consider using a pigtail catheter and Seldinger technique.
2. Sedate the patient if the procedure is non-emergent.
3. Raise head of bed to 45 degrees.
4. If time and conditions allow, prepare the skin with povidone-iodine and apply sterile drapes.
5. **Blind approach:** insert needle between the xiphoid process and left costal margin at 30-45 degree angle, aiming towards the left shoulder.

   Ultrasound guided approach: insert needle perpendicular to the skin at the left 4th or 5th intercostal space just lateral to the left sternal border.
6. Aspirate continually while advancing the needle.
7. Advance guidewire and pigtail catheter (if being used).

Penile Aspiration

1. Clean penis and mons with betadine.
2. Drape penis in sterile fashion.
3. Draw up 1% lidocaine without epinephrine.
4. Inject a total of 10-15 mL of 1% lidocaine in a "ring" completely around the base of the penis using 25G needle.
5. After anesthesia is achieved, locate the corpora cavernosa at the 3 o'clock and 9 o'clock positions.
6. Using a 19G or 21G butterfly needle and 20 mL syringe, aspirate blood from one side of penis until unilateral detumescence is achieved or bright red blood obtained (must avoid urethra).
7. Keep needle in place and inject 200 mcg phenylephrine - place 1 mL of 10 mg/mL phenylephrine in 100 mL of saline (100 mcg/mL) shake well, label and draw up 2 mL.
8. Repeat steps 5-7 on contralateral side.

