EMERGENCY MEDICINE

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INITIAL PATIENT ASSESSMENT AND MANAGEMENT

APPROACH
❑ 5 level triage (new Canadian Guidelines)
- I Resuscitation
- II Emergent
- III Urgent
- IV Less-urgent
- V Non-urgent

PRIORITIZED PLAN
1. Rapid Primary Survey (RPS)
2. Resuscitation (often occurs at same time as RPS)
3. Detailed Secondary Survey
4. Definitive Care

RAPID PRIMARY SURVEY (RPS)
Airway maintenance with C-spine control
Breathing and ventilation
Circulation (pulses, hemorrhage control)
Disability (neurologic status)
Exposure (complete) and **E**nvironment (temperature control)
❑ restart sequence from beginning if patient deteriorates

A. AIRWAY
❑ first priority is to secure airway
❑ assume a cervical (C-spine) injury in every trauma patient —> immobilize with collar and sand bags

Causes of Airway Obstruction
❑ decreased level of consciousness (LOC)
❑ airway lumen: foreign body (FB), vomit
❑ airway wall: edema, fractures
❑ external to wall: lax muscles (tongue), direct trauma, expanding hematoma

Airway Assessment
❑ assess ability to breathe and speak
❑ signs of obstruction
- noisy breathing is obstructed breathing until proven otherwise
- respiratory distress
- failure to speak, dysphonia
- adventitous sounds
- cyanosis
- agitation, confusion, "universal choking sign"

❑ think about ability to maintain patency in future
❑ can change rapidly, ALWAYS REASSESS

Airway Management
❑ goals
- achieve a reliably patent airway
- permit adequate oxygenation and ventilation
- facilitate ongoing patient management
- give drugs via endotracheal tube (ETT) if IV not available
  - **NAVEL**: **N**arcan, **A**tropine, **V**entolin, **E**pinephrine, **L**idocaine

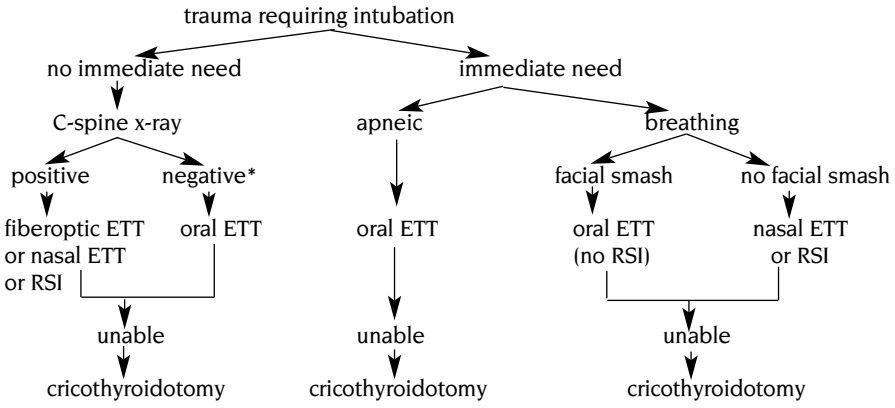
❑ start with basic management techniques then progress to advanced

1. Basic Management (Temporizing Measures)
❑ protect the C-spine
❑ chin lift or jaw thrust to open the airway
❑ sweep and suction to clear mouth of foreign material
❑ nasopharyngeal airway
❑ oropharyngeal airway (not if gag present)
❑ transtracheal jet ventilation (through cricothyroid membrane)
- used as last resort, if unable to ventilate after using above techniques

2. Definitive Airway
❑ endotracheal intubation (ETT) (see Figure 1)
- orotracheal +/– Rapid Sequence Intubation (RSI)
- nasotracheal - may be better tolerated in conscious patient
- does not provide 100% protection against aspiration

❑ indications for intubation
- unable to protect airway
- inadequate spontaneous ventilation
- O_2 saturation < 90% with 100% O_2
- profound shock
- GCS = 8
- anticipate in trauma, overdose, congestive heart failure (CHF), asthma, and chronic obstructive pulmonary disease (COPD)
- anticipated transfer of critically ill patients

❑ surgical airway (if unable to intubate using oral/nasal route)
- needed for chemical paralysis of agitated patients for investigations
- cricothyroidotomy



* note: clearing the C-spine also requires clinical assessment (cannot rely on x-ray alone)
* ETT (endotracheal intubation), RSI (rapid sequence intubation)

Figure 1. Approach to Endotracheal Intubation in an Injured Patient

B. BREATHING

LOOK mental status (anxiety, agitation), colour, chest movement, respiratory rate/effort
FEEL flow of air, tracheal shift, chest wall for crepitus, flail segments and sucking chest wounds, subcutaneous emphysema,
LISTEN sounds of obstruction (e.g. stridor) during exhalation, breath sounds and symmetry of air entry, air escaping

Oxygenation and Ventilation
❑ measurement of respiratory function: rate, pulse oximetry, ABG, A-a gradient, peak flow rate
❑ treatment modalities
- nasal prongs —> simple face mask —> oxygen reservoir —> CPAP/BiPAP
- Venturi mask: used to precisely control O_2 delivery
- Bag-Valve mask and CPAP: to supplement ventilation

C. CIRCULATION - see Shock section

Table 1. Estimation of Degree of Shock

Class	I	II	III	IV
Blood loss	< 750 cc (<15%)	750 - 1500 cc (15 - 30%)	1500 - 2000 cc (30 - 40%)	> 2000 cc (> 40%)
Pulse	< 100	> 100	> 120	> 140
Blood pressure (BP)	Normal	Normal	Decreased	Decreased
Respiratory Rate (RR)	20	30	35	> 45
Capillary refill	Normal	Decreased	Decreased	Decreased
Urinary output	30 cc/hr	20 cc/hr	10 cc/hr	None
CNS status	Anxious	Mild	Confused	Lethargic
Fluid replacement	Crystalloid	Crystalloid	Crystalloid + blood	Crystalloid + blood

Table 2. Estimated Systolic Blood Pressure (SBP) Based on Position of Palpable Pulse

	Radial	Femoral	Carotid
sBP (mmHg)	> 80	> 70	> 60

❑ stop major external bleeding
- apply direct pressure
- elevate profusely bleeding extremities if no obvious unstable fracture
- consider pressure points (brachial, axillary, femoral)
- do not remove impaled objects as they tamponade bleeding
- use tourniquet as last resort

❑ treatment
- 2 large bore peripheral IV's for shock (14-16 gauge)
- bolus with Ringer's lactate (RL) or normal saline (NS) (2 litres) and then blood as indicated for hypovolemic shock
- inotropes for cardiogenic shock
- vasopressors for septic shock

D. DISABILITY

❑ assess level of consciousness by **AVPU** method (quick, rudimentary assessment)
A - **A**LERT
V - responds to **V**ERBAL stimuli
P - responds to **P**AINFUL stimuli
U - **U**NRESPONSIVE

❑ size and reactivity of pupils
❑ movement of upper and lower extremities (UE/LE)

E. EXPOSURE / ENVIRONMENT

❑ undress patient completely
❑ essential to assess all areas for possible injury
❑ keep patient warm with a blanket +/– radiant heaters; avoid hypothermia

RESUSCITATION

❑ restoration of ABCs
❑ manage life-threatening problems as they are identified
❑ often done simultaneously with primary survey
❑ vital signs q 5-15 minutes
❑ ECG, BP and O_2 monitors
❑ Foley catheter and nasogastric (NG) tube if indicated
- Foley contraindicated if blood from urethral meatus or other signs of urethral tear (see Traumatology section)
- NG tube contraindicated if significant mid-face trauma or basal skull fracture
- may use orogastric tube

❑ order appropriate tests and investigations: may include CBC, lytes, BUN, Cr, glucose, amylase, INR/PTT, β-HCG, tox screen, cross + type

DETAILED SECONDARY SURVEY

❑ done after RPS problems have been corrected
❑ designed to identify major injuries or areas of concern
❑ head to toe physical exam and X-rays (C-spine, chest, pelvis - required in blunt trauma)

History

❑ "**AMPLE**": **A**llergies, **M**edications, **P**ast medical history, **L**ast meal, **E**vents related to injury

Head and Neck

❑ pupils
- assess equality, size, symmetry, reactivity to light
  - inequality suggests local eye problem or lateralizing CNS lesion
- reactivity/level of consciousness (LOC)
  - reactive pupils + decreased LOC —> metabolic or structural cause
  - non-reactive pupils + decreased LOC —> structural cause
- extraocular movements (EOM's) and nystagmus
- fundoscopy (papilledema, hemorrhages)

❑ palpation of facial bones, scalp
❑ tympanic membranes

Chest

❑ flail segment, contusion
❑ subcutaneous emphysema
❑ auscultate lung fields
❑ CXR

Abdomen

❑ inspection, palpation, percussion, auscultation
❑ immediate laparotomy if
- refractory shock with no other discernable cause
- obvious peritonitis
- increasingly distended abdomen
- positive diagnostic peritoneal lavage/CT scan

❑ rectal exam for gastrointestinal (GI) bleed, high riding prostate and anal tone
❑ bimanual exam in females

Musculoskeletal (MSK)
- ❑ examine all extremities for swelling, deformity, contusion, tenderness
- ❑ log rolled, palpate thoracic (T) and lumbar (L)-spines
- ❑ pelvis: palpate iliac crests and pubic symphysis, pelvic stability (lateral, AP, vertical)

Neurological Examination (see Neurosurgery Chapter)
- ❑ Glasgow Coma Scale (GCS)
- ❑ alterations of rate and rhythm of breathing are signs of structural or metabolic abnormalities
- ❑ progressive deterioration of breathing pattern implies a failing CNS
- ❑ full cranial nerve exam
- ❑ assessment of spinal cord integrity
  - conscious patient: assess distal sensation and motor ability
  - unconscious patient: response to painful or noxious stimulus applied to extremities
- ❑ signs of increased intracranial pressure (ICP)
  - deteriorating LOC (hallmark of increasing ICP)
  - deteriorating respiratory pattern
  - Cushing reflex (high BP, slow heart rate)
  - lateralizing CNS signs (e.g. cranial nerve palsies, hemiparesis)
  - seizures
  - papilledema (occurs late)

DEFINITIVE CARE
1. continue therapy
2. continue patient evaluations (special investigations)
3. specialty consultations including O.R.
4. disposition: home, admission, or another setting

Ethical Considerations

Adults
- ❑ Emergency Rule: consent not needed when patient is at imminent risk of suffering serious injury (i.e., severe suffering, loss of limb, vital organ or life)
  AND obtaining consent is either:
  a) not possible (eg., patient is comatose), OR
  b) would increase risk to the patient (e.g., time delay)
- ❑ any CAPABLE and INFORMED patient can refuse any treatment or part of treatment, even if it is life-saving
- ❑ in E.D. consider: is the patient truly capable? does pain, stress, psychological distress cloud their judgement?
- ❑ the emergency rule assumes that most people would want to be saved in an emergency
- ❑ EXCEPTIONS: Treatment can not be initiated if:
  1. a competent patient has previously refused the same or similar treatment and there is no evidence to suggest the patient's wishes have changed
  2. an advance directive is available
  3. a do not resucitate (DNR) order is available
  4. refusal for help in a suicide situation is NOT an exception; care must be given
- ❑ when in doubt, treat

Children
- ❑ treat immediately if patient is at imminent risk
- ❑ parents / guardians have right to make treatment decisions, however
- ❑ if parents refuse treatment that is life-saving or will potentially alter the child's quality of life, CAS is almost always contacted
- ❑ MDs cannot then treat without consent of Child Services

Jehovah's Witnesses
- ❑ refuse whole blood, packed red blood cells (PRBCs), platelets, plasma and WBCs even if life-saving
- ❑ should be questioned directly about the use of albumin, immunoglobulins, hemophillic preparations
- ❑ do not allow for autollogous transfusion unless there is uninterrupted extra corporeal circulation
- ❑ ask for the highest possible quality of care without the use of the above interventions (e.g., crystalloids for volume expansion, attempts at bloodless surgery)
- ❑ may carry a signed, witnessed, dated Medical Alert card +/– bracelet specifically identifying their religious affiliation and the procedures they will not consent to
- ❑ will generally sign hospital forms releasing medical staff from liability
- ❑ are consenting, capable adults and have the right to refuse medical treatment
- ❑ most legal cases involve children of Jehovah's Witnesses
- ❑ large centres may have policies surrounding care
- ❑ if life-saving treatment is refused (e.g., blood transfusion) CAS is contacted

PRE-HOSPITAL CARE

LEVEL OF PROVIDERS

- ❑ levels of providers not standard in every community
- ❑ first responders usually non-medical (i.e. firefighters, police)
  - administer CPR, O_2, first aid, assist ventilation, automatic defibrillation (firefighters)
- ❑ Level I Paramedic
  - basic airway management (oropharyngeal airway and suction), O_2 by mask or cannula, CPR, semi-automatic external defibrillation, basic trauma care
  - blood sugar kit, administer some drugs (nitro, salbutamol, epinephrine, ASA, glucagon)
- ❑ Level II Paramedic
  - start IV, ventilation of intubated patients, interpret ECGs, manual defibrillation
- ❑ Level III Paramedic
  - advanced airway management (intubation), cardioversion and defibrillation, emergency drugs (IV epinephrine, naloxone, dopamine), ACLS, needle thoracostomy
- ❑ base hospital physicians
  - provide medical control and verbal orders for Paramedics through line patch
  - ultimately responsible for delegated medical act and pronouncement of death in the field

APPROACH TO COMA

Definitions

- ❑ Coma - a sleep-like state, unarousable to consciousness
- ❑ Stupor - unresponsiveness from which the patient can be aroused
- ❑ Lethargy - state of decreased awareness and mental status (patient may appear wakeful)

GLASGOW COMA SCALE (GCS)

- ❑ designed for use on trauma patients with decreased LOC; good indicator of severity of injury
- ❑ often used for metabolic causes as well, but less meaningful
- ❑ most useful if repeated
  - changes in GCS with time is more relevant than the absolute number
  - patient with deteriorating GCS needs immediate attention

Table 3. Glasgow Coma Scale

Eyes Open		Best Verbal Response		Best Motor Response	
spontaneously	**4**	answers questions appropriately	**5**	obeys commands	**6**
to voice	**3**	confused, disoriented	**4**	localizes pain	**5**
to pain	**2**	inapproriate words	**3**	withdraws to pain	**4**
no response	**1**	incomprehensible sounds	**2**	decorticate (abnormal flexion)	**3**
		no verbal response	**1**	decerebrate (abnormal extension)	**2**
				no response	**1**

- ❑ best reported as a 3 part score: Eyes + Verbal + Motor = total
- ❑ provides indication of degree of injury
  - 13-15 = mild injury
  - 9-12 = moderate injury
  - < 8 = severe injury
- ❑ if patient intubated, GCS score reported out of 10 + T (T= tubed, i.e. no verbal component)

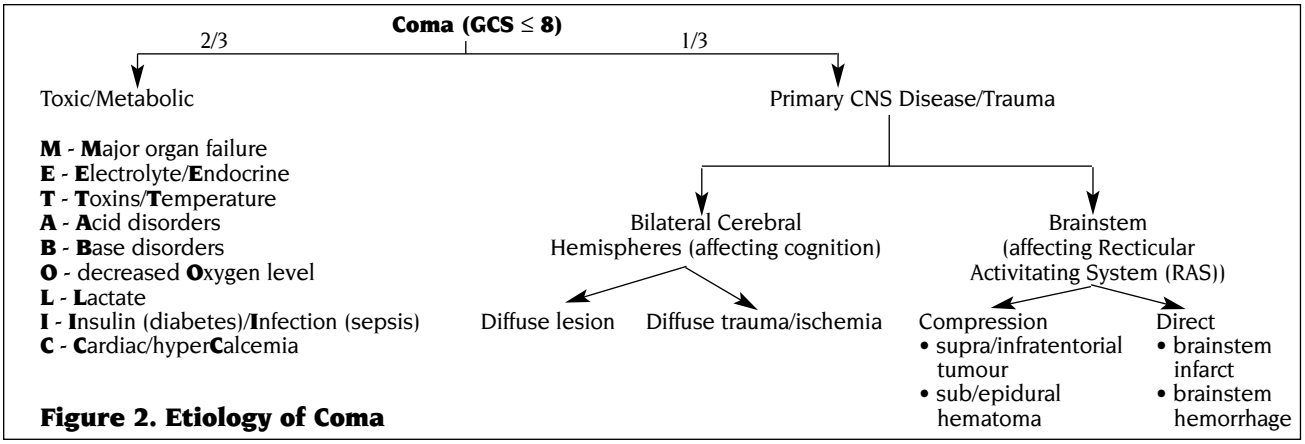


Figure 2. Etiology of Coma

MANAGEMENT OF THE COMATOSE PATIENT

❑ ABC's
❑ airway management should take into account
- probability of C-spine injury, high if:
  - major trauma
  - head or face trauma
  - history of fall or collapse
- likelihood of aspiration
- adequacy of ventilation
  - correct hypoxia and hypercarbia
- reversibility of the cause of the coma
  - hypoglycemia or narcotic overdose (OD) rapidly reversible therefore ETT may not be needed (controversial)
- need for maximizing oxygenation
  - carbon monoxide (CO) poisoning
  - raised ICP (usually requires ETT)

