

THE ABCs OF EMERGENCY MEDICINE

**Twelfth Edition
2012**

Divisions of Emergency Medicine
Faculty of Medicine, University of Toronto

Editors
Laura Hans, M.D.
St. Michael's Hospital

Yasmine Mawji, M.D.
Mt. Sinai Hospital

CONTRIBUTORS

Bryan Au, M.D.
St. Michael's Hospital

Mary Ann Badali, M.D.
Sunnybrook Health Sciences
Centre

Glen Bandiera, M.D.
St. Michael's Hospital

Mike Brzozowski, M.D.
Sunnybrook Health Sciences
Centre

Alan Campbell, M.D.
Trillium Health Centre-
Mississauga Site

David Carr, M.D.
University Health Network

Dan Cass, M.D.
St. Michael's Hospital

Jordan Chenkin, M.D.
Sunnybrook Health Sciences
Centre

Anil Chopra, M.D.
University Health Network

Jennifer Devon, M.D.
Scarborough Hospital

Sheila Dunn, M.D.
Women's College Hospital

Heather Farina, R.N.
Hospital for Sick Children

Steven Friedman, M.D.
University Health Network

Lynne Fulton, M.D.
Sunnybrook Health Sciences
Centre

Peter Glazer, M.D.
University Health Network

Brian Goldman, M.D.
Mt. Sinai Hospital

Paul Hannam, M.D.
Toronto East General Hospital

Laura Hans, M.D.
St. Michael's Hospital

Leah Harrington, M.D.
Hospital for Sick Children

Anton Helman, M.D.
Toronto East General Hospital

Walter Himmel, M.D.
North York General Hospital

Matthew Hodge, M.D.
University Health Network

Martin Horak, M.D.
St. Michael's Hospital

Cheryl Hunchak, M.D.
Mount. Sinai Hospital

Santosh Kanjeeval, M.D.
Toronto East General Hospital

Tom Kaul, M.D.
St. Michael's Hospital

Simon Kingsley, M.D.
St. Michael's Hospital

Kate Lazier, M.D.
University Health Network

Shirley Lee, M.D.
Mount Sinai Hospital

Eric Letovsky, M.D.
Credit Valley Hospital

Joel Lexchin, M.D.
University Health Network

Dave MacKinnon, M.D.
St. Michael's Hospital

Shauna Martiniuk, M.D.
Mount Sinai Hospital

Laurie Mazurik, M.D.
Sunnybrook Health Sciences
Centre

Nazanin Meshkat, M.D.
University Health Network

Andrew McDonald, M.D.
Sunnybrook Health Sciences
Centre

Maria McDonald, LLB

Howard Ovens, M.D.
Mount. Sinai Hospital

Rick Penciner, M.D.
North York General Hospital

Sev Perelman, M.D.
Mount. Sinai Hospital

Sara Pickersgill, M.D.
Credit Valley Hospital

George Porfiris, M.D.
Toronto East General Hospital

Jessica Potvin, R.N.
Women's College Hospital
Sexual Assault and Domestic
Violence Care Centre

Tim Rutledge, M.D.
North York General Hospital

Suzan Schneeweiss, M.D.
Hospital for Sick Children

Suzanne Schuh, M.D.
Hospital for Sick Children

Brian Schwartz, M.D.
Sunnybrook Health Sciences
Centre

Debbie Selby, M.D.
Sunnybrook Health Sciences
Centre

Dominick Shelton, M.D.
Sunnybrook Health Sciences
Centre

Kevin Skarratt, M.D.
Sunnybrook Health Sciences
Centre

Telisha Smith Gorvie, M.D.
University of Toronto

Leeor Sommer, M.D.
North York General Hospital

Kasia Stefanska, M.D.
Toronto East General Hospital

Heather Sues-McKay, M.D.
Mount Sinai Hospital

Peter Switakowski, M.D.
University Health Network

Fernando Teixeira, M.D.
St. Michael's Hospital

Caroline Thompson, M.D.
Scarborough Hospital

Margaret Thompson, M.D.
St. Michael's Hospital

Rajani Vairavanathan, M.D.
Toronto East General Hospital

Jeff Tyberg, M.D.
St. Michael's Hospital

Rick Verbeek, M.D.
Sunnybrook Health Sciences
Centre

Natalie Wolpert, M.D.
St. Michael's Hospital

Joel Yaphe, M.D.
University Health Network

PREFACE

Welcome to your Emergency Medicine Rotation!

We are pleased to present you with your manual, the ABC's of Emergency Medicine. This manual has been authored by many of the Emergency physicians with whom you will be working during your core rotation.

The manual is meant to accompany you on your shifts, to be a guide for the next month as well as a resource for future rotations. While the manual is not a comprehensive review of the listed topics, it will outline key issues, controversies and current practice for each topic.

This rotation is fast paced and exciting, you will see a broad variety of patients across all spectrum of ages from stable to critically ill. This rotation is beneficial to all medical students, as it will provide you a unique opportunity to see first

presentations of common medical problems and assist you in becoming more comfortable with the ill, undifferentiated patient.

We thank all the authors and chapter editors who have contributed their time and expertise. We are fortunate to have contributions from so many leaders in our community. Special thanks to Nancy Medeiros who has worked tirelessly to complete the 2012 update.

Enjoy your core Emergency rotation!

Laura Hans and Yasmine Mawji, editors 2012

Every effort has been made in preparing this manual to provide accurate and up-to-date information which is in accord with accepted standards and practice at the time of publication. Nevertheless, the authors and editors can make no warranties that the information contained herein is totally free from error, not least because clinical standards are constantly changing through research and regulation. The authors and editors therefore disclaim all liability for direct or consequential damages resulting from the uses of material contained in this manual. Readers are strongly advised to pay careful attention to information provided by the manufacturer of any drugs or equipment that they plan to use. This manual is intended for use by medical students. It is expected that the intended reader will consult with a supervising physician prior to initiation of treatment or management of a patient.

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1: AIRWAY

Dan Cass, Bryan Au

OBJECTIVES

1. To review basic airway management
2. To review indications for definitive airway management
3. To review rapid sequence intubation

INTRODUCTION

In the resuscitation of any patient, management of the airway is the first priority. One cannot continue in managing breathing or circulation problems if the patient does not have a patent airway. Even after airway management has taken place, in any patient who fails to improve, or who deteriorates, always start again with assessment and management of the airway.

ASSESSING THE AIRWAY

Before managing any patient's airway, it is important to quickly assess and identify those patients in whom you anticipate difficulty in ventilation and / or intubation. If you do – call for help.

Some key predictors of a difficult airway include:

Difficult Bag-valve Mask Ventilation - "BOOTS"

B = Beard

O = Obese

O = Older

T = Toothless

S = Snores / Stridor

Difficult Intubation - "MAP the Airway"

M = Mallampati Class and Measurements

Evaluate the Mallampati classification by asking patient to open their mouth (<http://bit.ly/bl8QE9>)

"3-3-2-1 rule"

- 3 fingers mouth opening
- 3 fingers distance from hyoid to chin
- 2 fingers distance from thyroid cartilage notch to floor of mandible
- 1 finger anterior jaw subluxation

A = Atlanto-occipital (neck) extension

- Normal = 35 degrees or more

P = Pathologic conditions

- Tumour, hematoma, trauma, etc.

REVIEW OF AIRWAY TECHNIQUES

Temporizing / Adjunctive Measures

- Chin lift/jaw thrust to open airway - caveat: no neck extension if suspected C-spine injury
- Bag-valve-mask ventilation – probably the most important, yet under-appreciated, skill of airway management. In the ED, when bagging, use a two-hands on the mask technique for a tight seal, and always use an oral airway
- Suctioning/removal of foreign bodies
- Nasal airway - generally well-tolerated by the temporarily obtunded patient (e.g. post-ictal, post-procedural sedation, intoxicated)
- Oral airway – aids in peri-intubation ventilation; not to be used in patient with intact gag reflex
- Laryngeal mask airway (LMA) – this device is inserted into the mouth and has a cuff that occludes the hypopharynx. It has a port through which ventilation can then occur. A variation is the Intubating LMA – this allows the insertion of an endotracheal tube via the ventilation port. The LMA is used both as a “rescue device” in failed intubation, and as a primary airway device
- Needle cricothyroidotomy - accomplished by inserting a needle in cricothyroid membrane, and oxygenating the patient using high pressure oxygen source

Definitive Airway

A definitive airway is the placement of a cuffed tube in the trachea. A cuffed endotracheal tube does not ensure that aspiration cannot occur, but does reduce the risk.

- Orotracheal / nasotracheal intubation
- Surgical airway – either percutaneous or open cricothyroidotomy with insertion of a cuffed tracheal tube

2 Section One RESUSCITATION

INDICATIONS FOR INTUBATION

The indications for intubation can be broken down into four main categories. These can be recalled as the **four P's**:

1. **Patency** - to obtain and maintain a patent airway in the face of obstruction. Examples include: decreased LOC, airway edema/burns, neck hematoma, tumour
2. **Positive-pressure ventilation** - to correct deficient oxygenation and/or ventilation. Examples include: pulmonary edema, COPD exacerbation
3. **Protection** - to protect the airway from aspiration in the event of decreased LOC
4. **Predicted deterioration** - in some situations, early intubation may be preferable to the potential need to urgently intubate in a less favourable environment (e.g. in CT scan), or when it may be significantly more difficult (e.g. progressive edema)

WHEN INTUBATION SHOULD BE ANTICIPATED

The following are several situations during which ED patients are commonly intubated:

- Trauma
- Overdoses on medications which cause rapid decrease in level of consciousness
- Severe congestive heart failure, asthma, COPD
- Head injured patients, or those who are comatose for non-traumatic reasons

SPECIAL CONSIDERATIONS IN THE ED

Airway management in the ED usually occurs on an urgent or emergent basis. The following are some things to keep in mind, as they will modify the plan of airway management:

- less time to assess airway, obtain past history, etc.
- less controlled than in the elective setting
- patients are frequently hemodynamically unstable
- all must be considered to have full stomachs, with the attendant risk of aspiration
- patients often have altered mental status, from markedly decreased to fighting/agitated due to alcohol, drugs, or head injury

Cervical spine injury and instability must be assumed in patients who have experienced major trauma, falls, or have an unknown history of injury.

- this consideration requires modification of airway techniques, both basic and advanced
- when intubating a patient with a known or suspected c-spine injury, remove the front of the cervical collar and have an assistant manually stabilize the neck ('in-line manual stabilization'). The collar can be replaced once tube placement is confirmed.

RAPID SEQUENCE INTUBATION

Rapid sequence intubation is defined as the simultaneous administration of a powerful sedative (induction) agent and a paralytic agent to facilitate intubation and decrease the risk of aspiration.

Although a detailed discussion of RSI is beyond the scope of this chapter, the basic steps are reviewed below. These can be recalled as the **six P's**.

1. **Preparation** – prepare all equipment, personnel, and medications
2. **Pre-oxygenation** – patient breathing 100% oxygen for 3-5 minutes or asking the patient to take 4-8 full breaths on 100% oxygen will wash out the nitrogen in the lungs, and prolong the time available for intubation before desaturation occurs.
3. **Pretreatment** – pretreatment with medications such as atropine in children, defasciculating doses of a non-depolarizing muscle relaxant prior to the administration of succinylcholine, and lidocaine in the setting of head injury is considered *optional*, given the lack of evidence for their benefit
4. **Paralysis with induction** – administration of a sedative agent (e.g. ketamine, propofol, etomidate) followed rapidly by the administration of a muscle relaxant (e.g. succinylcholine or rocuronium)
5. **Place the tube with proof** – intubate the patient, and confirm tube placement with end-tidal capnometry
6. **Post-intubation management** – chest x-ray, analgesia and sedation, further resuscitation

Relative contraindications to rapid sequence intubation include:

- anticipated difficult airway, especially difficult bag-valve mask ventilation. In this situation, an “awake” intubation with the patient maintaining respirations is preferred
- inadequate familiarity and comfort with the technique
- unnecessary (e.g. the patient in cardiac arrest or near-arrest)

THE TECHNIQUE OF LARYNGOSCOPY

Ensure that the proper preparations have been made, and the patient is positioned correctly in the “sniffing position”. The laryngoscope is held in the left hand, and introduced into the mouth on the right side of the tongue. Advance the laryngoscope *slowly* to the base of the tongue. Identification of the epiglottis is *crucial*. A common novice error is to rapid insert the blade too deeply, missing identification of the epiglottis.

Once the epiglottis is identified, seat the tip of the blade in the vallecula. Lift can now be applied to the laryngoscope in the direction of the handle. Do *not* lever the blade back. Once the epiglottis is lifted, the vocal cords should come into view. Without losing sight of the vocal cords, ask an assistant to hand you the endotracheal tube in your right hand. The tube is introduced into the right side of the patient’s mouth without obscuring your view of the cords. It is important to visualize the tip of the tube as it passes through the cords. As the tip passes, ask the assistant to remove the stylet, and place the tube in its final position. Inflate the cuff, and confirm end-tidal CO₂.

Routine post-intubation care including chest x-ray, analgesia and sedation, and further resuscitation will now follow.

For an excellent laryngoscopy video, go to <http://emcrit.org/airway/laryngoscopy>

TIPS AND TOOLS TO FACILITATE INTUBATION

A number of tips and tools exist that can make intubation easier, even in patients for whom a clear visualization of the vocal cords is not possible. Some of those commonly used in the ED include:

- ‘BURP’ technique – refers to application of ‘backward, upward, rightward pressure’ on the larynx to facilitate visualization of the cords during laryngoscopy. It is important to understand how this differs from ‘cricoid pressure’ which is applied in order to prevent aspiration
- Bougie or tracheal tube introducer – long, thin, flexible device inserted under the epiglottis during laryngoscopy. As it enters the trachea, “clicks” are felt as the bougie passes over tracheal rings, and it STOPS when it reaches a mainstem bronchus. If esophageal, no clicks are felt and the bougie advances into stomach. Once in trachea, advance ET tube over bougie.
- Video laryngoscopy (Glidescope) – a laryngoscope with a camera mounted on a more sharply-angled blade allows for improved visualization of the anterior larynx

SUMMARY

- Most patients' airways can be managed, at least temporarily, with simple airway maneuvers and a bag-valve mask device
- Familiarize yourself with assessing an airway
- Emergency patients have a number of special considerations regarding airway management
- In any patient who fails to improve, or who deteriorates, always start again with assessment and management of the airway

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2: BREATHING

Heather Sues McKay
Updated by Martin J. Horak

OBJECTIVES

1. To develop an organized approach to breathing problems
2. To use the history and physical examination to help identify the cause of breathing problems
3. To understand the utility of various investigations for breathing problems
4. To know the various treatment modalities related to breathing problems

INTRODUCTION

After airway, the next priority in resuscitation (ABC's) is assessment and management of breathing problems. The label 'Breathing' encompasses all problems related to shortness of breath (SOB) and respiratory dysfunction, and these are among the most common clinical problems encountered in the Emergency Department (ED).

Airway and breathing problems can be difficult to distinguish from each other initially, and are frequently assessed in tandem. Of course, airway management always comes first. 'Breathing' comes before 'Circulation' in resuscitation because there is no point in working on the pump part of the equation unless that pump is delivering oxygenated blood to the tissues.

APPROACH

The causes of respiratory distress or dyspnea are myriad. Rather than learn long lists of possible diagnoses, it is better to have a clear approach in which to organize all the information gathered from your history and physical exam. However, the following is a short list of immediately life-threatening diagnoses that must be rapidly identified and treated:

- Pulmonary Embolus
- Pulmonary Edema (CHF)
- Acute exacerbation of COPD
- Acute severe Asthma
- Tension Pneumothorax

Mnemonic: Breathing Poorly Can Cause A lot of Tension

After considering these immediately life-threatening diagnoses, an anatomical approach can be used to identify other causes of breathing difficulties:

Bronchi and Bronchioles

Asthma, COPD, Bronchiectasis

Lung Parenchyma

The etiologies listed with clinical examples cause problems by filling or blocking the alveoli and thus preventing gas exchange. Alveoli can be blocked by pus (infection), fluid (edema), blood and gastric contents (aspiration).

Blood: Pulmonary Contusion, Goodpasture's Syndrome, Bleeding Carcinoma

Fluid/Edema: CHF, ARDS, Neurogenic Pulmonary Edema, Toxin/Drug Induced Pulmonary Edema, High Altitude Pulmonary Edema

Pus/Infection: Bacterial Pneumonia, TB, Fungal

Gastric Contents: Aspiration

Diffusion Diseases: Amyloidosis, Interstitial Pulmonary Fibrosis

Vasculature and Blood

This category includes blockage of the pulmonary circulation and disorders of the content/chemistry of the blood.

Embolii: Clot, Fat, Air, Amniotic Fluid

Metabolic: Acidosis, Thyroid disease

Anemia

Methemoglobinemia

Pleural Space

The pleural space is a potential space between the lung pleura and the chest wall, usually devoid of any significant fluid/substance. Accumulation of exogenous material in the pleural space impedes normal respiratory function.

Air: Tension Pneumothorax, Simple Pneumothorax

Blood: Hemothorax

Fluid: Pulmonary Effusion

Pus/Infection: Empyema

Chest Wall & Diaphragm

When the chest wall, intercostal musculature or diaphragm is either damaged or non-functioning, the result is breathing impairment. Trauma, neurologic disease and congenital deformity are potential culprits.

Trauma: Flail Chest, Spinal Cord injury,

Diaphragmatic Rupture

Neurogenic Causes: Guilliane-Barré, Myasthenia Crisis and ALS

Congenital: Kyphosis, Scoliosis

Cardiac Causes

While many cardiac causes of dyspnea cause pulmonary edema, some cardiac disease increase pulmonary vascular pressures and decrease lung compliance, thus producing dyspnea. These include:

Myocardial Infarction

Cardiac Tamponade

Valvular and Congenital Heart Disease

Central Causes

Hypoventilation: over-sedation or CO₂ retainers

Fever

Psychogenic/Anxiety

HISTORY

Try to ascertain, even in the sickest patients, some historical features of the disease process. Important features of the history are:

- Onset of Symptoms
- Progression of Symptoms
- Severity of Symptoms
- Presence of Associated Symptoms – especially chest pain, fever, cough
- Exposure to Noxious Substances
- Exposure to Allergens
- Possible FB ingestions
- Past Medical History: This is particularly important as many respiratory and cardiac diseases like asthma, COPD, and CHF have a recurrent course.
- Risk Factors: It is also imperative to assess for the risk factors of such diseases as ischemic heart disease and pulmonary embolus.

PHYSICAL EXAMINATION

The physical exam can be very revealing and is based on the classic components of the physical

examination: inspection, palpation, percussion, and auscultation.

Once the airway is controlled, the rate and pattern of breathing are important clues to underlying diseases. Tachypnea is usual for most conditions - both intrapulmonary and extrapulmonary. Bradypnea is classic of opiate intoxication (as well as some, usually catastrophic, CNS events). Certain patterns of breathing, (eg. Kussmaul's or apneustic breathing) may be indicators of metabolic and neurogenic causes of respiratory dysfunction. Both hypoxia and hypercarbia may cause agitation, anxiety, and obtundation. Carefully observe the mechanics of breathing such as chest expansion, accessory muscle use, paradoxical breathing, indrawing, and number of words spoken per breath (if applicable). These signs indicate significant respiratory dysfunction and the need for prompt treatment. Cyanosis is a late and ominous sign (except in chronic intrapulmonary and intracardiac shunts). Look for surgical scars over the chest as clues to underlying pulmonary disease and impairment.

Palpation may reveal subcutaneous emphysema over the neck or chest, suggesting pneumomediastinum or pneumothorax. Check the position of the trachea; if it is not midline then something is causing it to shift, such as air, fluid or a mass lesion in the chest. Percussion helps to define what this could be. Hyperresonance is due to air and pneumothorax (+/- tension) is the likely cause. Percussion that is dull may be due to a pleural effusion or a hemothorax.

Auscultation may reveal normal, absent, or diminished breath sounds that help to delineate some of the underlying causes of respiratory dysfunction. Wheezing may be due to bronchospasm secondary to asthma, COPD, CHF or aspirated foreign body. Crackles may indicate CHF, pneumonia or chronic underlying lung pathology. Pleural friction rubs suggest pneumonia or pulmonary embolism.

Although clinical assessment of respiratory function is invaluable, adjunctive tests are often employed. These tests include pulse oximetry, blood gas determination, and pulmonary function testing.

PULSE OXIMETRY

Pulse oximetry provides continuous, immediate and non-invasive assessment of arterial oxygenation. It is of great value at the bedside in rapidly determining the patient's oxygenation status, and usually obviates an immediate need for blood gas testing. Pulse oximetry measures hemoglobin saturation, rather than

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pO₂, via spectrophotometric determination of the relative proportions of oxygenated versus deoxygenated hemoglobin in blood coursing through an accessible pulsatile capillary bed (usually the nailbed). Using the oxyhemoglobin dissociation curve, it is possible to estimate the pO₂ for any given oxygen saturation. An SaO₂ of 90% equals a pO₂ of 60 mmHg. Below this level of saturation you have hit the steep portion of the curve and pO₂ drops off precipitously. For this reason, we strive to keep the oxygen saturation well above 90%.

The accuracy of pulse oximetry is dependent on adequate pulsatile blood flow. Therefore, shock states, severe anemia, hypothermia, and use of vasopressor agents impairs accurate measurements. Jaundice, skin pigmentation and nail polish may also interfere with readings.

INVESTIGATIONS

The following investigations are valuable adjuncts to the assessment of the respiratory status of a patient:

- CBC- looking for evidence of infection or severe anemia
- Electrolytes- looking for evidence of anion gap acidosis
- Cardiac Enzymes- in patients with risk factors for ischemia
- D-dimer- frequently used to *rule out* the diagnosis of pulmonary embolism
- CXR- visualizes many forms of lung pathology
- Blood Gas - to assess oxygenation and ventilation

Arterial or Venous Blood Gases

Blood gases are a useful adjunct for a precise assessment of respiratory function, notably providing information on the adequacy of alveolar oxygenation (pO₂), ventilation (pCO₂), the acid-base status of the patient, and whether the respiratory condition is acute or chronic. Venous blood gases (VBG) provide a close approximation of pH, CO₂ and bicarbonate to the arterial blood gas. While arterial blood gases are slightly more accurate, they cause a great deal of pain to the patient and require more time to perform. Therefore a VBG is often measured first and may be sufficient in the clinical assessment.

Pulmonary function tests (PFT)

The most commonly used PFT in the ED is peak expiratory flow rate (PEFR). This is easily measured with a hand held peak flow meter, in the patient who is co-operative, to assess the severity of airflow limitation and response to treatment in asthma and

COPD. Forced expiratory volume in one second (FEV1) is another test sometimes used for this purpose.

ACUTE RESPIRATORY FAILURE

This is defined as hypoxia (pO₂<50 mmHg) with or without associated hypercapnia (pCO₂>45 mmHg). It is divided into two types:

Type I: respiratory failure without pCO₂ retention. This is characterized by marked V/Q mismatch and intrapulmonary shunting. Examples include diffuse pneumonia, pulmonary edema, ARDS.

Type II: respiratory failure with pCO₂ retention. This involves V/Q mismatch and inadequate alveolar ventilation. There are two categories of this type of respiratory failure:

- A.** Patients with intrinsically normal lungs but with inadequate ventilation due to disorders of respiratory control (e.g. overdose, trauma, CNS disease), neuromuscular abnormalities (e.g. muscular dystrophy, Guillain-Barre, myasthenia), and chest wall trauma.
- B.** Patients with intrinsic lung disease with V/Q mismatch and alveolar hypoventilation. Respiratory failure is precipitated by additional clinical insult, usually infection, which worsens the underlying disease. Examples include COPD, asthma, cystic fibrosis.

INDICATIONS FOR INTUBATION

1. Airway Protection

- decreased level of consciousness (ie. CNS bleed or overdose)
- *general rule of thumb is “GCS Eight – Intubate”*
- prevent aspiration

2. Respiratory Failure

- this may be a clinical assessment with bedside adjuncts such as pulse oximetry (blood gases NOT necessary to proceed to intubation)
- examples include hypoxic OR hypercarbic failure

3. Anticipated Course (Prophylactic Intubation)

- airway burn or significant neck trauma (airway compromise likely)
- ill patient that is CT or O.R.-bound
- transfer of critically ill patient to another facility

SPECIFIC TREATMENT MODALITIES

Nasal Prongs

Nasal prongs are usually a well-tolerated method of administering oxygen to the spontaneously breathing patient. With O₂ flows of 2-6 L/minute FiO₂ of 25-40% can be attained.

Face Mask

Use of a face mask requires a spontaneously breathing patient and can deliver up to 50-60% FiO₂ with a flow rate of 10L/minute. This FiO₂ may vary depending upon how well the mask fits, and what the patient's minute ventilation is; i.e. how much room air is entrained through the mask

CLINICAL PEARL

In the severely dyspneic and hypoxic patient in whom you believe you can treat the underlying problem without intubation and ventilation, (the classic example being the CHF patient who has been given diuretics and afterload reducers) one can use a double-flush system. With this system a face mask is hooked up to two O₂ outlets, thus delivering up to 30L/minute of pure O₂ and FiO₂ of nearly 100%.

Oxygen Reservoir Mask

Oxygen reservoir mask is essentially the same as the above set-up, except the mask has an attached inflatable bag that stores O₂ during expiration and from which O₂ is inspired. With a tight fit and low entrainment, FiO₂ of up to 90% can be obtained with O₂ flow of >10L/minute.

Bag-valve Mask Devices

These masks can be used to manually supplement the patient's respiratory effort in patients who are breathing spontaneously, but require respiratory assistance. The mask comes in various styles with the most common being the 'AMBU bag'. It consists of a rubber or inflatable plastic facemask, a connector bag

which contains O₂, and an O₂ reservoir attached to the bag and to the O₂ outlet. These devices can deliver up to 100% O₂ with high flow O₂ and proper bagging procedure. If tolerated, an oral or nasal airway can help facilitate ventilation of the patient.

Bag-valve mask ventilation can temporize patients in respiratory arrest until other therapeutic modalities take effect. However, the majority of patients needing this type of intervention will require intubation and mechanical ventilation. The decision to mechanically ventilate the patient in the ED is usually a clinical one. For patients in severe respiratory distress, do not wait for the blood gas to confirm what you should already know.

CPAP Masks/ BiPAP Masks

CPAP (continuous positive airways pressure masks) are a therapeutic modality option being increasingly used to treat patients in respiratory distress. The commonest and most studied uses are in the patient with CHF or severe COPD. This non-invasive mechanical ventilation temporizes the need for intubation, and may reduce the incidence of patients that need invasive respiratory support.

Other Therapeutic Modalities

Needle thoracostomy can relieve tension pneumothorax prior to chest tube insertion. Tube thoracostomy can relieve pneumo-/hemo-thoraces and drain pleural effusions.

Pharmacologic Therapy

Certain medical therapies may assist in specific diseases. Examples include bronchodilation (ie. salbutamol) in asthma/COPD, diuretics in CHF, antibiotics in pneumonia, anticoagulation/thrombolysis in MI/PE.

Summary

The prompt recognition of respiratory dysfunction, including the respective clinical signs and adjunctive testing, is critical in the ED. Knowledge of specific oxygenation/ventilation and pharmacologic therapies is paramount to prevent further clinical deterioration.

3: CIRCULATION

Joel Yaphe

OBJECTIVES

1. To recognize shock utilizing the physical examination
2. To understand the causes of shock
3. To review the management of different types of shock

INTRODUCTION

The circulatory system exists in order to supply cells with oxygenated blood and nutrients, and to remove waste products. Shock is defined as 'an abnormality of the circulatory system causing inadequate tissue

perfusion which, if not corrected, will result in cell death.'

CAUSES OF SHOCK

The circulatory system consists of two pumps connected in series (right and left heart), a system of conduits (blood vessels), and circulating fluid (blood). The causes of shock can be understood by looking at the various components of the circulatory system, and the disorders that affect them. The following table lists some of the circulatory disorders that may result in shock.

Table 1. Causes of Shock

COMPONENT	Causes of Shock		DISORDER
PUMPS (Cardiogenic)	Right Heart	Inflow obstruction	<ul style="list-style-type: none"> Pericardial Tamponade Tension Pneumothorax
		Chamber	RV failure (ischemic, secondary to LV failure)
		Outflow obstruction	Pulmonary Embolus
	Left Heart	Chamber	LV failure (ischemia, cardiomyopathy)
		Valves	Papillary muscle rupture
		Arrhythmias	<ul style="list-style-type: none"> Bradycardia Tachycardia
VESSELS (Vasogenic, Distributive)			<ul style="list-style-type: none"> Septic Shock Anaphylactic Shock Neurogenic Shock
BLOOD (Hypovolemic, Distributive)		Hypovolemic Shock (haemorrhage, dehydration) Toxins (carbon monoxide, cyanide)	

The mnemonic "SSSHOCK" commonly used for remembering the causes of shock can be reviewed in chapter 43.

RECOGNIZING SHOCK

Shock has many causes, and the clinical presentation varies. However, many features of hypoperfusion can be easily recognized by the bedside examination of the patient.

Mental Status

Early: Agitation due to increased sympathetic tone
Late: Obtundation due to decreased CNS perfusion

Pulse

Tachycardia is generally sensitive for acute losses in excess of 12-15% blood volume. Exceptions occur in primary bradyarrhythmias, in patients on beta blockers, and in some cases of intraperitoneal bleeding.

The presence of palpable pulses may give a rough indication of systolic blood pressure. If the radial pulse is palpable, systolic pressure exceeds 80mm Hg. If the femoral pulse is palpable, systolic pressure exceeds 70mm Hg. If the carotid pulse is palpable, systolic pressure exceeds 60mm Hg.

Blood Pressure

Hypotension is an insensitive marker for tissue hypoperfusion. In the case of hemorrhagic shock, a fall in blood pressure may not occur until there is blood loss in excess of 30% of total blood volume. (Similarly, hypotension can occur without shock.)

Orthostatic Vital Signs

An assessment of the change in pulse and blood pressure as a patient is moved from a supine to sitting or erect position has been used to identify mild

degrees of hypovolemia. There is no consensus as to what changes constitute a ‘positive response’ and the test is insensitive and nonspecific in the assessment of volume status. Orthostatic vital signs should never be performed in a potentially unstable patient.

Respiratory Rate

Tachypnea occurs in response to increased sympathetic tone and metabolic acidosis. It is an early sign of hypovolemic shock. The tachypneic response may be blunted in response to CNS depressants or head trauma.

Skin

The skin is cool and pale early on as blood is shunted to vital organs. Peripheral cyanosis may appear later. The exception to this rule is in vasogenic shock, when the skin may be warm and possibly flushed due to peripheral vasodilatation. In later stages of vasogenic shock, depression of cardiac output may cause the usual changes of skin hypoperfusion to become manifest.

Capillary Blanch Test: A positive test occurs when a compressed nail bed takes >2 seconds to ‘pink up’ and is said to occur when there is acute blood loss in excess of 15% of total blood volume.

Heart Sounds

Muffled heart sounds may be noted in cardiac tamponade.

Jugular Venous Pressure

Low: Hypovolemia, Sepsis
High: Left Ventricular Failure, Right Heart Problem

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Table 2. Assessment of Hemorrhagic Shock According to Presentation

	Class I	Class II	Class III	Class IV
Blood Loss (ml)	≤ 750	750-1500	1500-2000	≥ 2000
Blood Loss (%BV)	$\leq 15\%$	15-30%	30-40%	$\geq 40\%$
Pulse Rate	<100	>100	>120	≥ 140
Blood Pressure	Normal	Normal	Decreased	Decreased
Pulse Pressure (mmHg)	Normal	Decreased	Decreased	Decreased
Capillary Refill Test	Normal	Positive	Positive	Positive
Respiratory Rate	14-20	20-30	30-40	>35
Urine Output (ml/hr)	≥ 30	20-30	5-15	Negligible
CNS-Mental Status	Slightly anxious	Mildly anxious	Anxious, confused	Confused, lethargic
Fluid Replacement (3:1 rule)	Crystallloid	Crystallloid	Crystallloid + Blood	Crystallloid + Blood

BEDSIDE ULTRASOUND

ED ultrasound can be a useful tool in identifying the cause of shock. It can rapidly detect intraabdominal hemorrhage, hypovolemia (IVC filling), pericardial tamponade, or RV dysfunction (PE).