ESSENTIAL PROCEDURES

Lateral Canthotomy Pericardiocentesis Penile Aspiration

Anne Messman | Abdallah Ajani

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CRISIS PROTOCOLS

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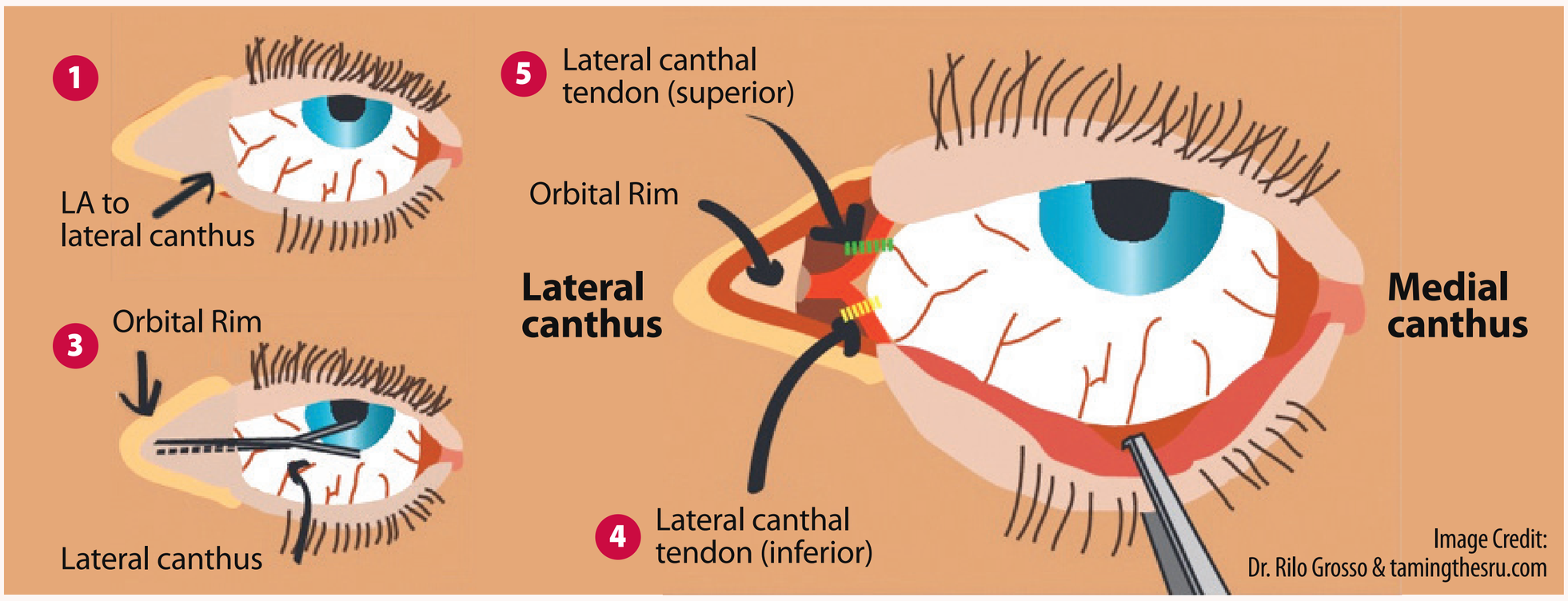
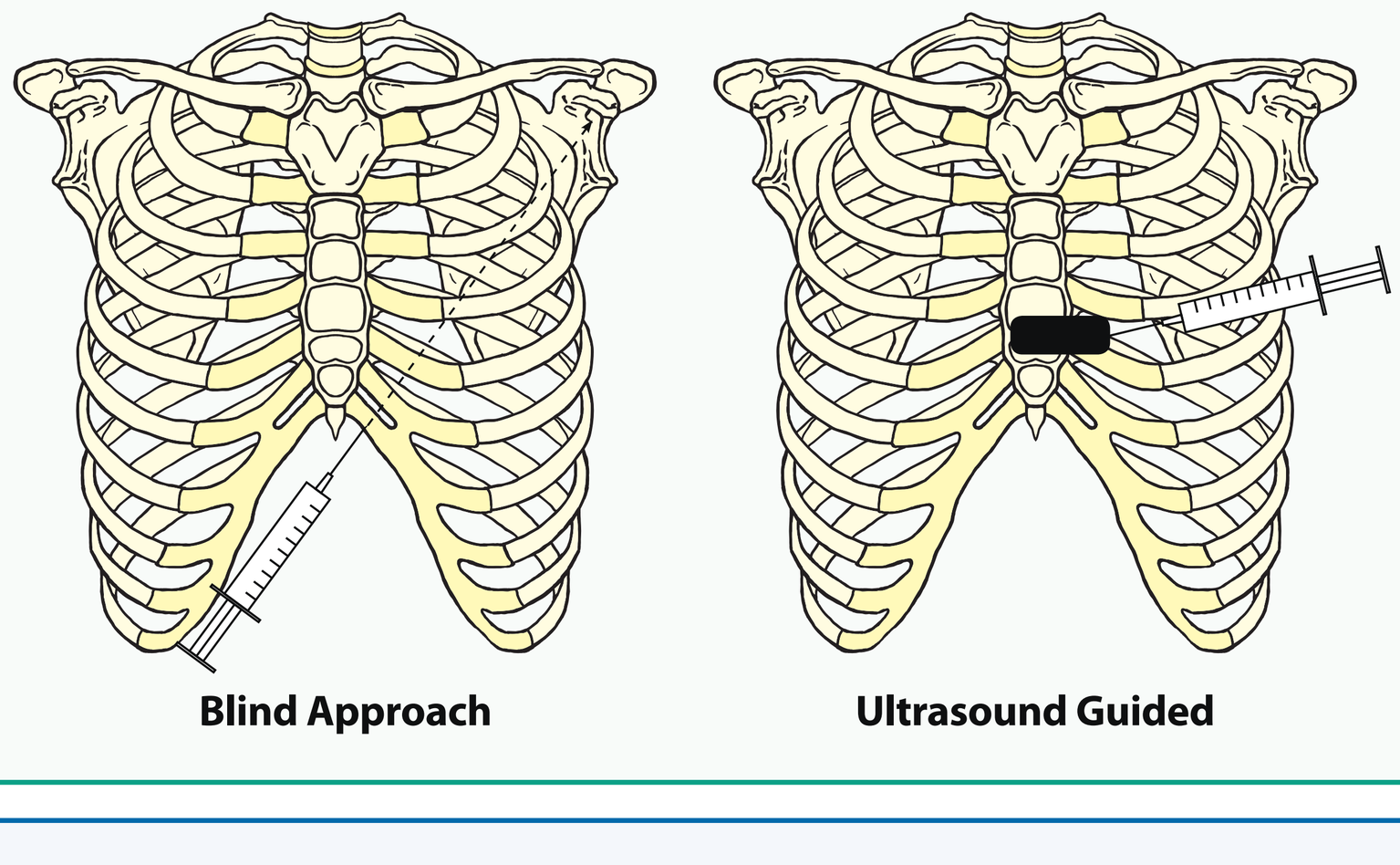
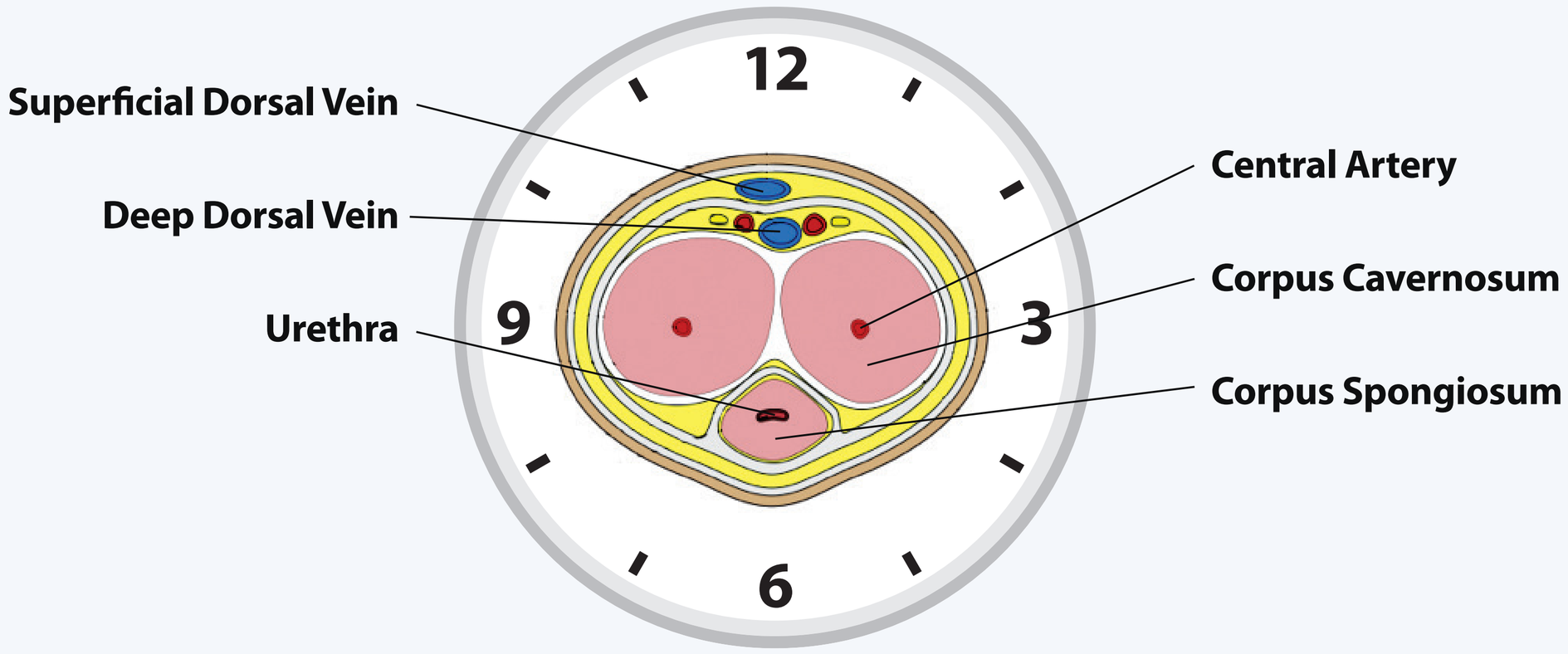
CRISIS PROCEDURES