Components of Resuscitation

❑ IV access
❑ rapid blood sugar, CBC, lytes, Cr and BUN, LFT's, glucose, serum osmolality, ABG's
❑ ECG
❑ universal antidotes
- thiamine 100 mg IM before glucose (if cachectic, alcoholic, malnourished)
- glucose: 50 cc of 50% (D50W) if glucose < 4 mmol/L (70 mg/dL) or rapid measurement not available
- naloxone 0.4-2.0 mg IV if narcotic toxidrome present (risk of withdrawal reaction in chronic opiate users, therefore use naloxone 0.4 mg in known users)

❑ drug levels of specific toxins if indicated
❑ rapid assessment and correction of abnormalities essential to prevent brain injury

Secondary Survey and Definitive Care

❑ focused history (from family, friends, police, paramedics, old chart, etc.)
❑ onset and progression
- abrupt onset suggests CNS hemorrhage/ischemia or cardiac cause
- progression over hours to days suggests progressive CNS lesion or toxic/metabolic cause

❑ condition prior to coma
- confusional/delerious states suggest toxic/metabolic cause
- antecedent trauma, seizure activity, fever
- medications, alcohol, or drugs

❑ past medical history (e.g. similar episode, depression)
❑ physical examination
- vitals including temperature, cardiac, chest, abdominal exam and inspection for 5 N's

❑ selected laboratory and imaging studies (x-ray and CT)

Inspection - The Five N's

❑ **N**oggin – e.g. Raccoon eyes, Battle's sign (appear ~8 hrs. after trauma)
❑ **N**eck – C-spine, neurogenic shock, nuchal rigidity
❑ e**N**t – otorrhea, rhinorrhea, tongue biting, odour on breath, hemotympanum
❑ **N**eedles – track marks of IV drug abuse
❑ **N**eurological – full examination essential but concentrate on
- GCS - follow over time
- respirations (rate and pattern)
  - apneustic or ataxic (brainstem)
  - Cheyne-Stokes (cortical, brainstem or toxic/metabolic)
- posture
  - decorticate: severe bilateral damage above midbrain
  - decerebrate: damage in midbrain, diencephalon
- movement
  - spontaneity, symmetry and seizure activity
- pupils - reactivity and symmetry (CN II, III), papilledema (increased ICP)
- reflexes
  - corneal reflex (CN V, VII)
  - gag reflex (CN IX, X)
  - oculocephalic reflex/doll's eye reflex (after C-spine clearance): test for brainstem integrity
  - oculovestibular reflex (rule out tympanic perforation and cerumen impaction first)
  - deep tendon reflexes and tone
  - plantar reflex
- caloric stimulation: normal response consists of ipsilateral slow gaze (brainstem mediated) and contralateral saccadic correction (cortically mediated); cannot be voluntarily resisted
- lumbar puncture (LP) after normal CT to rule out meningitis, subarachnoid hemorrhage (SAH) (increasing evidence that LP can be done as primary investigation if no evidence of increased ICP)

Diagnosis

❑ findings suggesting a toxic-metabolic cause
- dysfunction at lower levels of the brainstem (e.g. caloric unresponsiveness)
- respiratory depression in association with an intact upper brainstem (e.g. reactive pupils)
- see Tables 4 and 5

Table 4. Structural vs. Metabolic Coma

	Structural	Toxic-Metabolic
Pupillary Reaction	asymmetric or absent	pupils equal, round, regular reaction to light (see Table 5)
Extraocular movements	asymmetric or absent	symmetric or absent
Motor Findings	asymmetric or absent	symmetric or absent

Table 5. Toxic - Metabolic Causes of Fixed Pupils

Cause	Pupils	Characteristics	Treatment
Anoxia	dilated	antecedent history of shock, cardiac or respiratory arrest, etc.	100% O_2, expectant management
Anticholinergic Agents (e.g. atropine, TCA's)	dilated	tachycardia	physostigmine (for Atropine) warm, dry skin sodium bicarbonate (for TCA)
Cholinergic Agents (e.g. organo-phosphates)	small, barely perceptible reflex	diaphoresis, vomiting, incontinence, increased secretions	atropine
Opiates (e.g. heroin)	pinpoint, barely perceptible reflex (exception: meperidine)	needle marks	naloxone
Hypothermia	normal or dilated	history of exposure temperature < 35°C	warm patient (e.g. warm IV solutions, blankets)
Barbiturates	midsized to dilated	history of exposure positive serum levels confusion, drowsiness, ataxia shallow respirations and pulse	ABC's no specific antidote
Methanol (rare)	dilated	optic neuritis increased osmolal gap metabolic acidosis	ethanol ± dialysis

- ❑ it is essential to re-examine comatose patients frequently - can change rapidly
- ❑ diagnosis may only become apparent with the passage of time
  - delayed deficit after head trauma suggestive of epidural hematoma

Disposition

- ❑ readily reversible coma: discharge if adequate follow-up care available
- ❑ enduring decreased LOC: admit to service based on tentative diagnosis
- ❑ transfer patient if appropriate level of care not available

TRAUMATOLOGY

EPIDEMIOLOGY
- ❑ trauma is the leading cause of death in patients < 44 years
- ❑ trimodal distribution of death
  - minutes: lethal injuries; death usually at the scene
  - early: this period includes the "golden hour" (death within 4-6 hours, decreased mortality with trauma care)
  - days-weeks: death from multiple organ dysfunction, sepsis, etc.
- ❑ injuries generally fall into two categories
  - blunt
    - most common
    - MVC, pedestrian-automobile impact, motorcycle collision, fall, assault, sports, etc.
  - penetrating
    - increasing in incidence
    - gunshot wound, stabbing, impalement

CONSIDERATIONS FOR TRAUMATIC INJURY
- ❑ important to know the mechanism of injury in order to anticipate/suspect traumatic injuries
- ❑ always look for an underlying cause (alcohol, other drugs, seizure, suicide, medical problem)
- ❑ always inquire about head injury, loss of consciousness, amnesia, vomiting, headache and seizure activity

Motor Vehicle Collisions (MVC)
- ❑ weight and size of vehicle
  - inversely proportional to severity of injury
- ❑ speed of vehicle
- ❑ location of patient in vehicle
- ❑ type of crash and associated serious injuries:
  - lateral/T-bone: head, cervical spine, thoracic and abdominal injury
  - front end: head, cervical spine, thoracic, abdominal, pelvic and lower extremity
  - rear end: over-extension of cervical spine (whiplash injury to neck)
  - roll over: energy dissipated, less likely severe injury if victim restrained by seatbelt
  - ejection of patient from vehicle/entrapment of patient under vehicle
- ❑ degree of damage to vehicle, (especially if intrusion into passenger compartment)
- ❑ broken windshield (head and cervical spine injury), condition of steering wheel (chest injury), knees to dashboard (hip, femur injury)
- ❑ use and type of seatbelt
  - lap belt: spine and abdominal injury
  - shoulder belt: look for major vessel injury
- ❑ airbag deployment
- ❑ death of same vehicle occupant
- ❑ motorcycle collisions
  - motorcycle speed
  - site of anatomic impact
  - use of helmet

Pedestrian-Automobile Crash
- ❑ vehicle speed
- ❑ site of impact on car
  - children: tend to be run over
  - adults: tend to be struck in lower legs, impact again on car and ejected to the ground
    - look for triad of: 1. tibia-fibula or femur fracture, 2. truncal injury and 3. craniofacial injury

Falls
- ❑ distance of fall: 50% mortality at 4 stories and 95% mortality at 7 stories (1 story = 12 feet)
- ❑ position in which patient landed and type of surface
  - look for shock, lower extremity, spine and pelvic fractures

Assault
- ❑ weapon used
- ❑ strangulation
- ❑ sexual assault (see Common ER Presentations section)

Gunshot Wounds
- ❑ type of gun
  - handgun injuries: low or high velocity, extent of injury may be limited to a small area
  - hunting and rifle injuries: high velocity, widespread injury
- ❑ type of ammunition (e.g. hollow point bullets)
- ❑ range of shot
  - close range: massive tissue destruction at close range, deposition of wadding into wound
- ❑ route of entry

Stab Wounds
- ❑ route of entry, length of blade
- ❑ type of penetration (stab, slash, impalement)
- ❑ victim recollection and witness reports are often inaccurate and may not correlate with depth/severity of wound

SHOCK IN THE TRAUMA PATIENT (see Anesthesia Chapter)

❑ inadequate organ and tissue perfusion (brain, kidney, extremities)
❑ SHOCK IN THE TRAUMA PATIENT IS HEMORRHAGIC UNTIL PROVEN OTHERWISE

Classification

❑ hemorrhagic shock (most common) - see Table 6
❑ cardiogenic shock - e.g. blunt myocardial injury
❑ obstructive shock - e.g. tension pneumothorax, cardiac tamponade, pulmonary embolism
❑ distributive shock - e.g. spinal/neurogenic, septic and anaphylactic shock

Table 6. Classification of Hemorrhagic Shock (70kg male)

Class	Blood loss (mL)	BP	Pulse	Resp rate	Urine output
I	< 15% (< 750)	normal	<100	14-20	> 30 mL/hour
II	15-30% (750-1500)	normal	>100	20-30	0-30 mL/hour
III	30-40% (1500-2000)	↓	>120	30-40	5-15 mL/hour
IV	>40% (>2000)	↓↓	>140	> 35	0 mL/hour

Clinical Evaluation

❑ rapidly assess for other causes of traumatic shock
❑ clinical features of acute hemorrhage
- early: tachypnea, tachycardia, narrow pulse pressure, reduced urine output (U/O), reduced capillary refill, cool extremities and reduced central venous pressure (CVP)
- late: hypotension and altered mental status

Management of Hemorrhagic Shock

❑ secure airway and supply O_2
❑ control external bleeding (prompt surgical consultation for active internal bleeding)
❑ infusion of 1-2 L of NS or RL as rapidly as possible
- replace lost blood volume at ratio of 3:1 (maintain intravascular volume)

❑ if no response, consider ongoing blood loss (e.g. chest, abdomen, pelvis, extremities) —> operative intervention required
❑ blood transfusion
- indicated if: 1. severe hypotension on arrival, 2. shock persists following crystalloid infusion, 3. rapid bleeding
- packed RBC's (PRBCs)
  - cross-matched (ideal but takes time)
  - type-specific (provided by most blood banks within 10 min.) preferred to O-negative uncrossmatched blood if both available
  - O-negative (children and women of child-bearing age)
  - O-positive (everyone else) if no time for cross and match
  - consider complications with massive transfusions

Unproven or Harmful Treatments

❑ Trendelenberg position
❑ steroids (used only in spinal cord injury)
❑ MAST garments - non efficacious for treatment of shock; no longer used
❑ vasopressors during hemorrhagic shock

CHEST TRAUMA

❑ trauma to the chest accounts for, or contributes to 50% of trauma deaths
❑ two types
- immediately life-threatening
- potentially life-threatening

A. IMMEDIATELY LIFE-THREATENING CHEST INJURIES

❑ identified and managed during the primary survey
- airway obstruction
- tension pneumothorax
- open pneumothorax
- massive hemothorax
- flail chest
- cardiac tamponade

❑ 80% of all chest injuries can be managed non-surgically with simple measures such as intubation, chest tubes, and pain control

Table 7. Immediately Life-Threatening Chest Injuries

	Physical Exam	Investigations	Management
Airway Obstruction	• anxiety, stridor, hoarseness, altered mental status • apnea, cyanosis	• do not wait for ABG's to intubate	• definitive airway management • intubate early
Tension Pneumothorax • a clinical diagnosis • one-way valve causing accumulation of air in pleural space	• respiratory distress, tachycardia, distended neck veins, cyanosis, asymmetry of chest wall motion • tracheal deviation away from pneumothorax • percussion hyperresonnance • unilateral absence of breath sounds, hypotension	• non-radiographic diagnosis	• large bore IV needle, 2nd ICS mid clavicular line, followed by chest tube in 5th ICS, anterior axillary line • ICS = intercostal space
Open Pneumothorax • air entering chest from wound rather than trachea	• gunshot or other wound (hole > 2/3 tracheal diameter) ± exit wound • unequal breathsounds	• ABG's: decreased pO_2	• air-tight dressing sealed on 3 sides • chest tube • surgery
Massive Hemothorax • > 1500 cc blood loss in chest cavity	• pallor, flat neck veins, shock • unilateral dullness • absent breath sounds, hypotension	• upright CXR: costophrenic blunting	• restore blood volume • chest tube • may receive thoracotomy: > 1500 cc total blood volume ≥ 200 cc/hr continued drainage
Flail Chest • free-floating segment of chest wall due to > 4 rib fractures, each at 2 sites • underlying lung contusion (cause of morbidity and mortality)	• paradoxical movement of flail segment • palpable crepitus of ribs • decreased air entry on affected side	• ABGs: decreased pO_2, increased pCO_2 • CXR: rib fractures, lung contusion	• O_2 + fluid therapy + pain control • judicious fluid therapy in absence of systemic hypotension • positive pressure ventilation • +/– intubation and ventilation
Cardiac Tamponade	• penetrating wound (usually) • Beck's triad (hypotension, distended neck veins, muffled heart sounds) • pulsus paradoxus • Kussmaul's sign	• ECHO	• IV fluids • pericardiocentesis • open thoracotomy

B. POTENTIALLY LIFE-THREATENING CHEST INJURIES

❑ identified in secondary survey (CXR)
- **C** - **C**ontusion: pulmonary, myocardial
- **H** - **H**ernia: traumatic diaphragmatic
- **ES** - **ES**ophageal perforation
- **T** - **T**racheobronchial disruption/**T**raumatic aortic injury

❑ need to have high index of suspicion, usually dependent on mechanism of injury

Pulmonary Contusion

❑ history: blunt trauma to chest
❑ interstitial edema impairs compliance and gas exchange
❑ CXR: areas of opacification of lung within 6 hours of trauma
❑ management
- maintain adequate ventilation
- monitor with ABG, pulse oximeter and ECG
- chest physiotherapy
- positive pressure ventilation if severe

Blunt Myocardial Injury (Rare)

❑ history: blunt trauma to chest (usually in setting of multi-systemtrauma and therefore difficult to diagnose)
❑ physical examination: overlying injury, i.e. fractures, chest wall contusion
❑ investigations
- ECG: arrhythmias, ST changes
- patients with a normal ECG and normal hemodynamics never get dysrhythmias

❑ management
- O_2
- antiarrhythmic agents
- analgesia

Ruptured Diaphragm

❑ difficult to diagnose (often missed)
❑ more often diagnosed on left side since liver conceals defect on right
❑ history: blunt trauma to chest or abdomen (e.g. high lap belt in MVC)
❑ investigations
- CXR - abnormality of diaphragm/lower lung fields/NG tube placement
- CT scan and endoscopy - sometimes helpful for diagnosis

❑ management
- laparotomy for diaphragm repair and because of associated intra-abdominal injuries

Esophageal Injury
- ❑ history: usually penetrating trauma (pain out of proportion to degree of injury)
- ❑ investigations
  - CXR: mediastinal air (not always)
  - esophagram (Gastrografin)
  - flexible esophagoscopy
- ❑ management
  - early repair (within 24 hrs.) improves outcome but all require repair