GENERAL MANAGEMENT

ABCs + Monitoring

- Airway and breathing assessment occurs first
- Oxygen and saturation monitor
- Cardiac monitor
- Intravenous access and send blood to lab (crossmatch if hemorrhage is suspected); Lactate
- Control any external bleeding by applying pressure to the wound
- Foley catheter - monitor urine output
- Ongoing assessment of clinical parameters of tissue perfusion include:
 - Blood pressure, pulse, respirations, level of consciousness, skin
 - Invasive Monitoring: CVP measurement, Arterial line, ScvO₂

SPECIFIC MANAGEMENT

Treatment of Hemorrhagic Shock

- ABCs
- If active control of internal hemorrhage is needed, consult surgery (or occasionally GI) while you resuscitate - don't wait until the patient is stabilized and the blood work is back
- Restore circulating blood volume: Prompt restoration of circulating blood volume is felt to be a critical factor in the reversal of shock. In hemorrhagic shock, as in most other types, fluid resuscitation begins with aggressive intravenous infusions of (warmed) crystalloid. Use at least two short, large bore catheters (flow is inversely proportional to the length of the catheter, and proportional to the 4th power of the catheter radius). Pressure infusion devices may be used to increase flow rates. The chart above provides some guidelines for appropriate fluid management.

Peripheral access (preferred):

Equipment: 16-gauge angiocath or larger

Sites: forearm, antecubital

Central access:

Equipment: 8FR introducer inserted via Seldinger technique

Sites: femoral vein, internal jugular vein, subclavian vein

Fluids:

- Ringers lactate or normal saline
- 1-2 litres administered rapidly (20ml/kg in children)
- Expect to need approximately 3 times the estimated blood loss (3:1 rule)

Blood (packed RBC's):

- if no response or transient response to 2-3L fluids

Platelets and FFP

- In patients with significant blood loss, early transfusion of platelets and FFP may improve outcome. Many institutions have a "massive transfusion protocol".

Adequacy of fluid resuscitation is assessed by following the clinical parameters of tissue perfusion as well as urine output. Measurement of central venous pressure may be helpful. Adequate volume replacement is important, but administration of volume in excess of need is harmful. Watch for development of pulmonary edema (cardiogenic and non-cardiogenic).

Under investigation:

- Delayed volume resuscitation in acute hemorrhagic shock - immediate surgery

SPECIFIC MANAGEMENT NON-HEMORRHAGIC SHOCK

Anaphylactic Shock

Epinephrine (IM; IV if cardiovascular collapse)

Intravenous crystalloid

Antihistamines (H1 and H2 blockers)

Corticosteroids

Wheezing: Nebulized beta₂ agonists

Stridor: Nebulized epinephrine

Cardiogenic Shock

Inotropes, intra-aortic balloon pump, emergency angioplasty

Tension Pneumothorax

Needle thoracostomy followed by chest tube

Septic Shock

Intravenous crystalloid

Antibiotics

Goal directed therapy in the ED decreases mortality in sepsis: urine output >0.5 mL/kg/h

CVP 8 to 12 mm Hg

MAP 65 to 90 mm Hg

ScvO₂ >70%

Definitive therapy (drainage of closed space infections, surgery).

Cardiac Tamponade

Intravenous crystalloid, pericardiocentesis

Massive Pulmonary Embolus

Intravenous crystalloid, inotropes, thrombolysis or surgery

Arrhythmias

Specific anti-arrhythmic therapy

SUMMARY

- The causes of shock can be understood by looking at the various components of the circulatory system, and the disorders that affect them (pumps, vessels, blood)
- Many features of shock can be easily identified on physical examination by assessing mental status, pulse, blood pressure, respiratory rate, skin, jugular venous pressure and capillary refill
- Hemorrhagic shock can be classified into 4 categories depending on the estimated amount of blood lost and some of the above mentioned physical examination findings
- In fluid resuscitation of hemorrhagic shock, expect to need approximately 3 times the estimated blood loss (3:1 rule)
- It is important to rapidly identify the cause of shock and institute specific treatment as soon as possible

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4: ALTERED MENTAL STATUS & COMA

Brian Schwartz, Sara Pickersgill

OBJECTIVES

1. To understand the causes of Altered Mental Status (AMS)
2. To develop an approach to the AMS patient
3. To make an appropriate disposition in an altered patient

INTRODUCTION:

- altered mental status is a non-specific term referring to patients who have impaired responsiveness, or are unresponsive to external stimulation, and are either difficult to rouse or are unrouseable
- coma is a sleep-like state or “unrouseable unresponsiveness”
- lethargy, stupor and obtundation refer to the states between alertness and coma

Altered mental status is an acute, life-threatening emergency; it requires rapid intervention to preserve brain function and life.

CAUSES OF ALTERED MENTAL STATUS AND COMA:

A decrease in level of consciousness is caused by either:

- Diffuse CNS dysfunction (toxic-metabolic causes) majority of cases; these conditions impair oxygen or substrate delivery to the brain which alters cerebral metabolism or interferes with neuronal function; or
- Primary CNS disease or trauma – either:
 - Injury to bilateral cerebral hemispheres (diffuse trauma, ischemia); or
 - Structural injury to the reticular activation system (RAS) in the brainstem; caused by:
 - direct “hit” (hemorrhage, brainstem stroke)
 - indirectly from compression (tumour, subdural or epidural hematoma)

Pneumonic for possible causes of coma is “TIPS” AND “AEIOU”:

Trauma/Temperature

Infection (CNS or other)

Poisoning/Psychiatric

Space-occupying lesions/Stroke

Alcohol/Acidosis

Epilepsy (nonconvulsive status epilepticus, post-ictal state)/Endocrine

Insulin (hypoglycemia, hyperglycemia)

Oxygen (hypoxia)

Uremia

EMERGENCY EVALUATION AND MANAGEMENT OF AMS AND COMA

1. Initial Approach – ABCD:

1. Airway:

- if patient is deeply comatose, endotracheal intubation needs to be considered.
- indications for intubation include in the altered LOC patient include:
 - Airway protection in reduced LOC, GCS 8 or less
 - The need to provide positive pressure ventilation, poor oxygen saturation despite supplemental oxygen
 - Predicted deterioration, i.e. in cases of shock or head injury with signs of increased intracranial pressure

2. Breathing:

- supplemental oxygen should be given on all patients with AMS

3. Circulation/C-spine:

- adequate cerebral perfusion is critical in AMS patients; maintain systemic blood pressure at a mean arterial pressure (MAP) of at least 80 mm Hg
- ensure adequate IV access to facilitate above
- -ensure C-spine stabilization in potential trauma patient

4. Drugs:

- administer appropriate universal antidotes (“DON’T”):

Dextrose: one ampule D50W if low blood sugar

Naloxone: 0.4 – 2mg IV or IM if opiate use is suspected, (often lower initial doses are used)

Thiamine: 100mg IV if history of alcohol abuse or malnourished

Note: Flumazenil (a benzodiazepine antagonist) is not indicated in the treatment of an undiagnosed coma patient

2. Vital Signs:

- extreme hypertension may suggest hypertensive encephalopathy or hypertensive intracerebral/cerebellar/brainstem hemorrhage
- hypotension may reflect sepsis, hypovolemia, cardiac failure or drug ingestion
- hyperthermia usually signifies infection but consider heat stroke and Ingestions
- hypothermia may be caused by exposure or secondary due to adrenal failure, hypothyroidism, sepsis or drug/alcohol intoxication

3. Rapid Neurological Exam:

- A 3-5 minute focused neurological exam will provide enough information to determine whether the cause of AMS/coma is metabolic or structural
 - Level of Consciousness (GCS) – this is a useful index of the depth of impaired consciousness and is used for prognosis and communication among pre-hospital staff and trauma/neurosurgical consultants. *GCS does not aid in diagnosing coma.* GCS should be determined initially and repeated at regular intervals. A drop in the GCS score means a deteriorating condition and increased intracranial pressure secondary to an expanding mass lesion must be ruled out.

Table 1: Glasgow Coma Scale

EYE OPENING	Score
-spontaneous	4
-to voice	3
-to pain	2
-none	1

VERBAL RESPONSE	Score
-appropriate, oriented	5
-confused, disoriented	4
-syllables, expletives	3
-grunts	2
-none	1

MOTOR RESPONSE	Score
-spontaneous, obeys commands	6
-localizes pain	5
-withdraws to pain	4
-decorticate flexion	3
-decerebrate extension	2
-none	1

- Head and Neck:

- Examine scalp for lacerations, contusions, deformities

- Signs of basal skull fracture (check tympanic membranes for CSF leak or hemotympanum, nares for CSF leak, “raccoon eyes” and “battle sign”)

- Flex neck if trauma has been ruled out to look for meningismus

- Cranial nerve exam:

- Check pupils for reaction and symmetry (with the exception of certain toxic syndromes, pupils are typically spared in metabolic and toxic syndromes)
- Extraocular movements (have patient follow a finger if awake enough, Doll’s eye maneuver if no trauma)
- Check fundi for papilledema (late sign of increase ICP)

- Motor and Sensory:

- Ask patient to move each limb or give painful stimulus; record best response and any asymmetry
- Painful stimuli include nail bed pressure and sternal pressure
- Check reflexes for asymmetry

- With rare exceptions, focal findings means a structural lesion. In particular, a dilated pupil on one side with weakness of the contralateral limbs indicates an expanding mass lesion on the side of the larger pupil. This occurs when the mass lesion is supratentorial and pushes or herniates the uncus of the temporal lobe through the tentorium (called “coning”). The oculomotor nerve, which runs adjacent to this, is compressed causing paralysis of the constrictor mechanism of the ipsilateral pupil.

4. General Examination:

- feel the skin: warm, dry, diaphoretic
- smell breath: can be a clue to cause of altered LOC
- inspection/ skin exam: rashes (meningitis, sepsis), track marks, jaundice, evidence of trauma
- examination of the lungs, heart and abdomen (masses, bowel sounds) may provide clues to other organ system disease

5. Ancillary History:

- obtained from family, friends, police, paramedics, old chart, etc.
- onset and progression:
 - abrupt onset suggests CNS hemorrhage/ischemia or cardiac etiology
 - progression over hours/days suggests progressive CNS lesions or metabolic-toxic causes

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- preceding events:
 - patient's baseline LOC preceding deterioration
 - antecedent trauma, fever, seizure activity
- past medical history (comorbidities ie. COPD, cirrhosis, similar prior episode, mental health history, drug use)

6. Diagnostic Testing:

- the goal of diagnostic testing in a comatose patient is to identify treatable conditions (infection, metabolic abnormalities, seizure, intoxications/overdose, surgical lesions)
- because neurologic recovery is reliant on early treatment, testing must proceed rapidly alongside, and in response to clinical evaluation.
- examples of clinical clues which should prompt emergent diagnostic testing include:
 - papilledema or focal neurologic deficits suggesting a structural etiology require an emergent CT scan
 - fever suggesting bacterial meningitis or viral encephalitis mandates and urgent lumbar puncture and initiation of antibiotics

CLINICAL PEARLS

Lumbar punctures in a comatose or AMS patient with focal findings should not be performed until an expanding mass lesion with increased intracranial pressure has been ruled out, as there is a risk of precipitating herniation of the brain stem through the tentorium.

Other investigations include:

- rapid blood sugar, CBC, electrolytes, Cr, BUN, LFT's, glucose, serum osmolality, ABG's, INR/PTT, troponin, urinalysis
- ECG, CXR, EEG

DISPOSITION OF ALTERED MENTAL STATUS AND COMA:

- patients with readily reversible causes of coma, such as insulin-induced hypoglycemia, may be discharged if treatment is initiated, the patient returns to baseline mental status, the cause of the episode is clear, and the patient has reliable home care and follow-up
- in all other cases, admission is warranted for further evaluation and treatment

- transfer patient if appropriate level of care not available

SUMMARY OF ALTERED MENTAL STATUS AND COMA:

- causes of coma are diverse and include structural brain lesion and systemic disease; remember TIPS and AEIOU
- stabilization of airway, breathing and circulation is the top priority; endotracheal intubation may be indicated to protect the airway
- remember to maintain C-spine precautions in suspected trauma patients
- readily reversible causes such as hypoglycaemia, hypoxia and opiate overdose should be considered (use universal antidotes "DON'T" as appropriate)
- repeat GCS at regular intervals
- if elevated ICP is suspected, consult neurosurgery urgently
- if meningitis is suspected, initiate appropriate antibiotics immediately

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5: SYNCOPE

Eric Letovsky

OBJECTIVES

1. To understand what the important historical features, physical examination findings and diagnostic tools are for syncope
2. To know the most common causes of syncope
3. To make disposition decisions for patients with syncope

INTRODUCTION

Syncope is defined as a transient, self-limited loss of consciousness with an inability to maintain postural tone, followed by spontaneous recovery. It must be differentiated from seizures, coma, shock, or other states of altered consciousness. Patients presenting with syncope represent one of the most difficult diagnostic challenges facing Emergency Physicians. The problem is not insignificant; up to 3% of all Emergency Department (ED) visits, and up to 1% of all hospital admissions are accounted for by patients with syncopal episodes. The problem is that the spectrum of diseases that may cause syncope spans from common benign conditions to severe life threatening disorders.

It can be particularly difficult trying to distinguish a true syncopal attack from a seizure. As a general rule, if there is evidence of soft tissue injury at multiple sites (due to vigorous tonic clonic movements), if the event lasts more than 4 or 5 minutes, it is unlikely to be syncope and more likely to be a seizure. The corollary is that syncopal episodes themselves can also produce some jerking or twitching of extremities (because of cerebral hypoperfusion) which could mimic a seizure.

One of the other entities that sometimes gets confused with true syncope is a drop attack. A drop attack can be differentiated from true syncope by no loss of consciousness. A drop attack can be a sign of a TIA (vertebrobasilar insufficiency).

How often can a cause of syncope be established?

A definite cause of syncope can be established in only about 50% of the cases.

What are the best diagnostic tools?

The history and physical examination are the most helpful in establishing a cause of syncope. A 12-lead ECG is indicated in most patients with syncope and is the most useful test.

Initial laboratory studies (e.g. CBC, electrolytes) are rarely abnormal, and rarely establish a cause for the syncope. CT scans rarely establish a cause of syncope.

What are the most common causes of syncope?

Of all patients with syncope, approximately half of the patients may have an etiology - 1/4 due to cardiovascular causes, 1/4 due to non-cardiovascular causes. In the remainder of the patients, no cause can be established.

CARDIOVASCULAR CAUSES OF SYNCOPE

Ventricular Tachycardia
Myocardial Infarction
Sick Sinus Syndrome
Pacemaker Malfunction
Aortic Stenosis
Carotid Sinus Syncope
Supraventricular Tachycardia
Pulmonary Embolism
Heart Blocks
Pulmonary HTN

NON-CARDIOVASCULAR CAUSES OF SYNCOPE

1. Situational Syncope – reflex mediated mechanism and occurs during or immediately after coughing, micturition, defecation or swallowing. (the reason lots of folks have syncopal episodes in washrooms!)
2. Orthostatic Syncope: Diagnosis of Exclusion!
Can be associated with:
 - a. decreased intravascular volume (diarrhea, diuretics)
 - b. anemia
 - c. drug effects (vasodilators, opiates etc)
 - d. autonomic insufficiency (Parkinson's, DM)
3. Vasovagal syncope: is often associated with a sensation of increased warmth and may be

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accompanied by nausea. It may occur after exposure to an unexpected or unpleasant sight, sound or smell, fear, severe pain, emotional distress and instrumentation. It may also occur in association with prolonged standing or kneeling in a crowded or warm place.

What is the prognosis of patients with syncope?

Table 1. 12 Month Prognosis for Patients Presenting with Syncope

Group	Mortality	Incidence of Sudden Death
Cardiac related	30%	24%
Non cardiac related	12%	4%
Unknown causes	3%	3%

Patients with a cardiovascular cause of syncope tend to be male, older, with frequent history of ventricular arrhythmias, congestive heart failure, atrial fibrillation, and left ventricular hypertrophy. In addition, patients with a non-cardiovascular cause of syncope who die suddenly usually have a serious underlying disease. It is clear that the history and physical exam are the most useful aids in establishing a cause of syncope.

GUIDELINES FOR ASSESSMENT

History

- Presyncopal activity and position
- Presyncopal associated symptoms
- Precipitating factors for vasovagal syncope (e.g., mild trauma with pain, sight of blood, fear)
- Estimated DWP (duration of warning)
- Estimated loss of consciousness
- Reports from witnesses (including convulsions, loss of urine)
- Post syncopal residua
- Family history (unexplained death, deafness, congenital heart disease, metabolic disorders, MI at young age)
- Medications (certain drugs can cause prolongation of QT interval and predispose to Torsades de Points)

One of the most important pieces of information that should be obtained is an estimation of the duration of the warning period (DWP), either by the patient or by a witness. Brief DWP's are characteristic of cardiac, orthostatic, or micturition syncope. Patients with a vasovagal cause of syncope tend to have a longer prodrome before they pass out.

Physical

- Orthostatic Blood pressure/Pulse
- CVS exam (listen carefully for murmur of aortic stenosis)
- CNS exam
- Stool for OB

CLINICAL PEARL

About 20% of adults have an 'orthostatic drop'. This fact makes measuring the orthostatic blood pressure an indiscriminatory test for syncope.

WHAT'S NEW

A recent study identified several risk factors for short-term poor outcomes in patients with syncope: an abnormal ECG, concomitant trauma, absence of symptoms of impending syncope and male gender.

Investigations

ECG

ECG Monitoring (15-20% yield)

The ECG in syncope: what to look for:

- Arrhythmias (e.g., bradycardia, atrial fibrillation)
- Heart blocks
- Interval abnormalities: short PR segment (sign of WPW or other bypass diseases), QRS prolongation, prolonged QT interval (risk factor for Torsades de Points).
- ECG findings of PE: sinus tachycardia, S1Q3T3 pattern.
- Signs of ischemia (ST deviations)

See attached ECG's 1 and 2.

Predicting Adverse Outcomes

Overall, the 30 day incidence of death in patients presenting with syncope is low. The San Francisco Syncope Rule is a risk stratification tool derived and

validated to predict short term outcomes. It uses 5 features to predict serious outcome:

1. History of CHF
2. Low Hematocrit
3. Abnormal ECG
4. History of Dyspnea
5. Systolic BP < 90 at triage.

Causes of Sudden Death in Athletes

1. Hypertrophic cardiomyopathy (see below)
2. Congenital coronary anomalies
3. Arrhythmias (eg: prolonged QT syndrome)
4. Commotio Cordis (or Innocent Chest Blow)

Be very wary of syncope associated with exertion! You have to worry about hypertrophic cardiomyopathy - a heterogeneous genetic disorder that is seen in 1 of every 500 adults in the general population, and is the most common cause of sudden death during exercise. Patients with hypertrophic cardiomyopathy can remain asymptomatic throughout their lives; there is potential, however, for dyspnea, exercise intolerance, angina, syncope, and sudden death due to the mechanical effects of this disorder.

SUMMARY

A thorough history, detailed physical examination, and investigations limited to a 12 lead EKG +/- 24 hour ECG monitoring, is a rational and cost effective

approach to patients with syncopal attacks, and should allow a diagnosis in about half of all patients. It is reassuring to remember that in those patients in whom a diagnosis cannot be made, the incidence of sudden death is no higher than the general population. Therefore, the great majority of patients can be discharged from the ED after a brief period of observation, with appropriate out-patient referral. Older patients with only a brief duration of warning, those patients in whom a cardiac etiology is suspected, and patients with a high San Francisco Syncope score should be considered for admission.

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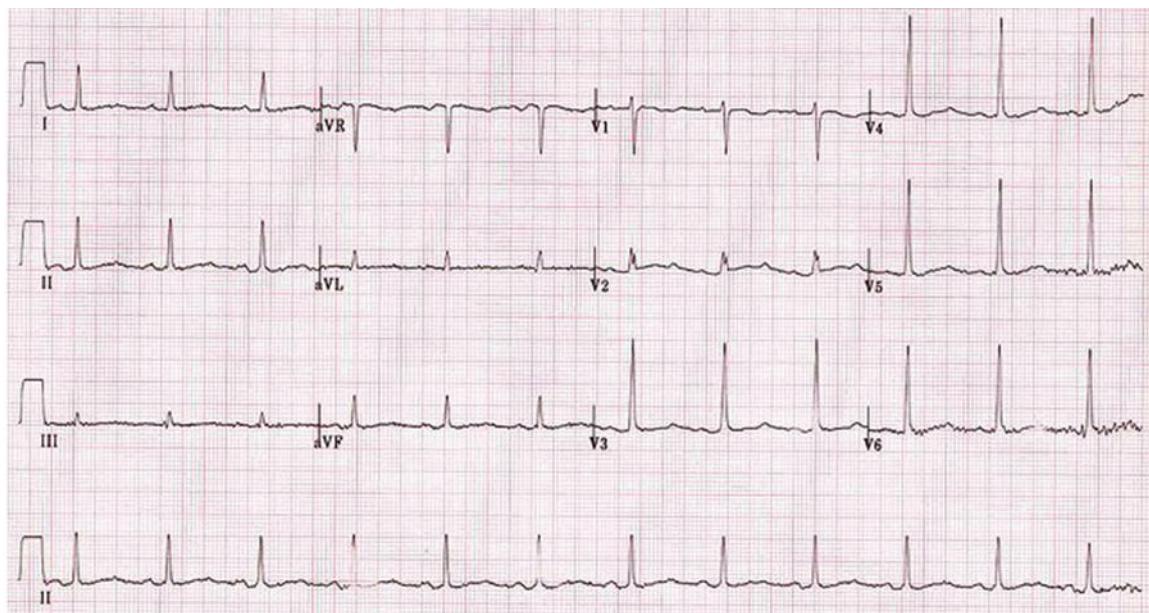
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50 year old, psychiatric history

Meds: quetiapine, potassium 2.6

Prolonged QT Interval: metabolic causes – hypokalemia, hypocalcemia, hypomagnesemia, medications- i.e. cardiac, psychiatric, cardiac causes- severe bradycardia, myocarditis, CNS causes- SAH, hypothermia

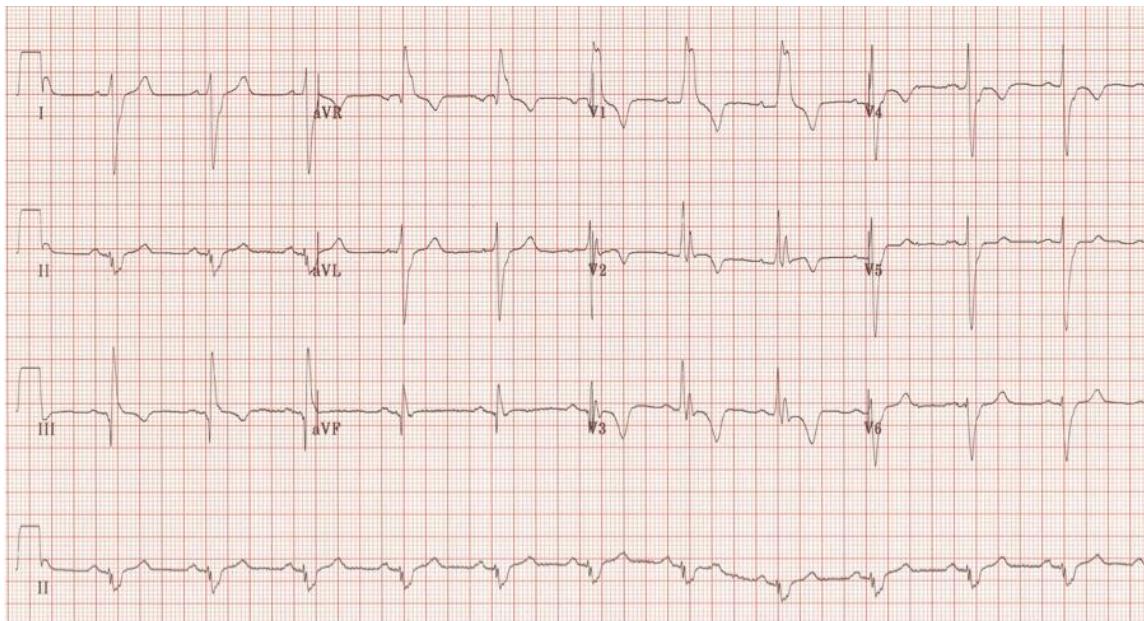
Prolonged QT : risk for torsades de pointes



60 year old man, syncopal episode caused by pulmonary embolus.

ECG findings seen in PE:

1. Sinus tachycardia
2. S1Q3T3: S wave in Lead 1, Q wave in lead 3, inverted T in lead
3. Inverted T waves in the precordial leads
4. RBBB indicating heart strain in large PE
5. Clockwise rotation with persistent S wave in V6, implies rotation of the heart secondary to right ventricular dilatation



6: HEADACHE

Dan Cass
Updated by Tom Kaul

OBJECTIVES

1. To review both common and dangerous causes of headache in the ED patient
2. To develop an approach to patients presenting to the ED with headache
3. To review therapy commonly instituted in the ED to relieve headache
4. To understand the workup and treatment of some of the serious causes of headache

INTRODUCTION

Headache is a frequent cause of Emergency Department visits. The challenge facing the Emergency Physician is to determine if the cause of the headache is relatively benign or due to a more sinister cause. Of patients presenting to the ER with headache, 50% have tension headaches, 10% migraines, 30% benign nonspecific headaches, and only 8% a headache emanating from a potentially serious cause. Less than 1% will have a life threatening reason for their headache such as a subarachnoid hemorrhage.

As with many topics in Emergency Medicine, it is helpful to consider the causes of headache under two headings: "the common" and "the deadly".

THE COMMON

The two most common causes of headache – migraine and tension-type headache - are, fortunately, the least dangerous. Even so, they may be quite disabling to the patient, either due to their severity, or to their frequency. There are now many therapies available to patients with these headaches, and often their pain may be relieved entirely in the Emergency Department.

Migraine

Migraine headaches are believed to result from changes in the reactivity of blood vessels inside and outside of the skull. Thus, they are often referred to as vascular headaches. "Classic migraine" involves a sensory or other aura which precedes the actual headache. "Common migraine" does not involve an aura. Because the diagnosis of migraine is so

dependent upon an appropriate history, it is vital to have a clear understanding of the typical symptoms of migraine:

- Usually gradual onset; may wake patient from sleep; some patients have sensory "aura" prior to headache
- Moderate to severe; often interferes with normal activities
- Typically unilateral (migraine literally means "half skull"); but, may be bilateral; rarely posterior/occipital
- Typically pounding or throbbing in character; may be constant
- May have associated nausea and vomiting, photophobia or phonophobia; less commonly neurologic symptoms before or during the headache
- May occur several times a week or very infrequently
- Often lasts 1-2 days; may last longer

The International Headache Society diagnostic criteria for migraine are shown below. Fulfilling all but one of these criteria is sufficient for the diagnosis of "probable migraine". Similar diagnostic criteria exist for "Classic Migraine with Aura".

International Headache Society Diagnostic Criteria for Common Migraine

A: At least 2 of the following:

1. Moderate or severe intensity
2. Pulsating quality
3. Unilateral location
4. Inhibits or prohibits daily activities

B: At least 1 of the following symptoms:

1. Nausea or vomiting
2. Phonophobia and photophobia

C: Attacks have occurred:

At least 5 times, AND
Lasting 4-72 h each

Triggers vary from patient to patient. Common triggers include: certain foods (alcohol [especially red wine], cheeses, chocolate, caffeine, MSG), stress, change in sleep patterns, oral contraceptives, certain phases of menstrual cycle, changes in weather)

Physical examination, aside from photophobia, is generally normal. In patients with “classic migraine”, focal sensory or other neurological findings may be present in the aura phase and may less frequently be present in the headache phase. The majority of migraine patients can tell you whether their current headache is “typical” for them.

A number of treatments have been tried for migraines and favored regimens vary regionally. Many medications are thought to work in migraine due to their actions on the central brain neurotransmitter receptors, particularly serotonin and dopamine. Indeed, the most effective migraine therapies act at least in part on these receptors. These include:

- Neuroleptics - chlorpromazine, haloperidol
- Dopamine antagonists – metoclopramide (maxeran), prochlorperazine (stemetil)
- Vasoactive medications – ergotamine (DHE), sumatriptan

In addition, non-neurotransmitter-based therapies can offer significant relief for many patients presenting with migraine-type headaches; these include anti-inflammatories (including ASA and NSAIDs,) as well as pure analgesics, such as acetaminophen and narcotics. An important point to stress is that, with such a broad array of therapies available, narcotics should be used only as a last resort in the treatment of the patient with migraine headache.

A common and evidence based therapeutic approach includes the use of 5-10 mg prochlorperazine, repeated once in 30-60 minutes if needed, with the addition of an intravenous NSAID such as ketorolac. Akathesias and other forms of EPS are the most prominent side effects to be wary of.

The use of steroids (such as dexamethasone) in migraine is controversial. Recent meta analyses show a benefit in prevention of migraine recurrence in the subsequent 24-48 hrs with a single dose of dexamethasone.

Tension-type Headache

These are the most common causes of headache. They may appear similar, in some ways, to the migraine; however, several key features help to differentiate them:

- Virtually never occur during sleep
- Often develop gradually over the course of a day
- May occur daily
- Often posterior/occipital
- Generally less disabling than a migraine

- significant association with stressors or prolonged work in one position (eg: typing)
- Rarely associated with nausea/vomiting, photophobia

The most effective therapy of tension (or muscular) headaches involves prevention, by addressing and modifying the triggers - stress management, properly designed workstations, etc. Acute headaches are treated with mild, generally non-narcotic analgesics and rest.

It is important to remember that there is overlap in the presentation of these two types of headaches; that is, patients with tension-type headaches may have some features typically associated with migraine headache and may respond to anti-migraine therapy, such as sumatriptan, and vice versa.

THE DEADLY

Life-threatening causes of headache are rare. However, they can be easily missed if the Emergency Physician does not take the approach that every headache is potentially deadly until proven otherwise. This “proof” generally comes in the form of an appropriately directed history and physical examination. The following are some key examples of life-threatening causes of headache, which should always be considered in any patient presenting to the emergency room with a chief complaint of headache.

CLINICAL PEARLS

Serious causes of non-traumatic headache

1. Subarachnoid Hemorrhage
2. Temporal Arteritis
3. Hypertensive Encephalopathy
4. Pseudotumour Cerebri
5. Carbon Monoxide Poisoning
6. Tumour
7. Meningitis/Encephalitis
8. Pre-eclampsia
9. Cervical Artery Dissection
10. Cerebral Venous Thrombosis
11. Acute angle closure glaucoma

Subarachnoid Hemorrhage

This is perhaps the most devastating cause of headache in the Emergency Department, mainly because missing the diagnosis has grave consequences. Unfortunately, the diagnosis is missed on initial presentation in 25-50% of cases.

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Features of such headaches include:

- Sudden onset
- Often occurs with exertion (exercise, intercourse)
- Usually severe “worst headache of my life”
- Most severe at onset
- Frequently associated with nausea and vomiting
- Look for: pupillary changes, meningismus, altered level of consciousness, focal deficits (NB: Initially may have normal neurological examination)

Any patient with a suspected subarachnoid hemorrhage (SAH) represents a true emergency. The danger with SAH is that of re-bleeding; the initial headache is often the warning of further events (“sentinel headache”).