HIGH PEAK PRESSURE
Volume Control Ventilation

Atif Farooqi

1. If SpO_2 low or falling, refer to *Crashing on Ventilator* 11.
2. Check for capnograph waveform and kinking of endotracheal tube or circuit.
3. Test the tube patency by passing a suction catheter and if it's difficult, consider bronchoscopic assessment or tube exchange.
4. Change tidal volume (TV) to ≤ 8 mL/kg predicted body weight.
5. Increase the ventilator *Peak Pressure Alarm Limit* until entire set volume is delivered without triggering.
6. Perform inspiratory hold on ventilator to assess plateau pressure.

If peak pressure and plateau pressure difference < 5 cm H_2O

7. Rule out dynamic hyperinflation by lowering the respiratory rate or disconnecting the ventilator.
8. If no dynamic hyperinflation, perform ultrasound or get CXR to exclude pneumothorax and main-stem tube position.
9. Consider atelectasis, pneumonia, mucus plugging or ARDS.
10. Try to reduce TV until the plateau pressure < 30 cm H_2O.

If peak pressure and plateau pressure difference > 5 cm H_2O

11. Auscultate to assess for obstructive airway disease and use bronchodilators if indicated.
12. Check each piece of ventilator circuit for obstruction.
13. Consider leaving peak pressure limit as high as necessary to deliver breaths if plateau pressure remains < 30 cm H_2O.