Penetrating Neck Trauma
- ❑ includes all penetrating trauma to the three zones of the neck (see Otolaryngology Chapter)
  - zone 1: below cricoid cartilage, extending to thoracic inlet
  - zone 2: between angle of mandible and cricoid cartilage
  - zone 3: area of neck above mandible
- ❑ management
  - injuries require further evaluation if deep to platysma (should not be explored in E.D.)
  - zone 1 and 3 injuries —> angiography
  - zone 2 injuries —> O.R. for exploration
- ❑ DON'T:
  - clamp structures (can damage nerves)
  - probe
  - insert NG tube (leads to bleeding)
  - remove weapon/impaled object

Airway Injuries
- ❑ always maintain a high index of suspicion
- ❑ larynx
  - history: strangulation, clothes line, direct blow, blunt trauma, any penetrating injury involving platysma
  - triad of:
    1. hoarseness
    2. subcutaneous emphysema
    3. palpable fracture, crepitus
  - other symptoms: hemoptysis, dyspnea
  - investigations
    - CXR
    - CT scan
    - arteriography (if penetrating)
  - management
    - airway - manage early because of edema
    - C-spine: may also be injured, consider mechanism of injury
    - surgical: tracheotomy vs. repair
- ❑ trachea/bronchus
  - frequently missed
  - history: deceleration, penetration, increased intra-thoracic pressure
  - complaints of dyspnea, hemoptysis
  - examination: subcutaneous air, Hamman's sign (crunching sound synchronous with heart beat)
  - CXR: mediastinal air, persistent pneumothorax or persistent air leak after chest tube inserted for pneumothorax
  - management
    - surgical repair if > 1/3 circumference

Aortic Tear
- ❑ 90% tear at subclavian (near ligamentum arteriosum), most die at scene
- ❑ salvageable if diagnosis made rapidly in E.D.
- ❑ history
  - sudden high speed deceleration (e.g. MVC, fall, airplane crash)
  - complaints of chest pain, dyspnea, hoarseness (frequently absent)
- ❑ physical examination: decreased femoral pulses, differential arm BP (arch tear)
- ❑ investigations: CXR, CT scan, transesophageal echo (TEE), aortography (gold standard)
- ❑ x-ray features
  - wide mediastinum (most consistent)
  - pleural cap
  - massive left hemothorax
  - indistinct aortic knuckle
  - tracheal deviation to right side
  - depressed left mainstem bronchus
  - esophagus (NG tube) deviated to right side
- ❑ management
  - thoracotomy (may treat other severe injuries first)

Late Causes of Death in Chest Trauma
- ❑ respiratory failure
- ❑ sepsis (adult respiratory distress syndrome (ARDS))

ABDOMINAL TRAUMA

❑ two mechanisms
- blunt trauma - usually causes solid organ injury
- penetrating trauma - usually causes hollow organ injury

Blunt Trauma

❑ two types of hemorrhage
- intra-abdominal bleed
- retroperitoneal bleed

❑ high clinical suspicion in multi-system trauma

❑ physical exam unreliable in multi-system trauma
- slow blood loss not immediately apparent
- other injuries may mask symptoms
- serial examinations are required

❑ inspection: contusions, abrasions, distension, guarding

❑ palpation: tenderness, rebound tenderness, rigidity

❑ diagnostic tests are indicated in patients with
- unexplained shock
- equivocal signs of abdominal injury
- unreliable physical exam (paraplegia, head injury, substance use)
- high likelihood of injury (pelvic/lumbar fracture, etc.)
- impending periods of non-observation (e.g. surgery)

❑ diagnostic tests include
- CXR
  - free air under diaphragm (if patient not supine)
  - diaphragmatic herniation
- ultrasound: FAST (focused abdominal sonogram for trauma)
  - to identify presence/absence of free fluid in the peritoneal cavity
  - NOT used to identify specific organ injuries
- CT scan: best investigation if patient stable enough
- IVP
- diagnostic peritoneal lavage (DPL)
  - tests for intra-peritoneal bleed
  - cannot test for
    - retroperitoneal bleed
    - discerning lethal from trivial bleed
    - diaphragmatic rupture
  - criteria for positive lavage:
    - > 10 cc gross blood
    - bile, bacteria, foreign material
    - RBC count > 100,000 x 10^6/L, WBC > 500 x 10^6/L, amylase > 175 IU

❑ management
- general: fluid resuscitation and stabilization
- surgical: watchful wait vs. laparotomy
- solid organ injuries: decision based on hemodynamic stability, not the specific injuries
  - hemodynamically unstable or persistently high tranfusion requirements —> laparotomy
  - all hollow organ injuries —> laparotomy

❑ note: seatbelt injuries may have
- retroperitoneal duodenal trauma
- intraperitoneal bowel transection
- mesenteric injury
- L-spine injury

Penetrating Trauma

❑ high risk of gastrointestinal (GI) perforation and sepsis

❑ history: size of blade, calibre/distance from gun, route of entry

❑ local wound exploration with the following exceptions:
- thoracoabdominal region (may cause pneumothorax)
- back or flanks (muscles too thick)

❑ management
- gunshot wounds —> always require laparotomy
- stab wounds - "Rule of Thirds"
  - 1/3 do not penetrate peritoneal cavity
  - 1/3 penetrate but are harmless
  - 1/3 cause injury requiring surgery
- mandatory laparotomy if
  - shock
  - peritonitis
  - evisceration
  - free air in abdomen
  - blood in NG tube, Foley catheter or on rectal exam

GENITOURINARY TRACT (GU) INJURIES

❑ diagnosis based on mechanism of injury, hematuria (gross or microscopic), and appropriate radiological studies

Renal

❑ etiology
- blunt trauma
  - contusions (parenchymal ecchymosis with intact renal capsule)
  - parenchymal tears
    - non-communicating (hematoma)
    - communicating (urine extravasation, hematuria)
- penetrating injuries
- renal pedicle injury due to acceleration/deceleration

❑ history: mechanism of injury, hematuria, flank pain
❑ physical exam: costovertebral angle (CVA) tenderness, upper quadrant mass, shock
❑ investigations
- CT scan (study of choice if hemodynamically stable)
- intravenous pyelogram (IVP) (during laparotomy)
- renal arteriography (if renal artery injury suspected)

❑ management
- 90% conservative (bedrest, analgesia, antibiotics)
- 10% surgical for
  - hemodynamically unstable or continuing to bleed > 48 hours
  - major urine extravasation
  - renal pedicle injury
  - all penetrating wounds
  - major lacerations
  - renal artery thrombosis
  - infection

Ureter

❑ etiology
- iatrogenic (most common)
- blunt (rare) at uretero-pelvic junction
- penetrating (rare)

❑ history: mechanism of injury, hematuria
❑ physical exam: findings related to intra-abdominal injuries
❑ investigations: retrograde ureterogram
❑ management: uretero-uretostomy

Bladder

❑ etiology
- blunt trauma
  - extraperitoneal rupture from pelvic fracture fragments
  - intraperitoneal rupture from trauma + full bladder
- penetrating trauma

❑ history: gross hematuria, dysuria, urinary retention, abdominal pain
❑ physical exam
- extraperitoneal rupture: pelvic instability, suprapubic tenderness from mass of urine or extravasated blood
- intraperitoneal rupture: acute abdomen

❑ investigations: urinalysis, CT scan, urethrogram, +/– retrograde cystography
❑ management
- extraperitoneal: minor rupture —> Foley drainage, major rupture —> surgical repair
- intraperitoneal: drain abdomen and surgical repair

Urethral

❑ etiology
- usually blunt trauma in men
  - anterior (bulbous) urethra damage with straddle injuries
  - posterior (bulbo-membranous) urethra with pelvic fractures

❑ history/physical
- anterior: blood at meatus, perineal/scrotal hematoma, blood and urine extending from penile shaft and perineum to abdominal wall
- posterior: inability to void, blood at meatus, suprapubic tenderness, pelvic instability, superior displacement of prostate, pelvic hematoma on rectal exam

❑ investigation: retrograde urethrography
❑ management
- anterior: if Foley does not pass, requires suprapubic drain
- posterior: suprapubic drainage, avoid catheterization

Contraindications to Foley Catheterization

1. blood at the urethral meatus
2. ecchymosis of the scrotum
3. "high riding" prostate on DRE of male patients

❑ if any of the above, digital rectal exam (DRE), a retrograde cystouretharogram indicated to rule out urethral tear or ruptured bladder

HEAD TRAUMA (see Neurosurgery Chapter)

- ❑ 60% of trauma admissions have head injuries
- ❑ 60% of MVC-related deaths are due to head injury
- ❑ first physician who sees patient has greatest impact on the outcome
- ❑ alteration of consciousness is the hallmark of brain injury

Assessment of Brain Injury

- ❑ history
  - pre-hospital state, mechanism of injury
- ❑ vital signs
  - shock (not present in isolated brain injury, except in infants)
  - Cushing's response to increasing ICP (bradycardia with hypertension)
  - hyperthermia
- ❑ level of consciousness
  - Glasgow Coma Scale (GCS)
- ❑ pupils: pathology = anisocoria > 1 mm (in patient with altered LOC)
- ❑ neurological exam: lateralizing signs - motor/sensory

Severe Head Injury

- ❑ GCS = 8
- ❑ deteriorating GCS
- ❑ unequal pupils
- ❑ lateralizing signs

Investigations

- ❑ CT scan
- ❑ skull x-rays
  - little value in the early management of obvious blunt head injury
  - for diagnosis of calvarium fractures (not brain injury)
  - may help localize foreign body after penetrating head injury

Specific Injuries

- ❑ skull fractures (diagnosed by CT of head)
  - linear, non-depressed
    - most common
    - typically occur over temporal bone, in area of middle meningeal artery (commonest cause of epidural hematoma)
  - depressed
    - open (associated overlying scalp laceration)
    - closed
  - basal skull
    - typically occur through floor of anterior cranial fossa (longitudinal more common than transverse)
    - clinical diagnosis superior (Battle's sign, racoon eyes, CSF otorrhea/rhinorrhea, hemotympanum)
- ❑ facial fractures (see Plastic Surgery Chapter)
- ❑ diffuse brain injury
- ❑ diffuse axonal injury
- ❑ concussion (brief LOC then normal)
- ❑ focal injuries
  - contusions
  - intracranial hemorrhage (epidural, acute subdural, intracerebral)

Management

- ❑ general
  - ABC's
  - treat other injuries e.g. shock, hypoxia
- ❑ early neurosurgical consultation to direct acute and subsequent patient management
- ❑ medical
  - seizure treatment/prophylaxis
    - steroids are of NO proven value
    - diazepam, phenytoin, phenobarbital
  - treat suspected raised ICP
    - 100% O_2
    - intubate and hyperventilate to a pCO_2 of 30-35 mmHg
    - mannitol 1 g/kg infused as rapidly as possible (reserved for head-injured patients who are showing evidence of increased ICP)
    - raise head of stretcher 20 degrees if patient hemodynamically stable
    - consider paralyzing meds if agitated/high airway pressures
- ❑ surgical

Disposition

- ❑ neurosurgical ICU admission for severely head-injured patients
- ❑ in hemodynamically unstable patient with other injuries, prioritize most life threatening injury

SPINE AND SPINAL CORD TRAUMA

- ❑ spinal immobilization (cervical collar, spine board) must be maintained until spinal injury has been ruled out
- ❑ vertebral injuries may be present without spinal cord injury, therefore normal neurologic exam does not exclude spinal injury
- ❑ if a fracture is found, be suspicious, look for another fracture
- ❑ spine may be unstable despite normal C-spine x-ray
- ❑ collar everyone except those that meet ALL the following criteria
    - no pain
    - no tenderness
    - no neurological symptoms or findings
    - no significant distracting injuries
    - no head injury
    - no intoxication
- ❑ note: patients with penetrating trauma (especially gunshot and knife wounds) can also have spinal cord injury

X-Rays

- ❑ full spine series for trauma
    - AP, lateral, odontoid
- ❑ lateral C-Spine
    - must be obtained on all blunt trauma patients (except those meeting above criteria)
    - must visualize C7-T1 junction (Swimmer's view or CT scan often required)
- ❑ thoracolumbar
    - AP and lateral views
    - indicated in
        - patients with C-spine injury
        - unconscious patients
        - patients with symptoms or neurological findings
        - patients with deformities that are palpable when patient log-rolled

Management of Cord Injury

- ❑ immobilize the entire spine with the patient in the supine position (collar, sand bags, padded board, straps)
- ❑ if patient must be moved, use a "log roll" technique with assistance
- ❑ if cervical cord lesion, watch for respiratory insufficiency
    - low cervical transection (C5-T1) produces abdominal breathing (phrenic innervation of diaphragm still intact)
    - high cervical cord injury —> no breathing —> intubation
- ❑ hypotension (neurogenic shock)
    - treatment: warm blanket, Trendelenberg position (occasionally), volume infusion, consider vasopressors

APPROACH TO SUSPECTED C-SPINE INJURY

Clearing the C-Spine

- ❑ cervical collar must stay on at all times until C-spine is cleared (see Figure 3)

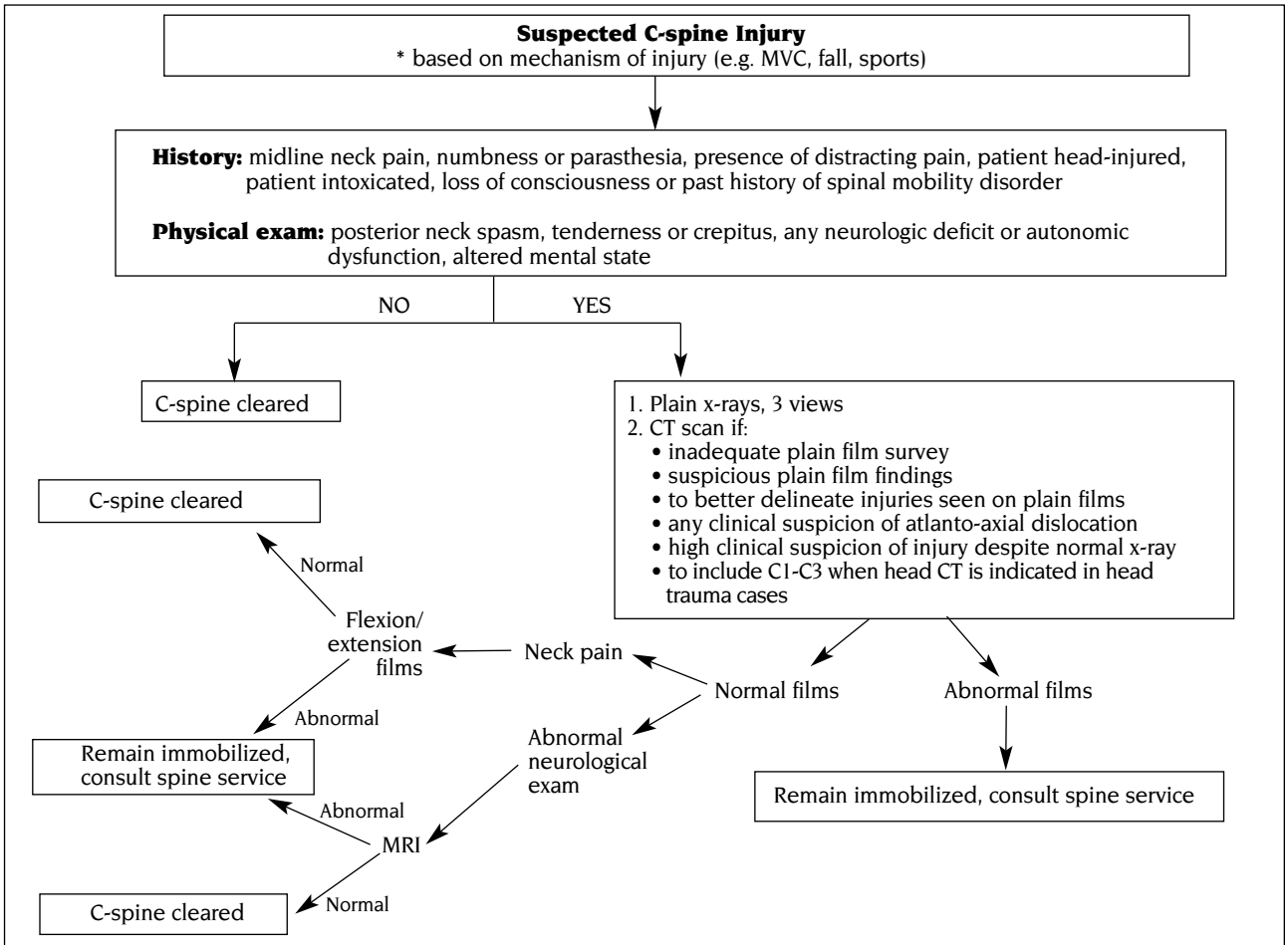


Figure 3. Approach to C-spine Clearing

C-Spine X-Rays
❑ 3-view C-spine series is the screening modality of choice
- AP
- lateral C1-T1 (± swimmer's view) - T2 not involved with neck movements
- odontoid (open mouth or oblique submental view)

Odontoid View (see Figure 5)
❑ examine the dens for fractures
- beware of artifact (horizontal or vertical) caused by the radiologic shadow of the teeth overlying the dens. Repeat view if unable to rule out fracture. If still unable to rule out fracture consider CT or plain film tomography.