Diagnosis often starts with CT scanning. Sensitivity of ever improving CT scans for SAH has long been a topic of controversy. A landmark paper (referenced at the end of this section) recently helped clarify this matter. Per this study, CT performed within 6 hours of onset of symptoms yielded 100% sensitivity for SAH when read by a qualified radiologist. However, the sensitivity of this tool decreases rapidly starting 12 to 24h after onset; by day five, the sensitivity of CT scanning for SAH falls to 58%. If a patient with a suspected SAH has a normal CT scan and is beyond the 6 hour window, then a lumbar puncture (LP) must be performed to look for blood in the cerebrospinal fluid (CSF).

There is growing evidence that it is both safe and potentially more effective to start with lumbar puncture in patients who have a normal mental status, no focal neurological signs, and no papilledema. There is virtually no risk of cerebral herniation in this sub-group of patients.

The timing of LP is controversial. Red cells can be seen in the CSF of patients within 2 hours of SAH; however, xanthochromia (pigment caused by the breakdown of RBCs) can take as long as 12h to be reliably present. Since xanthochromia is more specific than RBCs and is used to help distinguish between a traumatic LP and true SAH, some authors have recommended delaying the performance of LP until at least 12h after the onset of headache. However, this can delay the diagnosis and treatment of SAH, and many clinicians are uncomfortable waiting 12h to establish the diagnosis. The sensitivity of xanthochromia in the LP is effectively 100% when performed more than 12h after the onset of headache, and, unlike CT, remains 100% sensitive for up to 14 days.

All patients with SAH require urgent neurosurgical consultation. Treatment of SAH involves the usual general resuscitative measures as with any critically ill patient, with particular attention to the blood pressure. Excessive hypertension should be managed in order to decrease the likelihood of further bleeding.

Increased Intracranial Pressure

In the non-traumatized patient, raised intracranial pressure (ICP) usually occurs as a result of tumors, although it may also be due to brain abscess, intracerebral hemorrhage (especially with intraventricular bleeding causing secondary hydrocephalus) or chronic subdural hematoma. The symptoms generally come on gradually, over days to months, rather than acutely (with the exception of intracranial hemorrhage). Other features of raised ICP include:

- Worsening of the headache when supine or bending down; frequently worse in the a.m.
- Neurological symptoms and focal findings including cranial nerve abnormalities
- Subtle, gradual changes in mood, cognition, behaviour, memory
- Absence of vomiting and photophobia

The diagnosis of a space occupying lesion is accomplished with CT scanning. Often, a CT scan with intravenous contrast or an MRI is useful in order to better delineate small mass lesions which may enhance with contrast. This is particularly true of patients with HIV/AIDS with a new onset of headache and/or neurologic / cognitive changes.

Treatment involves stabilizing the patient, and, if appropriate, taking steps to lower the ICP as a temporizing measure. Definitive treatment generally involves neurosurgery.

Meningitis

The diagnosis and treatment of meningitis are discussed elsewhere in this manual. In brief, the features to look for in the headache patient are:

- Gradual onset of headache over hours to days
- Fever
- Altered level of consciousness
- Evidence of a focus of infection (intra or extra-cranial)
- Meningismus

Be especially suspicious of the diagnosis of meningitis in any immunocompromised patient (chronic illness, HIV/AIDS, malignancy, chemotherapy, steroid dependency), as well as any

patient who has undergone splenectomy as this predisposes to infection with encapsulated organisms such as Pneumococcus.

The diagnosis of meningitis is accomplished by lumbar puncture. As with SAH, LP may safely be performed without CT scanning in patients with normal mental status, no focal neurologic signs, and no papilledema.

The treatment of meningitis begins when it is first suspected. Antibiotics should be administered before obtaining a CT scan or awaiting LP results; standard of care is to administer antibiotics within one hour of suspecting the diagnosis of meningitis. The choice of antibiotics is discussed elsewhere.

There is still significant controversy regarding the role of steroids (IV dexamethasone) in the treatment of bacterial meningitis. There is fairly good evidence to support the use of steroids in adults with suspected meningitis with no contraindications (sepsis, immunosuppression, and those who have already received antibiotics). The benefits in children are less clear. If administered, steroids must be given before or with the first dose of antibiotics.

Temporal Arteritis

While not truly “deadly”, temporal arteritis is included here as a cause of headache which may result in significant morbidity, including blindness. The typical patient profile includes:

- Middle-aged to elderly
- Gradual onset of headache over days
- May have visual changes (but not universal)
- Localized headache and tenderness in the temporal region

Initial diagnosis is clinical supported by markedly elevated ESR (greater than 50 mm/h; often much higher). Definitive diagnosis requires temporal artery biopsy, although this is not 100% sensitive as a diagnostic tool. Treatment consists of oral or intravenous steroids, generally in consultation with Internal Medicine or Neurology. In a patient with suspected temporal arteritis, treatment should generally commence without awaiting biopsy results.

THE APPROACH

Benign causes of headache should be thought of as diagnoses of exclusion. That is, the more serious causes must be considered and then ruled out before these diagnoses may be considered. This does not,

however, mean that every patient with headache requires a full set of investigations, including CT scans, lumbar punctures, etc. A careful history and physical examination should answer the question “Is this a worrisome type of headache?” in the majority of cases. Certain features of the history and physical examination should set off alarms in the Emergency Physician:

History

- Sudden onset
- Most severe headache, or unlike previous headaches
- New onset of headache at age 50 or older
- Associated neurological symptoms

Physical

- Pupillary changes
- Altered level of consciousness
- Abnormal vital signs, especially temperature
- Meningeal irritation
- Focal neurologic signs

If in doubt regarding the etiology of the patient’s headache, the patient requires investigation and treatment directed towards the most likely sinister cause. Although frequently forgotten, appropriate pain relief should be considered for all patients with headache, including those with life-threatening causes of their pain. However, it is important to remember that response to therapy in the ED does not mean that the patient has a benign cause of headache. There are many case reports of patients who were given non-narcotic medications and had partial or complete relief of their headache, only to suffer catastrophic events such as SAH a few hours or days later. The best approach is to decide what investigations the patient needs first, and not be dissuaded by an improvement in the patient’s condition after treatment.

SUMMARY

- Every headache is potentially deadly until proven otherwise by history and physical examination, supplemented when necessary with radiological and laboratory investigations.
- Treatment of the patient with a suspected serious cause of headache should begin immediately, and not await the result of definitive investigations.
- Narcotics should be the last resort in the treatment of migraine headache.

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WHAT'S NEW

1. Commonly used and readily accessible antiemetics are becoming the initial treatment of choice for migraine in the ED
2. Dexamethasone plays a role in prevention of recurrent migraine following discharge in a subset of the population
3. A normal non contrast CT read by a radiologist may be sufficient to rule out SAH in patients presenting within 6 hours of the onset of their symptoms.

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7: CHEST PAIN

Steven Marc Friedman, Telisha Smith Gorvie

OBJECTIVES

1. To recognize emergent and non-emergent causes of chest pain.
2. To describe various laboratory, imaging and other diagnostic modalities used in the evaluation of the Emergency Department (ED) patient with chest pain.
3. To understand general management principles of selected causes of chest pain.

INTRODUCTION

Chest pain is one of the most common presenting complaints in the emergency department. Multiple organ systems may be the source of chest pain: heart, lungs, upper GI, muscle, bone, fascia, skin, abdomen, and cervical spine. The diagnosis is based primarily on history and physical examination. Ancillary tests are often not diagnostic and frequently misleading. Pain from visceral organs can be referred to a myriad of locations on the chest and abdomen. A patient may have more than one source of pain. Elderly patients often have serious illnesses, but present with vague or nonspecific atypical symptoms. Their clinical presentation is frequently not 'classic'.

SOMATIC VERSUS VISCERAL PAIN

Somatic pain

- Precisely located
- Sharp, piercing
- Arises from dermal pain fibres that enter the spinal cord at a single level

Visceral pain

- Poorly localized
- Less distinct
- Dull, aching, pressure
- Arises from internal organ pain fibres that enter the spinal cord at multiple levels (T1-T6 for organs of the thorax or upper abdomen)

MANAGEMENT PRIORITIES

ABC's (Airway, Breathing, Circulation) are always the first step in management. Resuscitation may be

prompted by obvious distress, abnormal vital signs, or deep visceral type pain. Provide supplemental oxygen, establish intravenous access, apply a cardiac monitor and obtain an oxygen saturation. Do not send unstable patients to X-Ray - get a portable film. Approach each patient with acute chest pain as a possible life-threatening situation. When in doubt, err on the side of caution.

Assess for emergent and potentially life-threatening causes of chest pain

- Cardiovascular: Acute Coronary Syndrome (unstable angina, myocardial infarction [MI]), aortic dissection, cardiac tamponade
- Respiratory Pulmonary embolus (PE), tension pneumothorax,
- Mediastinitis (eg: from esophageal rupture)
- Arrhythmias

Other causes

- CV: pericarditis, myocarditis, endocarditis, arrhythmias
- Resp: infections (pneumonia, bronchitis)
- GI: pancreatitis, biliary colic, GERD, esophageal spasm
- MSK: costochondritis, rib fractures
- Psych: panic attack (diagnosis of exclusion)
- Other: shingles

CLINICAL PEARLS

'The Big Five': Acute Life-Threatening Causes of Chest Pain

1. Myocardial Infarction
2. Pulmonary Embolism
3. Aortic Dissection
4. Tension Pneumothorax
5. Esophageal Rupture

HISTORY

- Elicit characteristics of the pain:
 - onset (sudden vs. gradual) and duration
 - constant vs. intermittent
 - location
 - quality (visceral vs somatic)
 - radiation (arm, back, jaw, abdomen)

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- aggravating factors (position, respiration, exertion, eating)
- relieving factors (position, rest, medications)
- associated symptoms (dyspnea, nausea/vomiting, cough, syncope, dizziness, diaphoresis, weakness, focal neurological symptoms)
- Any history of recent trauma?
- Was there any use of medications or drugs (especially cocaine) before the pain began?
- Have there been previous episodes of similar pain? Has the pattern changed – i.e. coming on after climbing fewer stairs, or after fewer holes of golf? Obtain copies of previous test results and medical records (eg: ECG, echo, stress test). Speak to the family physician or consultants involved.
- Cardiac risk factors should be assessed in patients without a known and prior history of coronary artery disease (CAD): personal history of hypertension, diabetes, hypercholesterolemia, positive family history of CAD (before age 45 for female and 55 for male 1° relatives)
- Relative risk factors are age, male sex, postmenopausal women, obesity and a sedentary lifestyle
- PE risk factors include prolonged immobilization, cancer, exogenous estrogen use, personal history of DVT/PE.
- The classic presentation of aortic dissection is a hypertensive middle-aged male with sudden onset of severe, tearing pain in the retrosternal or midscapular area. Presentation may include shock, MI, neurologic signs and symptoms
- Past and present significant illnesses, medications, allergies, drugs, smoking and alcohol use.
- Using clinical prediction scoring systems such as the PERC score (pulmonary embolism rule-out criteria) or the Wells score for DVT and PE are useful in stratifying risk and guiding further investigations.
- A friction rub may be heard with pericarditis.
- Listen for new cardiac murmurs: a mitral regurgitant murmur may be the result of a dysfunctional ischemic papillary muscle; an aortic regurgitant murmur may be heard in a dissection of the ascending aorta. Many elderly patients have systolic ejection murmurs due to aortic sclerosis. An S₃ and S₄ are associated with greater risk of cardiovascular complications.
- Check for evidence of DVT when considering the diagnosis of PE.
- Crackles may be associated with congestive heart failure exacerbation (CHF) or ACS complicated by CHF.
- In cardiac tamponade, there may be a combination of hypotension, elevated central venous pressure, muffled heart sounds, narrow pulse pressure, tachycardia and a pulsus paradoxus of >10 mmHg. The first three constitute Beck's triad.
- Decreased or absent breath sounds and hyperresonance in one hemithorax suggests pneumothorax. In addition, a deviated trachea and haemodynamic compromise (low bp, high JVP, tachycardia) suggest tension pneumothorax, which necessitates emergent resuscitation.

DIAGNOSTIC TESTS

ECG

- An ECG should be done on most, if not all patients with acute chest pain (see chapter on MI for discussion of ECG findings in patients with ischemia)
- ECG findings may suggest non-ischemic causes of chest pain, including PE (sinus tachycardia, flipped Ts in precordial leads, S1Q3T3, signs of right heart strain, RBBB) and pericarditis (diffuse ST elevation, PR depression in II, III, aVF and V4-6, or T wave inversion, electrical alternans if a large effusion is present).

Cardiac Markers

See chapter on MI

Chest X-ray

Look for evidence of:

- Pulmonary embolus: the chest x-ray is normal in over 50% of pulmonary emboli and most changes seen are non-specific. These include focal oligemia (Westermark's sign), a peripheral wedge-shaped opacity (Hampton's hump), atelectasis, pleural effusions and pleural based infiltrates.

PHYSICAL EXAMINATION

A thorough physical examination should be performed. However, findings are often nonspecific.

- Look for diaphoresis, apprehension, pallor, cyanosis and anxiety.
- A pulse deficit (minimal or no pulse on one side vs the other) and/or a difference of systolic blood pressure >20 mmHg between raises the suspicion of aortic dissection.
- Pulse oxymetry must be considered along with other vital signs.

- Aortic dissection: suggested by a widened mediastinum, left pleural effusion, an indistinct aortic knob and/or aortopulmonary window, separation >4mm of intimal calcification from the outer edge of the aortic shadow or a depressed mainstem bronchus.
- Pneumothorax: PA, lateral and expiratory views are usually diagnostic. CXR is less sensitive in supine views, look for a deep sulcus sign- a deep costophrenic angle on the affected side.
- Pneumomediastinum: may be seen in esophageal rupture, severe asthma
- Lung parenchymal disease
- Rib fractures

CT Thorax and Ventilation/perfusion Lung Scan

- Demonstrates: aortic root and thoracic aorta; small pneumothoraces and/or pneumomediastinum; pleural and pericardial effusions may also be revealed.
- Consider a spiral CT pulmonary angiogram (CTPA) or V/Q scan when PE is suspected. CT may be preferable in the patient with preexisting lung disease. V/Q scan is helpful only in suspected cases of PE when it is normal (good negative predictive value) or high probability (good positive predictive value), and the patient has a normal CXR. A patient with an intermediate or even low probability scan requires further investigations (eg: CTPA, bilateral leg dopplers or rarely, pulmonary angiography). Another benefit to a CTPA is to assess for other causes of chest pain (e.g. aortic dissection).

Emergency Department Echocardiography

ED ultrasound has been increasingly utilized by the emergency physician. Applications include assessing for pericardial effusion, cardiac activity, and pneumothorax.

Arterial Blood Gas

Look for hypoxemia, hypo or hypercapnea, acid-base abnormalities. A venous blood gas may also be used if an arterial sample is not obtained.

D-dimer

If a patient is determined to have a low pre-test probability of DVT/PE and a negative d-dimer, the need to pursue further testing for DVT/PE may be eliminated. However, d-dimers are nonspecific and may be elevated in other conditions (e.g infection,

pregnancy, malignancy), and so a positive d-dimer does not rule in PE.

MANAGEMENT OF EMERGENT DIAGNOSES

Acute Coronary Syndrome

See chapter on MI

Cardiac Tamponade

Haemodynamic instability mandates emergent treatment. A pericardiocentesis may be performed, either by blind technique or under direct ultrasound visualization.

Pneumothorax

- Thoracostomy with placement of one or more chest tubes, usually inserted in the 4th or 5th costal interspace at the anterior or mid axillary line
- If haemodynamic instability or respiratory failure is present, a needle decompression is performed at the 2nd intercostal space at the midclavicular line.

Aortic Dissection

- Reduce blood pressure and heart rate with IV medications; Prompt surgical consultation

Pulmonary Embolus

- Anticoagulants should be administered (provided no contraindications);
- Low molecular weight heparins increasingly viewed as preferable to unfractionated heparin.
- If respiratory failure should occur, aggressive airway management should proceed, including endotracheal intubation and mechanical ventilation. Thrombolysis for PE (intra-arterial and IV) is controversial.

WHAT'S NEW

Multislice computed tomography (MSCT) scanning has evolved as a potential new test to provide detailed diagnostic noninvasive imaging of the coronary anatomy for patients presenting to the emergency room with suspected acute coronary syndromes.

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DISPOSITION

- Patients with the following characteristics have a higher likelihood of being inappropriately discharged from the ED with an acute MI: women, younger than 55 years of age, non-Caucasian, atypical symptoms at presentation, normal or non-diagnostic findings on the ECG.
- Before discharge, feel confident that the life-threatening causes of acute chest pain have been excluded by the accumulated body of clinical evidence. In all patients discharged, close follow-up must be clear and the patient must understand events that mandate a return to the ED.
- If one remains unsure about the etiology of the acute chest pain after the ED assessment, then further observation or investigations may be indicated. Future standards for intradepartmental testing may include resting sestamibi testing, ECG or echo stress testing, CT or MRI of the coronary arteries, chest pain observation units and imaging of the patient who cannot exercise with pharmacological stress (adenosine, dipyramidole or dobutamine)

SUMMARY

- Nitrates can relieve both esophageal and myocardial pain; Patients also may respond to the placebo effect of this therapeutic trial. This test is neither sensitive nor specific.
- The use of ‘pink ladies’ (antacid mixed with xylocaine) as a therapeutic trial should not be used as a diagnostic test.
- Pleuritic or burning pain should not exclude the diagnosis of acute MI.
- Reproducible chest wall pain is seen in up to 15% of MI. Tenderness on forceful palpation does not rule out an ACS.
- Enquire about cocaine use, which increases the risk of acute MI and aortic dissection

- The elderly commonly have atypical presentations for acute MI, for example, confusion, weakness, syncope, vertigo, nausea or abdominal pain.
- History and physical examination are the most important diagnostic tools. If they are suggestive of serious illness, then consult early while ancillary tests are arranged. Unstable patients require immediate resuscitation, consultation and disposition to a definitive care setting (CCU, OR, ICU).
- Finding the cause of chest pain can sometimes be difficult, and certain conditions (eg: PE, aortic dissection) may not be suspected by history and physical. Be sure to keep such conditions in the back of your mind- if they are not thought of, they will not be looked for.

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8: ABDOMINAL PAIN

Rick Penciner

OBJECTIVES

By the end of this chapter, you will be able to:

1. Discuss a broad differential diagnosis for acute abdominal pain
2. List the life threatening causes of acute abdominal pain
3. Discuss the key elements of the history, physical examination and investigations in the management of a patient with abdominal pain
4. Describe the initial management of the patient presenting to the ED with abdominal pain

INTRODUCTION

Abdominal pain is one of the most common presenting complaints in the ED, accounting for up to 5% of all visits. In up to 25% of patients the cause is unknown. Most patients are discharged and only 10% require urgent surgery. The diagnostic possibilities range from trivial to life-threatening.

Two factors - gender and age, significantly modify the differential diagnosis in the ED. In children, the differential diagnosis is dependent on the age of the child. Non-specific abdominal pain and appendicitis account for the diagnosis in up to 95% of these cases. In the adult population, advancing age increases the risk of having a serious disease as the cause of abdominal pain. Elderly patients are also more likely to present without classic signs and symptoms. Herein lies the challenge.

When faced with a patient experiencing abdominal pain, one must be prepared to accept a relatively high level of diagnostic uncertainty, but life-threatening problems must be ruled out.

LIFE-THREATENING CAUSES OF ABDOMINAL PAIN

Intra-abdominal

- Ischemic bowel (middle age to elderly)
- Abdominal aortic aneurysm; dissection, leakage, rupture (middle age to elderly)
- Hepatic or splenic injury (blunt trauma)
- Perforated viscus

- Acute pancreatitis
- Intestinal obstruction
- Extra-abdominal
 - Ectopic pregnancy (female of child bearing age)
 - Myocardial infarction

ETIOLOGIES OF ABDOMINAL PAIN

The causes of abdominal pain can be classified according to location or system.

Intra-abdominal

Peritoneal inflammation

- caused by aseptic or bacterial origin
- primary causes seen in cirrhotics with ascites
- secondary causes (most common) due to disease or injury to abdominal or pelvic viscera

Obstruction of hollow viscus

- obstruction of intestine, biliary tree or ureter which typically leads to colicky abdominal pain, nausea and vomiting

Vascular disorders

- bowel (infarction/ischemia)
- aortic aneurysm (dissection, leakage, rupture)

Extra-abdominal

Abdominal wall

- contusions, hematomas, muscle strains
- usually traumatic

Pelvic

- ectopic pregnancy
- salpingitis, tubo-ovarian abscess
- ovarian cyst (torsion or rupture)
- testicular torsion

Intrathoracic

- myocardial infarction
- pneumonia, pulmonary embolus
- esophageal disease

Metabolic

- diabetic ketoacidosis
- sickle cell crisis

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Neurogenic

- pre-eruptive phase of herpes zoster
- spinal disk disease

DIAGNOSIS OF ABDOMINAL PAIN

The assessment of abdominal pain begins with a focused history and physical examination. There should be no delay in initiation of immediate resuscitative procedures if necessary. Remember, very young patients and the elderly may not have classic presentations. The history and physical examination are essential; additional investigations should be done only when indicated.

History

Inquire about the patient's pain onset, duration, location, radiation, severity, quality, aggravating factors, relieving factors, associated symptoms, and previous episodes.

Location often provides important clues:

- diverticulitis - left lower quadrant
- appendicitis - periumbilical, then right lower quadrant
- cholelithiasis, cholecystitis - right upper quadrant
- pancreatitis - epigastric, periumbilical

Symptoms that suggest peritonitis:

- pain increased with coughing, driving over bumps in the road

Associated symptoms

- nausea, vomiting
- fever, chills
- bowel movements (constipation, diarrhea, melena, hematochezia)
- urinary symptoms
- vaginal bleeding, discharge

Gynecological history

- pregnancy, infection, menstrual history, contraception, discharge
- patients should be given the opportunity to be interviewed without family or friends in the room.

Past surgical history

- previous surgery increases the risk of obstruction due to adhesions

Medications

- prescription and non-prescription drugs
- alcohol

Physical Examination

Always assess the patient in privacy and disrobed appropriately. Serial examinations may be helpful due to the evolving nature of abdominal pain.

General appearance & vital signs

- always assess all the vital signs, including temperature
- consider postural vital signs (hypotension should always suggest blood loss until proven otherwise)
- colour (pallor, icterus)
- movement (writhing movement of colic, quiet posture of peritonitis)
- avoid missing extra-abdominal causes of pain (examine all systems)

Inspection

- distention (obstruction, ascites)
- scars
- ecchymoses (Grey Turner's sign, Cullen's sign)

Auscultation

- presence and quality of bowel sounds (increased with obstruction, decreased with peritonitis)
- bruits

Palpation

- attempt to locate the site of maximal tenderness and elicit signs of peritonitis
- begin in areas less likely to be tender and slowly move to more tender areas
- look for involuntary guarding - a reflex muscle tensing to palpation
- look for cough tenderness, rebound tenderness, percussion tenderness
- check for organomegaly

Other

- check for inguinal and femoral hernias
- rectal examination
- pelvic exam in females with lower abdominal pain
- testicular exam in males

Investigations

Investigations are helpful, but cannot replace a careful history and physical examination.

Laboratory tests

Complete blood count (CBC)

- an elevated white cell count (WBC) may indicate an infectious or inflammatory process
- WBC count should not be used solely to determine therapeutic or disposition decisions

- hemoglobin and hematocrit do not necessarily reflect acute blood loss, but serial measurements may be of benefit

Urinalysis

- the presence of hematuria may indicate renal colic, but up to 10% will have no blood
- pyuria may indicate a urinary tract infection, but may also be present with cervicitis/vaginitis or when an inflammatory mass lies in close proximity to the urinary tract (such as appendicitis)

Serum amylase/lipase

- serum amylase is elevated in acute pancreatitis, but may also be elevated in a number of conditions including; biliary obstruction, cholecystitis, bowel obstruction, bowel infarction, viscera perforation, salpingitis, ectopic pregnancy, or from a salivary origin
- lipase is more specific for pancreatitis
- if elevated, usually indicates a serious disorder

Pregnancy test

- should be performed in all women of childbearing age
- can be qualitative (urine HCG) or quantitative (serum HCG)

Other blood tests

- electrolytes, creatinine, urea are important in patients with protracted vomiting and diarrhea
- alkaline phosphatase and bilirubin may be elevated with cholecystitis and obstructive biliary tract disease
- liver function tests are not routinely ordered but may reveal non-icteric hepatitis (AST, ALT, INR)
- Cross and type for blood is indicated in patients with GI bleeding, ruptured AAA, or unstable patients
- electrocardiogram (ECG) should be performed in all adults with upper abdominal pain if myocardial ischemia is a possibility and all unstable patients

Medical Imaging

Imaging should not delay definitive management. Unstable patients should not be sent out of the emergency department for imaging.

Standard views of the abdomen are of limited value except for:

- free air (perforation)
- gas pattern (dilated loops of bowel, air fluid levels in obstruction)

- foreign body

Ultrasonography

- ED ultrasound can be used to rapidly diagnose AAA, presence of an intrauterine pregnancy and presence of free fluid
- biliary tract disease
- renal disease (hydronephrosis; but insensitive to small renal stones, partial obstruction and renal function)
- valuable in evaluating women of childbearing age with lower abdominal pain (intrauterine and ectopic pregnancy, adnexal pathology)
- some value in the evaluation of acute appendicitis (specific, but not sensitive test)
- aortic abdominal aneurysm
- ascites

Computed tomography

CT is the imaging modality of choice for most significant causes of abdominal pain. Oral and IV contrast is often used. This test exposes patients to a significant amount of radiation (increasing their risk of lethal cancer significantly) – consider its use carefully in children and women of reproductive years. It may be used for:

- trauma
- suspected ruptured or leaking aortic aneurysm
- pancreatitis
- urolithiasis, renal colic (non-contrast)
- diverticulitis
- appendicitis

MANAGEMENT

Rapid Assessment and Stabilization

- Hemodynamically unstable patients or those with potentially life threatening causes should be triaged immediately to an acute care area of the ED
- Careful attention to airway, breathing, circulation
- Supplemental oxygen, IV, cardiac monitoring
- Volume repletion with crystalloid (normal saline or Ringer's lactate solution) and then colloid (blood products) if necessary
- Consider ED ultrasound if available

General

- Nothing per os (NPO)
- Intravenous (vascular access for volume replacement, route of administration of medications) normal saline or Ringer's Lactate

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- Nasogastric (NG) suction for obstruction, perforation, retracted vomiting
- Analgesia - appropriate use of analgesics combined with serial examination may in fact aid in the diagnosis
- Antibiotics - Regimens vary (gram negative and anaerobic coverage required). Indicated for perforation, sepsis, peritonitis
- Control of vomiting with antiemetics should be considered

Specific Diseases

Appendicitis

- Diagnostic history and physical exam - consult Surgery
- Questionable history: consider ultrasound or CT
- Antibiotics and analgesia

Cholecystitis

- Alkaline phosphatase, bilirubin, AST, ultrasound
- Surgical consult
- Antibiotics and analgesia

Diverticulitis

- CT
- Moderate to severe cases require surgical consult
- Antibiotics and analgesia

Pancreatitis

- Amylase or lipase, alkaline phosphatase, bilirubin, AST, calcium, oxygen saturation, INR
- CT
- Chest x-ray (for pleural effusion, ARDS)
- Surgical consult - may need intensive care unit (ICU)

Perforated Viscus

- Chest xray (for free air), 2 views abdomen
- Nasogastric suction
- Surgical consult, prepare for OR
- Antibiotics and analgesia

Suspected leaking aortic aneurysm

- 2 large bore intravenous catheters and volume resuscitation
- STAT crossmatch (6 units packed cells)
- STAT Surgical (vascular) consult
- No imaging studies outside the ED in an unstable patient
- ED ultrasound and/or CT

Small Bowel Obstruction

- Abdominal x-rays
- Nasogastric suction

- Surgical consult

CLINICAL PEARLS

Most Common Causes of 'Surgical Abdomen' in the Elderly:

1. Cholecystitis
2. Bowel Obstruction
3. Appendicitis

Disposition

Most patients are discharged from the ED. Prior to discharge, reassessment of pain and physical exam findings should be documented. Instruct the patient to return if increasing pain or other symptoms develop to suggest a serious or surgical cause of their pain. Analgesics can be prescribed for home use, but some practitioners believe that potent narcotics should be avoided in patients without a specific diagnosis because they may mask worsening or 'red flag' symptoms. Ensure appropriate and timely follow-up (reassessment in 24 hours). Discharge diagnosis should accurately reflect the findings. Consider "abdominal pain uncertain etiology" instead of guessing at a specific diagnosis.

PITFALLS

- Extra-abdominal pathology can have intra-abdominal manifestations
- Elderly patients may provide few cues but are more likely to have serious illness compared to younger patients
- Very young and elderly patients may not have classic presentations
- Immunosuppressed patients and alcoholics are more likely to have serious disease and often have atypical presentations.
- WBC lacks sensitivity and specificity to diagnose any cause of abdominal pain
- Prematurely attributing a diagnosis when the etiology is uncertain
- Assessing the patient in the hallway

SUMMARY

- Consider a broad differential diagnosis
- Life threatening causes must always be ruled out
- Diagnosis is made through a careful history, physical examination and selected investigations
- ED ultrasound, formal ultrasound and CT are the most important imaging modalities

- Unstable patients or those with potentially life threatening causes should be assessed and treated immediately

WHAT'S NEW

ED Ultrasound performed by emergency physicians is becoming increasingly utilized as a diagnostic tool for patients who present with abdominal pain. It has been described as 'the new stethoscope' in the initial evaluation of patients in the ED.

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9: BACK PAIN

Anton Helman

OBJECTIVES

1. To develop an approach to serious causes of acute back pain
2. To understand the ‘red flags’ of acute back pain
3. To understand why risk factor assessment is important in acute back pain
4. To review the indications for various imaging modalities in acute back pain
5. To review the treatment and disposition of patients with acute back pain

INTRODUCTION

As many as 90% of adults will have an acute back pain episode in their lifetime, often prompting a visit to the Emergency Department. The vast majority of these patients will have non-life-threatening causes, with lumbosacral strain being the most common. Despite the fact that many patients cannot be given a precise diagnosis, nearly all recover without major sequelae within 4-6 weeks.

However, there are approximately 5-10% of patients with acute back pain who do have life- or limb-threatening etiologies, which are often missed on the initial visit to a physician. As a result of delayed diagnosis, many of these patients go on to have poor outcomes. These unfortunate cases occur because most of these patients have non-classic presentations. It is therefore incumbent upon the astute clinician to approach each patient who presents with back pain with a wide differential diagnosis, to know the risk factors for specific etiologies and to pay particular attention to ‘red flags’ discovered on history and physical examination. This chapter will focus on the potentially catastrophic spinal causes of acute low back pain, which when diagnosed early, can prevent serious morbidity and mortality. Lumbosacral strain and sciatica will be covered briefly as well. Vascular causes of back pain are covered elsewhere in this manual.

APPROACH

Serious non-traumatic causes of acute back pain can be divided into 2 categories: spinal and vascular. While entities such as renal colic, herpes zoster and

spinal stenosis must be considered in patients presenting to the Emergency Department with back pain, they are typically not life-threatening and will not be discussed in particular here.

Spinal

1. Cauda Equina & Spinal Cord Compression
 - Cancer (Spinal Metastasis)
 - Epidural Abscess
 - Epidural Hematoma
 - Central Disc Herniation
2. Osteomyelitis
3. Transverse Myelitis

Vascular

1. Aortic Dissection
2. Rupturing Abdominal Aortic Aneurysm
3. Pulmonary Embolism
4. Myocardial Infarction
5. Retroperitoneal Bleed

HISTORY & PHYSICAL

The following lists the ‘red flags’ to elicit in the history and physical examination for serious spinal causes of back pain, with their corresponding diagnostic considerations.