HIGH PEAK PRESSURE
Volume Control Ventilation

Atif Farooqi

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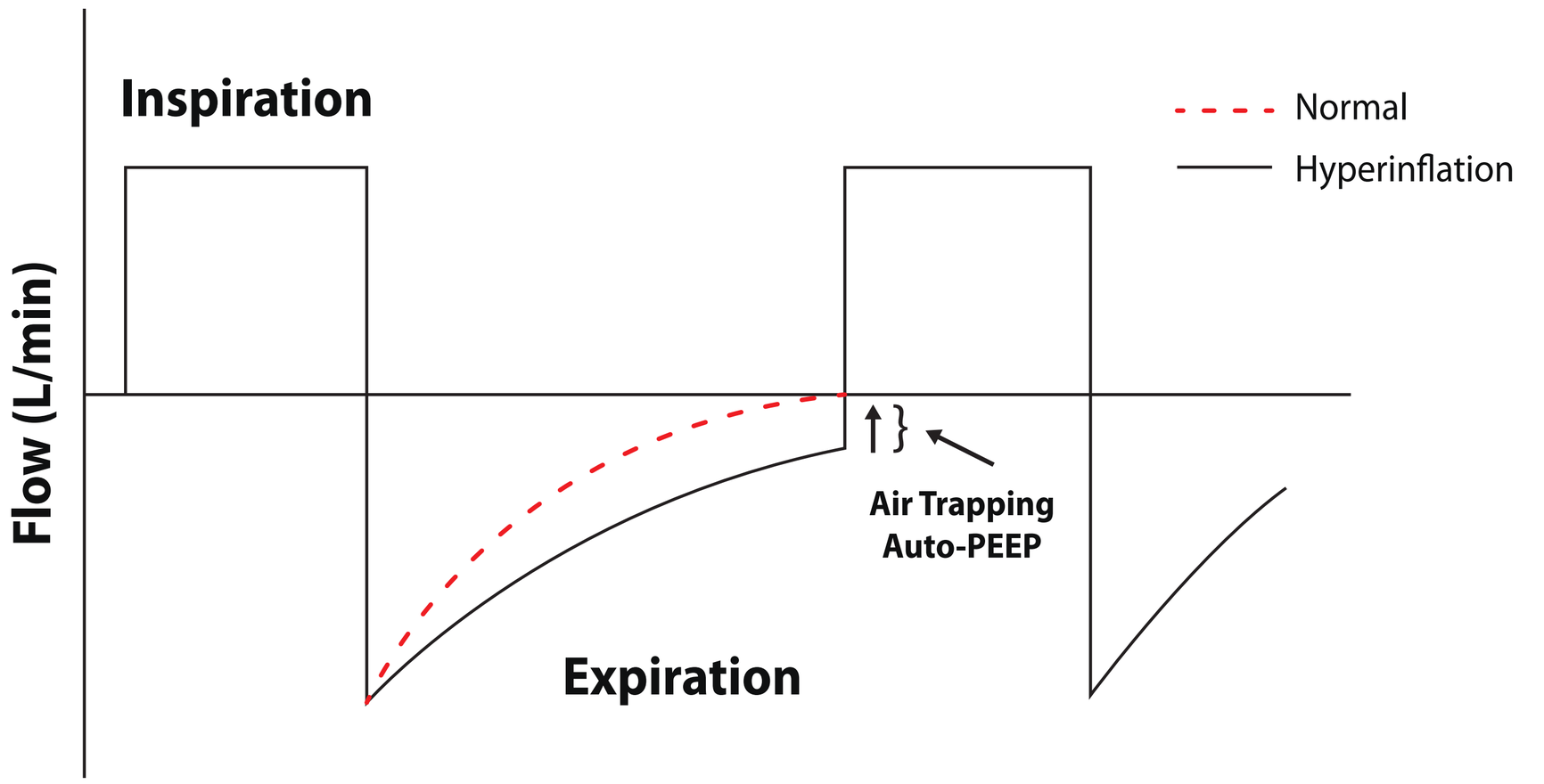
Raising the peak pressure limit on the ventilator will allow breaths to be delivered while troubleshooting.

Peak pressure = Airway Resistance Pressure + Compliance Pressure

Obtaining a plateau pressure by inspiratory hold on the ventilator allows for assessment of elevated compliance pressure and removes the factor of airway resistance.