❑ examine lateral aspects of C1
- odontoid should be centred between C1 lateral masses
- lateral masses of C1 and C2 should be perfectly aligned laterally. If not, suspect a fracture of C1
- lateral masses should be symmetrical (equal size)

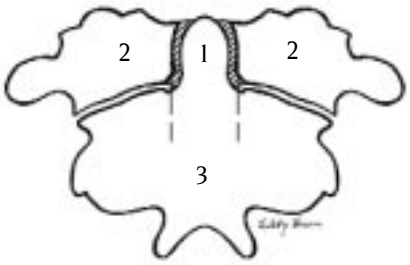
Anteroposterior View
❑ alignment of spinous processes in the midline
❑ spacing of spinous processes should be equal
❑ check vertebral bodies

Supine Oblique Views
❑ detects some injuries not visible on the usual three views
❑ better visualization of posterior element fractures (lamina, pedicle, facet joint)
❑ can be used to visualize the cervicothoracic junction

1. anterior vertebral line
2. posterior vertebral line (anterior margin of spinal canal)
3. posterior border of facets
4. laminar fusion line (posterior margin of spinal canal)
5. posterior spinous line (along tips of spinous processes)

Figure 4. Lines of Contour on a Lateral C-Spine X-Ray

Illustrated by Kim Auchinachie



1. Dens
2. C1 Lateral Mass
3. C2

To clear the x-ray ensure that:

A) the dens is centred between the lateral massess of C1
B) C1 and C2 are aligned laterally
C) the lateral masses of C1 are symmetrical in size

Figure 5. Odontoid of C-Spine X-Ray

Illustrated by Eddy Xuan

Lateral View: The ABCS

A - Alignment and Adequacy
- ❑ must see C1 to C7-T1 junction - if not - downward traction of shoulders, swimmer's view, bilateral supine obliques, or CT scan
- ❑ lines of contour (see Figure 4) (in children < 8 years of age: physiologic subluxation of C2 on C3, and C3 on C4, but the spinolaminal line is maintained)
- ❑ widening of interspinous space (fanning of spinous processes) suggests posterior ligamentous disruption
- ❑ widening of facet joints
- ❑ check atlanto-occipital joint:
  - line extended inferiorly from clivus should transect odontoid
- ❑ atlanto-axial articulation - widening of predental space (> 3 mm in adults, > 5 mm in children) indicates injury of C1 or C2

B - Bones
- ❑ height, width and shape of each vertebral body
- ❑ pedicles, facets, and laminae should appear as one - doubling suggests rotation

C - Cartilages
- ❑ intervetebral disc spaces - widening anteriorly or posteriorly suggests vertebral compression

S - Soft Tissues
- ❑ widening of retropharyngeal (> 7 mm at C1-4, may be wide in children less than 2 yrs. on expiration) or retrotracheal spaces (> 22 mm at C6-T1, > 14 mm in children < 15 years of age)
- ❑ prevertebral soft tissue swelling: only 49% sensitive for injury

Management Considerations
- ❑ immobilize C-spine with collar and sand bags (collar alone is not enough)
- ❑ injuries above C4 may need ventilation
- ❑ continually reassess high cord injuries - edema can travel up cord
- ❑ beware of neurogenic shock
- ❑ administer methylprednisolone within 8 hours of C-spine injury
- ❑ before O.R. ensure thoracic and lumbar x-rays are normal, since 20% of patients with C-spine fractures have other spinal fractures
- ❑ early referral to spine service

Sequelae of C-spine Fracture
- ❑ decreased descending sympathetic tone (neurogenic / spinal shock) responsible for most sequelae
- ❑ cardiac
  - no autoregulation, falling BP, decreasing HR, vasodilation
  - GIVE IV FLUIDS ± pressors
- ❑ respiratory
  - no cough reflex (risk of aspiration pneumonia)
  - no intercostal muscles +/– diaphragm
  - intubate and maintain vital capacity
- ❑ gastrointestinal
  - ileus, vasodilation, bile and pancreatic secretion continues (> 1L/day), risk of aspiration, GI stress ulcers
  - NG tube may be required for suctioning, feeding, etc.
- ❑ renal
  - hypoperfusion —> IV fluids
  - kidney still producing urine (bladder can rupture if patient not urinating)
  - Foley catheter may be required (measure urine output)

- ❑ skin
  - vasodilation, heat loss, no thermoregulation, atrophy (risk of skin ulcers)
- ❑ muscle
  - flaccidity, atrophy, decreased venous return
- ❑ penis
  - priapism

ORTHOPEDIC INJURIES (see Orthopedics Chapter)

❑ role of E.D.: identify injuries, restore anatomy (reduce and immobilize), administer antibiotics and tetanus prophylaxis

Physical Exam

❑ look: deformity, swelling, bleeding, bruising, spasm, colour
❑ feel: pulse, warmth, tenderness, crepitation, sensation, capillary refill
❑ move: range of motion (ROM) assessed actively (beware passive ROM testing)

Describing Orthopedic Injuries

❑ open vs. closed
❑ neurovascular status
❑ location of fracture
❑ type of fracture
❑ alignment: displacement, angulation

General Approach

- ❑ fractures
  - immobilize/traction/ice/analgesia
- ❑ open wounds
  - remove gross contamination, irrigate
  - cover with sterile dressing
  - definitive care within 6-8 hours
  - control bleeding with pressure (no clamping)
  - splint fracture
  - antibiotics - cefazolin (+/– gentamycin, metronidazole/penicillin in dirty injury)
  - tetanus prophylaxis (if none in last 10 yrs)
- ❑ joint injuries
  - orthopedic consultation
  - reduce dislocations after x-ray
  - immobilize

A. LIFE AND LIMB THREATENING INJURIES

- ❑ usually because of blood loss
  - pelvic fractures (up to 3.0L blood loss)
  - femur fractures (up to 1.5L blood loss per femur)
  - open fractures (double blood loss of a closed fracture)
- ❑ neurovascular compromise
- ❑ open fractures
- ❑ extensive soft tissue injuries
- ❑ amputations
- ❑ compartment syndrome

Life Threatening Injuries

❑ major pelvic fractures
❑ traumatic amputations
❑ massive long bone injuries
❑ vascular injuries proximal to knee/elbow

Limb Threatening Injuries

❑ fracture/dislocaton of ankle
❑ crush injuries
❑ compartment syndrome
❑ dislocations of knee/hip
❑ fractures with vascular/nerve injuries
❑ open fractures
❑ fractures above the knee or elbow

Assessment of Neurovascular Injury

❑ assess pulses before and after reduction
❑ diminished pulses should not be attributed to “spasm”
❑ angiography is definitive if diagnosis in doubt

Vascular Injuries Suggested by 6 P’s

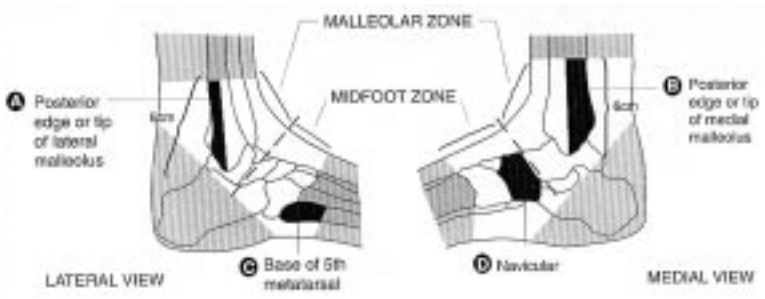
Pulse discrepancies
Pallor
Paresthesia/hypoesthesia
Paresis
Pain (especially when refractory to usual doses of analgesics)
Polar (cold)

Treatment of Vascular Compromise
- ❑ realign limb/apply traction
- ❑ recheck pulses (Dopplers)
- ❑ surgical consult
- ❑ consider measuring compartment pressures
- ❑ angiography

Compartment Syndrome (see Orthopedics Chapter)
- ❑ rise in interstitial pressure above that of capillary bed (30-40 mmHg)
- ❑ usually in leg or forearm
- ❑ often associated with crush injuries (extensive soft tissue damage)
- ❑ clinical diagnosis
- ❑ suspect when you find
  - excessive pain; worse with passive stretching of involved muscles
  - decreased sensation of nerves in that compartment
  - tense swelling
  - weakness, paralysis
  - pulse may still be present until very late
- ❑ hallmark: pain refractory to treatment with "usual" dose of analgesic agent
- ❑ management
  - compartment syndrome
    - remove constrictive dressings/casts
    - prompt fasciotomy

B. UPPER EXTREMITY INJURIES
- ❑ anterior shoulder dislocation
  - axillary nerve at risk
  - shown on lateral view: humeral head anterior to glenoid
  - reduce, immobilize, re-X ray, out-patient appointment with ortho
- ❑ Colle's fracture
  - from fall on the outstretched hand (FOOSH)
  - AP film: shortening, radial deviation, radial displacement
  - lateral film: dorsal displacement, volar angulation
  - reduce, immoblize with volar slab, out-patient with ortho (consider presentation)
  - if involvement of articular surface, emergent ortho referral
- ❑ scaphoid fracture
  - tender in snuff box, pain on scaphoid tubercle and pain on pressure on thumb (axial loading)
  - negative X -ray: thumb spica splint, re-X-ray in 1 week +/– bone scan
  - positive x-ray: thumb spica splint x 6-8 weeks
  - risk of avascular necrosis (AVN) of scaphoid if not immobilized

C. LOWER EXTREMITY INJURIES
- ❑ ankle and foot fractures
  - see Ottawa ankle rules (Figure 6)
- ❑ knee injuries
  - see Ottawa knee rules (Figure 7)
- ❑ avulsion of the base of 5th metatarsal
  - occurs with inversion injury
  - supportive tensor, below knee walking cast for 3 weeks
- ❑ calcaneal fracture
  - associated with fall from height
  - associated injuries may involve ankles, knees, hips, pelvis, lumbar spine



An ankle radiographic series is only required if there is any pain in malleolar zone and any of these findings:
1. bone tenderness at A
or
2. bone tenderness at B
or
3. inability to bear weight both immediately and in emergency department

A foot radiographic series is only required if there is any pain in midfoot zone and any of these findings:
1. bone tenderness at C
or
2. bone tenderness at D
or
3. inability to bear weight both immediately and in emergency department

Figure 6. Ottawa Ankle Rules

Reprinted with permission from Stiell et. al. JAMA 271:827-832,1994.

A knee x-ray examination is only required for acute injury patients with one or more of:

- age 55 years or older
- tenderness at head of fibula
- isolated tenderness of patella*
- inability to flex to 90°
- inability to bear weight both immediately and in the emergency department (four steps)**

*no bone tenderness of knee other than patella
**unable to transfer weight twice onto each lower limb regardless of limping

Figure 7. Ottawa Knee Rules

Reprinted with permission from Stiell et. al. JAMA 271:8, 611-615, 1995.

SOFT TISSUE INJURIES

Bruises

❑ tender swelling (hematoma) following blunt trauma
❑ is patient on anticoagulants? coagulopathy?
❑ acute treatment: "**RICE**"
Rest
Ice
Compression
Elevation

Tetanus Prophylaxis

❑ the following table provides guidance for tetanus prophylaxis in the E.D.

Table 8. Tetanus Prophylaxis

Immunization History	Non Tetanus Prone Wounds		Tetanus Prone Wounds[1]	
	Td[2]	TIG[3]	Td	TIG
Uncertain or < 3 doses	Yes	No	Yes	Yes
3 or more, none for > 10 years	Yes	No	Yes	No
3 or more, > 5 but < 10 years ago	No	No	Yes	No
3 or more, < 4 years ago	No	No	No	No

[1] wounds > 6 hours old, > 1 cm deep, puncture wounds, avulsions, wounds resulting from missiles, crush wounds, burns, frostbite, wounds contaminated with dirt, feces, soil or saliva
[2] tetanus and diptheria toxoids (Td), absorbed [3] tetanus immune globulin (TIG) - 250 units

Source: MMWr 2001; 50(20); 418, 427. MMWr 1991; 40(RR12); 1-52.

Abrasions

❑ partial to full thickness break in skin
❑ management
- clean thoroughly (under local anesthetic if necessary) with brush to prevent foreign body impregnation (tattooing)
- antiseptic ointment (Polysporin) or Vaseline for 7 days for facial and complex abrasions
- tetanus prophylaxis are per above table

Lacerations

❑ always consider every structure deep to a laceration severed until proven otherwise
❑ in hand injury patient, include following in history: handedness, occupation, mechanism of injury, previous history of injury
❑ physical exam
- think about underlying anatomy
- examine tendon function and neurovascular status distally
- x-ray wounds if a foreign body is suspected (e.g. shattered glass) and not found when exploring wound
- clean and explore under local anesthetic

❑ management
- irrigate copiously with normal saline
- evacuate hematomas, debride non-viable tissue, and remove foreign bodies
- secure hemostasis
- suture (Steristrip, glue, or staple for selected wounds) unless delayed presentation, a puncture wound, or animal bite
- in general, facial sutures are removed in 5 days, those over joints in 10 days, and everywhere else in 7 days; removal is delayed in patients on steroid therapy
- in children, topical anesthetics such as LET (Lidocaine, Epinephrine and Tetracain) and in selected cases a short-acting benzodiazepine (midazolam) for sedation and amnesia are useful
- DO NOT use local anesthetic with epinephrine on fingers, toes, penis, ears, nose
- maximum dose of lidocaine
  - 7 mg/kg with epinephrine
  - 5 mg/kg without epinephrine

Mammalian Bites

❑ important points on history
- time and circumstances of bite
- symptoms
- comorbid conditions
- allergies
- tetanus immunization status
- rabies risks

❑ on examination
- assess type of wound: abrasion, laceration, puncture, crush injury
- assess for direct tissue damage - skin, bone, tendon, neurovascular

❑ x-rays
- if bony injury or infection suspected check for gas in tissue
- ALWAYS get skull films in children with scalp bite wounds, +/– CT to rule out cranial perforation

❑ treatment
- wound cleansing and copious irrigation as soon as possible
- irrigate/debride puncture wounds if feasible, but not if sealed or very small openings - avoid hydrodissection along tissue planes
- debridement is important in crush injuries to reduce infection and optimize cosmetic and functional repair
- culture wound if signs of infection (erythema, necrosis or pus) - anaerobic cultures if foul smelling, necrotizing, or abscess
- notify lab that sample is from bite wound

❑ most common complication of mammalian bites is infection (2 to 50%)
- types of infections resulting from bites: cellulitis, lymphangitis, abscesses, tenosynovitis, osteomyelitis, septic arthritis, sepsis, endocarditis, meningitis
- early wound irrigation and debridement are the most important factors in decreasing infection

❑ rabies (see Infectious Diseases Chapter)
- virus is transmitted via animal bites
- reservoirs: warm-blooded animals except rodents, lagomorphs (e.g. rabbits)
- post-exposure vaccine is effective; treatment depends on local prevalence (contact public health)

❑ to suture or not to suture?
- the risk of wound infection is related to vascularity of tissue
- vascular structures (i.e. face and scalp) are less likely to get infected, therefore suture
- avascular structures (i.e. pretibial regions, hands and feet) by secondary intention