- Constitutional symptoms including fever (cancer, epidural abscess, osteomyelitis)
- IV drug use (epidural abscess, osteomyelitis)
- Immunocompromised status including corticosteroid use (epidural abscess, osteomyelitis)
- Pain worse at night (cancer)
- Urinary retention or incontinence; or fecal incontinence (cauda equina)
- History of cancer (spinal metastasis)
- Spinous process tenderness (spinal causes as above)
- ‘Saddle’ paresthesia or anesthesia (cauda equina syndrome)
- Bilateral neurologic deficit or complaint (cauda equina syndrome)
- Neurologic deficit at multiple nerve root levels
- Anticoagulant use or bleeding disorder (epidural hematoma)

- Recent invasive spinal procedure including epidural anaesthesia (epidural hematoma)

The history should elicit the location, radiation and nature of the pain as well as palliative and provocative factors. Typically, the description of back pain that has a benign cause is mild to moderate, dull, aching pain that is worse with movement, but improves with rest and lying still. Cough or Valsalva's maneuver that worsens the pain, usually signifies a musculoskeletal origin and may indicate a herniated disc in particular. Pain that is worse with flexion is consistent with lumbosacral strain and disc herniation. Pain that is worse at night, or is severe despite analgesics and lying still, often signifies a more serious cause. Associated neurologic symptoms such as sensory or motor deficits, gait disturbances and fecal or urinary incontinence should be sought after.

Risk factors for infection, cancer and vascular causes must be elicited to help heighten the suspicion for serious causes of back pain. Since the classic triad of spinal epidural abscess of fever, back pain and neurological deficits occurs in only 13% of confirmed cases, a history of IV drug use, chronic corticosteroid use and immunocompromised states must be asked about. Any patient with a known history of malignancy with new onset of back pain, should be considered to have spinal metastases until proven otherwise. Although prostate, lung and breast cancer are the most common culprits, any cancer can metastasize to the spine.

The physical examination is directed toward discovering 'red flags'. Examination of vital signs is of utmost importance with particular attention to fever. Inspect the skin for signs of infection and trauma. The abdomen should be palpated for aortic aneurysm, and the vertebral column as well as paraspinal muscles palpated for point tenderness. Perform a straight-leg test for sciatica. A positive straight-leg raise produces a pain that radiates below the knee into one or both legs. This radicular pain is improved by decreasing the elevation and worsened by ankle dorsiflexion. Radicular pain below the knee in the affected leg when lifting the asymptomatic leg constitutes a positive crossed straight-leg raise, which is highly specific for nerve root compression by a herniated disc.

The neurologic examination must be thorough and directed toward each of the spinal nerve roots. Sensation, power and reflexes in the lower extremities must be elicited. In particular, sensation in the 'saddle' area surrounding the anus and perineal area should be tested as well as rectal tone which may

both be decreased or absent in the setting of cauda equina syndrome. In addition, the anal wink reflex is elicited by gently stroking the skin lateral to the anus, which should cause a reflex of the external anal sphincter. The absence of sphincter contraction may indicate multiple sacral nerve root dysfunction.

CLINICAL PEARL

Any patient with a known history of malignancy with new onset of back pain should be considered to have spinal metastases until proven otherwise.

INVESTIGATIONS

For patients with the presumed diagnosis of lumbosacral strain or herniated disc in the absence of 'red flags', no investigations are required since the vast majority of these patients will be asymptomatic within a few weeks. An emergency department bedside ultrasound can rule out an abdominal aortic aneurysm with very good sensitivity. Spinal infection or cancer patients will often have an elevated WBC and/or ESR or C-reactive protein, although none have the sensitivity nor the specificity to rule in or out these diseases. A urinalysis and creatinine is done for patients suspected of renal pathology. Blood cultures should be obtained prior to antibiotic administration for suspected spinal infection.

Any patient displaying signs or symptoms consistent with cauda equina syndrome should have a post-void residual preformed. After the patient has voided, a catheter is inserted into the bladder and the urine volume is measured. If the volume of urine is >200ml then this is considered a positive test and increases the suspicion for cauda equina. A post-void residual of <100ml makes the diagnosis less likely.

Plain X-rays of the spine should be obtained if 'red flags' raise one's suspicion for cancer, infection or fracture. Specific indications for plain films include: age >70 years, unexplained weight loss, pain worse at rest, history of prolonged steroid use, cancer, IV drug use and osteoporosis. However, the sensitivity of plain films for spinal metastases is only 60%, while 17% of patients with epidural spinal cord compression due to metastasis have normal X-rays. In addition, the overall sensitivity and specificity of plain X-rays for osteomyelitis or epidural abscess is 82% and 57% respectively.

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It is thus incumbent upon the clinician who suspects cauda equina syndrome to obtain an MRI of the spine as soon as possible. MRI is the imaging modality of choice for spinal cord compression. This is one of the few instances in which an MRI is indicated on an emergent basis. Plain CT of the spine may aid in the diagnosis since it can show bony detail, but like X-ray films, it does not have sufficient sensitivity and specificity to diagnose cauda equina and spinal cord compression syndromes with certainty. CT Myelogram is an alternative option for patients who are unable to undergo MRI, however, it is an invasive procedure that has the potential to transmit infection into the subarachnoid space.

TREATMENT & DISPOSITION

Lumbosacral Strain & Disc Herniation

- Continue daily activities as much as tolerated, using pain as the activity limiting factor
- No/minimal bed rest (bed rest has been proven to prolong recovery and time back to work)
- Consider acetaminophen, NSAIDs and opioid analgesics
- Spinal traction, diathermy, ultrasonography, biofeedback and acupuncture have no proven benefit
- There is some evidence that heat application, massage therapy and physiotherapy may improve symptoms
- Follow-up in 4-6 weeks with primary care physician if pain persists
- Return if 'red flags' of impending spinal cord compression

Spinal Infection

- Prompt IV antibiotics (e.g. 3rd generation cephalosporin +/- ampicillin +/- vancomycin)
- Consult internal medicine or infectious diseases and neurosurgery (if signs of cord compression)

Cauda Equina & Spinal Cord Compression

- IV opioids
- IV dexamethasone, especially if cord compression from tumour is suspected

- Consult neurosurgery early

SUMMARY

- Risk factor assessment is important in identifying those patients with serious causes of back pain since presentations are often not classic
- A thorough neurological examination including assessment for 'saddle' anaesthesia and rectal tone is required to identify patients with potential spinal cord compression
- A post-void residual assessment is indicated in patients suspected of spinal cord compression
- X-rays have limited utility in the work-up of patients with acute back pain, but should nonetheless be considered in patients with risk factors for serious causes
- MRI is the imaging modality of choice for patients suspected of having spinal cord compression
- Lumbosacral strain and sciatica are clinical diagnoses treated with activity as tolerated, analgesics, heat application, massage and physiotherapy, appropriate follow-up and specific instructions including 'red flag' education
- Antibiotics should be administered promptly for patients suspected of having spinal infection
- Corticosteroid administration and early neurosurgical consultation are indicated for patients with spinal cord compression

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10: PAIN

Brian Goldman

OBJECTIVES

1. To understand the various types of pain and how to treat them
2. To assess severity of pain in emergency patients
3. To review the principles of good pain management
4. To understand the indications, advantages and disadvantages of various pain medications
5. To identify and manage drug seeking behaviour
6. To treat pain appropriately in common emergency illnesses

INTRODUCTION

Acute pain is one of the most common presenting complaints of emergency patients. However, pain continues to be managed poorly. Suboptimal pain management in hospital is sometimes referred to as ‘oligoanalgesia’. Factors that contribute to ‘oligoanalgesia’ include inadequate training and experience, inappropriate choices, dosages and routes of administration of analgesics, as well as the fear that the administration of opioid analgesics will lead to addiction. Improved pain management increases comfort and patient satisfaction, and may help prevent chronic pain.

DEFINITIONS

Acute pain is defined as a ‘complex, unpleasant experience with emotional and cognitive, as well as, sensory features that occur in response to tissue trauma’. Acute pain is either nociceptive, neuropathic, or a combination of the two.

Nociceptive pain arises from tissue damage and passes through intact nerves to the brain. Conditions associated with nociceptive pain include fractures, burns, appendicitis and renal colic. Nociceptive pain tends to be aching or burning in character.

Neuropathic pain occurs because of disease or damage to structures in the peripheral and/or central nervous system. Examples include complex regional pain syndrome, trigeminal neuralgia and pain associated with multiple sclerosis. Neuropathic pain is burning or lancinating in character; the location and

radiation of pain corresponds to the structure diseased or damaged. Neuropathic pain tends to be under-recognized.

Chronic pain is defined as pain that persists for 6 months or more or pain that lasts beyond the expected point of resolution of tissue damage. Chronic pain usually occurs because of processes in the peripheral and central nervous system that facilitate the transmission of pain signals. In general, it is not thought to be a symptom of malingering or of an underlying psychiatric condition.

Acute Pain is usually a symptom of a disease or injury. Chronic pain is considered a disease or disorder in and of itself.

ASSESSING THE PAIN

The assessment of pain should begin at triage. Pain scoring at triage leads to substantially faster provision of initial analgesia with long-term benefits.

Determine the location and radiation of the pain, onset, a description of the pain, the intensity, the effect of the pain on sleep and activities, associated symptoms such as nausea and sweating, as well as treatments and their effectiveness. Distinguish between nociceptive and neuropathic pain, as this helps guide treatment (see below). Ask about associated anxiety; managing anxiety associated with acute pain can have a beneficial effect on its perceived severity.

Determine the severity of pain by the patient’s subjective self-report. If done properly, subjective pain scales are sensitive and give consistent results. These include:

- **The Numeric Pain Distress Scale** is the easiest to use. Patients are asked to rate their pain on a scale from zero to ten, with zero signifying no pain, and ten signifying the worst pain ever.
- **The visual analog scale (VAS)** is a 100 mm line on which the patient is asked to rate his or her pain. VAS ratings of greater than 75 mm indicate severe pain, 45–74 indicates moderate pain, and 5–44 mm indicates mild pain.

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- **Categorical scales** use words to describe the severity of pain. This scale is useful for patients who have difficulty using the NPDS or VAS.

In practical terms, a 30 mm reduction in pain score on the VAS or a reduction of three digits (for example, from nine out of ten to six out of ten) in the Numeric Pain Distress Scale would be considered the minimum change associated with a ‘clinically meaningful’ reduction in pain.

On physical examination of a patient with nociceptive pain, check for point tenderness and other evidence of tissue damage. Features on physical examination that suggest neuropathic pain include hyperalgesia (increased response to a normally painful stimulus), allodynia (pain due to a stimulus that is normally not painful), areas of hypoesthesia, as well as regional autonomic features such as changes in temperature, colour and abnormal sweating.

Investigations are guided by the suspected cause of the pain. In general, there is no need to delay or withhold analgesics and other modalities of pain management pending the results of investigations, including abdominal pain. Far from obscuring the diagnosis, there is evidence that pain management enhances diagnostic accuracy. For instance, morphine administration has been shown to have no effect on diagnostic accuracy in the assessment and initial management of patients with abdominal pain.

MANAGEMENT OF ACUTE PAIN SYNDROMES

General Principles

Acute pain of mild or moderate intensity can usually be managed with a combination of appropriate oral analgesics as well as non-pharmacologic modalities. Acetaminophen and NSAIDs are indicated for the management of mild pain; opioid analgesics are indicated for the management of moderate and severe pain. In cases of moderate to severe pain, there may be value in combining opioid and non-opioid analgesics, as well as adjuvant drugs.

Severe pain is often managed in an emergency setting, and the assessment of pain in the emergency department is increasingly becoming a priority in terms of triage. The National Triage and Acuity Scale incorporates pain into the standardized triage assessment.

There are four principles of good pain management:

1. Find and treat the source of tissue damage or inflammation.
2. Manage pain preventatively. That means giving analgesics before doing painful procedures such as reduction of fractures and dislocations as well as incision and drainage of abscesses. In patients with spontaneous pain (for example, renal colic) preventative pain management means treating pain as early as possible following arrival in the ED.
3. Use multimodal approaches. The idea behind multimodal pain management is to take advantage of the additive effect of analgesics and with fewer side effects. Appropriate combinations might include an NSAID, acetaminophen as well as an opioid analgesic.
4. Manage pain continuously. That means scheduled doses of analgesics rather than ‘prn’ dosing for patients with constant pain; reserve ‘prn’ dosing for patients with intermittent pain.

NON-PHARMACOLOGICAL PAIN MANAGEMENT

Acute pain is managed through a combination of non-pharmacologic and pharmacological modalities. However, due to factors such as severity of pain, time pressure and patient anxiety, pharmacotherapy remains the mainstay of pain management in the ED.

PHARMACOLOGICAL AGENTS

Nociceptive pain can be managed with one or a combination of acetaminophen, NSAIDs (including COX-2 inhibitors), opioid analgesics as well as local anaesthetics. **Neuropathic pain** is managed usually with opioid analgesics in combination with tricyclic antidepressants or antiepileptic drugs (AEDs). Sometimes, neuropathic pain requires higher doses of opioid analgesics.

Acetaminophen

This medication is both analgesic and antipyretic, and can be administered orally or rectally. Single doses of acetaminophen are effective in the treatment of postoperative pain. Acetaminophen is also a useful adjunct to opioid therapy. Opioid analgesic requirements are reduced by 20-30% when combined with scheduled acetaminophen dosages.

Acetaminophen has fewer adverse effects than NSAIDs, and can be used in patients when NSAIDs

are contraindicated. The recommended oral dosage for acute pain management is 1000 mg qid. The dosage should be reduced in patients with alcohol-related and other forms of active liver disease, and patients with glucose-6-phosphate dehydrogenase deficiency. Exceeding the recommended dosage can result in hepatocellular necrosis and hepatic failure.

Nonsteroidal Anti-inflammatory Drugs (NSAIDs)

NSAIDs are indicated for the management of mild to moderate nociceptive acute pain as well as pain due to inflammatory conditions such as arthritis.

The lone intravenous NSAID available in Canada is ketorolac (Toradol). Ketorolac is effective for pain due to fractures, inflammatory conditions, renal colic, migraine headaches and sickle cell pain. The dosage of ketorolac is 30-60 mg/dose IM. Although not approved in Canada for intravenous administration, there is evidence to support the use of ketorolac IV at an initial dosage of 30 mg and 15 mg q3h thereafter.

NSAIDs have a dose ‘ceiling’, and should therefore not be titrated beyond the maximum recommended dose. NSAIDS used continuously have an annual 4 percent risk of gastrointestinal perforations, ulcers or bleeds. Taking an NSAID for two months or more carries a one in five risk of an endoscopically proven ulcer and a one in one hundred and fifty risk of a bleeding ulcer.

Other significant side effects include hypertension, increased risk of cardiovascular outcomes and flash pulmonary edema. The cardiovascular risk varies with the agent. The risk is increased with indomethacin, meloxicam and Diclofenac. Naproxen is not cardioprotective but carries a neutral risk; the data regarding ibuprofen is inconclusive.

Contraindications to the use of NSAIDs include known allergy or sensitivity to NSAIDs, ASA-induced asthma, rhinitis or urticaria, easy bruising, recent peptic ulcer, active inflammatory bowel disease, renal dysfunction, and pregnancy.

Cyclo-oxygenase-2 Selective Inhibitors (COX-2 inhibitor)

COX-2 inhibitors provide comparable efficacy to NSAIDs with a lower risk of peptic ulcers and other gastrointestinal adverse effects. However, this class of medications increases the risk of cardiovascular events such as heart attack and stroke.

Opioid Analgesics

Opioid analgesics are the most important agents in managing moderately-severe to severe acute pain in the ED. Opioids are effective in managing acute nociceptive as well as neuropathic pain.

Opioids can be titrated to analgesic efficacy or unmanageable adverse effects. There is tremendous inter-individual variation in the analgesic and adverse effect profile of opioid agents. Thus, opioids should be titrated to suit each patient. Likewise, there is little data to suggest that one opioid is superior to another; however, some opioids may have superior efficacy with fewer side effects in some patients.

The intravenous (IV) route of administration is preferred for rapid titration of opioid analgesics. Intramuscular (IM) administration of opioid analgesics produces delayed and often variable absorption.

Morphine is a mainstay of parenteral pain management. Morphine-6-glucuronide (M6G) is the active metabolite of morphine, while morphine-3-glucuronide (M3G) may be responsible for neurotoxic adverse effects such as hyperalgesia and myoclonus. Both metabolites are excreted by the kidney. Both metabolites accumulate with higher doses, renal impairment, oral route of administration and increasing age. Hydromorphone is approximately five to eight times more potent than morphine; the effective dosage of hydromorphone tends to be much lower than that of morphine. **Deaths due to respiratory depression have been reported when clinicians have failed to appreciate that hydromorphone is far more potent than morphine.**

For rapid control of acute pain, titrate morphine by intermittent IV bolus doses at 2-3 mg every 5 minutes as needed with no limitation on the number of bolus doses given. Hydromorphone may be administered at a dosage of 0.25-0.5 mg IV q 5 minutes as needed. Alternatively, a dose of hydromorphone of 1-2 mg IV and repeated q3-4 h prn relieves severe pain in most adult patients.

The use of meperidine in the ED is discouraged because it has a relatively short duration of action, and causes more nausea than other opioid analgesics. As well, repeated administration of meperidine over 48-96 hours causes accumulation of the normeperidine, a neuro-excitatory metabolite of meperidine that can cause seizures. The combination of meperidine with SSRI drugs can trigger the serotonin syndrome.

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Fentanyl has an extremely brief duration of action (20 minutes) and has no active metabolites. It is very useful for procedures such as incision and drainage of abscesses and reduction of fractures. Moreover, fentanyl causes little, if any, nausea or histamine release. The dosage of fentanyl is 0.5µg/kg q5-10 mins until the patient is comfortable. Fentanyl is useful in patients who have a true allergy to morphine.

Respiratory depression is the most significant side effect caused by opioid analgesics in the acute setting. It tends to occur when an opioid-naïve patient receives a dose of analgesic that far exceeds the patient's analgesic requirement. The greatest risk of opioid-induced respiratory depression occurs approximately 8 to 10 minutes after intravenous administration or 30 to 45 minutes after oral administration. Careful titration plus the use of a pulse oximeter can greatly reduce the risk of this complication. The best early measure of impending respiratory depression is increasing sedation.

Opioids can induce nausea and vomiting, which is relieved through the use of anti-nauseants such as:

- dimenhydrinate (Gravol®) 25-50 mg IV or 50 mg IM q4h PRN
- ondansetron (Zofran ®) 8 mg or 0.15 mg/kg IV
- prochlorperazine (Stemetil®) 10 mg IV or by suppository, or 10-20 mg IM q8h PRN

Ondansetron and prochloroperazine are less sedating than dimenhydrinate.

Constipation is also common, and should be managed prophylactically using agents such as senna and lactulose. Sedation tends to occur with opioid-naïve patients. Such patients should be observed in the ED until sedation passes or until a responsible caregiver can accompany the patient home.

Puritus is a known adverse effect of opioid analgesics, but true allergic reactions to opioid analgesics are rare. Urinary retention occurs rarely.

The risk of iatrogenic addiction arising from the administration of parenteral opioid analgesics in the management of severe acute pain appears to be very small. Nevertheless, ask about a history of current or past alcohol or drug abuse. The administration of opioid analgesics in patients with a history of opioid abuse is not contraindicated but does merit caution.

Oral Opioids

Codeine, and tramadol are indicated for the management of mild to moderate acute pain and as discharge medications. For codeine to be effective, it

must be metabolized into morphine. Six to ten percent of individuals are unable to metabolize codeine and are thus unable to derive analgesic benefit from the drug.

Codeine is available in non-compound forms. However, it is usually prescribed in combination with acetylsalicylic acid (ASA) (282s, 292s) or acetaminophen (Tylenol #2, Tylenol #3, Empacet-30). Dosage titration of these products is limited by the dosage of acetaminophen or ASA. In otherwise healthy patients, this generally means not exceeding 12 acetaminophen-containing combination tablets (4 grams) per day.

Oxycodone should be reserved for patients with moderately severe to severe pain as well as patients with pain unresponsive to codeine. As with codeine, oxycodone is available as a single agent but is more commonly prescribed in compound form (Percocet and Oxycocet).

The abuse liability of oxycodone is well known. At present, there is an epidemic of opioid abuse in many parts of Canada, along with a dramatic increase in opioid-related deaths. **Therefore, when considering a prescription of oral oxycodone for outpatient management of severe acute pain, it is important to ask about a history of past or current alcohol and/or drug abuse and to prescribe opioids with caution in such cases. The potential benefit of opioid analgesics must always be balanced against the risk of addiction and/or abuse.**

In 2012, to help prevent opioid abuse, Ontario's Narcotics Strategy was implemented. Ontarians are now required to provide identification to their physician, dentist and in certain cases to their pharmacist in order to receive prescription narcotics and other controlled substance medications .

Opioids and Elderly Patients

In adult patients, age is regarded as a reliable predictor of opioid requirements. Elderly patients often require much lower doses of opioids than younger patients. Studies show that the sensitivity of the brain to fentanyl is increased by 50% in elderly patients, and the dosage of morphine and fentanyl dosage may be 2-4 fold times lower in that age group.

PAIN MANAGEMENT vs DRUG SEEKING BEHAVIOUR

The doctor-patient relationship is founded on trust. Unfortunately, a small number of patients exploit that trust by pretending to be in pain in order to obtain

opioid pain relievers from unsuspecting physicians. Some of these individuals feign illnesses and obtain fraudulently the medical records of bona fide pain patients to justify their claim to receive opioid analgesics. A growing number of people in society use opioid analgesics for non-medical purposes.

It's important for emergency physicians to be vigilant to the possibility of drug seeking behaviour and to consider the following steps:

- Ask unfamiliar patients for identification (see above).
- Verify objectively the presenting complaint and observe for drug-seeking behaviours. These include a story that doesn't make sense, a patient who claims to be allergic to codeine and to NSAIDs, a patient who makes constant eye contact with the physician, presenting at times when the patient's regular physician can't be contacted, and individuals who use street drug terminology.
- Attempt to contact the patient's regular prescriber.
- Be a safe prescriber by prescribing the smallest number of pills necessary to treat the problem, by dispensing tablets instead of writing a prescription, and by considering non-opioid therapies where appropriate.

OTHER AGENTS

Calcitonin

Calcitonin has been shown to relieve pain associated with osteoporotic vertebral compression fractures. The recommended dosage is 200 IU per day IM or 400 IU per day by nasal spray (1x 200 IU spray per nostril).

Local and Topical Anesthetics

Intra-dermal administration of 1-2% lidocaine has been used for years for local anesthesia prior to suturing as well as incision and drainage of abscesses. As well, lidocaine is used for hematoma blocks prior to reduction of some fractures.

Intra-articular lidocaine can be as effective as intravenous analgesia and sedation with drugs such as opioids and midazolam or opioids and propofol, but with faster recovery.

Recently, nebulized lidocaine (4 cc 10%) has been shown to be effective; however, its use has been shown to be associated with an increased risk of epistaxis, which may be due to trauma to insensate turbinates. Another option is to use topical lidocaine gel instead.

Nerve blocks can be very effective. For example, a '3 in 1' femoral nerve block using bupivacaine with IV morphine has been shown to be more effective than IV morphine alone with faster onset of analgesia in patients with a fractured femoral neck.

SPECIFIC SITUATIONS

Arthritis

Inflammatory arthritis is managed acutely with NSAIDs, opioid analgesics, and the judicious use of systemic as well as intra-articular corticosteroids. Osteoarthritis can be managed acutely with acetaminophen, NSAIDs, opioid analgesics, as well as the occasional administration of intraarticular corticosteroids. Opioid analgesics should be used where NSAIDs are either ineffective or contraindicated.

Chronic Pain Patient

There are several reasons why patients with chronic non-cancer pain may present to the emergency department for treatment. These include disease progression, analgesic tolerance, or a new acute source of pain. A subset of such patients may be abusing their medication or diverting it for purposes of selling it.

Some chronic pain patients who seek analgesics in the ED lack a family physician. Others have a prescribing physician, and are presenting because of acute or chronic pain.

Physicians should attempt to contact the patient's regular physician or obtain a consultation with an expert in pain management. In general, it is not considered appropriate to manage chronic pain on an ongoing basis in the emergency setting. However, treating a new and acute pain problem by adding a modest incremental dosage of analgesic to the patient's usual opioid regime is usually appropriate.

Herpes Zoster

The pain of herpes zoster can be managed with topical ASA as well as oral NSAIDs and opioid analgesics. Antiviral agents (including acyclovir, valaciclovir and famciclovir) given within 72 hours of onset of the rash hastens resolution of pain and reduces the risk of post-herpetic neuralgia. Amitriptyline started at the onset of the rash and continued for 90 days has been shown to reduce the prevalence of pain at 6 months. Pregabalin (Lyrica ®) is indicated for the management of post-herpetic neuralgia.

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Sickle Crisis

Pain due to vaso-occlusive sickle episodes can be severe and may require hospitalization. A pain management plan for patients with frequent painful crises is highly recommended. Patients are usually managed with parenteral opioid analgesics. NSAIDs may be effective, but do not reduce opioid requirements.

Ureteral Colic

Pain due to ureteral colic should be managed with a combination of opioid analgesics and renal prostaglandin-specific NSAIDs. Specific NSAIDs for this indication include ketorolac, diclofenac and indomethacin. The latter two should be administered by rectal suppository. Tamsulosin (Flomax ®) may be prescribed to help calculi pass more quickly.

Vertebral Compression Fractures

Compression fractures occur commonly in elderly patients, causing severe pain. Calcitonin administered in a nasal aerosol (Miacalcin ®) or by IM injection can decrease pain by accelerating osteoblast activity. Its use has been shown to improve the mobility of patients in a matter of days.

SUMMARY

- Acute pain should be measured using the Numeric Pain Distress Scale (NPDS) or the Visual Analog Scale (VAS).
- A ‘clinically-meaningful’ reduction in pain is a 30 mm decrease in the VAS or a 2-3 digit decrease in the NPDS
- Acute pain should be managed pre-emptively, continuously, and with multimodal technique

- Acute undiagnosed abdominal pain may be managed with opioid analgesics pending investigations
- Meperidine use in acute pain is discouraged
- The dosage of opioid analgesics is typically reduced in elderly patients
- When administering and/or prescribing opioid analgesics, always be mindful of the risk of addiction, abuse, and drug-seeking behaviour.

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11: MYOCARDIAL INFARCTION

Eric Letovsky

OBJECTIVES

1. To recognize the typical and atypical presentations of myocardial infarction (MI)
2. To understand the utility of ECG and cardiac enzymes in the diagnosis of MI
3. To know the treatment priorities in MI
4. To understand some of the prognostic factors in MI

INTRODUCTION

Emergency physicians play a pivotal role in the diagnosis and treatment of acute MI. This is often a difficult diagnosis to make in the Emergency Department because, at present, there is no individual diagnostic test that has 100% sensitivity or specificity. The symptoms of this life threatening disease entity are often vague; the physical examination is often not very revealing; the ECG is often non diagnostic; the initial cardiac enzymes may not be helpful in ruling in or ruling out a diagnosis of acute MI. This accounts for the rather somber statistic that up to 4% of all MI's are mistakenly sent home from Emergency Departments. These patients have a higher morbidity and mortality than those patients admitted to hospital.

In Canada, tens of thousands of people every year suffer myocardial infarctions, and approximately 40% of those patients die suddenly before reaching medical care. Those patients who do reach hospital are at risk for dysrhythmias, heart failure, and cardiogenic shock. Fortunately, the mortality of those patients who do reach hospital has decreased steadily over the past twenty years, due primarily to aggressive pharmacological therapy with thrombolytic agents and early PCI (percutaneous intervention). Reperfusion therapy (pharmacological or mechanical) decreases mortality by 25 to 50 %, and improves left ventricular function. It is imperative to remember that the magnitude of the mortality reduction is directly related to the time from symptom onset to treatment; hence, the critical role of the Emergency Physician.

When assessing patients in the Emergency Department (ED) with myocardial ischemia, most of the above are done concurrently; i.e., the Emergency Physician may be doing a focused physical

examination while taking a history; the ED nurses may be completing an ECG and then initiating therapy. The components are broken down in sequential fashion in this chapter only for the purposes of discussion and teaching.

SYMPTOMS AND HISTORY

The 'classic' textbook description of acute MI is one of crushing retrosternal chest pain, with radiation of the pain to the jaw and down the left arm, associated with diaphoresis. While this presentation certainly occurs, large numbers of infarctions present in a more atypical fashion. For example, a significant proportion of patients with MI have the following descriptions of chest pain: "crushing, pressure, tightness, burning, indigestion, chest ache, sharp, stabbing." Therefore, one must consider atypical pains as possibly being ischemic in origin.

Radiation of the chest pain is actually an insensitive, moderately specific indicator of acute MI. Interestingly, while textbooks often describe radiation of pain to the left arm as a feature of myocardial ischemia; radiation of pain to the right arm is actually a more specific indicator of acute MI. Other symptoms most specifically associated with acute MI are nausea/vomiting, diaphoresis, and dyspnea.

The presentation of acute MI in the elderly is often non-classical. It is often without pain and diaphoresis, but with an increased frequency of neurological symptoms such as syncope, stroke, acute confusion, and weakness as the presenting complaint. In addition, dyspnea is a more common presenting symptom than chest pain in the elderly.

Other critical diagnoses must be considered when assessing a patient with possible myocardial ischemia. Aortic dissection is characterized by a sudden ripping or tearing pain in the back. Pericarditis should be suspected if the pain has pleuritic or positional components. Pulmonary embolism produces a sudden, often pleuritic pain associated with dyspnea. The pain of myocardial infarction usually begins gradually and worsens over a short period of time.

Risk factors for myocardial ischemia must be documented. The risk factors include smoking, diabetes, hypertension, family history and

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hypercholesterolemia. Their presence increases the likelihood that the pain, even if atypical, may be ischemic.

PHYSICAL EXAMINATION

The physical examination in patients with possible ischemic chest pain helps to rule out other diagnoses, establish any complications of ischemia, and provides important information to direct pharmacological and possibly mechanical intervention. In particular, attention should be directed to the appearance, vital signs (including BP in both arms), JVP, chest auscultation, cardiac exam, and peripheral pulses. Inspiratory crackles and an S3 are associated with left ventricular failure. Right-sided heart failure is characterized by an elevated JVP and peripheral edema. A new systolic murmur may indicate a ventricular septal defect or acute mitral regurgitation. An attempt should be made to determine the hemodynamic status as follows:

Killip I: well perfused, chest clear

Killip II: some pulmonary congestion (orthopnea, crackles)

Killip III: pulmonary edema (severe shortness of breath, crackles above scapulae)

Killip IV: poor perfusion, cardiogenic shock

All patients with acute inferior MI's, in particular, should be examined for signs of right ventricular infarction. These signs include low BP, clear chest, distended JVP.

ECG

The standard 12 lead ECG remains the most definite early test to document an acute MI. It should be obtained as soon as possible in patients with any type of chest pain, epigastric pain, dyspnea, or other symptoms which may accompany an acute MI (diaphoresis, nausea, vomiting, acute confusion or stroke in the elderly). Unfortunately, the ECG may be either completely normal or may only show nonspecific changes when the patient first presents. If the initial ECG is not diagnostic, repeat ECGs should be done in the ED, particularly during repeated episodes of pain, or if the patient describes a worsening of the pain while in the department.

A 15-lead ECG is a standard 12-lead ECG with 3 additional leads - V4R, V8, and V9. The V4R lead is particularly important to diagnose right ventricular infarctions and should be done on all patients with inferior wall infarctions. The V8 and V9 leads offer a

direct look at the posterior wall of the myocardium. This allows one to diagnose a true posterior wall infarction (ST elevation) when there is 'indirect' evidence on a standard 12-lead ECG (a tall R wave, ST segment depression, and an upright T wave in leads V1 and V2).