Dynamic Hyperinflation

Lowering the respiratory rate can give adequate time for full exhalation before the next breath. The flow/time curve on the ventilator can be assessed for dynamic hyperinflation, and will look similar to the image below:



Pneumothorax, mucus plugging, and main-stem intubation can all cause increase peak and plateau pressures, as one lung receives a greater amount of tidal volume.

ARDS can diminish lung compliance and requires PEEP adjustment to recruit more alveoli. Refer to the ARDSNet PEEP Table at **www.ardsnet.org** for step-by-step guidance on ventilator adjustments in this setting.

Situations that have high peak pressures with low plateau pressures are typically associated with bronchial/bronchiolar narrowing, tracheal obstruction or endotracheal tube narrowing. Ventilator tubing should also be tested for patency or obstruction.

Bronchoscopy is a useful bedside tool providing rapid diagnostic support.

RAPID SEQUENCE INTUBATION RSI

Atif Farooqi

1. If the airway assessment indicates potential difficulty, consider awake intubation 44.

2. Place all supplies for RSI, failed airway, and post-intubation management at the bedside.

3. Run through checklist in a call-response manner.

4. Ensure all missing equipment or missed steps from the checklist have been remedied.

5. Proceed to RSI.

6. If intubation attempts fail, refer to *Failed Intubation* 13.

7. When successfully intubated, complete the post-intubation checklist 47.

RAPID SEQUENCE INTUBATION RSI

Checklist

Plan	Physiology considered (shock, hypoxemia, acidosis)
	Induction agent/muscle relaxant
	Post intubation analgesia/sedation
	± Push-dose epinephrine
	Failed plan verbalized
	Cricoid evaluation

Patient	Denitrogenated ≥ 3 min
	NC > 15 L/min
	SpO_2 = 100% or CPAP placed
	Check mouth, dentures, neck mobility
	Positioning
	Pulse oximetry visible/audible, not on BP arm
	IV access - reliable and tested

Equipment	Equipment on table
	BVM (+ PEEP valve) on flush-rate oxygen
	Waveform capnograph on BVM
	Videolaryngoscope
	Backup laryngoscope
	OPA, bougie, SGA, scalpel
	Suction x 2

Team	ELM/head elevation/collar briefing
	Eye/face protection

EMERGENCY AWAKE INTUBATION

Lauren Maloney

Dry and pretreat 10-15 min prior

1. If >10 min for prep, give IV glycopyrolate 4 mcg/kg (max 400 mcg).
2. Suction and pad mouth dry with gauze.
3. If using nasal route, prep with 2 puffs of phenylephrine 0.5% spray.

Topicalize

4. Paint the back of the tongue with 5% lidocaine paste using a tongue depressor.
5. Spray posterior pharynx and back of tongue using EZ Atomizer device with 10 mL of 4% lidocaine.
6. Angle tip to allow spraying of epiglottis and periglottic structures.
7. If using the nasal route, spray the desired nare.

Analgesia/sedation

8. If not perfectly topicalized use midazolam 1-2 mg or ketamine 10 mg aliquots IV, or remifentanil infusion.

Intubate

9. Preoxygenate using nasal cannula at high flow and either a nonrebreather mask or CPAP.
10. Intubate with preferred device (video bronchoscope/laryngoscope).
11. Confirm placement with waveform $EtCO_2$.
12. Only then administer analgesia, sedation ± muscle relaxant.

EMERGENCY AWAKE INTUBATION

Lauren Maloney

If 5% lidocaine paste is not available, use 2% viscous lidocaine.

If EZ-Atomizer is not available, the LMA MADGic device may be substituted.

Systemic and pulmonary absorption from spraying atomized 4% lidocaine in the throat/pharynx is low, but be careful spraying through the glottis with bronchoscope - limit volume to 2-3 mL.

Patients will often cough. Wear a face shield and mask.

Make sure all backup airway supplies are at the bedside, including RSI medications, failed airway equipment and a cricothyrotomy kit.

Consider having an assistant (or the patient) use a gauze sponge to pull on the tongue while topicalising the posterior pharynx and tongue area.

HEMODYNAMICALLY UNSTABLE INTUBATION

Rory Spiegel

1. Ensure adequate pre-oxygenation.
2. Perform a RUSH exam to screen for correctable causes of hypotension acutely exacerbated by sedation/anesthesia.
3. Correct any obstructive causes of shock prior to intubation.
4. Infuse fluids or blood products via large bore peripheral IV.
5. If hypotension is significant start a vasopressor infusion.
6. Have 10 mL of 10 mcg/mL epinephrine ready at the bedside.
7. Induce with 0.5 mg/kg ketamine.
8. Paralyse with 2 mg/kg rocuronium or succinylcholine IV.
9. If reoxygenation is required, use low tidal volume breaths at a rate of 6-8 /min and monitor with waveform $EtCO_2$.