❑ high risk factors for infection
- puncture wounds
- crush injuries
- wounds greater than 12 hours old
- hand or foot wounds, wounds near joints
- immunocompromised patient
- patient age greater than 50 years
- prosthetic joints or valves

Prophylactic Antibiotics

❑ widely recommended for all bite wounds to the hand
❑ should be strongly considered for all other high-risk bite wounds
❑ 3-5 days is usually recommended for prophylactic therapy
❑ dog and cat bites (pathogens: *Pasteurella multocide*, S. *aureus*, S. *viridans*)
❑ 80% of cat bites, 5% of dog bites become infected (NEJM 1999, AnEm1994)
- 1st line: amoxicillin + clavulinic acid
- 2nd line: tetracycline or doxycycline
- 3rd line: erythromycin, clarithromycin, azithromycin

❑ human bites (pathogens: E*ikenella carrodens*, S. *aureus*, S. *viridans*, oral anaerobes)
- 1st line: amoxicillin + clavulinic acid
- 2nd line: erythromycin, clarithromycin, azithromycin
- 3rd line: clindamycin

❑ althoug antibiotic prophylaxis is frequently given following any mamallian bite Cochrane Review (2000) only shows decreased rate of infection following human bites; not cat or do

ENVIRONMENTAL INJURIES

Burns (see Plastic Surgery Chapter)

❑ immediate management
- remove noxious agent
- resuscitation
  - 2nd and 3rd degree burns: Parkland Formula: Ringer's lactate 4cc/kg/%BSA burned (not including 1st degree); give 1/2 in first 8 hours, 1/2 in second 16 hours
  - at 8 hours: FFP or 5% albumin: if > 25% BSA give 3-4 U/day for 48 hours
  - second 8 hours: 2/3-1/3 at 2cc/kg/%BSA
  - urine output should be 40-50 cc/hr or 0.5 cc/kg/hr
  - avoid diuretics
- continuous morphine infusion at 2 mg/hr with breakthrough bolus
- burn wound care - prevent infection, cover gently with sterile dressings
- escharotomy or fasciotomy for circumferential burns (chest, extremities)
- systemic antibiotics infrequently indicated
- topical - silver sulfadiazene; face - polysporin; ears - sulfomyalon

❑ guidelines for hospitalization
- 10-50 years old with 2nd degree burns to > 15% TBSA or 3rd degree to greater than 5% TBSA
- less than 10 years old or > 50 years old with 2nd degree to > 10% TBSA or 3rd degree to > 3% TBSA
- 2nd or 3rd degree on face, hands, feet, perineum or across major joints
- electrical or chemical burns
- burns with inhalation injury
- burn victims with underlying medical problems or immunosuppressed patients (e.g. DM, cancer, AIDS, alcoholism)

Inhalation Injury

❑ carbon monoxide (CO) poisoning - see Toxicology section
- closed environment
- cherry red skin/blood (usually a post-mortem finding, generally unreliable)
- headache, nausea, confusion
- pO_2 normal but O_2 sat low
- true O_2 sat must be measured (not value from pulse oximeter nor calculated value based on a blood gas)
- measure carboxyhemoglobin levels
- treatment: 100% O_2 +/– hyperbaric O_2

❑ thermal airway injury
- etiology: injury to endothelial cells and bronchial cilia due to fire in enclosed space
- symptoms and signs: facial burns, intraoral burns, singed nasal hairs, soot in mouth/nose, hoarseness, carbonaceous sputum, wheezing
- investigations: CXR +/– bronchoscopy
- treatment: humidified oxygen, early intubation, pulmonary toilet, bronchodilators

Hypothermia

❑ predisposing factors: old age, lack of housing, drug overdose, EtOH ingestion, trauma (incapacitating), cold water immersion, outdoor sports

❑ diagnosis: mental confusion, impaired gait, lethargy, combativeness, shivering

❑ treatment on scene
- remove wet clothing; blankets + hot water bottles; heated O_2; warmed IV fluids
- no EtOH due to peripheral vasodilating effect
- vitals (take for > 1 minute)
- cardiac monitoring; no chest compressions until certain patient pulseless > 1 minute, since can precipitate ventricular fibrillation
- NS IV since patient is hypovolemic and dehydrated secondary to cold water diuresis and fluid shifts
- note: if body temperature < 32.2ºC, you may see decreased heart rate, respiratory rate, and muscle tone, dilated + fixed pupils (i.e. patient appears "dead")
- due to decreased O_2 demands, patient may recover without sequelae

❑ treatment in hospital
- patient hypovolemic and acidotic
- rewarm slowly with warm top + bottom blankets (risk of "afterdrop" if cold acidotic blood of periphery recirculated into core)
- at body temperature < 30ºC risk of ventricular fibrillation therefore warm via peritoneal/hemodialysis or cardiopulmonary bypass

❑ PATIENT IS NOT DEAD UNTIL THEY ARE WARM AND DEAD!

Frostbite

❑ ice crystals form between cells

❑ classified according to depth - similar to burns (1st to 3rd degree)

❑ 1st degree
- symptoms: initial paresthesia, pruritus
- signs: erythema, edema, hyperemia, NO blisters

❑ 2nd degree
- symptoms: numbness
- signs: blistering, erythema, edema

❑ 3rd degree
- symptoms: pain, burning, throbbing (on thawing)
- signs: hemorrhagic blisters, skin necrosis, edema,decreased range of motion

❑ management
- remove wet and constrictive clothing
- immerse in 40-42ºC water for 10-30 minutes
- leave injured region open to air
- leave blisters intact
- debride skin gently with daily whirlpool immersion (topic ointments not required)
- surgical intervention may be required to release restrictive escars
- never allow a thawed area to re-freeze

PEDIATRIC TRAUMA

❑ priorities remain the same

Airway

❑ "sniffing position"
❑ short trachea (5 cm in infants, 7.5 cm at 18 months)
❑ orotracheal tube diameter = age/4 + 4
❑ uncuffed ETT under age 8
❑ surgical cricothyroidotomy NOT indicated
❑ needle cricothyroidectomy with jet ventilation if unable to intubate

Breathing

❑ stethoscope not as useful for diagnosing problems - noting tachypnea is important

Circulation

❑ normal blood volume = 80 ml/kg
❑ fluid resuscitation
- bolus crystalloid 20 ml/kg
- repeat x 1 if necessary
- blood replacement if no response to 2nd bolus of crystalloid

❑ venous access
- intraosseous infusion if unable to establish IV access in < 30 seconds
- venous cutdown (medial cephalic, external jugular, great saphenous)

Thermoregulation

❑ children prone to hypothermia
❑ blankets/external warming/cover scalp

Table 9. Normal Vitals in Pediatric Patients

	HR	sBP	RR
Infant	< 160	80	40
Preschool	< 140	90	30
Adolescent	< 120	100	20

TRAUMA IN PREGNANCY

❑ treatment priorities the same
❑ the best treatment for the fetus is to treat the mother

Hemodynamic Considerations

❑ near term, inferior vena caval compression in the supine position can decrease cardiac output by 30-40%
- use left lateral decubitus (LLD) positioning to alleviate compression and increase blood return

❑ BP drops 5-15 mmHg systolic in 2nd trimester, increases to normal by term
❑ HR increases 15-20 beats by 3rd trimester

Blood Considerations

❑ physiologic macrocytic anemia of pregnancy (Hb 100-120)
❑ WBC increases to high of 20,000

Shock

❑ pregnant patients may lose 35% of blood volume without usual signs of shock (tachycardia, hypotension)
❑ however, the fetus may be in "shock" due to contraction of the uteroplacental circulation

Management Differences

❑ place bolster under right hip to stop inferior vena cava compression
❑ fetal monitoring (Doppler)
❑ early obstetrical involvement
❑ x-rays as needed (C-spine, CXR, pelvis)
❑ consider need for RhoGAM if mother Rh^-

APPROACH TO COMMON ER PRESENTATION

ABDOMINAL PAIN

Origins of Abdominal Pain

1. GI: appendicitis, diverticulitits, bowel obstruction, perforation, hepatitis, cholecystitis, organ lasceration
2. Urinary: cystitis, pyelonephritis, ureteral calculi
3. Genital
   Female: ectopic pregnancy, pelvic inflammatory disease (PID), endometriosis, salpingitis / tubo-ovarian abscess, ovarian torsion / cyst
   Male: referred from testicles
4. Vascular: AAA, bowel or splenic infact (be aware of sickle cell status)
5. Other: diabetic ketoacidosis (DKA), Herpes Zoster Virus (HZV), intra-abdominal abscess, MI, pneumonia, lead poisoning, glaucoma

Life-Threatening Causes

- ❑ ectopic pregnancy
- ❑ ischemic bowel, aortic rupture/dissection (pain >> findings)
- ❑ hepatic/splenic injury
- ❑ perforated viscus
- ❑ myocardial infarction (MI)

History

- ❑ pain: onset, location, character, severity, aggravating and alleviating factors, associated symptoms (nausea, vomiting, diarrhea, vaginal bleeding)
- ❑ past medial/surgical/gynecological history, drugs, travel history

Physical Exam

- ❑ general appearance (diaphoretic, jaundiced, lying very still vs. writhing in pain), vital signs (including posturals)
- ❑ inspection: distention, scars, ecchymoses
- ❑ auscultation: bowel sounds, bruits
- ❑ palpation: peritoneal signs (shake, cough, rebound tenderness and guarding), organomegaly, hernias, pelvic and testicular exams

Tests

- ❑ DO NOT DELAY CONSULTATION if patient unstable
- ❑ CBC, U/A, amylase, β-HCG, LFTs, renal function, lytes
- ❑ AXR: calcifications, free air, gas pattern
- ❑ U/S: biliary tract, ectopic pregnancy, AAA (abdominal aortic aneurysm)
- ❑ CT: trauma, AAA, pancreatitis but UNSTABLE PATIENTS SHOULD NOT BE SENT FOR IMAGING

Management

- ❑ NPO, IV, NG tube, analgesics (communicate with consultants in advance)
- ❑ growing evidence that SMALL amounts of narcotic analgesics improve diagnostic accuracy of physical exam of surgical abdomen
- ❑ consults: general surgery, vascular, gyne as necessary

ALCOHOLIC EMERGENCIES (see Psychiatry Chapter)

- ❑ EtOH LEVELS CORRELATE POORLY WITH INTOXICATION

Acute Intoxication

- ❑ may invalidate informed consent
- ❑ slurred speech, CNS depression, disinhibition, incoordination
- ❑ nystagmus, diplopia, dysarthria, ataxia —> coma
- ❑ frank hypotension (peripheral vasodilation)
- ❑ obtundation - must rule out
  - head trauma + intracranial hemorrhage
  - associated depressant/street drugs
    - synergistic —> respiratory/cardiac depression
  - hypoglycemia: must screen with bedside glucometer
  - hepatic encephalopathy
    - precipitating factors: GI bleed, infection, sedation, electrolyte abnormalities, protein meal
  - **W**ernicke's encephalopathy ("**WACO**")
    Ataxia
    Coma
    Ocular findings: nystagmus, CN VI paresis ocular findings (may be absent at time of presentation)
    - give thiamine 100mg IV
  - post-ictal state, basilar stroke

Seizures

- ❑ associated with ingestion and withdrawal
- ❑ withdrawal seizures
  - occur 8-48 hr. after last drink
  - typically brief generalized tonic-clonic seizures
    - if >48 hr., think of delerium tremens (DT) (see Table 10)

Treatment
❑ diazepam 10mg PO q1h until calm
❑ thiamine 100mg IM then 50-100mg/day
❑ admit patients with DT
❑ withdrawal signs – see Table 9

Table 10. Alcohol Withdrawal Signs

Time since last drink	Syndrome	Description
6-8 hr.	Mild withdrawal	- generalized tremor, anxiety, agitation but no delerium - autonomic hyperactivity, insomnia, nausea, vomiting
1-2 days	Alcoholic hallucinations	- visual and auditory hallucinations - vitals often normal
8 hr. - 2 days	Withdrawal seizures	- see above
3-5 days	Delirium Tremens	- 5% of untreated withdrawal patients - severe confusional state - agitation, insomnia, hallucinations/delusions, tremor - tachycardia, hyperpyrexia, diaphoresis

Cardiovascular Diseases (see Cardiology Chapter)
❑ hypertension (HTN)
❑ cardiomyopathy: shortness of breath (SOB), edema
❑ arrhythmias ("holiday heart")
- atrial fibrillation, atrial flutter, premature ventricular contraction (PVC), premature atrial contraction (PAC), supraventricular tachycardia (SVT), ventricular tachycardia (VT)

Metabolic Abnormalities
❑ alcoholic ketoacidosis
- history of chronic alcohol intake with abrupt decrease in intake
- malnourished, abdominal pain with nausea and vomiting (N/V)
- anion gap (AG) metabolic acidosis, urine ketones, low glucose and normal osmolality EtOH level zero
- treatment: dextrose, thiamine and NS; resolves in 12-24 hr

❑ abnormal alcohols (see also Toxicology section)
- ethylene glycol —> CNS, CVS, renal findings
- methanol
  - early: lethargy, confusion
  - late: headache, visual changes, N/V, abdominal pain, tachypnea
- both produce severe metabolic acidosis with AG and osmolal gap
- EtOH co-ingestion is protective
- treatment
  - IV 10% EtOH bolus and drip to achieve blood level of 20 mmol/L
  - alcohol loading may be done PO
  - fomepizole (4-mp) if available
  - urgent hemodialysis required

❑ other abnormalities
- hypomagnesemia
- hypophosphatemia
- hypocalcemia
- hypoglycemia

Gastrointestional (GI) Abnormalities
❑ gastritis
- common cause of abdominal pain and GI bleed in chronic alcohol users

❑ pancreatitis
- serum amylase very unreliable in patients with chronic pancreatitis
- hemorrhagic form (15%) associated with increased mortality

❑ hepatitis
- AST/ALT ratio > 2 suggests alcohol as the cause as well as elevated GGT with acute ingestion

❑ peritonitis
- occasionally accompanies cirrhosis
- leukocytosis, fever, generalized abdominal pain
- paracentesis for diagnosis

❑ GI bleeds
- most commonly gastritis or ulcers, even if patient known to have varices
- must consider Mallory-Weiss tear
- often complicated by underlying hematologic abnormalities
- cirrhosis

Miscellaneous Problems
- ❑ rhabdomyolysis
  - presents as acute weakness associated with muscle tenderness
  - usually occurs after prolonged immobilization
  - increased creatinine kinase (CK), hyperkalemia
  - myoglobinuria - may lead to acute renal failure
  - treatment: IV fluids, forced diuresis (mannitol)
- ❑ increased infections - due to host defences, immunity, poor living conditions
  - atypical pneumonias (Gram negatives, anaerobes, TB)
  - meningitis
  - peritonitis with ascites - E.*coli*, K*lebsiella*, Strep
  - bacteremia - after urinary tract infection (UTI), soft tissue infections
  - usually require admission and IV antibiotics

ANAPHYLAXIS AND ALLERGIC REACTIONS

Etiology
- ❑ requires: 1. exposure 2. latent period 3. re-exposure
- ❑ exaggerated immune response to antigens
- ❑ mediated by IgE, released histamine, bradykinins, leukotrienes
- ❑ most common: penicillin, stings, nuts, shellfish
- ❑ anaphylactoid: non-IgE mediated, direct trigger, may occur with first exposure (e.g. radiocontrast dyes), treatment similar to anaphylaxis

Symptoms and Signs
- ❑ cardiovascular collapse (shock), arrhythmia, MI
- ❑ marked anxiety and apprehension
- ❑ skin - generalized urticaria, edema, erythema, pruritus
- ❑ respiratory compromise, choking sensation, cough, bronchospasm or laryngeal edema, wheezes and stridor
- ❑ allergies and prior episodes important
- ❑ patients on β-blockers and H2 blockers may develop more severe reactions and be refractory to standard therapies