Early and subtle ECG findings in acute MI include the following:

- increase in R wave voltage, usually in the precordial leads.
- hyperacute T waves, usually in the precordial leads. A normal T wave is asymmetric with steeper descending than ascending limb. An abnormal T wave is symmetric and prominent (usually over 10 mm in precordial leads). Other causes of hyperacute T waves include LBBB, hyperkalemia, LVH, and subarachnoid hemorrhage.
- ST segment elevation, suggesting a current of injury and acute transmural ischemia. There are other causes of ST segment elevation: pericarditis, benign early repolarization, hyperkalemia, LV aneurysm, LBBB, LVH, and hypothermia. In acute transmural MI, ST segment elevation occurs almost immediately and helps localize the area of infarction:

II, III, AVF:	inferior MI
V1-V3:	anteroseptal
1, AVL, V4 –V6:	lateral
V1-V6:	anterolateral
V8,V9:	posterior

In addition, the amount and distribution of ST elevation can be used to quantify the infarct. The higher the ST elevation and the more leads involved, the worse the prognosis. Greater benefits from treatment are found with extensive infarcts.

Q waves are a sign of extensive myocardial ischemia and frank necrosis. Whereas ST segment elevation occurs almost immediately in acute transmural infarction, Q waves usually require several hours to develop. Nontransmural (or subendocardial) infarction may result in ST segment depression and T wave inversion without Q wave development. Alternatively, ST segment depression may represent 'reciprocal' changes of a current of injury in another site of the heart.

Example ECGs, illustrating various myocardial infarctions can be found at the end of this chapter (see Figures 1-7).

INVESTIGATIONS

An early Chest X-ray (CXR) is not a priority in the assessment and treatment of patients with acute MI. When aortic dissection is a possibility in a patient presenting with chest pain, a CXR should be done immediately to look for a widened mediastinum and a pleural effusion. In MI, the CXR is usually normal, but may confirm clinical evidence of congestive heart failure.

Trying to rule out myocardial ischemia by giving the patient a ‘pink lady’ in the ED is fraught with hazard and must be avoided. A diagnostic challenge looking for relief of epigastric pain with an antacid/viscous lidocaine preparation is unreliable, insensitive, and nonspecific. Sending patients home based on their response to such a challenge is a classic medical-legal disaster.

Positive cardiac enzymes continue to be the mainstay of our ability to rule in or rule out a diagnosis of myocardial infarction when the initial ECGs are normal or non-specific. Cardiac troponin assays are widely available, but rises in troponin will not occur until at least 2 to 6 hours after the onset of symptoms; therefore, a single negative cardiac enzyme in a patient who presents with chest pain of recent duration cannot be used to rule out myocardial infarction. Serial serum troponin values, done at least 6 to 9 hours after initial presentation to the Emergency Department, approaches 100% sensitivity in ruling out myocardial infarction (but not unstable angina) in patients without diagnostic ECG changes. Troponin levels peak in 12 to 24 hours after infarction. The troponin testing is also limited by poor specificity. Renal failure and sepsis are examples of common conditions that can elevate troponin levels. The sensitivity of serial troponin testing is always better than obtaining a single sample.

The prime benefit of the cardiac troponins seems to be for prognostic value and for risk stratification. Patients with either unstable angina or myocardial infarction who present with higher troponin levels have higher short and long term mortality rates.

COMPLICATIONS OF ACUTE MI

- Arrhythmias - out of hospital mortality is almost entirely due to arrhythmia.
- Conduction disturbances - AV blocks
- LV pump failure - cardiogenic shock, assess Killip status

- Mechanical defects - cardiac rupture, VSD, papillary muscle dysfunction
- Thromboembolism - can be prevented with heparin SC or IV depending on the clinical scenario
- Pericarditis - an acute form of pericarditis occurs 5-7 days following an acute MI; the post-myocardial infarction syndrome (Dressler's) occurs much later.

CLINICAL PEARLS

Treatment Priorities in ST Elevation MI (STEMI)

1. ASA
2. Thrombolysis or PCI ASAP
3. Heparin or LMWH or Fondaparinux
4. Clopidogrel (or Ticagrelor)

MANAGEMENT

Stabilize

Oxygen, IV, cardiac monitor, ECG ‘STAT’

Pharmacotherapy

ASA

- 2-4 baby aspirin chewed
- up to 21% decrease in mortality

Nitroglycerin (NTG)

- increases coronary blood supply
- decreases preload, afterload
- use SL (sublingual) initially, 0.3mg sl x 3
- IV NTG **only** if persistent pain, CHF, hypertensive
- Slow infusion if hypotension occurs

Caution Regarding the Use of Nitroglycerin in Inferior MI

Right ventricular infarctions occur in approximately 1/3 of all inferior MI's, suggesting more proximal occlusions of their right coronary artery. Right ventricular (RV) ischemia results in decreased RV stroke volume, impairing filling of the left ventricle. Cardiac output is reduced, and systemic BP drops. It is therefore critically important to avoid drugs that result in venodilatation if the patient might have RV infarction. These patients are preload dependent, and drugs such as NTG may precipitate dramatic hypotension. Suspect RV infarction on all patients with inferior MI's. Do right-sided precordial leads (specifically V4R) looking for ST elevation.

Reperfusion therapy: Thrombolysis

Time is a crucial factor in the treatment of patients with acute myocardial infarction because of the demonstrated benefit of early treatment with thrombolytic therapy within the first hour or two after the onset of symptoms. Although thrombolytic therapy may be beneficial up to 12 hours after symptom onset, early therapy achieves maximal myocardial salvage and helps preserve left ventricular function. Since ‘time is muscle’, the primary objective should be to initiate thrombolysis within 30 minutes of presentation to the ED on all eligible patients. Thrombolytics should be administered by the Emergency Physician as soon as eligibility is determined, in centres that do not have facilities for PCI, and are far from a centre that can perform PCI in a timely fashion.

Thrombolytics activate plasminogen to lyse clot. They are indicated for all acute infarcts with ST elevation >1mm in two contiguous leads. The role of thrombolytics in the presence of a left bundle branch block (LBBB) is controversial. It may be acceptable to initiate lysis (or PCI) if the LBBB is new and the clinical suspicion is high. It is not indicated if the ECG is normal or shows ST segment depression. Thrombolysis is indicated routinely up to 12 hours post onset of pain; however, the earlier the administration, the greater the benefit. There is no age limit (in the elderly, although there is a greater hazard associated with thrombolytic therapy during the early stages of treatment, the later benefit is also greater, resulting in greater absolute benefit).

Contraindications to Thrombolytic Therapy

- recent major surgery or any active internal bleeding
- history of hemorrhagic strokes, or any recent stroke
- severe uncontrolled hypertension
- known intracranial neoplasm, AVM, or aneurysm
- suspect aortic dissection

Complications of Thrombolysis

Stroke: Risk factors for intracranial hemorrhage include

- age over 65
- body weight less than 70 kg
- BP > 165/95 on admission

Arrhythmias: 50% of patients who reperfuse have accelerated idioventricular rhythm, which usually requires no treatment.

Tenecteplase (TNK) is presently the most commonly used thrombolytic agent. It has the advantage of single bolus administration.

Reperfusion Therapy: Primary Angioplasty (PCI)

Recent studies have shown a trend toward lower mortality, less reinfarction, and fewer strokes in patients treated with immediate angioplasty compared to thrombolysis. This benefit is probably restricted to those patients with longer (> 3 hours) duration of symptoms because of the very good efficacy of thrombolytics when the chest pain has been of short duration. It is preferred over thrombolysis in those facilities capable, and if immediately available. In addition, emergent angioplasty should be considered early on all patients with AMI in cardiogenic shock. Survival rates are approximately 50% if performed within the first 18 hours of symptom onset. In patients with cardiogenic shock there is no proven benefit to thrombolysis, probably due to the lack of coronary perfusion.

Recent ACC/AHA guidelines made the following recommendations regarding reperfusion therapy:

1. Patients presenting to a hospital with PCI capability should be treated with primary PCI within 90 minutes of first medical contact.
2. Patients presenting to a hospital without PCI capability and who cannot be transferred to a PCI centre and undergo PCI **within 90 minutes** of first medical contact should be treated with fibrinolytic therapy within 30 minutes of hospital presentation. (Many hospitals within the GTA now have protocols called “Code STEMI” which allows for immediate transfer of a STEMI patient by EMS to a centre that can perform emergent PCI)

Unfractionated Heparin

Following TNK, heparin is indicated to prevent re-thrombosis. It should be initiated immediately following the thrombolytic. Traditionally, IV heparin has been used but it is reasonable to use low molecular weight heparin (LMWH), and perhaps even preferable.

Heparin may be indicated in other infarctions where thrombolysis may not be indicated; e.g., “old” infarctions (more than 12 hours old), non-Q wave MI, and patients with unstable angina. In these patients however, low molecular weight heparin is preferred.

Low Molecular Weight Heparins (LMWH)

There are pharmacologic/pharmokinetic advantages over unfractionated heparin:

- more predictable anticoagulant effect
- no need for PTT monitoring
- lower rates of thrombocytopenia
- better bioavailability

LMWH, (such as enoxaparin or dalteparin) should be used instead of unfractionated heparin in patients with unstable angina and non-Q-wave MI.

Fondaparinux, a synthetic factor Xa inhibitor, has also been shown to be effective as anticoagulant therapy following reperfusion therapy and may be associated with decreased risk of serious bleeds.

Beta Blockers

Beta blockers decrease oxygen demand and catecholamine effects. Trials done in the pre-thrombolytic era suggested they reduced mortality. A recent large trial suggested that beta blockers reduce arrhythmias such as ventricular fibrillation, but may increase the incidence of shock.

- CAUTION: asthmatic/bronchospasm, CHF, bradycardia, heart block, hypotensive, RV

infarcts, any patient with possible low output state or incipient shock.

Clopidogrel/ Ticagrelor/Prasugrel

Clopidogrel is a potent antiplatelet agent that works differently than ASA. It has proven benefits in ACS with new ECG changes or new ECG findings. It has proven efficacy in reducing events in ST elevation MI (STEMI) when added to ASA.

- Dose for patients 75 and under: 300 mg as adjunctive therapy to TNK; 600 mg as adjunctive therapy to PCI
- Dose for patients over 75: 75 mg a day (no load). The earlier the clopidogrel is given, the better
- Ticagrelor/Prasugrel: alternative antiplatelet agents which have uses in certain situations.

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Figure 1: Acute antero-lateral wall infarction. Dramatic convex ST elevation in leads V₁₋₄, I, aVL; reciprocal ST depression in leads II, III, aVF.

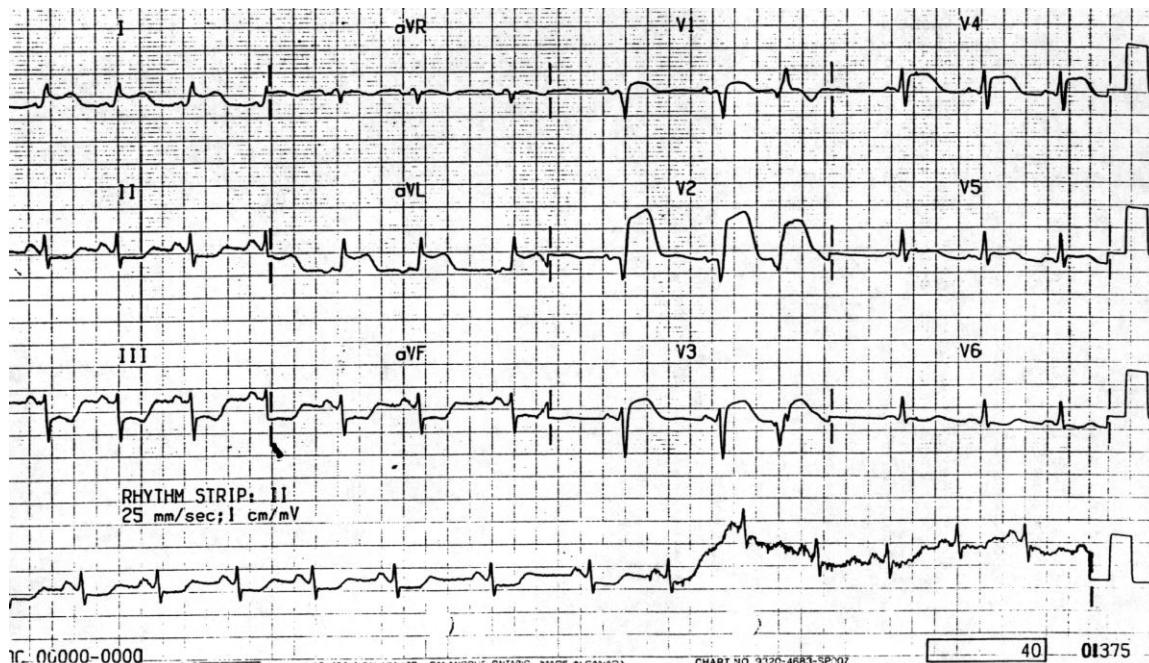


Figure 2: Acute anterior wall infarction. Striking ST segment elevation in leads V₂ through V₅. ST segment score (sum of ST elevation in precordial leads) approximately 17 mm: “extensive” infarct. The more extensive the infarct, the greater benefit from reperfusion (either mechanical or pharmacologic)

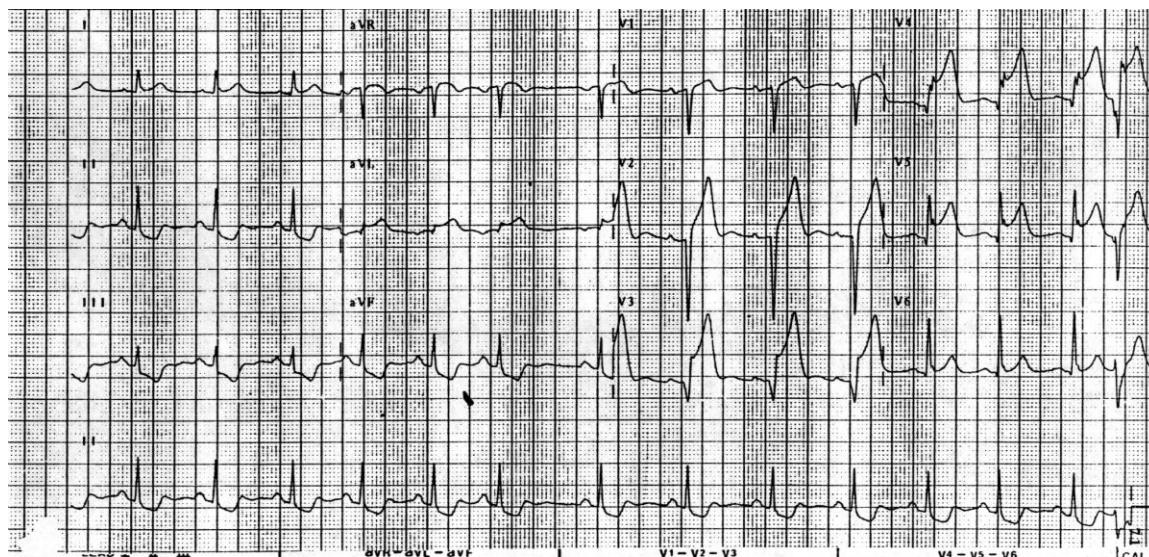


Figure 3: Acute inferior wall infarction. ST segment elevation in II, III, aVF with ST depression in leads V₁₋₃. Patients presenting with inferior infarctions associated with such precordial ST segment depression have larger myocardial infarctions than patients without ST segment depression.

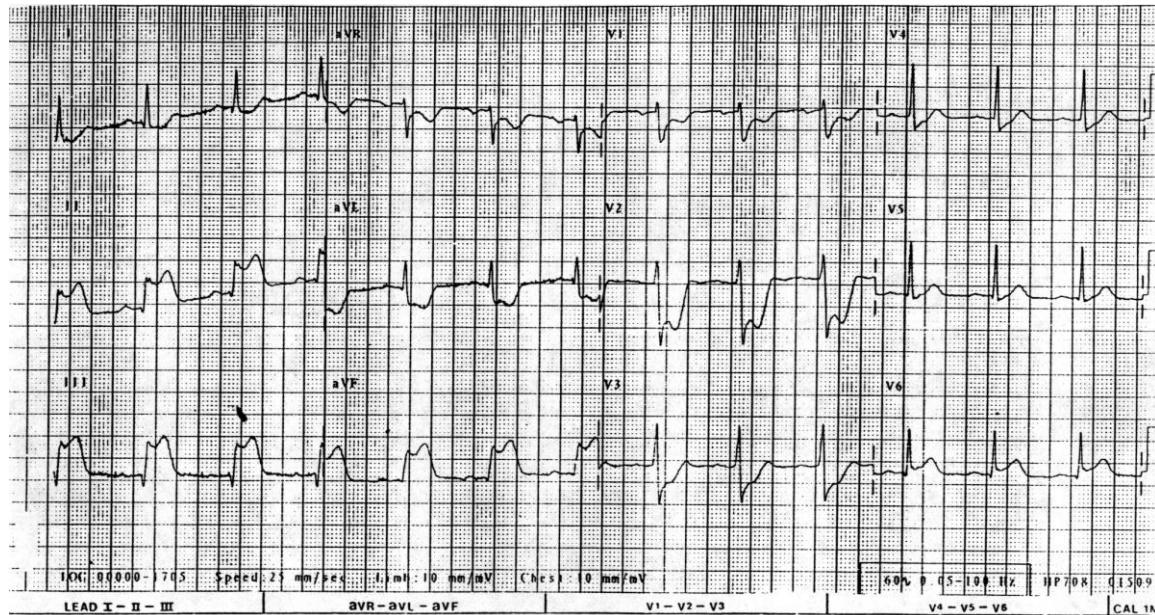
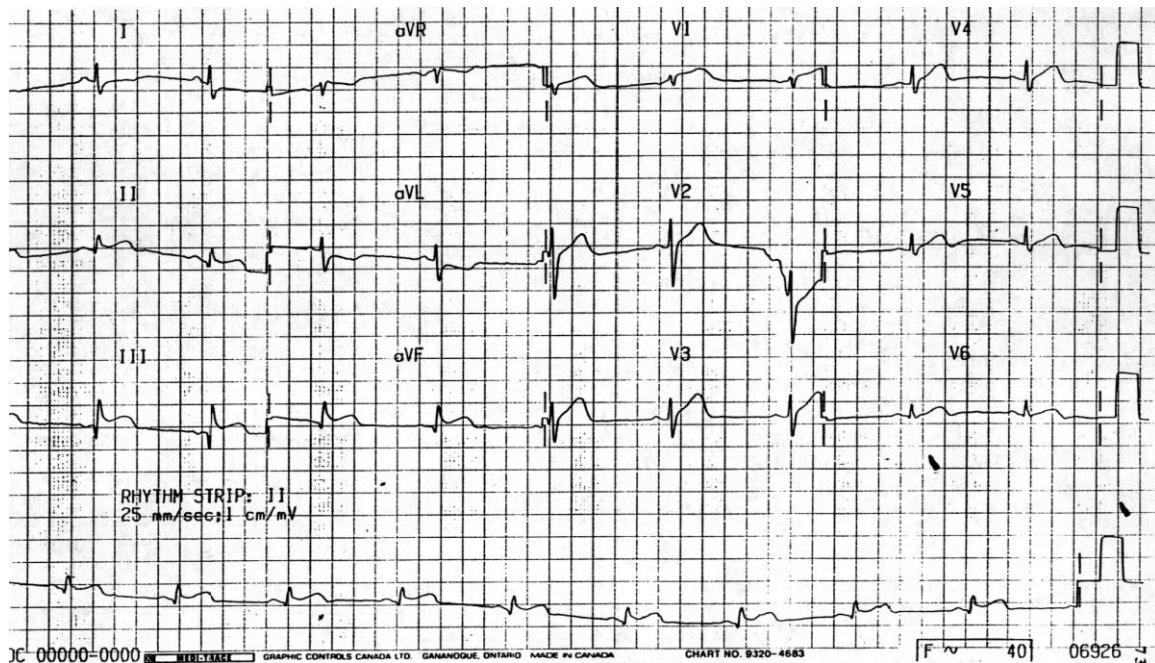


Figure 4: Acute inferior wall infarction. Note subtle ST elevation in leads II, III and aVF with no reciprocal ST depression.



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Figure 5: Acute inferior wall infarction with complete heart block. There is a markedly increased in-hospital mortality in patients whose inferior MI's are complicated by the development of second or third degree (high degree) heart block.

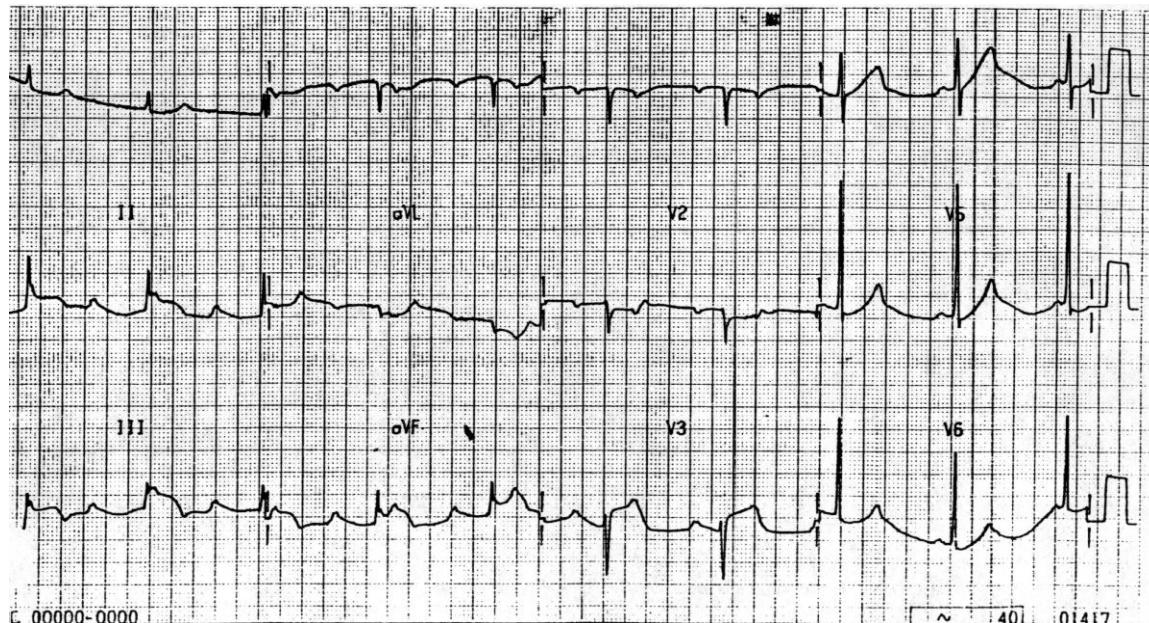


Figure 6: 15 lead ECG on same patient as Figure 5. The V₄, V₅ and V₆ leads have now been shifted to the V_{4R}, V₈ and V₉ positions and the ECG repeated. Note the ST elevation in lead V_{4R}, reliably identifying right ventricular infarction. It signifies proximal occlusion of the right coronary artery, and in consequence, a high incidence of AV block. This patient benefits dramatically from aggressive pharmacologic or mechanical reperfusion strategies.

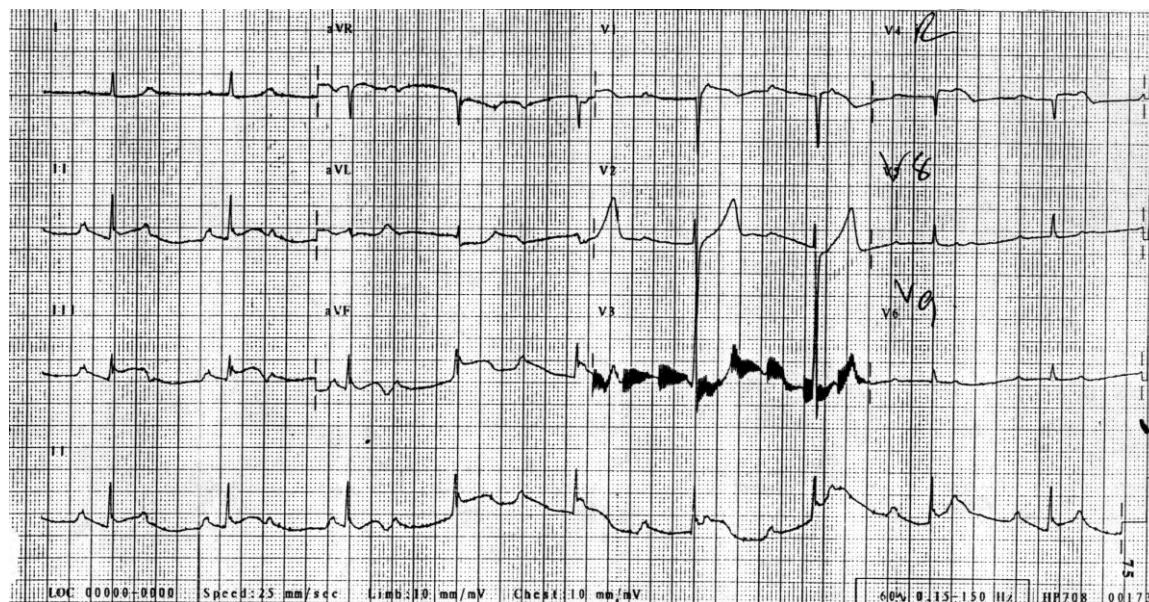
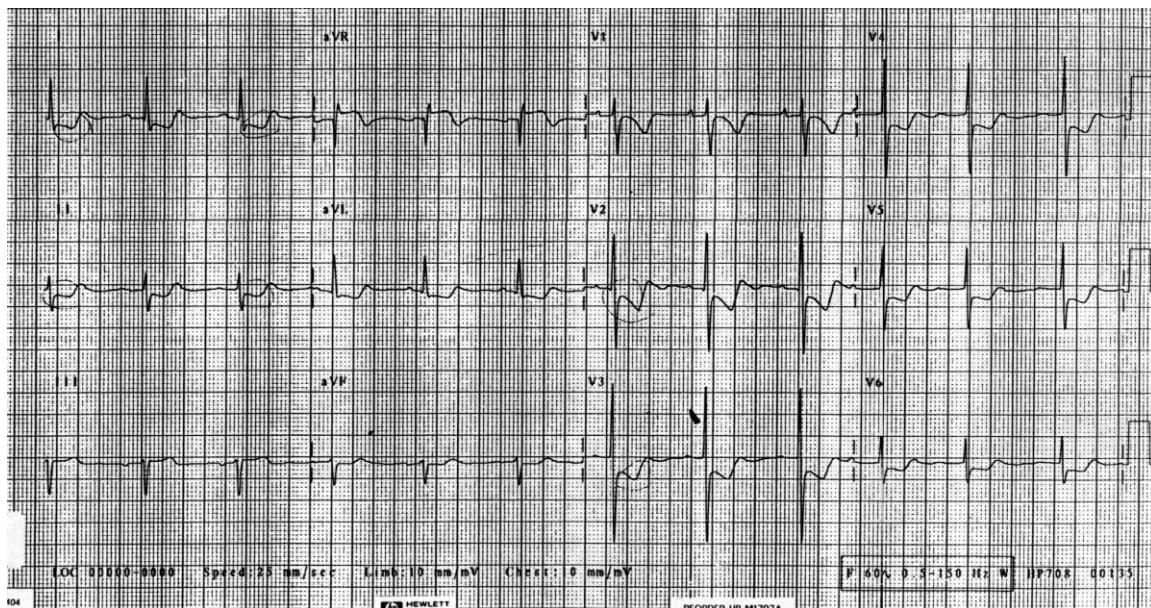


Figure 7: ST segment depression in a 68 year old male with chest pain. The patient was admitted to the CCU with a diagnosis of “unstable angina” and started on IV heparin and IV nitroglycerin. The prominent R wave, ST depression and upright T waves in leads V₁ and V₂ actually suggest posterior wall infarction. Had a 15 lead ECG been done, it may have revealed ST segment elevation in leads V₈ and V₉.



12: ACUTE CONGESTIVE HEART FAILURE

Dominick Shelton

OBJECTIVES

1. To understand the physiologic basis of how acute congestive heart failure (CHF) occurs
2. To understand the difference between causes of CHF and precipitants of acute CHF
3. To describe the clinical presentation, investigations and management of acute CHF

INTRODUCTION

CHF is a clinical syndrome that occurs when cardiac pump function does not adequately meet the circulatory demands of the body. It can be categorized according to the rapidity of onset (acute vs. chronic), which has implications for management. Pulmonary edema should be conceptualized as an extreme on the continuum of severity of CHF and is often an acute worsening of chronic heart failure. CHF has a significant mortality rate, and recurrences requiring hospitalization are frequent. It is therefore no surprise that emergency physicians encounter patients with CHF on a regular basis.

PATHOPHYSIOLOGY

As cardiac output decreases, reflex arterial vasoconstriction redistributes blood flow so that the brain and heart remain well perfused. The resulting drop in renal blood flow activates the renin-angiotensin-aldosterone system, causing increased sodium and fluid retention. Decreased cardiac output also results in an increase in left atrial and left ventricle filling pressures, which is in turn reflected in elevated pulmonary capillary pressures. The consequence of these elevated pressures is transudation of protein-poor fluid into the pulmonary interstitium and alveolar spaces. As the right ventricular volume and pressure increases, the systemic venous and capillary pressures rise and fluid transudates from the vascular to the interstitial space causing peripheral edema.

Heart failure can be subdivided into systolic and diastolic dysfunction. Systolic dysfunction is characterized by a dilated left ventricle with impaired contractility. Diastolic dysfunction refers to an increase in ventricular stiffness (reduced compliance)

and impaired relaxation that impedes ventricular filling during diastole. The net effect of diastolic dysfunction is an increase in end-diastolic pressure.

ETIOLOGY

Remember that acute CHF is not the primary problem; it is the consequence of an acute decompensation due to an underlying cause. Therefore, if possible, identify and correct the precipitating event while treating acute CHF.

Causes of CHF

- Coronary artery disease
- Hypertension
- Valvular heart disease
- Cardiomyopathy (dilated, hypertrophic, restrictive)
- Pericardial disease
- Metabolic disorders (e.g. hypothyroidism)
- Viral myocarditis
- Toxins

Precipitants of Acute CHF

Cardiac

- Ischemia/infarction
- Tachy- or bradyarrhythmias
- Mechanical complication of acute MI (e.g. papillary muscle rupture)

Medications

- Negative inotropes (beta-blocker, calcium channel blocker)
- NSAIDS
- Steroids
- Pharmacological non-compliance

Dietary

- Increased sodium intake

High output

- Anemia
- Infection
- Pregnancy
- Hyperthyroidism
- AV fistula

Other

- Iatrogenic fluid overload (e.g. blood transfusion)
- Renal failure
- Hypertensive crisis

- Drug or alcohol abuse

Symptoms of CHF

- Dyspnea
- Orthopnea, paroxysmal nocturnal dyspnea
- Cough, wheezing
- Fatigue, generalized weakness
- Edema of lower limbs, increased abdominal girth

CLINICAL PEARL

A complete cardiovascular assessment should be done on any patient suspected of having CHF, including a review of systems and physical exam of the cardiovascular system.

Signs of CHF

- Tachypnea, tachycardia, hypertension or hypotension, hypoxia
- Crackles, wheezes
- S3, S4
- Jugular venous distension, hepatojugular reflux
- Hepatomegaly
- Dependent edema
- Weak peripheral pulses, cool extremities, i.e. hypoperfusion

INVESTIGATIONS

Chest X-Ray

CXR is the most useful and readily accessible tool for diagnosing CHF. The following are radiographic findings in CHF: cardiomegaly, increase in prominence of upper lung fields (pulmonary vascular redistribution), interstitial edema and obliteration of vasculature structures, kerley B lines (1-2 cm lines perpendicular to the pleura and continuous with it, representing edematous septal lines), pleural effusions (usually absent in acute pulmonary edema).