HEMODYNAMICALLY UNSTABLE INTUBATION

Rory Spiegel

Patients at risk of hypotension during intubation are not always identified. It occurs in approximately 20% of intubations in the emergency setting and no single predictor is accurate enough for bedside use.

Clinicians should be prepared for peri-intubation hypotension in the majority of emergency department intubations.

Obstructive causes of shock including pneumothorax and pericardial tamponade should be corrected before proceeding.

> A **RUSH** exam (or some equivalent sonographic assessment of the common causes of hypotension) should be performed prior to intubation when time permits. This allows identification of obstructive pathologies that can be made significantly worse by the addition of positive pressure ventilation.

Patients with **significant IVC collapse** on bedside US prior to intubation can be at risk. However the absence of IVC collapse does not rule out the risk of peri-intubation hypotension in the sick emergency department population.

Patients who receive push-dose pressors just prior to intubation are less likely to experience hypotension.

The pharmacokinetics of sedative and paralyzing agents are altered in patients with hemodynamic compromise and most sedative agents work at a significantly lower dose than required for the normotensive patient. Ketamine, the most cardiac stable, should be dosed at 0.5 mg/kg but even with this modified dosing strategy, clinicians should still be prepared for worsening hemodynamics following intubation.

Conversely because of the decreased cardiac output, paralysing agents require a higher dose to achieve optimal intubating conditions. Rocuronium is normally given at 0.6-1.2 mg/kg but should be dosed at 2 mg/kg to reduce cognitive load during crises.

Positive pressure ventilation increases intrathoracic pressure resulting in decreased venous return.

If patients require reoxygenation it should be done at a low respiratory rate and with low tidal volume breaths, monitored with waveform $EtCO_2$.

NEUROPROTECTIVE INTUBATION

Rory Spiegel

1. *Ensure emergent intubation is not indicated* [48].

2. Preoxygenate in head-up position and plan to intubate with at least 20 degrees of head elevation.

3. When preparation complete give 5 mcg/kg fentanyl IV 3 min prior to induction.

4. Give 250 mL of 3% NaCl before induction if elevated ICP.

5. Give etomidate 0.3 mg/kg and succinylcholine 1.5 mg/kg (or rocuronium if immediate post-intubation exam not required).

6. Intubate with minimal laryngeal manipulation using an experienced operator and videolaryngoscopy.

7. Bolus nicardipine 0.25 mg IV as rescue medication for any blood pressure spikes in response to intubation.

8. If reoxygenation is required bag gently at 8-10 breaths/min ensuring adequate $EtCO_2$ response.

9. Start fentanyl at 1 mcg/kg/hr, propofol at 15 mcg/min and review *Post Intubation Checklist* [46].

NEUROPROTECTIVE INTUBATION

Rory Spiegel

If clinical circumstances dictate emergent intubation this checklist is not appropriate. It should only be utilized in patients who are hemodynamically stable and being intubated for an expected clinical course.

With all patients, care should be taken to ensure they receive adequate pre-oxygenation and denitrogenation.

Head elevation is the preferred position for intubation as it augments cerebral venous drainage and reduces spikes in ICP.

Using both an experienced operator and IV fentanyl should help reduce the hypertensive response. Supporting ventilation may be necessary from resultant respiratory depression.

In patients where acute spikes in blood pressure are potentially harmful as in unsecured SAH or aortic dissection, esmolol may be given before intubation. **The risk of hypotension should be balanced against benefits of blood pressure control based on clinical circumstances.**

The nicardipine bolus effect should be observed within 1-2 min and typically last approximately 5-7 min.

Mannitol is an alternative to 3% NaCl for ICP control, but monitor urine output carefully and replace losses caused by osmotic diuresis.

Succinylcholine is the preferred paralyzing agent for neuroprotective intubation, to allow for a reliable neurological examination shortly after. If rocuronium is used, ensure this examination is performed prior to intubation.

The increased intrathoracic pressure from positive pressure ventilation can inhibit venous drainage and increase in ICP. If patients require reoxygenation it should be done at a low respiratory rate and with low tidal volume breaths. $EtCO_2$ should be utilized to monitor both ventilation and respiratory rate.