Treatment
- ❑ stop the cause, ABC's
- ❑ on scene - 'epi-pen' (injectable epinephrine) if available
- ❑ MODERATE signs and symptoms (minimal airway edema, mild bronchospasm, cutaneous reactions)
  - adult: 0.3 -0.5 ml of 1:1000 solution IM or SC epinephrine
  - child: 0.01 ml/kg/dose up to 0.4 mL/dose 1:10 000 epinephrine
- ❑ SEVERE signs and symptoms (laryngeal edema, severe bronchospasm and shock)
  - epinephrine via IV or ETT starting at 1 ml of 1:10 000
  - cardiac monitoring, ECG
- ❑ diphenhydramine 50 mg IM or IV(Benadryl) q 4-6h
- ❑ methylprednisolone 50-100 mg IV dose depending on severity
- ❑ salbutamol via nebulizer if bronchospasm present
  - glucagon (for those on β-blockers) 5-15 µg q 1min IV

Angioedema / Urticaria (see Dermatology Chapter)
- ❑ cutaneous IgE-mediated reaction
- ❑ more severe form: erythema multiforme (EM)
- ❑ Treatment: epinephrine, antihistamines, steroids

ANALGESIA

Table 11. Summary of Analgesics

Drug	Dose	Indications	Side Effects
Aspirin	300-900 mg PO q 4-6h	headache, MSK, dysmenorrhea	interaction with warfarin, exacerbate asthma
Acetaminophen ± codeine	0.5-1g PO q 4-6h	similar to aspirin	liver and renal damage codeine: constipation, respiratory depression
NSAIDS			
• Ibuprofen • Diclofenac	• 0.4-0.6 g PO q 3-4h • 75 mg IM injection	MSK pain	gastric irritation, GI bleed, interaction with diuretics, warfarin and lithium
OPIOIDS			
• Morphine • Demerol	• 2-10 mg IV titrate up • 12.5-25 mg IV, titrate up	trauma, pulmonary edema in left ventricular failure (LVF), severe pain	nausea and vomiting (give with Gravol)

ASTHMA (see Respirology Chapter)

Etiology
- ❑ exposure to a "trigger"
- ❑ bronchospasm
- ❑ airway inflammation leading to airway edema

Differential Diagnosis
- ❑ foreign body aspiration
- ❑ bronchiolitis
- ❑ pneumonia
- ❑ cystic fibrosis (CF)
- ❑ congestive heart failure (CHF)

History
- ❑ onset, duration, severity
- ❑ management prior to coming to hospital
- ❑ past medical history
- ❑ asthma, cystic fibrosis (CF)
- ❑ previous visits to the E.D. and admissions for the same problem
- ❑ associated symptoms
- ❑ fever, productive cough, orthopnea, chest pain

Physical Exam
- ❑ general appearance
  - pale, cyanotic, diaphoretic, altered LOC, distressed, unable to speak in full sentences
- ❑ inspection
  - use of accessory muscles, tachypnea
- ❑ auscultation of the chest
  - Silent – Emergency!
  - prolongued expiratory phase +/– wheezes
  - crackles/ rhonchi/ rubs
  - heart murmurs, S3 / S4
- ❑ Investigations
  - O_2 Sat
  - peak clow meter
  - routine blood work +/- ABG's
  - CXR

Table 12. Asthma Assessment and Treatment

Classification	Assessment	Treatment
Near Death	- exhausted, confused, diaphoretic, cyanotic - silent chest, ineffective respiratory effort - decreased HR - O_2 sat <90%	- 100% O_2, cardiac monitor, IV access - intubate - β-agonist: MDI 4-8 puffs q 20 min x 3 OR nebulizer 5 mg q 20 min x 3 - anti-cholinergics: MDI 4-8 puffs q 20 min x 3 OR nebulizer 0.25 mg q 20 min x 3 - IV steroids: methylprednisolone 125 mg IV hydrocortisone 500 mg IV - CXR, ABG
Severe Asthma	- agitated, diaphoretic, laboured respirations - difficulty speaking - no relief from β-agonist - O_2 sat <90% - FEV_1 <40%	- anticipate need for intubation - similar to above management
Moderate Asthma	- SOB at rest, cough, congestion, chest tightness - nocturnal symptoms - inadequate relief from β-agonists - FEV_1 40-60%	- O_2 - β-agonist - systemic steroids: prednisone 40-60 mg PO - anticholinergics
Mild Asthma	- exertional SOB/cough with some nocturnal symptoms - good response to β-agonist - FEV_1 > 60%	- β-agonist - monitor FEV_1

- ❑ admit if FEV1 < 25% (pre-treatment) or FEV_1 < 40% (post treatment)
- ❑ discharge plans
  - β-agonist: x 2-3 days
  - steroids x 1-2 weeks
  - patient education on triggers, medication use, etc.

COPD (see Respirology Chapter)

Etiology
- ❑ emphysema: destruction of alveoli can lead to tachypnea and dyspnea
- ❑ chronic bronchitis: chronic cough and sputum production
- ❑ both usually co-exist

Exacerbations
- ❑ worsening dyspnea or tachypnea
- ❑ acute change in frequency, quantity and colour of sputum production
- ❑ history
  - premorbid health status
  - trigger: pneumonia, urinary tract infection (URTI), pulmonary embolism (PE), CHF, drugs
- ❑ tests
  - CXR, ECG, ABG, FEV_1
- ❑ treatment
  - keep O_2 sat > 90% (BEWARE OF CO_2 RETAINERS)
    - bronchodilators + anticholinergics
    - steroids: IV methylprenisolone 125 mg or prednisone PO 40 mg (tapered over 3 weeks)
    - antibiotics: Septra, cephalosporins, quinolones
    - ventilation: chance of ventilation dependency
  - admit if co-morbid illness
  - discharge on antibiotics, bronchodilators and taper steroids

CHEST PAIN

Must Rule Out Life-Threatening Causes
- ❑ unstable angina/acute MI
- ❑ thoracic aortic dissection
- ❑ pulmonary embolism (PE)
- ❑ spontaneous pneumothorax/tension pneumothorax
- ❑ esophageal rupture
- ❑ pericarditis/cardiac tamponade

Additional Differential Diagnosis
- ❑ stable angina
- ❑ GI disorders: peptic ulcer disease (PUD), pancreatitis, cholecystitis, esophagitis, etc.
- ❑ pneumonia
- ❑ MSK
- ❑ spontaneous pneumothorax (young, thin, tall)
- ❑ psychogenic (diagnosis of exclusion)

Initial Resuscitation and Management
- ❑ O_2, IV, cardiac monitoring, portable CXR

History
- ❑ compare with previous episodes
- ❑ pain characteristics
- ❑ risk factors
  - cardiac: hypertension, family history, smoking, cholesterol, DM
  - pulmonary embolism: immobility, cancer, use of estrogen, family history
  - aortic dissection: hypertension
- ❑ classic presentations (but presentations seldom classic)
  - aortic dissection - sudden severe tearing pain, often radiating to back
  - pulmonary embolism (PE) - pleuritic chest pain (75%), dyspnea, anxiety, tachycardia
  - pericarditis - anterior precordial pain, pleuritic, relieved by sitting up and leaning forward
  - acute coronary artery disease (CAD) - retrosternal squeezing/pressure pain, radiation to arm/neck, dyspnea, nausea/vomiting, syncope
  - esophageal - frequent heartburn, acid reflux, dysphagia, relief with antacids
- ❑ more likely to be atypical in females, and > 80 years

Physical Exam
- ❑ vitals
  - tachypnea (may be the only sign of PE)
  - BP in BOTH arms: = 20 mm Hg difference suggests thoracic aortic dissection
- ❑ palpate chest wall for tender points but not a good discriminator since 25% of patients with acute MI have chest wall tenderness
  - accept only if fully reproduces pain symptoms and more serious causes excluded
  - may result from pleural inflammation
- ❑ cardiac exam
  - jugular venous pressure (JVP)
  - heart sounds: friction rub, muffling
  - new murmurs
  - mitral regurgitation murmur in acute MI (papillary muscle dysfunction)
  - aortic insufficiency murmur in aortic dissection
- ❑ respiratory exam
  - percuss and auscultate all the lung fields
- ❑ peripheral vascular exam - abdomen, extremities

Investigations
- ❑ ECG
  - cardiac + non-cardiac causes
  - PE, acute MI may have NORMAL ECG in up to 50% of cases
  - always compare with previous
- ❑ CXR
  - pulmonary embolism (PE)
    - 50% completely NORMAL
    - atelectasis, elevated hemidiaphragm, pleural effusion
    - Westermark's sign, Hampton's hump
  - aortic dissection
    - mediastinal widening, bulging aortic arch, separation of intimal calcification from edge of aortic shadow, depressed left main bronchus
    - change from previous CXR is the most accurate finding
    - CXR is normal in 20% of thoracic dissection
  - pneumothorax
    - need inspiration and expiration views
- ❑ ABGs - NORMAL in 20% of patients with PE
  - serial cardiac enzymes (see Cardiology Chapter)
  - normal CK does NOT rule out MI
  - troponin I more sensitive (but positive later than CK-MB; can have false positives in renal failure)
- ❑ V/Q scan and helical CT if PE suspected

HEADACHE (see Neurology Chapter)
- ❑ key principles
  - brain is anesthetic (most headaches arise from surrounding structures such as blood vessels, periosteum, muscle)
  - every headache is serious until proven otherwise
- ❑ THE COMMON
  - migraine (no aura)/classic migraine (involves aura)
    - gradual onset, unilateral, throbbing
    - nausea/vomiting, photophonophobia
    - **treatment**: analgesics, neuroleptics, vasoactive meds
  - tension /muscular headache
    - never during sleep, gradual over 24 hours
    - posterior/occipital
    - increased with stressors
    - **treatment**: modify stressor, local measures, NSAIDS
- ❑ THE DEADLY
  - subarachnoid hemorrhage (SAH)
    - sudden onset, increased with exertion
    - "worst" headache, nausea and vomiting
    - **diagnosis**: CT, LP (5-15% of patients with negative CT have SAH)
    - urgent neurosurgery consult
  - increased ICP
    - worst in morning, supine, or bending down
    - physical exam: neurological deficits, cranial nerve palsies
    - **diagnosis**: CT scan
    - consult neurosurgery
  - meningitis
  - temporal arteritis (not immediately deadly but causes great morbidity)

HYPERTENSIVE EMERGENCIES
Varon J, Marik P. The Diagnosis and Management of Hypertensive Crises. Chest. 2000:118(1):214-227.

Hypertensive Emergencies (aka Hypertensive Crisis)
- ❑ definition: acute elevation of systolic and diastolic BP associated with end-organ damage of the CNS, the heart, or the kidneys
- ❑ treatment: lower blood pressure to "normal" within 30-60 minutes

Hypertensive Urgencies
- ❑ definition: severely elevated blood pressure (usually dBP > 115) with no evidence of end-organ damage
- ❑ most commonly due to non-compliance with medications
- ❑ treatment: gradually reduce pressure over 24-48 hours to a level appropriate for the patient

Pathophysiology
- ❑ the majority of hypertensive emergencies occur in patients with preexisting chronic hypertension. However, fewer than one percent of hypertensive patients will develop a hypertensive emergency
- ❑ related to acute increase in systemic vascular resistance likely induced by humoral vasoconstrictors
- ❑ endothelial injury and fibrinoid necrosis of the arterioles occurs following severe elevation of blood pressure
- ❑ vascular damage induces platelet deposition and fibrin formation resulting in tissue ischemia and further release of vasoactive substances

Evaluation of Patient With Severe Hypertension

- ❑ goal is to differentiate hypertensive emergencies from hypertensive urgencies
- ❑ history
  - prior hypertensive crises
  - antihypertensive medications prescribed and BP control
  - monoamine oxidase inhibitors (MAOIs)
  - street drugs (cocaine, amphetamines, phencyclidine, etc.)
- ❑ physical examination
  - blood pressure measurement in all limbs
  - fundoscopic exam (hemorrhages, papilledema, etc.)
- ❑ lab
  - CBC, electrolytes, BUN, creatinine, urinalysis
  - peripheral blood smear – to detect microangiopathic hemolytic anemia
  - CXR – if shortness of breath (SOB)
  - ECG – if chest pain
  - head CT – if neurological findings

Hypertensive Emergencies

- ❑ Hypertensive Encephalopathy
  - pathophysiology: cerebral hyperperfusion due to blood pressure in excess of the capacity for cerebral autoregulation
  - signs and symptoms: headache, nausea, vomiting, mental status changes (lethargy to coma), fundoscopic changes (hemorrhage, exudates, cotton wool spots, papilledema, sausage linking)
  - treatment: sodium nitroprusside or labetalol. Avoid clonidine and pure B blockers
  - NB: with CNS manifestations of severe hypertension is often difficult to differentiate causal relationships (ie: hypertension could be secondary to primary cerebral event [Cushing effect])

- ❑ Pregnancy Induced Hypertension (PIH) (see Obstetrics Chapter)
- ❑ Cardiovascular Emergencies
  - left ventricular failure (LVF)
    - pathophysiology: decreased LV function due to increased afterload, increased oxygen demand and decreased coronary blood flow may cause angina, MI, or pulmonary edema
    - signs and symptoms: chest pain, SOB
    - treatment: goal is to decrease preload and afterload (iv nitroprusside and nitroglycerin)
      - diuretics for volume overload
      - avoid diazoxide, hydralazine, minoxidil as these drugs increase oxygen demand
  - thoracic aortic dissection (see Cardiac and Vascular Surgery Chapter)

- ❑ Hypertensive Renal Emergencies
  - renal failure can be either the cause or effect of a hypertensive emergency
  - hypertension associated with deteriorating renal function is considered an emergency
  - hypertension in the setting of chronic renal failure is due to sodium and water retention by the diseased kidney and increased activation of the renin-angiotensin system
  - diagnosis: proteinuria, RBC and RBC casts in urine, elevated BUN and creatinine
  - treatment: IV calcium channel blockers, +/- emergent ultrafiltration

- ❑ Catecholamine Induced Hypertensive Emergencies
  - etiology: discontinuation of short-acting sympathetic blocker (e.g. clonidine, propranolol)
    - pheochromocytoma
    - sympathomimetic drugs (cocaine, amphetamines, phencyclidine)
    - MAOI in combination with sympathomimetics or tyramine containing foods (cheese, red wine)
  - treatment: readminister sympathetic blocker if due to withdrawal (e.g. clonidine, propranolol)
    - avoid use of pure beta-blockers as they inhibit beta mediated vasodilation and leave alpha-adrenergic vasoconstriction unopposed
    - best agents are nicardipine, verapamil, fenoldopam. Phentolamine and nitroprusside are possible alternatives

STATUS EPILEPTICUS (see Neurology Chapter)

- ❑ a single seizure/series of seizures that lasts > 30 min
  - generalized: tonic-clonic, tonic, clonic, myoclonic, absence
  - partial: simple, complex
- ❑ etiology
  - drugs (anticonvulsant withdrawal, EtOH withdrawal),metabolic disorders, cerebrovascular disorders, infection, idiopathic
- ❑ investigations
  - CBC, lytes, BUN, Cr, glucose, anticonvulsant levels, tox screen, prolactin (8), CT +/– MRI, EEG
- ❑ treatment
  - diazepam 5-20 mg IV
  - Dilantin 15 mg/kg, given over 30 mins
  - phenobarbital 15 mg/kg IV
  - if above fail, Lidocaine 1.5 mg/kg IV; Pentothal 3 mg/kg IV OR midazolam and intubate
  - emergency EEG if no response after 15-20 minutes

SYNCOPE

❑ sudden, transient loss of consciousness and postural tone with spontaneous recovery

Etiology

❑ usually caused by generalized cerebral hypoperfusion
❑ cardiogenic: arrhythmia, outflow obstruction, MI
- non-cardiogenic: peripheral vascular (hypovolemia), vaso-vagal, cerebrovascular disorders, seizure disorders

History

❑ gather details from witnesses
❑ distinguish between syncope and seizure (see Neurology Chapter)
- signs and symptoms of precyncope, syncope and postsyncope
- past medical history, drugs

Physical

❑ BP and pulses in both arms, posturals
❑ cardiovascular exam and neuro exam

Investigations

❑ ECG
❑ CBC, lytes, BUN, creatinine, glucose, ABG's, Troponin, CKMB, Mg, Ca

Disposition

❑ cardiogenic syncope: admit to medicine/cardiology
❑ non-cardiogenic syncope: discharge with follow-up of Holter or echo study

SEXUAL ASSAULT AND DOMESTIC VIOLENCE

❑ involve local/regional sexual assault team
❑ 1 in 4 women and 1 in 10 men will be sexually assaulted in their lifetime