ECG

An ECG may identify underlying conditions that predispose to CHF, such as left ventricular hypertrophy, left atrial abnormalities, myocardial ischemia, or the presence of an arrhythmia (e.g. atrial fibrillation).

Troponin

In the setting of acute CHF, an elevated troponin cardiac marker can occur with myocardial necrosis due to an acute MI. However, troponins can also be elevated in acute CHF without ischemia. Elevated

cardiac troponin levels are associated with an increased risk of morbidity and mortality in both acute and chronic CHF.

Echocardiogram

Two-dimensional and Doppler echocardiography enables assessment of ventricle size, wall motion, ejection fraction, systolic and diastolic function, valvular function, and pericardial disease. It can be used to estimate right atrial, pulmonary artery and pulmonary capillary wedge pressures. Echo results may be available from previous studies, or occasionally may be performed in the emergency department.

Natriuretic Peptides

Brain natriuretic peptide (BNP) and amino-terminal fragment of the prohormone (NT-proBNP) are elevated in heart failure and have been established as biomarkers which aid in the diagnosis and prognosis of heart failure. These tests are most useful in patients whose clinical assessment and CXR are suggestive, but not clearly diagnostic of heart failure.

Renal Function

Renal dysfunction is prognostic of adverse outcome in patients with acute CHF. In the presence of severe fluid overload, renal dysfunction may improve with diuresis. Alternatively, diuretic therapy may further worsen renal function in patients with baseline renal insufficiency.

Sodium

Hyponatremia due to volume overload is common among CHF patients and the degree of reduction in serum sodium parallels the severity of CHF. As a result, a low serum sodium is an adverse prognostic indicator.

Coronary Angiography

Early coronary angiography and revascularization is indicated in patients with acute CHF and acute coronary syndrome.

Swan-Ganz Catheterization

This investigation involves pulmonary artery catheterization and measures pulmonary artery pressure, pulmonary artery wedge pressure, central venous pressure and cardiac output. It is used in critical care units in patients with pulmonary edema and hypotension who require titration of inotropic and vasopressor agents. It is not routinely used in patients with acute CHF.

MANAGEMENT

General

- ABC's
- Place patient in upright position
- 100% oxygen non-rebreather facemask
- Cardiac monitor, oxygen saturation monitor
- IV access
- +/- Foley catheter
- Diuresis
- Vasodilator therapy

Specific

First line therapy:

- Nitroglycerin 0.4 mg sublingual q 5 min. (if sBP > 100) +/- Nitrodur 0.4 - 0.8 mg/hr topical
- Furosemide 40 - 100 mg IV (if sBP > 100)

If inadequate response to first line therapy:

- Double furosemide dose
- Nitroglycerin infusion (use especially if angina or hypertensive), start at 5 – 10 mcg/min and titrate rapidly to response
- Consider CPAP or Bi-PAP if respiratory distress or hypoxic on 100% O₂
- If decreased LOC or impending respiratory failure, intubate and ventilate
- If sBP < 90 (and persistent after holding medications) and no signs of shock, then start dobutamine 2 mcg/kg/min and titrate to response
- If sBP < 90 and signs of shock, then start dopamine at 5 mcg/kg/min and titrate to response
- If severe/refractory hypertension, start nitroprusside at 0.5 mcg/kg/min and titrate to response

Diuretics and vasodilators are the mainstay of treating acute congestive heart failure. The aggressive use of each therapy depends on the patient's volume and hemodynamic status. For example, patients with flash pulmonary edema due to hypertension require aggressive vasodilatory therapy. Patients with normal blood pressure and volume overload best respond to a combination of diuretic and vasodilator. Patients with hypotension and intravascular overload may respond to diuretics alone or in combination with vasopressors and inotropes.

CLASSES OF MEDICATIONS

Nitrates

Nitroglycerin is one of the first line medications for acute CHF since it works the fastest to relieve pulmonary congestion. Nitroglycerin decreases

pulmonary vascular pressure by causing venodilation, which results in decreased preload. Higher doses of nitrates cause arterial vasodilation, therefore also decreasing afterload. A decrease in afterload, by increasing cardiac output, further relieves pulmonary congestion. Nitroglycerin has the additional benefit of relieving cardiac ischemia, which may be a precipitating factor in acute CHF. Nitroprusside provides more arterial vasodilation than nitroglycerin and is used in patients with severe hypertension associated with pulmonary edema or inadequate response to nitroglycerin. Nitrate use is contraindicated after use of a PDE-5 inhibitor such as sildenafil.

Diuretics

By reducing intravascular volume, diuresis will eventually lower pulmonary and systemic pressures. Peak diuresis typically occurs 30 minutes after administration. IV loop diuretics also cause an early (5 – 30 min) venodilation that can decrease pulmonary congestion prior to the onset of diuresis. The IV dose should be equal to or greater than (up to 2.5 times) their maintenance oral dose.

Inotropes/Vasopressors

Positive inotropes are used for patients with hypotension and pulmonary edema. This class of drugs improves cardiac contractility, thus improving cardiac output and systemic perfusion. Inotropes also have the potential to increase ventricular arrhythmias and decrease pulmonary and systemic vascular resistance (thus lowering BP). Dobutamine is recommended in patients with hypotension who do not have clinical evidence of shock. Milrinone is a phosphodiesterase inhibitor that increases myocardial inotropy. It is used when a patient is on beta blockers, since dobutamine requires the beta receptor for its inotropic effects.

Dopamine is recommended in patients with hypotension who also have clinical evidence of shock. Dopamine has both inotropic and vasopressor effects which occur in a dose dependent manner. Thus dopamine can increase both cardiac output and blood pressure. A disadvantage of dopamine is that it is more arrhythmogenic and raises the heart rate, which increases the risk of myocardial ischemia.

Calcium Channel Blockers

Diltiazem may be used in acute CHF when rapid atrial fibrillation is also present. It is given as a dose of 0.25 mg/kg IV bolus over 2 min. If no response after 10 – 15 min., 0.35 mg/kg over 2 min. is administered. Verapamil should be avoided due to its potent negative inotropic effect.

Nesiritide

Nesiritide is recombinant human brain natriuretic peptide. It causes vasodilation and is used in selected patients who remain symptomatic despite routine therapy.

ADJUNCTIVE THERAPIES**CPAP or Bi-PAP**

Non-invasive positive pressure ventilation should be considered for patients with acute CHF in respiratory distress (tachypnea, laboured breathing, accessory muscle use) or persistent hypoxia despite 100% supplemental oxygen. This method of ventilation creates positive intrathoracic pressure that decreases both preload and afterload and improves oxygenation. It often avoids the need for intubation, permitting the time necessary for medications to work.

Intubation & Mechanical Ventilation

Endotracheal intubation may be warranted if non-invasive positive pressure ventilation fails to improve tissue oxygenation, if the patient has a decreased level of consciousness, or if the patient has experienced respiratory failure.

Ultrafiltration

For patients in severe and refractory pulmonary edema, ultrafiltration is an alternative method of fluid removal and is required in patients with severe renal insufficiency.

Mechanical Cardiac Assistance

The two mechanical modalities used are intra-aortic balloon pump and an internally implanted left ventricular assist device. These modalities are reserved for patients with severe pulmonary edema who are also in cardiogenic shock, refractory to standard treatment previously outlined and who may be considered heart transplant candidates.

ADDITIONAL MEDICATIONS NOT ROUTINELY USED**Digoxin**

Digoxin is used to treat chronic CHF and is most effectively used in CHF to control the ventricular rate when atrial fibrillation is also present. It has a slow onset of action and is not routinely used in the emergency department.

Beta Blocker

Beta blockers have proven benefit in the management of chronic CHF. In patients with systolic dysfunction, they have the potential to worsen acute heart failure.

ACE Inhibitor & Angiotensin Receptor Blocker

These medications have proven benefit in the management of chronic CHF. IV ACEI (enalaprilat) may have deleterious effects in patients with an acute MI, especially when complicated by heart failure. Early use in the management of acute CHF is not recommended. These medications are contraindicated in patients with hypotension and renal insufficiency.

Morphine

Morphine reduces patient anxiety, decreases work of breathing and reduces both preload and afterload. It should be used with caution in patients with impending airway compromise since it may result in the need for intubation due to respiratory depression.

RISK STRATIFICATION

Using the Acute Decompensated Heart Failure National Registry of U.S. patients hospitalized with acute CHF, 33,046 hospitalizations were analyzed to develop a model and then the model was validated using data from 32,229 subsequent hospitalizations. The best single predictor for mortality was high BUN ($>15.35 \text{ mmol/L}$), followed by low admission systolic BP ($<115 \text{ mmHg}$) and then by high levels of serum creatinine ($>243 \text{ mcg/L}$). All 3 prognostic factors were determined at the time of emergency department presentation.

HOSPITALIZATION

Hospital admission is recommended for patients with acute CHF and the following clinical features:

- Severe decompensated CHF
- Hypotension
- Worsening renal function
- Altered mentation
- Dyspnea at rest
- Hypoxia
- Hemodynamically significant arrhythmia
- Acute coronary syndrome
- Major electrolyte disturbance
- Comorbid conditions (e.g. pneumonia, PE, DKA)
- TIA or stroke
- ICD firings
- Previously undiagnosed CHF

SUMMARY

- Acute CHF occurs when cardiac pump failure is severe enough to cause increased pulmonary pressures, which results in increased lung fluid
- If possible, identify and correct the precipitating event while treating acute CHF
- Patients with pulmonary edema present in respiratory distress
- CXR reveals many characteristic features of pulmonary edema
- In patients presenting with dyspnea whose clinical assessment and CXR are suggestive, but not

clearly diagnostic of CHF, the BNP level is often helpful in ruling in or ruling out CHF

- Management of patients presenting in pulmonary edema requires stabilization of ABC's, diuresis, and vasodilator therapy
- Patients who are in acute CHF and hypotensive require inotropes and/or vasopressors

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13: CARDIAC DYSRHYTHMIAS

Laurie Mazurik
Updated by Peter Switakowski

OBJECTIVES

1. To learn an approach to reading a rhythm strip
2. To recognize basic cardiac rhythms
3. To classify cardiac rhythms as stable or unstable
4. To know when to call a ‘code’

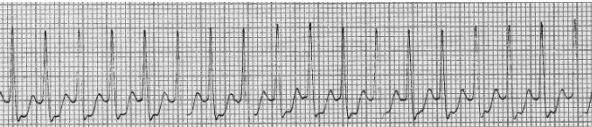
INTRODUCTION

This brief overview of cardiac dysrhythmias is not intended as a substitute for the more complete instruction offered by the American and Canadian Heart Associations' ACLS course. The latest Advanced Cardiac Life Support algorithms were published in 2010 in Circulation.

BASIC RHYTHM RECOGNITION

Some dysrhythmias are easier to recognize than others. For those that are more difficult to assess, be consistent and systematic. There are five questions that need to be answered when looking at a rhythm strip in order to determine if a dysrhythmia is present and to identify the particular arrhythmia.

1. Wide or narrow?

	<p>Even at a distance you can usually have a sense if the QRS complex is wide or narrow. A wide QRS is >0.12 seconds and means either the patient has a bundle branch block or a ventricular dysrhythmia. If the patient is stable, a 12 lead ECG may help determine if it is ventricular tachycardia (VT). All wide complex tachycardias should be considered as VT until proven otherwise.</p>
	<p>Narrow complex. In general narrow complex rhythms are benign, but some may require treatment.</p>

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2. Fast (tachycardia) or slow (bradycardia)?

In general we classify abnormal rates as being tachycardic (>100) or bradycardic (<60). In an adult at rest, sinus tachycardia seldom goes above 140 beats per minute (bpm), and sinus bradycardia seldom falls below 50 bpm. Cardiac dysrhythmias usually cause symptoms because of their rate. Although many machines will tell you the rate, you should know how to measure it. One method is the “Rule of 300” - count the number of large boxes between two R waves and divide that number into 300. Take a look at the examples below.

	<p>There are just over 2 large boxes between 2 R waves; therefore the ventricular rate is just under 150 bpm – approximately 140 bpm. Likely sinus tachycardia.</p>
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If the rhythm is slow and/or irregular, look at the number of complexes in a 6 second interval and multiply by 10.

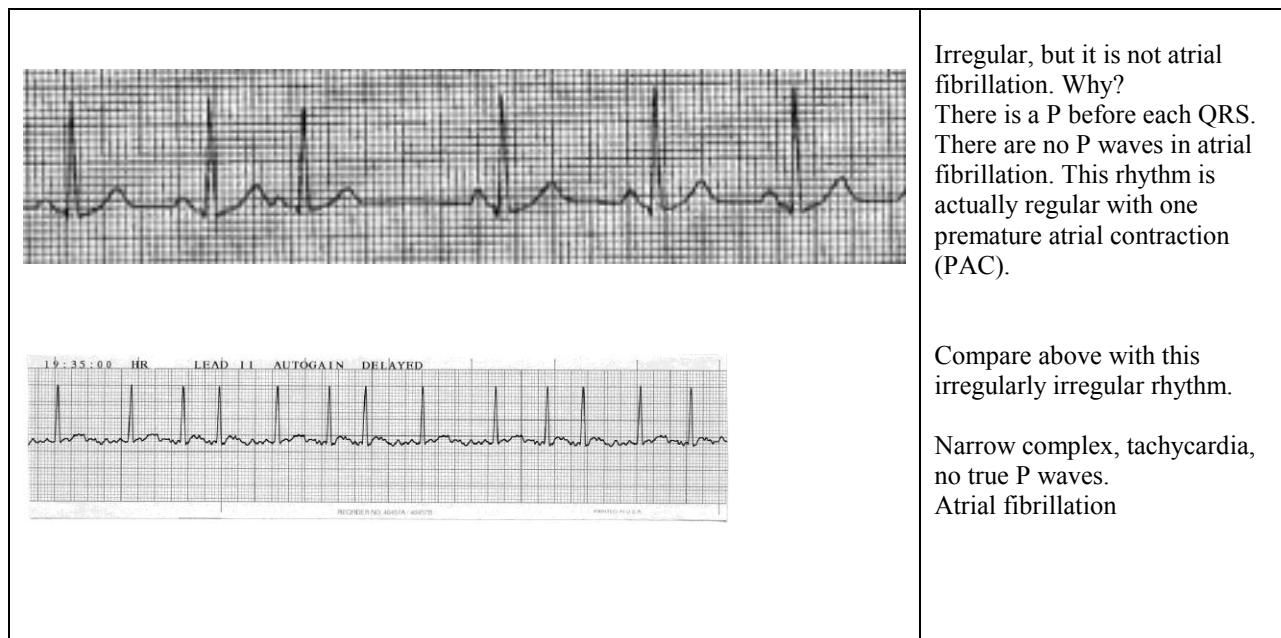
	<p>If this is a 6 second interval, there are 17 complexes in it. $17 \times 10 =$ ventricular rate of 170 bpm.</p>
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You can also apply the “Rule of 300” to P wave rates (provided you can see the P’s). This helps determine if there is a heart block. For example, 2 P’s for each QRS = 2:1 block. Evaluate the following strip for P wave rate

	<p>2 P waves have 1 large block between them $=300/1=300$. The P wave rate is 300. Each QRS has approximately 4 large squares between them $=300/4=75$. Conduction block is 4:1 (4 P’s for every QRS). 1 P must be hidden in the QRS and one is on the T-wave. Note the additional “peak” on the first wave form following the QRS.</p>
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3. Regular or irregular?

Very few rhythms are irregular. Atrial fibrillation is the most common and is irregularly irregular. Is the rhythm below regular or irregular?



4. Are there P waves?

Sometimes the P waves are obvious.

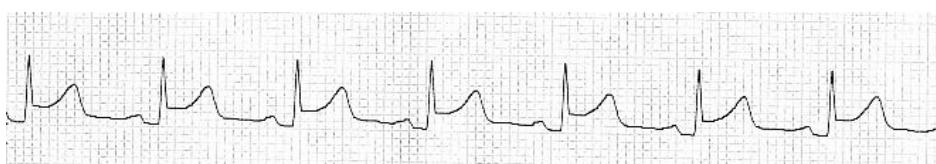


There are P waves seen before each complex.
Sinus rhythm

Sometimes they are hard to see.



These P's are difficult to see, but there appears to be a tiny inverted P just after the S and another inverted P just before the Q. The P wave rate is approximately 300. The ventricular rate is approximately 150. This patient is in atrial flutter with a 2:1 block.

5. Are the P waves related to the QRS, (i.e. are they conducted)?

In the first strip there are P waves seen before each complex, and a QRS after every P wave. This is sinus rhythm. A 12 lead ECG showed diffuse ST elevation. Patient has pericarditis.



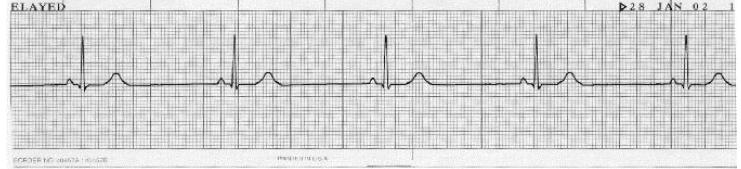
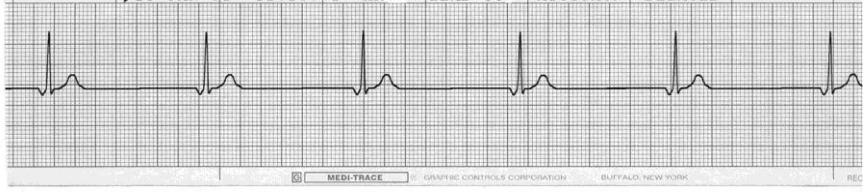
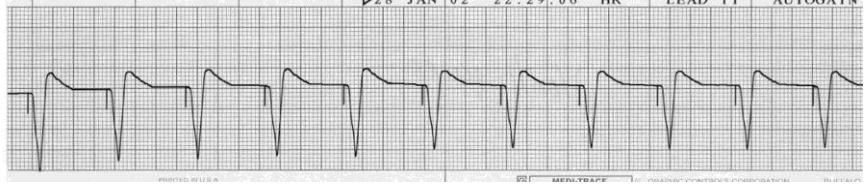
In the second strip, there are P waves but a QRS does not follow them in a consistent manner. They march through unrelated to the QRS - AV dissociation. The P-P and R-R intervals are constant (some P's are hidden in the QRS or T wave). The atria and ventricles are firing independently, but there is no relationship between the P's and QRS - 3rd degree heart block.

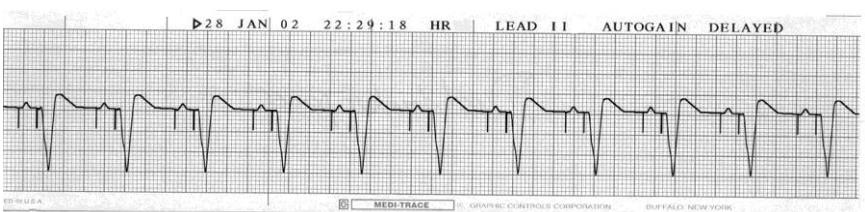
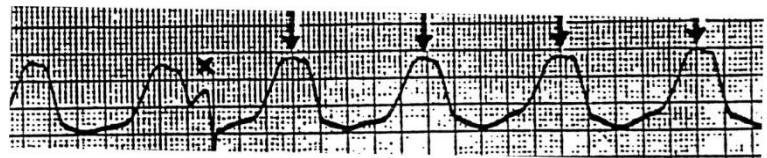
Analyze The Following Rhythm Strips:

Cover the right side of the page and reveal the correct answers as you proceed.

	Check the leads to make sure they are attached. If they are, this is asystole. If they are not attached, re-apply to get the correct rhythm.
	Wide complex tachycardia, regular, no P's seen. Differential Diagnosis: Ventricular tachycardia (VT) vs. Supraventricular tachycardia with aberrant conduction. Treat as VT until otherwise proven.
	Wide complex rhythm, irregular, no P waves seen. Coarse ventricular fibrillation
 NO PULSE!	Regular rhythm, P's before each QRS, rate ~ 100. Patient has no pulse = pulseless electrical activity (PEA). Know the differential diagnoses for this problem (H's and T's). This patient has ST elevation and cause for PEA is Myocardial infarction.
	Wide complex, slow =300/6=50, P's seen but don't appear to be conducted or related to the QRS. Third degree heart block
	Narrow complex, variable rate, irregular, P's seen but not all conducted, constant PR interval with dropped QRS. Second degree heart block, Mobitz type 2 = malignant arrhythmia Can convert to 3rd degree heart block.

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	<p>Narrow complex, variable rate, irregular, P's seen but not all conducted. Progressive lengthening of PR until QRS dropped and progressive shortening of RR Second degree heart block, Mobitz Type I = Wenckebach</p>
	<p>Narrow complex, slow (300/8~38), regular with a P before each QRS. Normal PR interval. Sinus bradycardia. Need to know BP!</p>
	<p>Narrow complex, fast ~180, regular, can't clearly see P's because of rapid rate. Most likely rhythm is paroxysmal supraventricular tachycardia (PSVT). Generally benign. Start with vagal maneuvers such as Valsalva or carotid sinus massage. If no success, try adenosine. Still no success, electrical cardioversion.</p>
	<p>Narrow complex, fast (up to 300 bpm in certain parts of strip), irregularly, irregular, unable to see P waves. Most consistent with rapid atrial fibrillation. At rapid rates can be difficult to distinguish from PSVT. Look for irregularly, irregular pattern. In most rapid atrial fibrillation a normal AV node provides a greater level of block (i.e. ventricular rate seldom goes much above 160). If ventricular rate > 200, think atrial fibrillation with bypass tract (i.e. Wolf Parkinson White = WPW).</p>
	<p>Narrow complex, slow (300/7=~45), regular. Is there an inverted P before R or is it a Q that is seen instead?</p> <p>Junctional bradycardia</p>
	<p>Wide complex, normal rate (300/4 =75), no P's but consistent vertical line before each QRS. Ventricular paced rhythm</p>

	Wide complex, rate 300/4=75 bpm, a P before each QRS, a vertical line before each P and each Q. AV sequential pacemaker.
	CPR

STABLE VERSUS UNSTABLE

Now that you have an approach to help you recognize basic dysrhythmias, it is also important to distinguish the stable from the unstable patient. A stable patient shows no evidence of hemodynamic or cardiac compromise. An unstable patient has clinical evidence of hemodynamic compromise.

Clinical Indicators of Instability

- Hypotension (BP<90)
- Congestive heart failure (or pulmonary edema)
- Chest pain (which may represent coronary ischemia)
- Altered level of consciousness (which may indicate a shock state)

Other signs, such as pallor, diaphoresis and anxiety may be associated with a shock state. This is where experience plays a role. If a

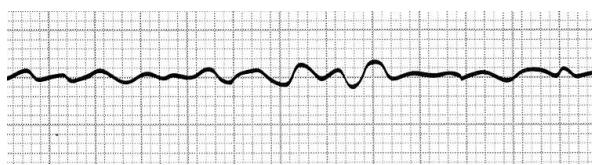
patient's SBP is >90 mmHg but the patient looks ill, an urgent assessment is indicated. Shock is not defined by a blood pressure; rather, it is a state of inadequate end-organ perfusion.

Three Degrees of Urgency In Treatment Response

- Emergent - drop everything and see patient immediately
- Urgent - see within several minutes to prevent an emergent situation
- Deferrable - can wait

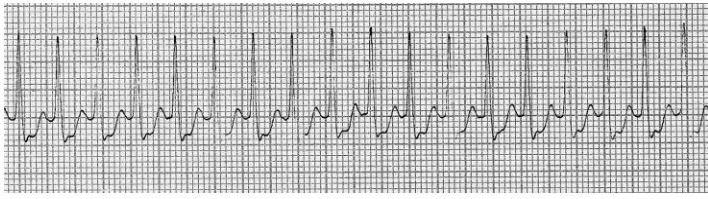
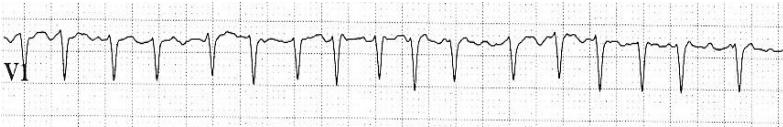
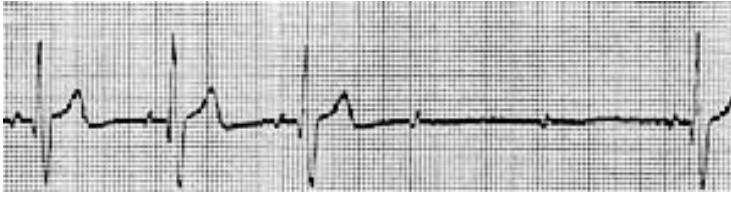
In the following cases identify the rhythm and determine whether the patient's dysrhythmia is stable or unstable. Ask yourself whether treatment response is emergent, urgent or deferrable. Cover the right side of the page and reveal the correct answers after you have decided on these issues for each case.

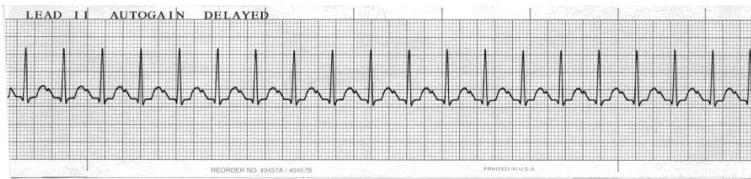
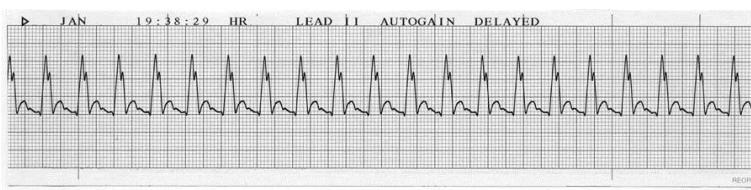
Here's an easy one to start. This patient is pulseless.



Ventricular fibrillation
Unstable
Emergent
Do CPR 2 minutes before defibrillation if unknown down time.

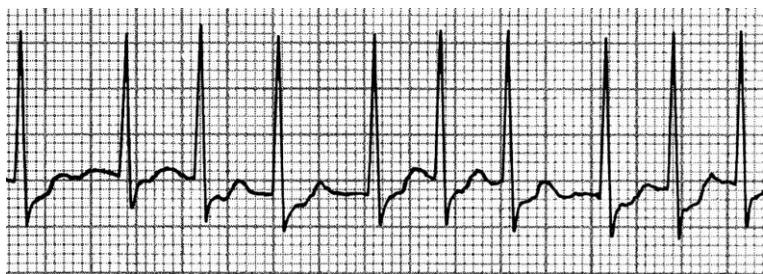
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<p>70 year old male. SBP 50 mmHg, florid pulmonary edema</p> 	<p>Wide complex tachycardia Assume VT Unstable Emergent Emergently treat with synchronized electrical cardioversion. Remember to press the 'sync' button before shocking!</p>
<p>16 year old male. SBP 80 mmHg, lightheaded, no chest pain, chest clear, sensorium normal.</p> 	<p>PSVT Unstable Urgent Although hypotensive, this patient is virtually asymptomatic and at his age, unlikely to have CAD. He will be able to tolerate this BP for a short time. If valsalva or carotid sinus massage fails, IV adenosine or diltiazem will likely work. The requirement for electrical cardioversion is extremely rare with PSVT.</p>
<p>60 year old woman presents with palpitations which began 12 hours ago and increasing SOB. Pulse 170 BP 90/50. Bilateral crackles.</p> 	<p>Irregularly irregular tachycardia with no definite P waves Rapid atrial fibrillation with heart failure Urgent Using drugs to cardiovert will be too slow. Treat with synchronized electrical cardioversion.</p>
<p>75 year old male, chest pain, diaphoretic, lightheaded, nauseated, BP 80/60 mmHg, chest clear, known coronary artery disease.</p> 	<p>2nd degree heart block Mobitz type 2. Unstable Urgent Treat with transcutaneous pacing until definitive treatment with transvenous pacemaker.</p>
<p>Same 75 year old male above. He vomits as the nurse is starting an IV and becomes confused and less responsive. BP 60 mmHg.</p> 	<p>3rd degree heart block Unstable Emergent treatment Needs emergent pacing</p>

<p>17 year old girl confused and agitated. Pulse 150 BP 200/120. Pupils dilated, chest clear. Admits using crack cocaine.</p> 	<p>Sinus Tachycardia Note P wave partially hidden by T wave Unstable. Urgent. She will need what medications? (Benzodiazepines)</p>
<p>15 year old boy comes to ED complaining of palpitations for 1 hour. He has had this intermittently for years but never investigated. He recently immigrated from a developing country. BP 110/60 Pulse 180, chest clear.</p> 	<p>Wide complex tachycardia. VT until proven otherwise. Stable. Urgent - even though a patient is stable, an arrhythmia that is potentially unstable should be assessed within minutes. What else could this be? What clinical, rhythm strip and 12 lead ECG findings will help you distinguish the two? This boy was converted to normal sinus rhythm with amiodarone. A 12 lead ECG revealed a short PR and a delta wave. What abnormality does he have with his heart? Wolff-Parkinson-White (WPW) syndrome.</p>

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48 year old woman with known mitral stenosis secondary to rheumatic heart disease presents with a “funny” fluttering sensation in her chest for past 10 hours. Pulse 150 BP 120/70, chest clear, patient looks well. Otherwise healthy. She is on the monitor in acute room reading a book.



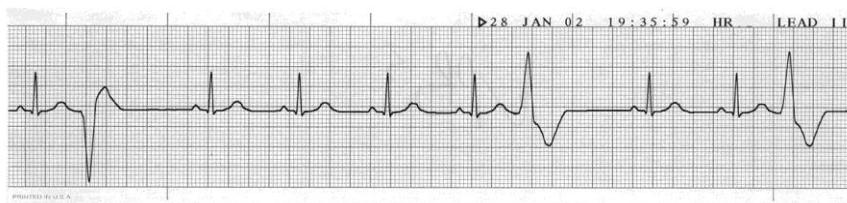
New onset rapid atrial fibrillation. Stable.

Deferrable - but should certainly be seen within the hour as this woman is a candidate for cardioversion (electrical or chemical with procainamide or amiodarone).

If she had had her symptoms for 3-4 days instead of 10 hours, she would not be a candidate for cardioversion because of the risk that a thrombus has formed in her heart. Converting her to normal sinus rhythm may lead to a cardioembolic event (stroke). If you are unsure of how long the patient has been in the rhythm or if it is > 48 hours, the treatment of choice is rate control and anticoagulation.

What if this patient had atrial flutter. Does your management change?

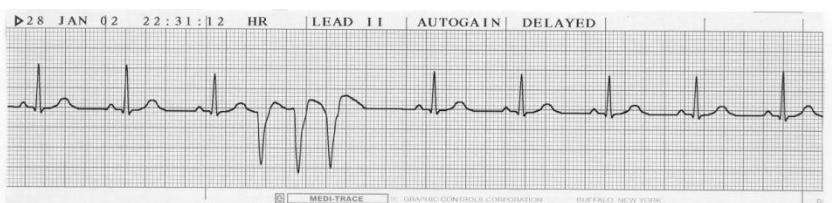
68 year old male took 1 spray of nitroglycerin for his chest pain. BP 140/80, chest clear, looks well.



Multifocal PVC's
Unstable (because of chest pain)
Urgent

In this case, the multifocal PVC's are due to the ischemia. Treat the ischemia and they will resolve.

His chest pain worsens, becomes nauseated and diaphoretic. Pulse 100 BP 100/70.



Brief run of VT
This man's ischemia is worsening.
Repeat the ECG and aggressively treat the ischemic chest pain
Have crash cart nearby and consider amiodarone if he gets sustained runs of VT.