Analgesia and sedation with fentanyl/propofol combination should follow intubation, titrated to allow a neurological exam when needed.

POST INTUBATION CHECKLIST

Kit Tainter

1. Confirm endotracheal tube placement is correct.
2. Provide pain control and additional sedation if needed.
3. Evaluate pCO_2 using VBG or ABG and check against $EtCO_2$.
4. X-ray to assess tube depth and exclude complicatons.
5. Place oro/nasogastric tube to decompress stomach.
6. Elevate the head 15-30 degrees.
7. Set a lung-protective ventilation strategy to maintain a plateau pressure < 30cm H_2O.
8. Humidify and assess the need for bronchodilators.
9. Establish endotracheal tube cuff pressure of 20-30 cm H_2O.
10. Ensure appropriate staffing - 1:1 or 1:2 nurse-to-patient ratio for ventilated patients.

POST INTUBATION CHECKLIST

Kit Tainter

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Use waveform $EtCO_2$ (gold standard), $EtCO_2$, ultrasound or esophageal detector device to confirm tube placement.

Correlate the gas pCO_2 with $EtCO_2$ for ongoing monitoring of ventilation.

Remember that residual muscle relaxation from RSI may mask signs of discomfort when titrating infusions.

Oro/nasogastric tubes may be required, especially if there is high suspicion for insufflation.

Keeping the head end slightly elevated decreases gastric reflux and ventilator-associated events like pneumonia.

Humidification reduces insensible fluid losses and dried secretions which in turn decreases the likelihood of mucus plugging.

Many patients may develop bronchoconstriction on the ventilator, even if not the primary indication for intubation. Consider bronchodilator therapy if indicated.

In-line suction eliminates the need to disconnect the circuit and removes a barrier to appropriate suction frequency.

Carefully monitor cuff pressures as higher pressures may lead to secondary injury and tracheomalacia.

Drug Infusions	
Analgesia	
fentanyl	1.5 mcg/kg bolus followed by 25-200 mcg/hr
morphine	0.1 mg/kg bolus followed by 1-10 mg/hr
dilaudid	1-2 mg bolus followed by 0.5-3 mg/hr
Sedation	
propofol	5-50 mcg/kg/min
dexmedetomidine	0.2-1.4 mcg/kg/min
lorazepam	10-100 mcg/kg/hr
midazolam	0.25-1 mcg/kg/min

Non-benzodiazepine sedatives are preferred.

POST CARDIAC ARREST MANAGEMENT

Joshua Reynolds

1. Confirm endotracheal tube position with waveform $EtCO_2$.
2. Request ECG, CXR, laboratory screen and diagnostic scans.
3. Test brainstem reflexes and motor response.
4. Use short-acting agents for sedation and analgesia.
5. Target a core temperature of 32-36°C and suppress shivering.
6. Aim for MAP 80 mmHg using one or more of vasopressors, inotropes or fluids if indicated.
7. Keep $PaCO_2$ 35-45 mmHg (use pH-stat) and SaO_2 94-98%.
8. Maintain blood glucose at 140-180 mg/dL (7.5-10 mmol/L).
9. Transfer to cardiac catheter lab for STEMI, STEMI equivalent or if the cause remains unknown.
10. Apply continuous EEG monitoring if available.
11. Admit or transfer to regional cardiac center.

POST CARDIAC ARREST MANAGEMENT

Joshua Reynolds

The following questions can help direct post arrest management
Was the event witnessed? Was there bystander CPR?
What was the duration without CPR (no-flow) and with CPR (low-flow)?
What were the preceding events and clinical context?

If patient is awake and following commands, identify the etiology (5Hs5Ts).
Laboratory screen should include CBC, blood chemistry, lactic acid and ABG.

Imaging
Non-contrast brain CT, bedside ultrasound and CT chest angiography (including coronary arteries if available), will assist diagnosis and provide prognostic information.

Hypothermia
If patient follows commands, maintain strict fever suppression. If not, actively induce targeted temperature management immediately after ROSC. Central venous or esophageal temperature monitoring are the most accurate.

Supression of shivering
Begin with 650 mg acetaminophen NGT q6hr unless allergic and 30 mg buspirone q8hr (unless on MAO inhibitor), then add the following in order until control established.