General Management Principles

❑ ABC's
❑ ensure patient is not left alone and ongoing emotional support provided
❑ set aside adequate time for exam (usually 1 1/2 hours)
❑ obtain consent for medical exam and treatment, collection of evidence, disclosure to police (notify police as soon as consent obtained)
❑ Sexual Assault Kit (document injuries, collect evidence)
❑ samples —> labeled immediately —> passed directly to police
❑ offer community crisis resources (e.g. shelter, hotline)
❑ do not report unless victim requests (legally required if <16 years)

History

❑ who? how many? when? where did penetration occur? what happened ? any weapons or physical assault?
❑ post-assault activities (urination, defecation, change of clothes, shower, douche, etc.)
❑ gynecologic history
- gravity, parity, last menstrual period (LMP)
- contraception
- last voluntary intercourse (sperm motile 6-12 hours in vagina, 5 days in cervix)

Physical Exam

❑ evidence collection is always secondary to treatment of serious injuries
❑ never retraumatize a patient with the examination
❑ general examination
- mental status
- sexual maturity
- patient should remove clothes and place in paper bag
- document abrasions, bruises, lacerations, torn frenulum/broken teeth (indicates oral penetration)

❑ pelvic exam and specimen collection
- ideally before urination or defecation
- examine for seminal stains, hymen, signs of trauma
- collect moistened swabs of dried seminal stains
- pubic hair combings and cuttings
- speculum exam
  - lubricate with water only
  - vaginal lacerations, foreign bodies
  - Pap smear
  - oral/cervical/rectal culture for gonorrhea and chlamydia
- posterior fornix secretions if present or aspiration of saline irrigation
  - immediate wet smear for motile sperm
  - air-dried slides for immotile sperm, acid phosphatase, ABO group

❑ others
- fingernail scrapings
- saliva sample from victim
- VDRL - repeat in 3 months if negative
- serum β-HCG
- blood for ABO group, Rh type, baseline serology (e.g. hepatitis, HIV)

Treatment
- ❑ medical
  - suture lacerations
  - tetanus prophylaxis
  - gynecology consult for foreign body, complex lacerations
  - assumed positive for gonorrhea and chlamydia
    - azithromycin 1g PO x 1 dose or cefixime 400 mg PO x 1 dose + doxycycline 100 mg BID PO x 7 days
  - may start prophylaxis for hepatitis B and HIV
  - pre and post counselling for HIV testing
  - pregnancy prophylaxis offered
    - Ovral 2 tabs STAT and 2 tabs in 12 hrs (within 72 hrs post-coital) with Gravol 50mg
- ❑ psychological
  - high incidence of psychological sequelae
  - have victim change and shower after exam completed
  - follow-up with MD in rape crisis centre within 24 hours
  - best if patient does not leave E.D. alone

Domestic Violence
- ❑ IDENTIFY THE PROBLEM (need high index of suspicion)
  - suggestive injuries
  - somatic symptoms (chronic and vague complaints)
  - psychosocial symptoms
- ❑ if disclosed, be supportive and assess danger
- ❑ management
  - treat injuries
  - ask about sexual assault and children at home
  - document findings
  - plan safety
- ❑ FOLLOW-UP: family doctor/social worker

VIOLENT PATIENT
- ❑ SAFETY FIRST - yourself, patient, staff, other patients
- ❑ always consider and rule out organic causes (as they can be fatal)
- ❑ leading organic causes are EtOH, drugs, and head injuries

Differential Diagnosis
- ❑ organic
  - drugs/toxins/withdrawal
  - metabolic (electrolyte abnormalities, hypoglycemia, hypoxia)
  - infections (sepsis, encephalitis, brain abscess, etc.)
  - endocrine (Cushing's, thyrotoxicosis)
  - CNS (head injuries, tumour, seizure, delirium and dementia)
- ❑ functional/psychiatric
  - situational crisis
  - schizophrenia, bipolar disorder (manic), personality disorder

Prevention
- ❑ be aware and look for prodromal signs of violence
  - prior history of violence or criminal behaviour
  - anxiety, restlessness, defensiveness, verbal attacks
- ❑ try to de-escalate the situation early
  - address the patient's anger
  - empathize

Restraints
- ❑ physical
  - present option to patient in firm but non-hostile manner
  - demonstrate sufficient people to carry it out
  - restrain supine or on side
  - suction and airway support available in case of vomiting
- ❑ pharmacologic
  - often necessary - may mask clinical findings and impair exam
  - Haldol 5-10 mg IM (be prepared for dystonic reactions, especially with multiple doses of neuroleptics over a short period) + lorazepam 2 mg IM/IV
  - look for signs of anticholinergic OD first (see Toxicology section)
- ❑ once restrained, search person/clothing for drugs and weapons

History
- ❑ antecedent and precipitating events and locale
- ❑ drugs: prescription, over the counter (OTC) (antihistamines, anticholinergics, stimulants), recreation/abuse/steroids, withdrawal reaction
- ❑ past medical history (especially DM)
- ❑ past psychiatric history and past legal history
- ❑ patient's insight
- ❑ speak to family/friends

Physical Exam
❑ vitals
- temperature often increased in delirium or toxic psychosis
- hypothermia may have altered mental status

❑ signs of trauma - especially head and neck
❑ neurologic exam, including brief mental status
❑ signs of drug toxicity and needle marks
❑ signs of hypoglycemia

Investigations
❑ screening bloodwork: CBC, lytes, glucose, creatinine, BUN, osmolality
❑ selective drug and toxin screen (see Toxicology section)
❑ CT head if necessary

TOXICOLOGY

APPROACH TO THE OVERDOSE PATIENT

History Taking
1. How much? How long ago? What method? (ingestion, inhalation, dermal, occular, environmental, IV ?)
2. accidental vs non-accidental exposure

Physical Exam
1. focus on: BP, HR, pupils, LOC, airway

Principles of Toxicology
❑ "All substances are poisons ... The right dose separates a poison from a remedy"
❑ 5 principles to consider with all ingestions
1. resuscitation (ABCs)
2. screening (toxidrome? clinical clues?)
3. decrease absorption of drug
4. increase elimination of drug
5. is an antidote available?

❑ suspect overdose when:
- altered level of consciousness/coma
- young patient with life-threatening arrhythmia
- trauma patient
- bizarre or puzzling clinical presentation

ABCs OF TOXICOLOGY
❑ basic axiom of care is symptomatic and supportive treatment
❑ can only address underlying problem once patient is stable

A **A**irway (consider stabilizing the C-spine)
B **B**reathing
C **C**irculation
D1 **D**rugs
- ACLS as necessary to resuscitate the patient
- universal antidotes

D2 **D**raw bloods
D3 **D**econtamination (decreased absorption, increased elimination)
E **E**xpose (look for specific toxidromes)/**E**xamine the Patient
F **F**ull vitals, ECG monitor, Foley, x-rays, etc.
G **G**ive specific antidotes, treatments

Go back and reassess.
CALL POISON CONTROL CENTRE
OBTAIN CORROBORATIVE HISTORY FROM FAMILY/FRIENDS IF PRESENT

D1 - UNIVERSAL ANTIDOTES
❑ treatments which will never hurt any patient and which may be essential

Oxygen
❑ do not deprive a hypoxic patient of oxygen no matter what the antecedent medical history (i.e. even COPD and CO_2 retention)
❑ if depression of hypoxic drive, intubate and ventilate
❑ only exception: paraquat or diquat (herbicides) inhalation or ingestion

Thiamine (Vitamin B1)
- ❑ 100 mg IV/IM to all patients prior to IV/PO glucose
- ❑ a necessary cofactor for glucose metabolism, but do not delay glucose if thiamine unavailable
- ❑ to prevent Wernicke-Korsakoff syndrome **WACO**
  - **W**ernicke's encephalopathy - **A**taxia, **C**onfusion, **O**phthalmoplegia)
  - untreated, may progress to Korsakoff's psychosis (disorder in learning and processing of new information)
  - treatment: high dose thiamine (1000 mg/day x 3 days)
  - most features usually irreversible
- ❑ populations at risk for thiamine deficiency
  - alcoholics
  - anorexics
  - hyperemesis of pregnancy
  - malnutrition states
- ❑ in E.D., must assume all undifferentiated comatose patients are at risk

Glucose
- ❑ give to any patient presenting with altered LOC
- ❑ do dextrostix prior to glucose administration
- ❑ adults: 0.5-1.0 g/kg (1-2 mL/kg) IV of D50W
- ❑ children: 0.25 g/kg (2 mL/kg) IV of D25W

Naloxone
- ❑ antidote for opioids: diagnostic and therapeutic (1 min onset of action)
- ❑ used in the setting of the undifferentiated comatose patient
- ❑ loading dose
  - adults
    - 2 mg initial bolus IV/IM/SL/SC or via ETT
    - if no response after 2-3 minutes, progressively double dose until a response or total dose of 10 mg given
    - known chronic user, suspicious history, or evidence of tracks 0.01 mg/kg (to prevent acute withdrawal)
  - child
    - 0.01 mg/kg initial bolus IV/IO/ETT
    - 0.1 mg/kg if no response and narcotic suspected
- ❑ maintenance dose
  - may be required because half-life of naloxone much shorter than many narcotics (half-life of naloxone is 30-80 minutes)
  - hourly infusion rate at 2/3 of initial dose that produced patient arousal

D2 - DRAW BLOODS
- ❑ essential bloods
  - CBC, electrolytes, urea, creatinine
  - glucose (and dextrostix), INR, PTT
  - ABGs, measured O2 sat
  - osmolality
  - acetylsalicylic acid (ASA), acetaminophen alcohol levels
- ❑ potentially useful bloods
  - drug levels
  - Ca^{2+}, $Mg2^{+}$, PO_4^{3-}
  - protein, albumin, lactate, ketones and liver tests

Serum Drug Levels
- ❑ treat the patient, not the drug level
- ❑ negative toxicology screen only signifies that the specific drugs tested were not detectable in the particular specimen at the time it was obtained (i.e. does not rule out a toxic ingestion)
- ❑ generally available on serum screens (differs by institution)
  (screen is different from drug levels - screen is very limited)
  - acetaminophen*
  - ASA*
  - barbituates and other sedative/hypnotics
  - benzodiazepines (qualitative only)
  - ethanol
  - ethylene glycol*
  - methanol*
  - tricyclic antidepressant (TCA) (qualitative only)

  * significant if in "toxic" range

- ❑ urine screens also available (qualitative only)

Toxic Gaps (see Nephrology Chapter)

Anion gap (AG) = $Na^+ - (Cl^- + HCO_3^-)$
- normal range 10-14 mmol/L
- unmeasured cations: Mg^{2+}, Ca^{2+}
- unmeasured anions: proteins, organic acids, PO_4^{3-}, sulfate

Plasma osmolal gap (POG) = (measured - calculated) osmoles
- normally POG < 10 mOsm/L
- calculated osmolality = 2 Na^+ + BUN + blood glucose (mmol/L)

Metabolic acidosis

Increased AG: differential of causes (*toxic)
"MUDPILES CAT"
Methanol*
Uremia
Diabetic ketoacidosis/Alcoholic ketoacidosis
Phenformin*/Paraldehyde*
Isoniazid*/**I**ron*
Lactate (anything that causes seizures or shock)
Ethylene glycol*
Salicylates*

Cyanide*
Arsenic*
Toluene*

Decreased AG
1. error
2. electrolyte imbalance (increased $Na^+/K^+/Mg^{2+}$)
3. hypoalbuminemia (50% fall in albumin ~ 5.5 mmol/L decrease in the AG)
4. Li, Br elevation
5. paraproteins (multiple myeloma)

Plasma Osmolal Gaps

Increased osmolal gap: "**MAE DIE**"
Methanol
Acetone
Ethanol
Diuretics (glycerol, mannitol, sorbitol)
Isopropanol
Ethylene glycol

Oxygen saturation gap: (measured – calculated) O_2 saturation
(1) measured by absorption spectrophotometry (pulse oximetry)
(2) calculated from Hb/O2 saturation curve

Increased O_2 saturation gap
(3) carboxyhemoglobin
(4) methemoglobin
(5) sulfhemoglobin

Normal AG
(1)K^+: pyelonephritis, obstructive nephropathy, renal tubular acidosi (RTA), IV, TPN
(2)K^+: small bowel losses, acetazolamide, RTA I, II

Table 13. Use of the Clinical Laboratory in the Initial Diagnosis of Poisoning

Test	Finding	Selected Causes
ABGs	• hypoventilation (↑ P_{CO2}) • hyperventilation	• CNS depressants (opioids, sedative-hypnotic agents, phenothiazines, and EtOH) • salicylates, CO, other asphyxiants
Electrolytes	• anion-gap metabolic acidosis • hyperkalemia • hypokalemia	• "MUDPILES CAT" • digitalis glycosides, fluoride, K^+ • theophylline, caffeine, β-adrenergic agents, soluble barium salts, diuretics
Glucose	• hypoglycemia	• oral hypoglycemic agents, insulin, EtOH
Osmolality and Osmolar Gap	• elevated osmolar gap	• "MAE DIE" **M**ethanol, **A**cetone, **E**thanol, **D**iuretics, **I**sopyropyl alcohol, **E**thylene glycol
ECG	• wide QRS complex • prolongation of QT interval • atrioventricular block	• TCAs, quinidine, other class Ia and Ic antiarrhythmic agents • quinidine and related antiarrhythmics, terfenadine, astemizole • Ca^{2+} antagonists, digitalis glycosides, phenylpropanolamine
Abdominal x-ray	• radiopaque pills or objects	• "**CHIPES**" **C**alcium, **C**hloral hydrate, **C**Cl_4, **H**eavy metals, **I**ron, **P**otassium, **E**nteric coated **S**alicylates, and some foreign bodies
Serum Acetaminophen	• elevated level (> 140 mg/l 4 hours after ingestion)	• acetaminophen (may be the only clue to a recent ingestion)

D3 - DECONTAMINATION

❑ PROTECT YOURSELF FIRST
❑ CALL POISON CONTROL CENTRE IF ANY UNCERTAINTY

Ocular Decontamination
❑ saline irrigation to neutral pH
❑ alkali exposure requires ophthalmology consult

Dermal Decontamination (wear protective gear)
❑ remove clothing
❑ brush off toxic agents
❑ irrigate all external surfaces

Gastrointestinal Decontamination
❑ activated charcoal (AC)
- indications
  - single dose will prevent significant absorption of many drugs and toxins
- contraindications
  - acids, alkalis, cyanides, alcohols, Fe, Li
- dose = 1 g/kg body weight or 10 g/g drug ingested
- odourless, tasteless, prepared as slurry with H_2O
- cathartics rarely used (risk electrolyte imbalance)

❑ multi-dose activated charcoal (MDAC)
- absorption of drug/toxin to charcoal prevents availability and promotes fecal elimination
- without charcoal, gut continuously absorbs toxins; MDAC (multidose activated charcoal) interrupts the enterohepatic circulation of some toxins and binds toxin diffusing back into enteral membrane from the circulation
- MDAC can increase drug elimination (potentially useful for phenobarbitol, carbamazepine, theophylline, digitoxin, others)
- dose
  - various regimes
  - continue until nontoxic or charcoal stool

❑ whole bowel irrigation
- flushes out bowel
- 500 mL (child) to 2000 mL (adult) of balanced electrolyte solution/hour by mouth until clear effluent per rectum
- indications
  - awake, alert patient who can be nursed upright
  - delayed release product
  - drug/toxin not bound to charcoal
  - drug packages - if any evidence of breakage —> emergency surgery
  - recent toxin ingestion (up to 4-6 hours)
- contraindications
  - evidence of ileus, perforation, or obstruction

❑ surgical removal
- indicated for drugs
  - that are toxic
  - that form concretions
  - that are not removed by conventional means

E - EXAMINE THE PATIENT

❑ vital signs (including temperature), skin (needle tracks, colour), mucous membranes, odours and CNS
❑ head-to-toe survey
- C-spine
- signs of trauma
- signs of seizures (incontinence, “tongue biting”, etc.)
- signs of infection (meningismus)
- signs of chronic alcohol abuse
- signs of drug abuse (track marks, nasal septum erosion)
- mental status