CALL A “CODE”

A “code blue” is called in the hospital if a patient is profoundly hypotensive, pulseless and/or apneic unless the patient has a confirmed “do not

resuscitate” order (DNR). Remember DNR does not mean do not treat; it is generally meant to mean “if a patient has a cardiac arrest do not resuscitate them”. Calling a “code” brings a resuscitation team running to the bedside and implies the patient has or

is about to arrest. Emergency physicians working in the ED would not call a code but instead call for help to begin immediate resuscitative measures.

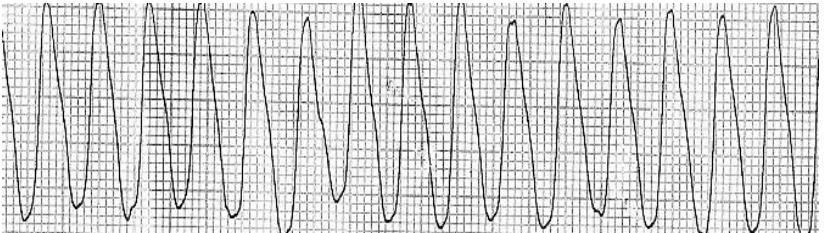
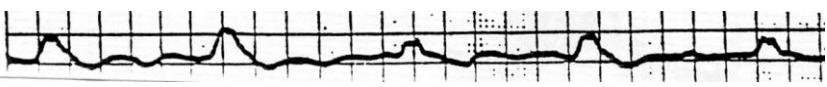
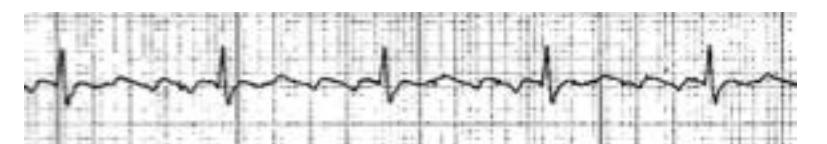
CPR

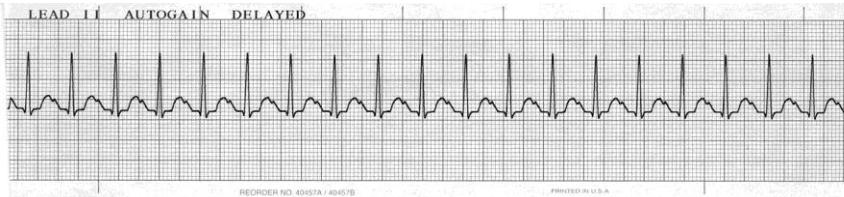
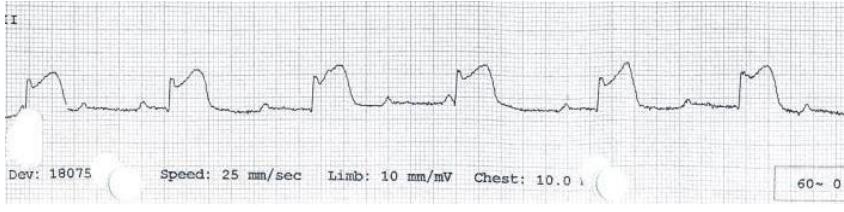
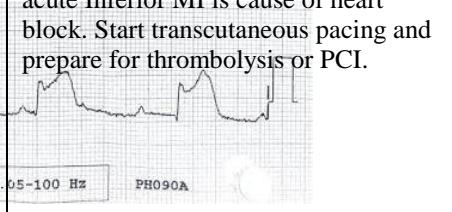
Remember that CPR is push hard (>5 cm) and fast ($>100/\text{min}$) and allow complete chest recoil. Minimize interruptions in compressions and avoid excessive ventilation. As well, remember post arrest

care includes hypothermia (in comatose patient), treatment of hypoxia and hypotension.

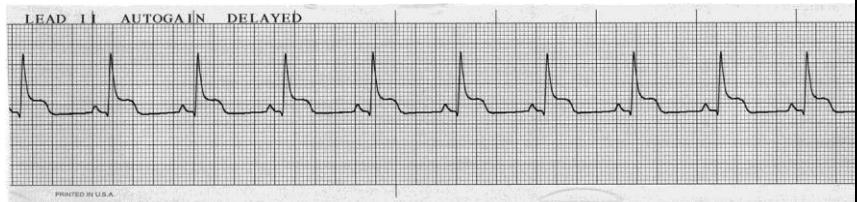
Review the following cases. Decide what the rhythm is, whether the patient is “stable” or “unstable”, if you should “call a code” and what interventions may be required (e.g. drugs or cardioversion). Assume all patients are on O_2 , cardiac monitor and have an IV established.

Cover the right side of the page and reveal the correct answers as you proceed.

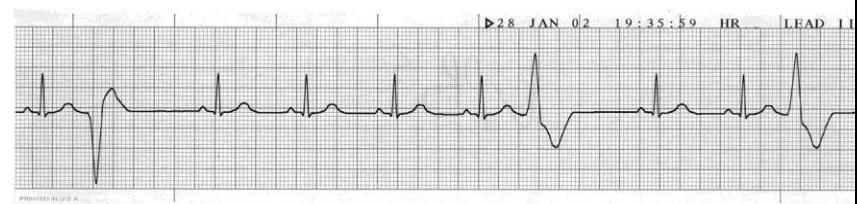
<p>Patient is pulseless and apneic. Action?</p> 	<p>V-Tach – Unstable Call a “code”. If unknown down time, perform 2 minutes CPR before going on to defibrillation. Defibrillation at 200 joules biphasic (360 joules monophasic) Epinephrine 1 mg IV q 3-5 minutes Amiodarone 300 mg initial dose, 150 mg in 5 minutes. Minimize interruptions in CPR</p>
<p>Code already called. CPR and ACLS drugs for 30 minutes in 80 year old man.</p> 	<p>Agonal rhythm Widening of the QRS and loss of amplitude combined with periods of asystole Represents the diminished electrical activity of a dying heart If there is no return of circulation after 20-30 minutes of CPR, it's time to stop.</p>
<p>This 66 year old woman presents with palpitations that started several hours ago. Pulse 76 BP 150/70 RR 22, chest clear.</p> 	<p>Atrial flutter – atrial rate 300 bpm and ventricular rate 75 bpm (4:1 block) Stable - don't call a “code” Patient should be cardioverted. Atrial Flutter does not respond well to chemical cardioversion. Patient can be electrically cardioverted. As with atrial fibrillation, no cardioversion if unclear how long patient has been in this rhythm or if longer than 48 hours, because of risk of stroke.</p>
<p>20 year old man presents to ED after taking an overdose of father’s digoxin medication. He complains of palpitations. BP 132/80, Pulse 110.</p> 	<p>Junctional tachycardia - no P waves Stable for now Don't call a “code” This patient needs to be admitted and kept on a monitor.</p>

<p>20 year old stabbed in left lateral chest. Unconscious, distended neck veins, absent breath sounds on left, no pulse.</p> 	<p>Sinus tachycardia but no pulse Pulseless electrical activity (PEA)</p> <p>Unstable Call a “code”</p> <p>Tension pneumothorax. Needle decompression 2nd intercostal space midclavicular line, followed by a chest tube</p> <p>What is your differential diagnosis for PEA? (5 H's and 5 T's) Hypovolemia H+ (acidosis) Hypothermia Hypo/Hyperkalemia Hypoxia</p> <p>Tension pneumothorax Toxins Tamponade cardiac Thrombosis cardiac Thrombosis pulmonary</p>
<p>Previously well 80 year old male had a syncopal episode while waiting for a bus. He feels light headed and slightly short of breath. BP 70/60, Pulse 40, RR 24, ashen, diaphoretic, chest is clear.</p> 	<p>Third degree heart block Unstable Do not need to call a “code”, but patient needs acute treatment. ST elevation in inferior leads reveals that acute Inferior MI is cause of heart block. Start transcutaneous pacing and prepare for thrombolysis or PCI.</p> 

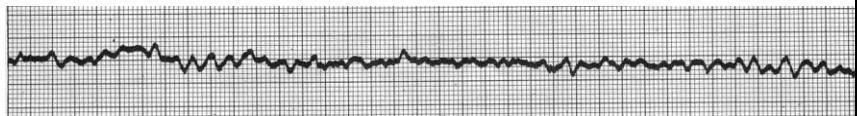
52 year old firefighter developed chest pain 1 hour ago while unloading hoses during a fire. He has a 50 pack year smoking history and his father had an MI at age 53. He is diaphoretic, short of breath, pulse 80 bpm BP 110/60 RR 22.



While looking at the fireman's 12 lead ECG, you notice he is having an acute inferior MI. He is given ASA, tenecteplase, enoxaparin and his first dose of metoprolol *. 10 minutes later he develops this, but tells you his chest pain is easing.



Suddenly the he moans, and becomes unconscious. You cannot find a pulse.



You take action and this results:



Unstable ...not due to the rhythm (it is NSR). He is having an MI. Although you can see ST elevation in rhythm strips, they are not reliable. You must do a 12 (or 15 lead ECG).

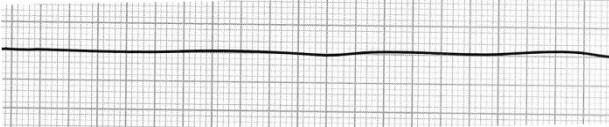
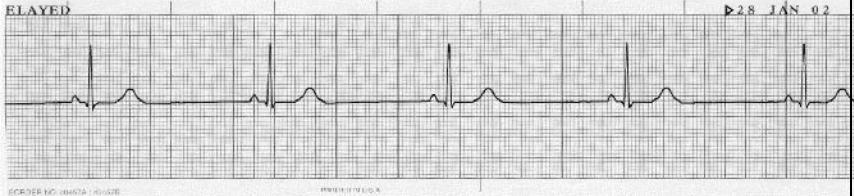
[*NOTE: If available, Percutaneous Coronary Intervention is first line therapy in addition to heparin, clopidogrel, and aspirin. If PCI is not available, thrombolysis is a good alternative with this patient. If the patient is out of hospital, advanced care paramedics may do a 12 lead ECG, identify the STEMI and bypass the ED right to the cath lab.]

You decide to monitor closely as it is normal to have some reperfusion dysrhythmias after thrombolysis.

Call a "code". Witnessed ventricular fibrillation (VF). Immediately defibrillate at 200J biphasic (360J monophasic). If fails to convert, begin CPR and intubate. Give epinephrine 1 mg IV and again, every 3-5 min. After 2 min of continuous CPR, defibrillate again at 200 J. If fails, amiodarone 300 mg IV, CPR for 2 min, then defibrillate at 200 J. Can repeat amiodarone once more 150 mg IV. Then continue pattern of CPR, epinephrine, and defibrillation.

In this case the patient converted on the first shock and when he quickly awoke he asked "why did you hit me in the chest?" Rhythm now NSR with first degree heart block.

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Patient is unresponsive, apneic, and pulseless. Unidentified, middle aged male. 	Aystole Check leads to confirm they are attached. Check leads to make sure they are attached and start CPR. Epinephrine 1 mg IV q 3-5 minutes No maximum for epinephrine
5 year boy old fell through the ice in a pond. Pulled out by firefighters. Time underwater approximately 25 minutes. CPR in progress. Temperature 28 C. 	Sinus bradycardia Call a "code". This is a hypothermic arrest. CPR with aggressive re-warming is indicated. Only when the child is re-warmed and still has vital signs absent do you consider pronouncing death. (i.e. they are not dead until they are warm and dead). This child survived.

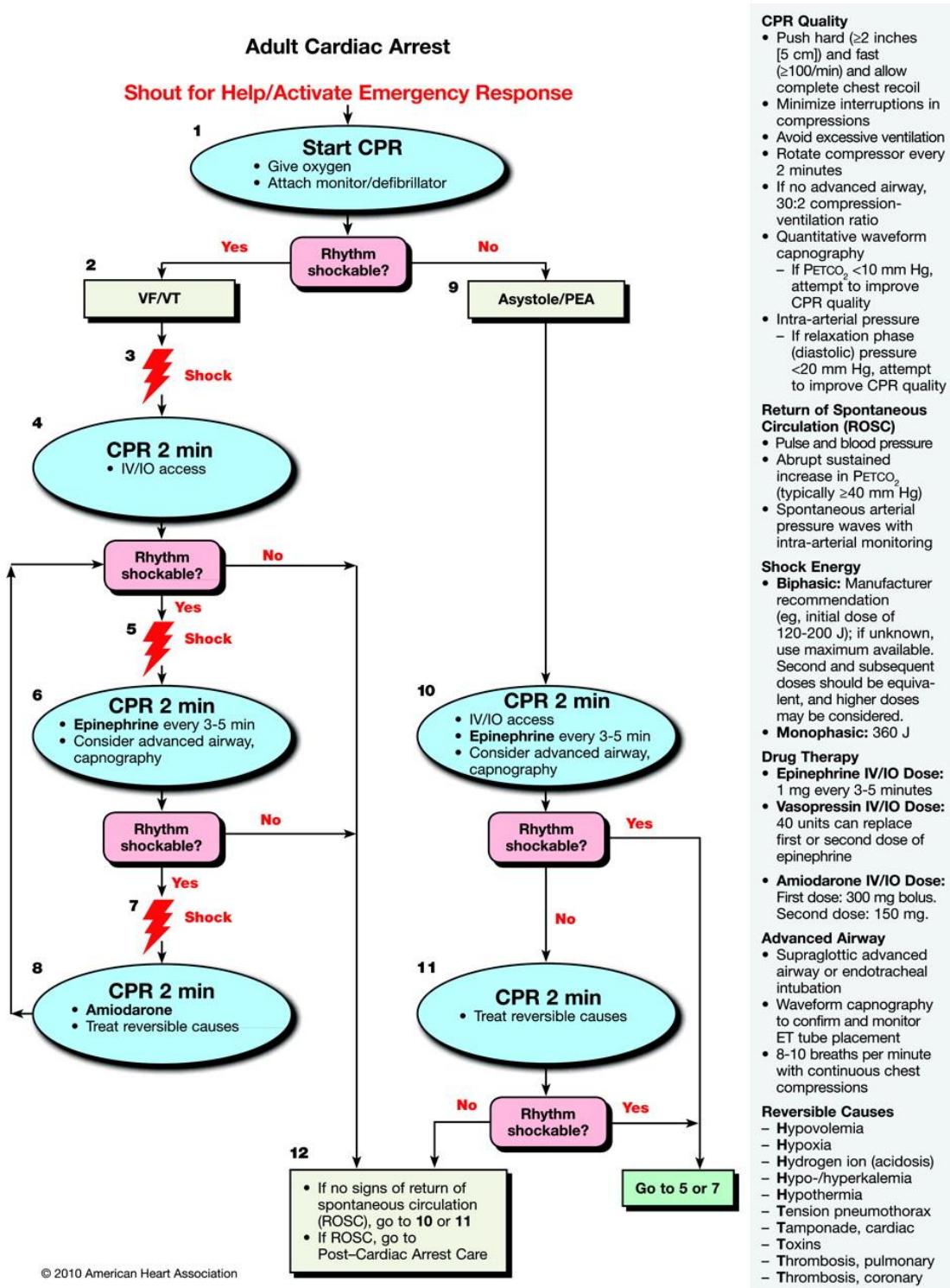
WHEN TO PRONOUNCE DEATH

Some patients will die despite your best efforts. The first time this happens to you, expect to experience a host of responses. In Emergency Medicine, we deal with sudden and unexpected death more frequently than is the case in any other specialty. We use our clinical judgement and respect of human dignity as part of the decision making process as to when resuscitative efforts should stop. Knowing when to

stop CPR comes with experience. There are guidelines and attempts to establish legal standards; but the final decision is that of the arrest team leader. If a patient has been confirmed as apneic and pulseless for approximately 20- 30 minutes, and is not hypothermic, termination of treatment should be considered.

ACLS ALGORITHMS

Figure 1. Pulseless Arrest Algorithm



**Figure 2. Tachycardia Algorithm
Adult Tachycardia
(With Pulse)**

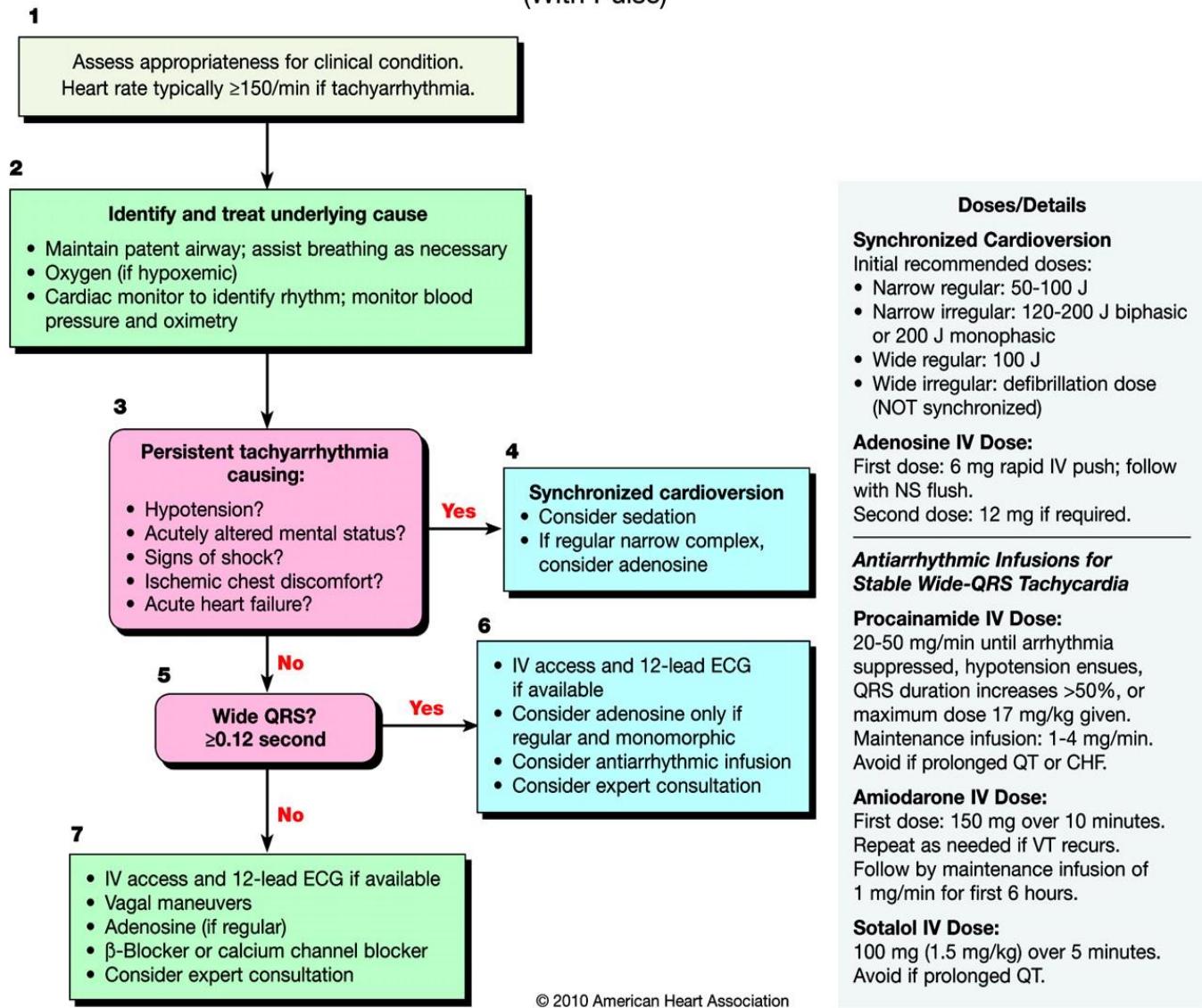
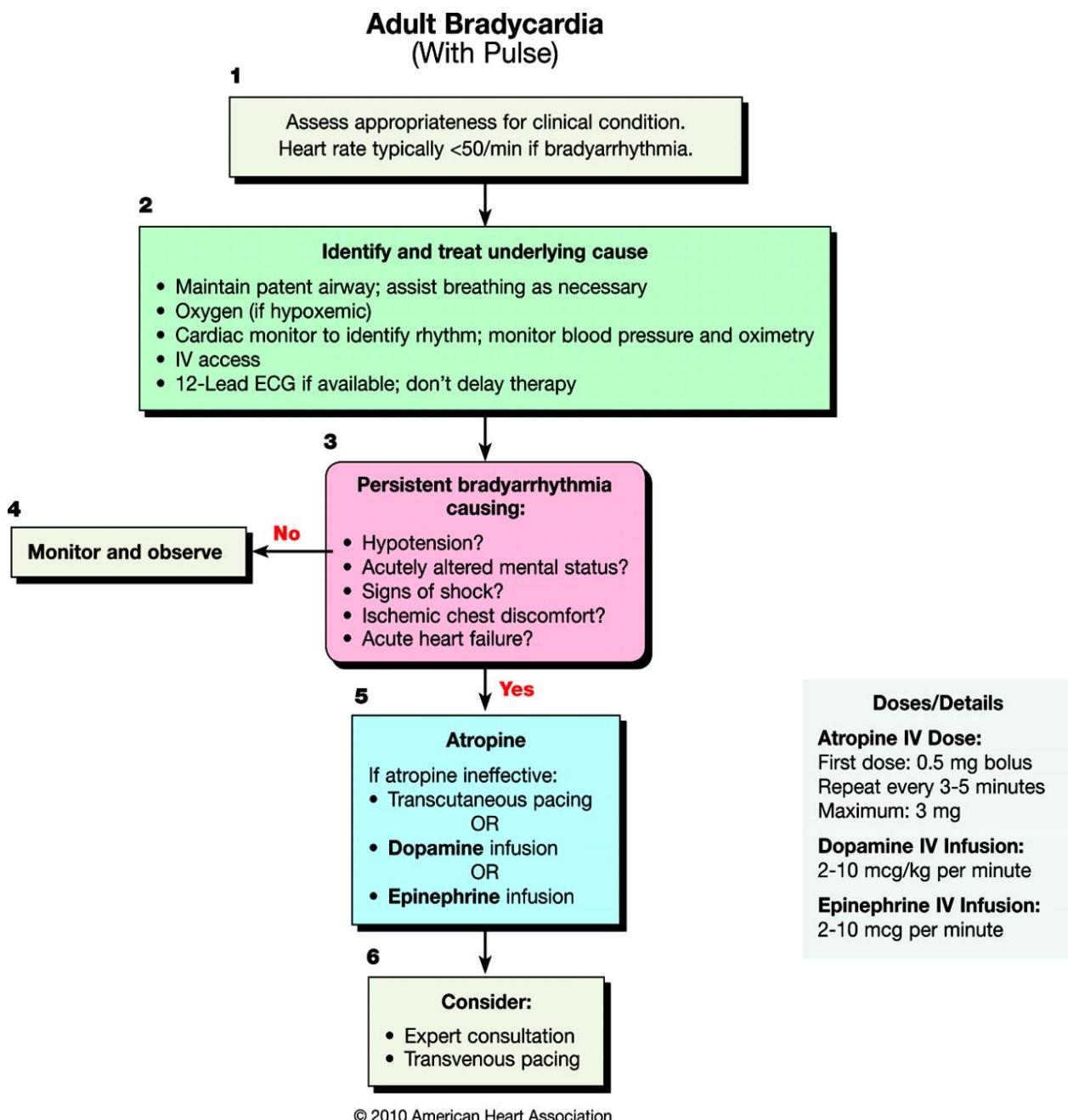


Figure 3. Bradycardia Algorithm

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Neumar R W et al. Circulation 2010;122:S729-S767

14: VASCULAR EMERGENCIES

Paul Hannam

OBJECTIVES

1. To recognize and manage aortic dissection
2. To recognize and manage ruptured abdominal aortic aneurysm
3. To recognize and manage acute arterial occlusion

INTRODUCTION

This chapter will focus on emergencies of the aorta and peripheral arteries. These diagnoses can be elusive, and there is still truth to Sir William Osler's observation that "there is no disease more conducive to humility than aneurysm of the aorta". Since definitive management of these conditions frequently requires operative intervention, the emergency physician is most likely to affect the outcome through early recognition.

AORTIC DISSECTION

The aortic wall is comprised of three layers, the intima, the media, and the adventitia. Aortic dissection occurs when arterial blood crosses the intimal layer and expands into the potential space of the media, splitting the aortic wall and shearing off branch arteries in the process. Aortic dissections are most commonly described using the Stanford classification. Stanford type A dissections involve the ascending aorta, where type B dissections are more distal. Type A dissections are more common and are associated with increased mortality.

Mortality from aortic dissection is reported at 1 to 5 per 100 000, although true incidence and prevalence are difficult to estimate due to under-reporting. Early diagnosis is particularly difficult with 38% of cases being missed at the initial presentation to a physician. Aortic dissection is a time-sensitive diagnosis, and mortality increases with each hour to reach 80% by two weeks.

Risk Factors

Populations at risk for aortic dissection can be divided into three major areas:

Normal aging, degenerative changes:

- Male
- Elderly (>65 years)
- History of hypertension

Predisposition to weakened aortic wall (any age):

- Pregnancy
- Family history of dissection
- Previous operative repair of aneurysm or dissection
- Connective tissue disease (Marfan's, or Ehlers-Danlos syndromes)

Increased stress on a normal aortic wall:

- Cocaine use
- Congenital bicuspid aortic valve

History

Sudden onset of pain is the most common symptom associated with aortic dissection. Pain is most likely to be maximal at onset and originate in the chest. However, the classic description of tearing/ripping with radiation into the back is uncommon. An awareness of the signs and symptoms resulting from branch vessel involvement are likely to help you make this diagnosis. Any of the 'chest pain with...' descriptions below should be seen as red flags:

- Chest pain with neurologic features (syncope, central or cord ischemia)
- Chest pain with (or migrating to) abdominal pain
- Chest pain with ischemic symptoms to a lower extremity

Physical Exam

Physical examination is likely to show a hypertensive patient, although hypotension may occur later in the course due to hypovolemia or pericardial tamponade. A pulse differential between the carotid, radial, or femoral arteries is helpful to rule in dissection although may not be present (has poor sensitivity). Blood pressure differential of >10mmHg between arms is a less useful finding given that it is nonspecific and may be found in 20% of people without this condition. Cardiac auscultation may reveal a murmur of aortic insufficiency, significant if confirmed to be a new finding. Physical examination should also include screening neurologic and vascular examinations to rule out more subtle presentations of branch artery ischemia, such as an anterior cord syndrome.

Investigations

Investigations for dissection primarily rely on imaging studies. Chest x-ray findings may include left pleural effusion, wide mediastinum, or aortic calcification >5mm from the outside of the expanded

aortic contour. These findings are often subtle and 12% of patients with dissections have a normal chest x-ray. Point of care ultrasound examination by the emergency physician is an effective method to quickly rule out pericardial effusion due to proximal dissection.

CT scanning with IV contrast is diagnostic in almost all cases, and is likely to be required for planning definitive management. Hypotension and/or renal insufficiency are common in the setting of dissection, and therapy to decrease the risk of contrast-induced nephropathy should be considered if time permits. Trans-esophageal echocardiography is both sensitive and specific and is the procedure of choice in patients who are unstable, although it is operator dependent and may have limited availability, depending on the facility.

Adjunct testing is unlikely to assist in making the diagnosis of dissection. Non-specific findings such as hematuria or leukocytosis may distract the clinician from the diagnosis of dissection. The use of d-dimer assays to rule out aortic dissection has been the focus of recent research. This test is most likely to be useful for patients with a low pretest probability, but there are no validated clinical prediction rules to support such a practice at this time.

It may be difficult to differentiate aortic dissection from acute coronary syndrome on ECG. Proximal dissection may affect the right coronary artery, resulting in inferior/posterior ischemia. In spite of this association, failure to identify dissection occurs in only 0.1-.0.2% of patients receiving revascularization treatment for myocardial infarction. Isolated myocardial infarction is a far more common condition than aortic dissection, and if there are no red flags for dissection on history or physical exam, it is appropriate to treat the MI.

Complications

Complications associated with aortic dissection include the following:

- Myocardial ischemia or infarction
- Acute aortic valve insufficiency
- CHF
- Pericardial effusion/tamponade
- Ischemic stroke
- Mesenteric ischemia
- Renal failure
- Lower extremity paralysis
- Ischemic limb
- Aortic rupture

Management

Initial management of suspected aortic dissection should consist of analgesia and blood pressure control. Labetolol is a beta blocker with both beta and alpha blocking activity, and can be titrated easily to a target blood pressure of 120/80 mmHg. Sodium nitroprusside may also be used as an adjunct in achieving blood pressure control. Close monitoring of vital signs, neurologic status, and urine output are also appropriate while definitive management is being arranged.

Definitive management of aortic dissection is classically surgical for Stanford A dissections (involving ascending aorta) and medical for Stanford B (distal). These distinctions are not absolute, however, and may be altered by patient factors as well as local expertise. Endovascular approaches to treatment have been described, and may be appropriate depending on your setting.

CLINICAL PEARL

In patients presenting to the Emergency Department with sudden onset of chest or back pain, an awareness of the symptoms and signs resulting from branch vessel involvement of aortic dissection, are more likely to help you make this sometimes elusive diagnosis.

ABDOMINAL AORTIC ANEURYSM

Abdominal aortic aneurysm (AAA) most commonly results from chronic degenerative changes to the aortic wall. Although there is an association with atherosclerosis, the exact mechanism of this degeneration is not well defined for the majority of patients. As the aneurysm expands the wall force increases exponentially, leading to increased risk of rupture and subsequent hemorrhage. Ruptured AAA is a devastating illness - the overall mortality approaches 80-90%, decreasing to 50% for those who survive to reach the operating room.

Risk Factors

AAA is relatively common, and is present in 5-10% of men over 65. Risk factors for AAA include:

- Hypertension
- Age greater than 50
- Male
- Peripheral vascular disease and/or coronary artery disease

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- Family history of aneurysm*
- Smoking
- Diabetes
- Connective tissue disease

*Family history of aneurysm is a significant risk factor. In one study 39% of patients diagnosed with AAA had a first degree relative with the same diagnosis.

History

Ruptured AAA commonly presents as sudden onset abdominal, back, or flank pain. It is the speed of onset rather than the location of the pain which is most suggestive of rupture. Atypical presentations include sudden onset of pain radiating to the groin, thigh, or inguinal areas.

Syncope often occurs with ruptured AAA due to transient hypotension. A patient with risk factors for AAA who presents with syncope should be treated with a great deal of caution regardless of the initial blood pressure reading in the emergency department, since the hemodynamic compensation for hypovolemia is likely to be transitory.

Atypical presentations can lead to delayed diagnosis of ruptured AAA and subsequent poor outcomes. The following diagnoses should prompt consideration of AAA on the differential diagnosis:

- Renal colic or diverticulitis if >65 years old
- Unexplained hypotension or syncope
- MSK back pain with risk factors for AAA
- Previous endovascular repair of AAA (with subsequent leak)

Physical Exam

The majority of AAAs arise in the infrarenal portion of the aorta, and may be palpable just superior to the umbilicus. There is no risk of rupture associated with palpation. Femoral pulses may be diminished, and the lower extremities should be examined for associated popliteal aneurysms or signs to suggest distal micro-embolism – ‘blue toe syndrome’. Less common presentations may result from aorto-enteric fistula (unexplained or massive GI bleeding) or arterio-venous fistula (CHF with lower extremity ischemia).

Investigations

Point of care ultrasound in the emergency department has the potential to diagnose the presence or absence of AAA within minutes. An outside diameter of >3cm confirms the diagnosis of AAA. Technically adequate ultrasound studies have high sensitivity and specificity, allowing for early management decisions.

Emergency department ultrasound is limited in that it cannot determine whether or not an AAA has ruptured, since most ruptures will occur into the retroperitoneal space.