+ fentanyl infusion
+ propofol infusion
+ forced air warming device (for counterwarming on both arms)
+ $MgSO_4$ 2 g IV, then 0.5-1 g/hr for target serum Mg 3 mg/dL
+ dexmedetomidine infusion
+ ketamine 0.5 mg/kg IVP, may start drip at same dose per hour

Only then consider cisatracurium 0.15 mg/kg IV q1hr PRN

Analgesia/Sedation
Many sedatives result in hypotension, so consider the hemodynamics when selecting drugs to sedate or suppress shivering. Consider using only short-acting agents that don't accumulate.

Prognosis
Decisive assessment resulting in withdrawal of life-sustaining therapies should not be made until at least 72 hr after return of spontaneous circulation (cases with advanced directives or terminal comorbid conditions notwithstanding).

TRANSVENOUS PACEMAKER INSERTION

Chidi Nwakanma | Charles Bruen

In hemodynamically significant bradyarrhythmia and if *hyperkalemia* has been excluded start external pacing at 50-60 bpm.

Placement

1. Use sterile conditions and ultrasound guidance.
2. Insert sheath introducer in right IJV (preferred) or left subclavian vein.
3. Place sterile sleeve over pacer wire and slide proximally.
4. Test balloon at the distal catheter tip and then fully deflate.
5. Advance pacing wire to 15 cm, inflate balloon and lock valve.
6. Connect electrode adapters to the wire's distal ends (leads) and hand to a non-sterile assistant.
7. Assistant inserts the ***proximal*** lead (+) to the ***positive*** port on the pacer generator and the distal lead (-) to negative port.
8. Turn on and set rate to 60 bpm and output to maximal mA.
   If already externally pacing the patient, set the rate to 100.

Capture

9. Advance pacer wire until electrical capture achieved - pace spikes will correspond to wide QRS at set rate.
10. Confirm mechanical capture by SpO_2 waveform, arterial line or echocardiographic rate equal to pacemaker setting.
11. Dial output down to minimum threshold mA needed to maintain capture then set output at twice (2x) this level.
12. Deflate balloon.
13. Turn off transcutaneous pacer and decrease transvenous pacing rate to 60-80 bpm.
14. Extend sterile sleeve along the length of wire, lock both ends and secure sheath introducer with suture.
15. Confirm position with follow up CXR.

TRANSVENOUS PACEMAKER INSERTION

Chidi Nwakanma | Charles Bruen

Indications for emergent transvenous pacemaker placement

- hemodynamically significant bradycardia from high grade heart block or sinus node dysfunction
- stable bradycardia with high grade heart block or sinus node dysfunction - to protect against decompensation (when close monitoring or cardiological intervention not immediately available)
- overdrive pacing of severe QT prolongation - to treat or avoid Torsade de Pointes

Transcutaneous pacing should be utilized first in hemodynamically unstable patients.

Transvenous pacing is not usually effective poisoning bradycardias.

The hypothermic bardycardic patient is generally unresponsive to electrical stimulation (and atropine). The bradycardia is appropriate at those temperatures.

Pacing generators

Many types are available. Clinicians should be familiar with the device in their own department before it is required.

Set sensitivity to asynchronous while floating the wire.

The rate should be set to 100 if the patient is receiving transcutaneous pacing, then reduced to 60-80 once capture is established and then transcutaneous stopped.

If amperage > 5 mA is required for capture, the pacemaker tip is probably not fully engaged with ventricular wall and may require advancing.

After placement, sensitivity threshold should be adjusted to avoid R on T phenomena.

Potential complications

- pacer wire misplacement/movement
- atrial or ventricular perforation leading to pericardial tamponade
- induced dysrhythmias (oversensing p or T waves)

The RESUSCITATION CRISIS MANUAL

The Resuscitation Crisis Manual is a practical quick reference handbook in aviation checklist format, giving step-by-step instructions for the management of Emergency and ICU resuscitation events.

- 49 crisis management and crisis procedure protocols cover the major scenarios requiring immediate therapeutic intervention to prevent a catastrophic outcome. These include life-threatening cardiovascular, respiratory, airway, neurological, trauma, obstetric and metabolic events.
- A crisis procedure section includes commonly performed life-saving procedures as well as referral criteria for ECG findings and ECMO.
- Color-coded, bulleted and numbered lists enhance memory recall in stressful conditions.
- The tabbed layout enables quick and easy navigation for use in the midst of a crisis.
- Water resistant and durable, suitable for attachment to code and resus carts.

A must have manual for all levels of healthcare professionals working in the Emergency, ICU and Resuscitation areas.

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