SPECIFIC TOXIDROMES

Narcotics, Sedatives/Hypnotics, Alcohol Overdose
❑ signs and symptoms
- hypothermia
- bradycardia
- hypotension
- respiratory depression
- dilated/constricted pupils
- CNS depression

Drug / Substance Withdrawal

❑ withdrawal state generally opposite to the physiological effect of the drug
❑ signs and symptoms of sedative withdrawal
- increased temperature
- tachycardia
- hypertension
- dilated pupils
- diaphoresis
- agitation
- tremor
- hallucinations
- seizures

❑ drugs
- alcohol
- benzodiazepines
- barbituates
- antihypertensives
- opioids

Drug Class	Overdose Signs and Symptoms	Examples of Drugs
Anticholinergics	• hyperthermia "Hot as a Hare" • dilated pupils "Blind as a Bat" • dry skin "Dry as a Bone" • vasodilatation "Red as a Beet" • agitation/hallucinations "Mad as a Hatter" • ileus "The bowel and bladder • urinary retention lose their tone and the • tachycardia heart goes on alone"	Anticholinergic drugs antidepressants Flexeril Tegretol antihistamines (e.g. Gravol, diphenhydramine) antiparkinsonians antipsychotics antispasmotics belladonna alkaloids (e.g. atropine, scopolamine)
Cholinergics	"**DUMBELS**" **D**iaphoresis, **D**iarrhea, **D**ecreased blood pressure **U**rination **M**iosis **B**ronchorrhea, **B**ronchospasm, **B**radycardia **E**Emesis, **E**xcitation of skeletal muscle **L**acrimation **S**alivation, **S**eizures	cholinergics (nicotine, mushrooms) anticholinesterases (physostigmine, organophosphate insecticides)
Extrapyramidal	dysphonia rigidity and tremor dysphagia torticollis laryngospasm trismus (deviation of eyes in all directions) oculogyric crisis	major tranquilizers
Hemoglobin Derangements	increased respiratory rate decreased level of consciousness seizures cyanosis (unresponsive to O_2) lactic acidosis	carbon monoxide poisoning (carboxyhemoglobin) drug ingestion (methemoglobin, sulfhemoglobin)
Metal Fume Fever	abrupt onset of fever, chills, myalgias metallic taste in mouth nausea and vomiting headache fatigue (delayed respiratory distress)	fumes from heavy metals (welding, brazing, etc.) amphetamines • caffeine • cocaine • ephedrine (and other decongestants) • LSD
Sympathomimetics	increased temperature CNS excitation (including seizures) tachycardia nausea and vomiting hypertension diaphoresis dilated pupils	• PCP • theophylline • thyroid hormone •ASA toxicity looks like sympathomimetic overdose • sedative/hypnotic withdrawal (including alcohol) also similar

G - GIVE SPECIFIC ANTIDOTES AND TREATMENTS

❑ note: hemodialysis can be considered for all drugs

Table 14. Toxins and Antidotes

Toxin	Antidote/Treatment
Acetaminophen	N-acetylcysteine (Mucomyst)
Anticholinergics	*Physostigmine
Benzodiazepines	flumazenil (Romazicon/Anexate)
β-blockers	atropine, isoprotenerol, glucagon
Calcium channel blockers	calcium chloride or gluconate
Carbon Monoxide	100% oxygen, hyperbaric O_2
Cyanide	Lilly kit (amyl nitrite, then sodium nitrite): Na thiosulfate
Digoxin	stop dig, use FAB fragments (Digibind), restore K^+
Heparin	protamine sulfate
Insulin	glucose/glucagon
Iron	deferoxamine
Methanol/Ethylene glycol	ethanol or fomepizole, leucovorin (for methanol), thiamine and pyrudoxine for EG
Nitrites	methylene blue
Opioids	naloxone (Narcan)
Organophosphates	atropine, pralidoxime
Salicylates	alkalinize urine, restore K^+
TCA's	sodium bicarbonate bolus
Warfarin	vitamin K; (FFP if necessary)
	* available through special access programme only

SPECIFIC TREATMENTS

Acetaminophen Overdose

acetaminophen = paracetamol = APAP

❑ acute acetaminophen OD —> metabolized by Cytochrome P450 —> saturation of pathway —> toxic metabolite (NAPQI) scavenged by glutathione (an antioxidant) (in non-overdose situations)
- in OD: exhaustion of glutathione stores —> NAPQI accumulates —> binds hepatocytes and hepatic necrosis

❑ toxic dose of acetaminophen > 150 mg/kg (~7.0 g)
❑ increased risk of toxicity if chronic EtOH and/or anti-convulsant drugs
❑ clinical: no symptoms
- serum acetaminophen level
- evidence of liver/renal damage (delayed > 24 hours)
- increased AST, INR
- decreased glucose, metabolic acidosis, encephalopathy (indicate poor prognosis)

❑ management
- decontamination
- serum acetaminophen level 4 hours post ingestion
- measure liver enzymes and INR, PTT
- use the Rumack-Matthew Nomogram for acetaminophen hepatotoxicity
- N-acetylcysteine (Mucomyst)
  - substitutes for glutathione as anti-oxidant to prevent liver damage
  - use according to dosing nomogram
  - best effect if started within 8 hrs post-ingestion, but therapy should be initiated regardless

ASA Overdose

❑ acute and chronic (elderly with renal insufficiency)
❑ clinical
- hyperventilation (central stimulation of respiratory drive)
- increased AG metabolic acidosis (increased lactate)
- tinnitus, confusion, lethargy
- coma, seizures, hyperthermia, non-cardiogenic pulmonary edema, circulatory collapse

❑ ABG's possible: 1. respiratory alkalosis
2. metabolic acidosis
3. respiratory acidosis

❑ management
- decontamination
- 10:1 charcoal:drug ratio
- whole bowel irrigation (useful if enteric-coated ASA)
- close observation of serum level, serum pH
- alkalinization of urine as in Table 14 to enhance elimination and to protect the brain (want serum pH 7.45-7.55)
- may require K^+ supplements for adequate alkalinization
- consider hemodialysis when
  - severe metabolic acidosis (intractable)
  - increased levels
  - end organ damage (unable to diurese)

Table 15. Urine Alkalinization in ASA Overdose

Plasma pH	Urine pH	Treatment
alkaline	alkaline	D5W with 20 mEq KCl/L + 2 amps HCO_3/L at 2-3 cc/kg/hr
alkaline	acid	D5W with 40 mEq KCl/L + 3 amps HCO_3/L at 2-3 cc/kg/hr
acid	acid	D5W with 40 mEq KCl/L + 4 amps HCO_3/L

Anticholinergic Overdose (e.g. antihistamines)
- ❑ physostigmine salicylate: reversible acetylcholinesterase inhibitor
- ❑ supportive therapy

Benzodiazepine (BZ) Overdose
- ❑ flumazenil (Anexate): specific benzodiazepine antagonist
- ❑ indications
  - iatrogenic BZ oversedation
  - to reverse BZ anesthesia
- ❑ contraindications
  - known seizure disorder
  - mixed OD (especially if tricyclic antidepressant (TCA) suspected)
  - BZ dependence or chronic use
- ❑ dose
  - adult: 0.3 mg IV (q5mins to maximum 1.0 g)
  - child: 10 µg/kg (as above, maximum 0.3 mg)
- ❑ CAUTION - most BZ have prolonged half life compared to flumazenil
  - if re-sedation occurs, repeat doses or IV infusion may be indicated

Beta Blockers
- ❑ symptoms within 2 hours of ingestion
- ❑ atropine or isoprotenerol if severe
- ❑ glucagon
  - works as non-β-adrenergic receptor agonist to increase production of cAMP, thereby increasing contractility
- ❑ glucagon 50-100 µg/kg (5-10 mg for adults) slow IV push, then IV at 70 µg/kg/hour

Calcium Channel Blockers
- ❑ order ECG, lytes (Ca^{2+}, Na^{+}, Mg^{2+}, K^{+} especially)
- ❑ calcium chloride 1-4 g of 10% solution IV if hypotension
- ❑ atropine or isoprotenerol if severe
- ❑ glucagon
- ❑ inotrope/aggressive supportive therapy

CO Poisoning
- ❑ 100% O_2 therapy by face mask critical for CO poisoning
- ❑ hyperbaric O_2 disputed benefits; no evidence for therapy in mild-moderate cerebral dysfunction (may prevent neurological sequelae)
- ❑ coma is an undisputed indication for hyperbaric O_2 therapy (still the standard of care)
- ❑ hyperbaric O_2 (efficacy unclear) suggested for pregnant patients

Digoxin Overdose
- ❑ Digibind = digoxin-specific antibody fragments (Fab)
- ❑ use in combination with activated charcoal
- ❑ indications
  - life threatening arrhythmias unresponsive to conventional therapy (ventricular fibrillation, ventricular tachycardia, conduction block)
  - 6 hr serum digoxin >19 nmol/L (> 15 ng/mL)
  - initial serum K^{+} > 5 mmol/L
  - history of ingestion > 10 mg for adult, > 4 mg for child
- ❑ dose
  - 1 vial = 40 mg Digibind neutralizes 0.6 mg digoxin
  - cost of one vial = $200
  - empirically: 20 vials if acute ingestion 5-10 vials if chronic ingestion (90-95% of cases)
  - onset of action 20-90 minutes
  - renal elimination half life 20-30 hours

Acute Dystonic Reaction
- ❑ benztropine (Cogentin), antihistamines, benzodiazepines effective
- ❑ benztropine has euphoric effect and potential for abuse
- ❑ for acute dystonic reaction
  - 1-2 mg IM/IV benztropine then 2mg PO bid x 3 days
    OR
    diphenhydramine 1-2 mg/kg IM/IV then 25 mg PO qid x 3 days

Hydrogen Fluoride Burns
❑ Ca^{2+} gluconate gel topical or intradermal or both
❑ intravenous calcium chloride for systemic hypocalcemia, hyperkalemia

Insulin/Oral Hypoglycemic Overdose
❑ glucose can be given IV, PO or via NG
❑ glucagon (if no access to glucose)
- 1-2 mg IM

Ethanol Overdose
❑ very common in children
❑ mouthwash > 70% EtOH, perfumes/colognes 40 – 60% EtOH
❑ dehydrogenase pathway is less active in children
❑ presentation: flushed face, dilated pupils, sweating, GI distress, hypothermia, hypoventilation, hypotension
❑ order: serum EtOH level, glucose
❑ manage: airway, circulation, glucose levels
❑ folate up to 50 mg q4h IV
❑ gastric decontamination if ingestion < 1hr ago
❑ hemodialysis if serum EtOH > 500 mg/dL

Methanol or Ethylene Glycol Overdose
❑ ethanol PO/IV used to block metabolism of methanol and ethylene glycol preventing toxicity
❑ Fomepizole (4-methylpyrazole) 15 mg/kg initial dose, a competitive inhibitor of alcohol dehydrogenase, available for ethylene glycol intoxication (~$2000 a day); recent evidence for use in methanol intoxication
❑ folic acid (Leucovorin) 50 mg IV q4h for several days to potentiate folate-dependent metabolism of formic acid in methanol poisoning
❑ dialysis if ethanol treatment unsuccessful, visual impairment (MetOH), renal failure (ethylene glycol) or uncorrectable metabolic acidosis
❑ thiamine 100 mg IV q6h plus pyridoxine 100 mg IV q6h to enhance alternative metabolism to non-toxic products in ethylene glycol poisoning

Organophosphates/Cholinergic Overdose
❑ atropine
- anticholinergic / antimuscarinic
- for anticholinesterase poisonings and cholinergic poisonings with muscarinic symptoms
- 0.03 mg/kg to max 2 mg/dose (may repeat q 10-15 min until secretions dry) (may need hundreds of milligrams)

❑ pralidoxime (Protopam, 2-PAM)
- reactivates acetylcholinesterase, reverses nicotinic effects
- organophosphate poisonings only
- most beneficial if given within 24-36 hours
- 25-50 mg/kg over 5 min IV q6h, up to 1-2 g for adults

Tricyclic Antidepressants (TCAs) Overdose
❑ cardiac monitoring mandatory because of cardiac toxicity
❑ QRS complex width predicts outcome
- > 100 ms: increased risk of seizures
- > 160 ms: increased risk of arrythmias

❑ evidence unavailable regarding when to give bicarbonate (HCO_3^-)
❑ dose: 1-2 mEq/kg q 10-15 min bolus (2-4 amps)

Warfarin/Rat Poison Overdose
❑ Vitamin K (see Table 16)

Table 16. Protocol for Warfarin Overdose

INR	Management
< 5.0	• reduce maintenance dose +/– hold dose x 1
5.0-9.0	• if no risk factors for bleeding, hold Coumadin x 1-2 days and reduce maintenance dose OR • if rapid reversal required, Vit K 2-4 mg PO, repeat INR in 24 h • additional Vit K 1-2mg PO if INR still high (onset 4-6 h)
9.0-20.0	• Vit K 3-5 mg PO, INR in 24h and additional Vit K if necessary
> 20.0	• fresh frozen plasma (FFP) 10-20 mL/kg • Vit K 10 mg IV over 10 min (IV Vit K only if life-threatening hemorrhage); onset ~ 2 h • increased Vit K dosing (q4h) • phenobarbital (little evidence)

pH ALTERATION – see Table 14
- ❑ if toxin has potential for ion-trapping at physiologically achievable pH
- ❑ urine alkalinization
  - urine pH 7.5-8.0
  - potentially useful for salicylates, phenobarbital
  - evidence for phenobarbital is equivocal

EXTRA-CORPOREAL DRUG REMOVAL (ECDR)

Criteria for Hemodialysis
- ❑ toxins that have
  - water solubility
  - low protein binding
  - low molecular weight
  - adequate concentration gradient
  - small volume of distribution (Vd) or rapid plasma equilibration
- ❑ removal of toxin will cause clinical improvement
- ❑ advantage is shown over other modes of therapy
- ❑ greater morbidity from prolonged supportive care
- ❑ predicted that drug or metabolite will have toxic effects
- ❑ impairment of normal routes of elimination (cardiac, renal, or hepatic)
- ❑ clinical deterioration despite maximal medical support
- ❑ useful for toxins at the following blood levels:
  - alcohols
    - methanol: > 15.6 mmol/L (> 25-50 mg/dL)
    - ethylene glycol: > 8 mmol/L (> 50 mg/dL)
  - salicylates
    - acute (within 6 h): > 7.2-8.7 mmol/L (> 100 mg/dL)
    - chronic: > 4.3-4.8 mmol/L (> 60 mg/dL)
  - lithium
    - acute (within 6 h): > 4.0 mmol/L
    - chronic: > 2.5-4.0 mmol/L
  - bromine: > 15 mmol/L
  - phenobarbital: 430-650 mmol/L
  - chloral hydrate (—> trichloroethanol): > 200 mg/kg

Criteria for Hemoperfusion
- ❑ as for hemodialysis
- ❑ absorbent has greater drug binding capacity than protein or tissue
- ❑ useful for
  - theophylline: > 330 mmol/L (chronic), > 550 mmol/L (acute)
  - short acting barbiturates (secobarbital)
  - non-barbiturate sedative-hypnotics
  - (phenytoin, carbemazepine, disopyramide, paraquat, methotrexate, Amanita phalloides)

DISPOSITION FROM THE EMERGENCY DEPARTMENT (E.D.)
- ❑ discharge home vs. prolonged E.D. observation vs. admission
- ❑ methanol, ethylene glycol
  - delayed onset
  - admit and watch clinical and biochemical markers
- ❑ TCA's
  - prolonged/delayed cardiotoxicity warrants admission to monitored (ICU) bed
  - if asymptomatic and no clinical signs of intoxication: 6 hour E.D. observation adequate with proper decontamination
  - sinus tachycardia alone (most common finding) with history of OD warrants observation in E.D.
- ❑ hydrocarbons/smoke inhalation
  - pneumonitis may lag 6-8 hours
  - consider observation for repeated clinical and radiographic examination
- ❑ ASA, acetaminophen
  - if borderline level, get second level 2-4 hours after first
- ❑ oral hypoglycemics
  - admit all patients for minimum 24 hours if hypoglycemic

Psychiatric Consultation (see Psychiatry Chapter)
- ❑ once patient medically cleared, arrange psychiatric intervention if required
- ❑ beware - suicidal ideation may not be expressed

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