CT scan is virtually 100% accurate in diagnosing AAA as well as the resulting complications. The primary limitation of CT scanning relates to the time it takes to transfer the patient to and from the scanner, which is not an option for the unstable patient. Early consultation with the receiving surgeon is important since the patient may deteriorate quickly despite initial improvement.

Management

AAA rupture can occur at any size, but is most likely to occur when the diameter is greater than 5cm. A newly diagnosed AAA in an otherwise asymptomatic individual should have follow-up arranged for serial ultrasound examinations and/or elective intervention as appropriate. Mortality following elective repair of AAA approximates 5%, in contrast to the poor outcomes associated with rupture.

When a patient presents with a clinical picture consistent with rupture, urgent operative intervention is the primary consideration. Appropriate consultation should take place early so that transfer to the operating room can occur as efficiently as possible. Preparations should include IV access with at least two large bore IVs, and blood work should include cross-matching in accordance with a massive transfusion protocol. While awaiting transfer, the initial fluid resuscitation of the patient with ruptured AAA should be administered with the goal of maintaining end-organ perfusion. Although the optimal strategy for resuscitation is still debated, using normal saline boluses to target a systolic blood pressure of 90 mmHg is reasonable.

ACUTE ARTERIAL OCCLUSION

Acute arterial occlusion is a true emergency since irreversible damage can occur within 6-8 hours of onset. Acute occlusion may occur due to acute embolus from a proximal source, or due to arterial thrombosis in a narrowed or damaged arterial wall (e.g. advanced atherosclerosis). Even with appropriate treatment, 10-15% of patients will require amputation.

Risk Factors

Arterial Embolism - cardiac thromboembolism is most common, and occurs with:

- MI (with subsequent LV thrombus)

- Atrial Fibrillation
- Valve stenosis and/or replacement

Arterial Thrombosis:

- Occlusion of previous stent or graft
- Peripheral vascular disease (atherosclerosis)

Less common sources of acute embolism are associated with following:

- AAA (atherosclerotic plaque)
- Metastatic Cancer (tumor debris)
- Recent surgery (air embolism)
- Bacterial endocarditis (septic embolus)
- IV drug use (foreign body)

History

The most important features associated with arterial occlusion are the six P's:

- Paresthesia
- Pain
- Pallor
- Pulselessness
- Poikilothermia (cool to touch)
- Paralysis

Physical Exam

In the case of embolism, the acutely ischemic limb will appear pale and have a sharply demarcated line relative to normal tissue. Pulses to the affected limb should be assessed using a vascular Doppler probe if they are not palpable. If flow is audible by Doppler, a blood pressure cuff may be placed proximally. A perfusion pressure of less than 50 mmHg (or completely inaudible pulse) indicates ischemia. Pain and tenderness to palpation of the affected area will develop as the ischemia progresses. Cyanosis is a late finding, as are neurologic features such as decreased sensation or paralysis. The examination should also include a search for a source (e.g. cardiac) if embolus is suspected.

Investigations

Acute arterial occlusion is primarily a clinical diagnosis. Formal Doppler ultrasound in a vascular lab poses little risk to the patient, although the benefits should be weighed against the time it will take to obtain the results. Angiography is the gold standard for the diagnosis of peripheral vascular disease but is more appropriate in the non-acute setting. In both cases, the decision to proceed with investigations in the emergency room should be made in consultation with the vascular surgeon since there are significant risks to delaying treatment of acute ischemia.

Management

Where acute ischemia is suspected, unfractionated heparin IV is the only therapy likely to have an effect in the ER. Early consultation with vascular surgery will facilitate definitive management, which may include a combination of surgical thrombectomy, angioplasty, or intra-arterial thrombolysis.

WHAT'S NEW

Recent research has suggested that a blood pressure differential between arms of 10 -15 mmHg may be associated with an increased risk of peripheral vascular disease, cardiovascular events, and all cause mortality in asymptomatic patients with hypertension. Although there may be an association there is insufficient evidence to make a clear recommendation regarding management at this time.

SUMMARY

- A minority of patients with aortic dissection will present with classic symptoms. Recalling the “chest pain with...” features will help to prompt consideration of this diagnosis.
- CT chest with contrast or trans-esophageal echo have very good sensitivity and specificity for aortic dissection.
- Initial management of suspected aortic dissection should consist of analgesia and blood pressure control.
- Ruptured AAA commonly presents as sudden onset abdominal, back, or flank pain. It is the speed of onset rather than the location of the pain which is most suggestive of rupture.
- Point of care ultrasound in the Emergency Department has the potential to diagnose the presence or absence of AAA within minutes.
- For a patient presenting with a clinical picture consistent with ruptured AAA, time to operative intervention is the primary consideration.
- Acute arterial occlusion is a true emergency since irreversible damage occurs within 6-8 hours of onset.
- The most important diagnostic features of arterial occlusion are the six P's: Paresthesia, Pain, Pallor, Pulselessness, Poikilothermia, and Paralysis.
- Emergency Department treatment of suspected acute ischemic limb consists of IV heparin and consultation for definitive management.

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15: DVT & PULMONARY EMBOLUS

Anil Chopra

OBJECTIVES

1. To risk stratify a patient suspected of having a DVT or PE into low, moderate and high risk
2. To choose the most appropriate non-invasive test(s) to detect and rule out a venous clot
3. To know the specific issues related to pregnancy, heparin-induced thrombocytopenia (HIT), and calf DVTs
4. To choose a safe management strategy to treat a patient with DVT or PE

INTRODUCTION

The true incidence of deep venous thrombosis (DVT) is unknown given that most of these clots are occult. The most common fatal consequence of DVT is pulmonary embolism (PE) with a historical estimated seven-day mortality of 25%, though the mortality is now significantly lower with early recognition and treatment. The accurate diagnosis and management of a known or presumed venous thrombosis is essential to improving outcomes from local and thromboembolic sequelae.

RISK ASSESSMENT

Venous clots form in the presence of at least one of Virchow's triad of factors: venous stasis, injury to the vessel wall and hypercoagulable state. The clinical risk factors for venous thrombosis can be remembered by the mnemonic '**thrombosis**':

T	Trauma, travel
H	Hypercoagulable, hormone replacement
R	Recreational drugs (IV drugs)
O	Old (age > 60)
M	Malignancy
B	Birth control pill
O	Obesity, obstetrics
S	Surgery, smoking
I	Immobilization
S	Sickness (CHF/MI, nephrotic syndrome, IBD, vasculitis)

The most significant risk factors for venous clots are major surgery or trauma, permanent immobilization, malignancy or other hypercoagulable state and prior thromboembolic disease.

Clinical symptoms and signs are unreliable for the detection and exclusion of DVT. Less than half of patients with DVT have the classical constellation of pain, redness, swelling, warmth and tenderness. A suspicion of DVT alone is sufficient to instigate objective investigations even with a negative physical examination. Clinical tools have been developed to risk stratify patients and help guide investigations. One such clinical model developed and validated by Wells has been shown to be reliable to predict the probability of DVT. It divides patients into three pretest risk categories: low (zero points), moderate (1 to 2 points) and high (3 or more points) with a likelihood of DVT of 5, 33 and 85 % respectively. A symptomatic DVT will be at or proximal to the popliteal vessels in more than 80% of cases. A calf DVT will extend proximally only 20% of the time.

DEEP VENOUS THROMBOSIS

Predictors of DVT ('Well's Criteria')	Points
Active cancer (treatment ongoing, palliative)	1
Paralysis, paresis, or recent plaster immobilization of lower extremities	1
Recently bedridden more than 3 days or major surgery within 4 weeks	1
Localized tenderness along the distribution of the deep venous system	1
Entire leg swollen	1
Calf swelling 3 cm > asymptomatic side (10 cm below tibial tuberosity)	1
Pitting edema confined to the symptomatic leg	1
Collateral superficial veins (non-varicose)	1
Alternative diagnosis as likely or greater	-2

Diagnosis Of DVT

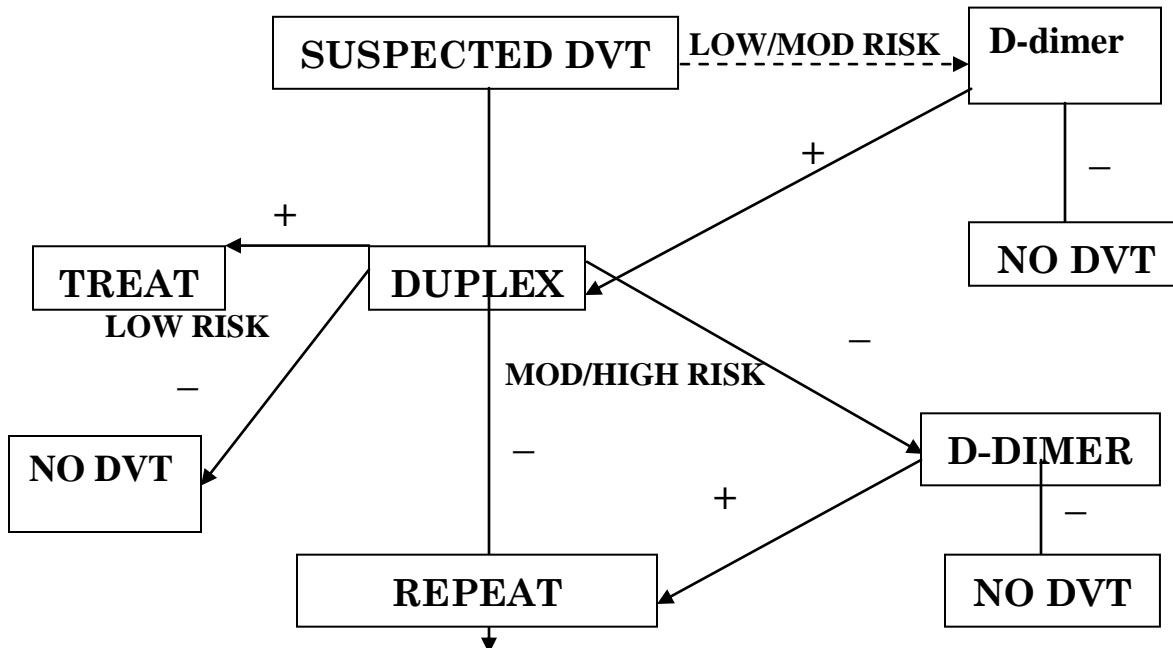
Diagnostic tests for DVT include a plasma D-dimer assay, Doppler ultrasound (duplex), contrast venography, computed tomographic venography (CTV) and magnetic resonance venography (MRV). Some of these tests are rarely used or needed due to invasiveness (contrast venography) or lack of availability (MRI).

D-dimer fragments are degradation products of fibrin and an elevated level is an indicator of activation of the clotting mechanism. A D-dimer test is only useful if it is negative. A negative sensitive D-dimer assay excludes the presence of a venous clot in low to moderate risk patients (by Well's criteria) with a negative predictive value of over 99%. However, the D-dimer is only negative in about one third of unselected patients with suspected DVT or PE. An elevated D-dimer level can be noted in the elderly (specificity of 10% if age > 85) and in patients with infections, recent surgery, trauma, hemorrhage, pregnancy and other medical conditions such as MI, stroke, liver disease, cancer, and collagen vascular diseases. It follows, then, that a D-dimer assay should

generally only be done initially to exclude venous clots in younger patients unlikely to have a false positive test. The ELISA D-dimer test has historically been the gold standard amongst the available D-dimer assays with a sensitivity of about 97% for proximal DVT. An immunoturbidimetric D-dimer test also has a comparable sensitivity to the ELIZA.

The choice test for diagnosis and exclusion of DVT in North America is the duplex scan. With a high sensitivity of 97% and specificity of 94% for proximal clots, it is a reliable, non-invasive and a practical test. However, its sensitivity for calf DVT is only 73% and it cannot distinguish an acute from chronic DVT accurately. Ultrasound can also detect alternative disease such as Baker's cyst, hematoma, arterial aneurysms, abscess, lymphadenopathy and superficial thrombophlebitis. One would only consider an alternative test to ultrasound such as the D-dimer when ultrasound is not readily available. A single negative ultrasound excludes a proximal DVT in low risk individuals, but a repeat duplex should be done in 5 to 7 days in higher risk patients to exclude an extending calf clot.

Figure 1. Investigation of a Patient Suspected to Have DVT



Management of DVT

The management of a proven DVT is anticoagulation to prevent clot extension while waiting for the intrinsic fibrinolytic pathways to break down the clot. Historical recommendations including bedrest, continuous leg elevation and compressive stockings are of unproven benefit when used along with anticoagulation. Early ambulation with appropriate analgesia is a practical and safe approach. Low-molecular-weight heparin (LMWH) or fondaparinux are the preferred agents for treatment of DVT and have several advantages over unfractionated heparin. Both have a predictable anticoagulation effect, are easily administered as once-daily subcutaneous injections, do not require monitoring by blood tests and have a lower incidence of major bleeding and Heparin-Induced Thrombocytopenia (HIT). LMWH or fondaparinux are not recommended in renal failure patients unless anti-Xa levels are closely monitored. Common LMWH treatment protocols for DVT include: dalteparin 200 U/kg q24h, enoxaparin 1.5 mg/kg q24h and tinzaparin 175 U/kg q24 h. Fondaparinux is given as a 7.5 mg injection once daily for average weight patients. Warfarin is started at the same time as LMWH in the absence of contraindications (e.g. pregnancy). An initial dose of 10 mg per day is given for the first two days followed by dosing based on INR and adjusted to keep the INR between 2 and 3. LMWH is continued daily with warfarin until the INR has been therapeutic for two consecutive days. New oral anticoagulants such as dabigatran and rivaroxaban may in the future replace warfarin therapy with the advantage of fixed dosing and a lack of need for any routine laboratory monitoring. The duration of anticoagulation treatment needs to be individualized, but typically is required for at least 3 months for patients with transient coagulopathy (birth control pill, leg cast, surgery, etc.). Patients with unprovoked venous thrombosis require at least 6 months of therapy and patients with ongoing coagulopathy (e.g. cancer, immobility) will need life-long anticoagulation.

Patients with HIT cannot be treated with heparin for a venous thrombosis. For these patients, a hematology consultation is indicated to consider use of alternative agents including fondaparinux, argatroban, lepirudin or danaparoid.

The vast majority of DVT patients can be discharged with outpatient follow-up. Patients who are unable to ambulate, have poor social supports, lack reliable follow up, or need lytic or invasive therapy may have

to be admitted to hospital. Recent guidelines suggest not treating all isolated distal (calf) DVTs. For distal DVT without severe leg symptoms or risk factors for extension (e.g. recent stroke), serial leg ultrasound studies to look for extension without any anticoagulation are preferred. For those patients with distal DVT provoked by surgery or another risk factor, anticoagulation for 3 months is recommended to diminish the risk of proximal extension and embolization.

There are no clear guidelines for thrombolysis or mechanical therapy of DVT. No controlled studies have shown a survival benefit from these aggressive measures in comparison with anticoagulation alone. There is some evidence to suggest a lower incidence of postthrombotic syndrome in patients treated with thrombolysis, but at a significantly higher risk of complications such as bleeding. Thrombolytics can be considered for treatment for DVT in patients with extensive iliofemoral thrombosis if there is a concern for limb compromise, particularly in patients with a low bleeding risk. Catheter-directed lysis is preferred to systemic thrombolysis. An inferior vena cava filter and/or surgical thrombectomy can be considered in patients who have absolute contraindications to anticoagulation.

PULMONARY EMBOLISM

The diagnosis of PE is one of the most challenging tasks faced by emergency physicians. Only a minority of patients with this disease will present with the classical triad of pleuritic chest pain, dyspnea and hemoptysis. Autopsy studies show that about 30% of PE is clinically not recognized. As with DVT, a risk stratification strategy is also necessary in the diagnosis of PE. Clinical judgment by experienced physicians to risk stratify patients suspected of PE appears to be valid, but many recommend a less subjective approach. Wells developed a clinical model to help predict the likelihood of PE and found a prevalence of PE of 3% in the low-probability group (< 2 points), 28% in the intermediate-probability group (2-6 points) and 78% in the high-probability group (> 6 points):

Predictors of PE (‘Well’s Criteria’)	Points
Clinical signs of DVT	3.0
Alternative diagnosis less likely than PE	3.0
Heart rate > 100	1.5

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Immobilization or surgery within 4 weeks 1.5

Previous DVT/PE 1.5

Hemoptysis 1.0

Malignancy (ongoing treatment/palliative) 1.0

Kline suggested a pulmonary embolism rule out criteria ('PERC' rule) which can be applied to patients who have already been risk stratified to low risk to help identify patients who require no further testing. This rule has been validated now in subsequent studies and can be applied to a patient population where the prevalence of the disease is lower than 15%. The eight low risk criteria are outlined in the chart below. If all of these criteria are negative **and** the physician's assessment of the risk of PE is <15%, Kline recommends no objective testing for PE as the pretest clinical probability is less than 2%.

Diagnosis of PE

After a pretest clinical probability assessment, an ECG and CXR are useful to detect alternative diagnosis such as pericarditis, acute coronary syndrome or pneumonia, though they rarely help to diagnose PE. Sinus tachycardia and evidence of right ventricular strain is often seen on an ECG of patients with large PE. The classical pattern of a deep S wave in lead I with a deep Q wave and inverted T wave in lead III (S1Q3T3) is seen in only a minority of patients. The presence of simultaneous T wave inversions in both the anterior and inferior leads is highly suggestive of PE in patients not known to have heart disease, but only observed in less than 10% of PE patients. Similarly, a Hampton's Hump (pleural based triangular density indicating infarction) or Westermark sign (focal oligemia indicating decreased pulmonary arterial flow) are not usually seen on a CXR of PE patients, though non-specific findings of atelectasis, parenchymal disease or pleural effusion may often be seen.

CLINICAL PEARLS

Pulmonary Embolism Rule-Out Criteria ('PERC' Rule):

1. age < 50
2. heart rate < 100
3. oxygen saturation of > 94% on room air
4. no previous history of DVT or PE
5. no recent surgery
6. no hemoptysis
7. no estrogen use
8. no clinical signs of DVT

To be applied only on patients with <15% chance of having a PE (low risk)

In low to moderate probability patients, a negative highly sensitive D-dimer assay excludes PE with a 3-month thromboembolic rate of about 1%, which is similar to patients with a negative pulmonary angiogram. Patients with a positive D-dimer, high-probability patients or patients who did not undergo D-dimer testing should have either a ventilation-perfusion lung (V/Q) scan or a helical CT with contrast depending on the presence or absence of significant abnormalities on CXR (which will likely result in a nondiagnostic V/Q scan), availability and contraindications to contrast material. CT scanning is rapidly becoming the choice test to detect and exclude PE in North America in patients with adequate renal function and with no dye allergy, particularly due to greater availability, improving accuracy and ability to detect alternative diagnosis (e.g. consolidation, mass) in a quarter of scanned patients. Even the higher generation CT scans, however, may miss very small and subsegmental clots and for this reason, a negative scan requires further testing to exclude a venous clot in high risk individuals. A pulmonary angiogram is rarely needed to diagnose or exclude a PE except in situations when there is a high clinical probability, but non-invasive tests have not been diagnostic and it is considered unsafe to withhold anticoagulation. Pulmonary angiography is associated with 0.5% mortality and 2% serious morbidity. MRI lacks the sensitivity to be useful in the routine investigation of the patient suspected of having PE.

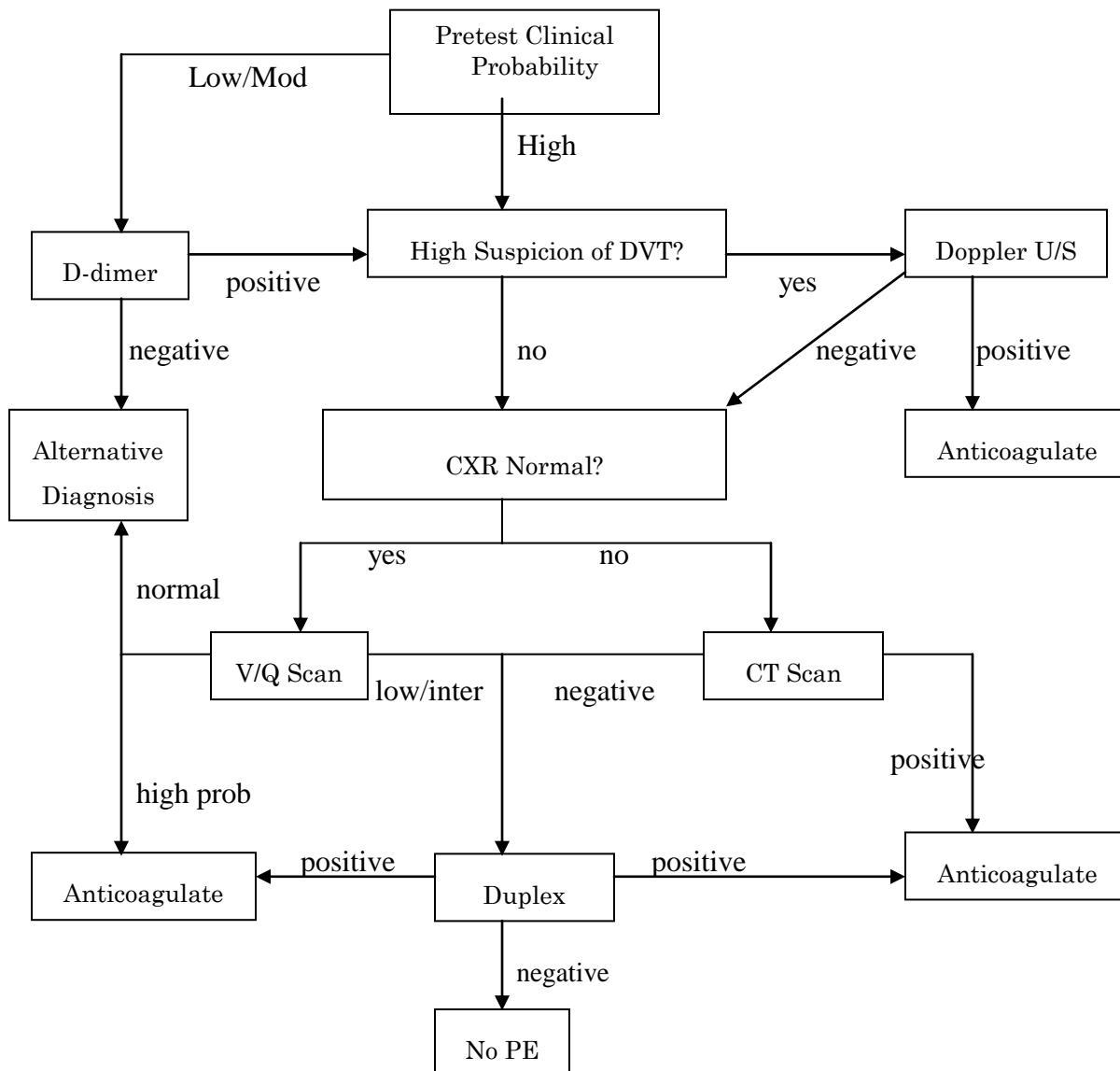
Combinations of tests are sometimes required to exclude PE. A low to intermediate V/Q scan followed by negative serial duplex scans is associated with a 0.6% risk of PE within 3 months which is the same risk as with a normal V/Q scan. It is reasonable to use Duplex to look for a leg DVT in these patients given that 90% of all PE arise from a leg clot, but only about 40% of patients with acute PE have a positive leg Duplex.

In pregnant patients, the D-dimer levels increase normally as the pregnancy progresses, but a sensitive D-dimer assay may still be useful in the first trimester as at least 50% of these patients will have a negative test which safely excludes venous thromboembolism. The utility of this assay declines in the second trimester where about 75% of the assays will be positive and this test is not useful in the third trimester as nearly all tests are positive. Imaging pregnant patients is also troublesome given the concerns for radiation exposure for the fetus and mother. Recent American Thoracic Society guidelines suggest that a V/Q scan be the first test of choice to investigate pregnant patients suspected of having a

PE if the CXR is normal. To minimize the radiation dose, the test should be modified as a perfusion only test with lower dose of contrast. For patients with significant abnormalities on CXR, a CT scan is recommended with appropriate shielding of the mother and fetus. A duplex scan is not suggested as a first choice test given it will be negative in about 98% of patients tested in the ED unless they have clinical

evidence of DVT on examination. An MRI may be considered in very difficult patients, but a lack of sensitivity and availability makes it a less useful study. If imaging is not possible or the results of imaging are ambiguous, the non-low risk patient can be treated with LMWH safely in pregnancy while waiting for a definite diagnosis.

Figure 2. Investigation of a Patient Suspected to Have PE



Management of PE

The treatment of PE with anticoagulation with LMWH or fondaparinux, and warfarin is the same as for DVT, as there is no evidence that a longer course of treatment is needed. If investigations have to be delayed, non-low risk patients should be treated with

LMWH or fondaparinux while waiting for these tests. Thrombolysis should be considered in patients with a massive PE and hemodynamic instability or cardiogenic shock. Typical dosing recommendation for intravenous thrombolysis is tPA 100 mg iv over 2

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hours, though it can be given as a 0.6 mg/kg iv STAT bolus (typically 50 mg iv) for near death scenarios.

Over the last decade, there is an increasing trend to treat patients with PE as outpatients.

Hemodynamically stable patients who do not require supplemental oxygen, have good social supports, no major comorbidities such as renal failure or CHF or advanced COPD, adequate pain control and no contraindication to anticoagulation are excellent candidates for outpatient therapy. Catheter-directed thrombolysis or surgical embolectomy is rarely needed except in patients with a massive PE or an absolute contraindication to anticoagulation.

SUMMARY

- risk factors for venous thrombosis: mnemonic “thrombosis”
- Well’s criteria or PERC rule to risk stratify patients
- a negative sensitive D-dimer test excludes DVT and PE in low to moderate risk patients
- U/S Doppler is choice test to detect a proximal DVT but may miss a calf clot
- CT chest is choice test to detect PE in patients with adequate renal function
- LMWH or fondaparinux, plus warfarin is the choice treatment for patients with DVT or PE (thrombolysis should be considered for patients who are hemodynamically unstable or have limb compromise)
- in pregnant patients, a sensitive D-dimer test in the first trimester or imaging (V/Q scan if CXR normal or CT scan if abnormal CXR) are choice initial tests to exclude and detect DVT and PE

WHAT'S NEW

Oral direct thrombin inhibitors such as dabigatran and oral anti-Xa agents such as rivaroxaban have been shown to be comparable in efficacy and safety to warfarin for venous thromboembolism, but with the added benefit of a fixed dosing regimen and without the need for any lab monitoring.

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16: ASTHMA & COPD

Rick Verbeek, Kasia Stefanska

OBJECTIVES

1. To make a rapid assessment of the severity of asthma or COPD exacerbation
2. To understand the utility of pulmonary function tests in the emergency department
3. To know the treatment priorities for different severities of acute asthma and COPD
4. To understand the importance of discharge instructions for patients with asthma and COPD

ASTHMA

Introduction

Asthma is a chronic inflammatory disorder of the airways, associated with hyperresponsiveness and reversible airflow limitation. Exacerbations, commonly triggered by viral infections and environmental allergens or irritants, are characterized by a progressive increase in SOB, cough, wheezing, or chest tightness, and by a decrease in expiratory airflow. Severity ranges from mild to life threatening, and guides therapeutic interventions.

In any patient presenting with SOB and wheezing, consider the differential diagnosis: COPD, congestive heart failure, upper airway obstruction, pneumonia, acute coronary syndrome, and pulmonary embolism.

Assessment

Determine the risk for severe asthma.

HISTORY

- Poor asthma control
- History of hospital/ICU admissions
- Previous need for mechanical ventilation
- Multiple ER visits for asthma
- History of previous hypercapnic attacks
- Poor compliance with medications
- Excessive reliance on short-acting bronchodilators
- Underuse of inhaled steroids
- History of need for oral steroids
- Monotherapy with long acting β -adrenergic bronchodilator
- Continued smoking
- Consider comorbidities

PHYSICAL SIGNS

- Accessory muscle use
- Tachycardia
- Tachypnea
- Difficulty speaking because of dyspnea or fatigue
- Altered level of consciousness
- Quiet chest
- Diaphoresis
- Inability to lie supine because of dyspnea
- Pulsus paradoxus
- Peak expiratory flow <30% predicted or FEV1 <25% predicted (1-2hrs after initial therapy)
- Oxygen sat <90%
- cyanosis

INVESTIGATIONS

- obtain objective measurement of severity of airflow obstruction with peak expiratory flow rate – helps determine severity and guide therapy
- consider CXR
- consider ECG/cardiac workup
- ABG if deteriorating (pCO₂ level indicates severity and fatigue)

Severity

Severity of attack can be classified into 4 categories.

MILD ASTHMA

- Exertional dyspnea/cough +/- nocturnal symptoms
- Increased use of B₂ agonist with good response
- FEV₁ or PEF >60% predicted
- O₂ sat >95%

MODERATE ASTHMA

- Dyspnea at rest, cough, congestion, chest tightness, nocturnal symptoms
- Partial relief from B₂ agonist or required more than every 4 hours
- FEV₁ or PEF 40-60% predicted
- O₂ sat >95%

SEVERE ASTHMA

- Labored respiration
- Tachycardia
- Agitated, diaphoretic
- Difficulty speaking
- No relief from B₂ agonist

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- FEV1 or PEF <40% predicted or unable to do
- O₂ sat 90-95%

POTENTIALLY FATAL

- Exhausted, confused, diaphoretic, cyanotic
- Failing heart rate
- Silent chest
- Decreased respiratory effort
- FEV1 or PEF – unable to do
- O₂ sat <90%

CLINICAL PEARLS

Treatments to Consider for Acute Asthma

1. Oxygen
2. Salbutamol (Ventolin)
3. Ipratropium (Atrovent)
4. Steroids (Prednisone, Methylprednisolone)
5. Magnesium
6. Assisted ventilation

Treatment

based on severity of attack

SUPPLEMENTAL OXYGEN

- Continuous monitoring by oximetry
- Titrate O₂ to achieve sat of at least 92%

RAPID-ACTING INHALED BRONCHODILATORS

- B₂ adrenergic bronchodilators are first line therapy in acute asthma
- Likely additional benefit when add anticholinergic bronchodilator in combination
- Optimal dose is undefined
- Titrate to clinical response and PEF
- MDI and nebulizers equally effective
- **Salbutamol**
 - MDI 4-8 puffs (100 ug/puff) q15-20 min via spacer device x 3
 - may increase to 1 puff q30-60 sec (up to 20 puffs) in severe asthma
 - nebulizer 5.0 mg q15-20 min x 3 or continuous nebulizer as necessary
- **Ipratropium bromide**
 - MDI 4-8 puffs (20ug/puff) via spacer q15-20 min x 3
 - May increase to 1 puff q30-60 sec (up to 20 puffs)
 - nebulizer 250-500ug q15-20 min x 3 or continuous nebulizer as necessary
- Inhaled bronchodilator route as effective and safer than parenteral route.

- Consider IV salbutamol or IV/SC epinephrine if response to inhaled bronchodilators is poor and patient is moribund. Evidence does not support routine use.

CORTICOSTEROIDS

- Systemic steroids for all patients with moderate/severe asthma
- Slow onset of action – administer promptly!
- No evidence that IV better than PO route
- IV often given in severe asthma where patient unable to tolerate PO (severe dyspnea, nausea and vomiting, may require intubation)
- Optimal dose not determined
- Usual starting dose:
 - prednisone 40-60 mg PO
 - methylprednisolone 125 mg IV
- May consider adding high dose inhaled steroid to systemic steroid for severe asthma but additional benefit not yet demonstrated (inhaled fluticasone 500ug q10 min x 1 hour)

MAGNESIUM SULFATE

- Effective for patients with no response to first line therapy and with severe airflow obstruction
- Given as bolus - 2 g IV over 20 min

HELIOX-OXYGEN MIXTURES

- Insufficient evidence. May be of benefit for severe obstruction and failure of first-line therapy
- No role for routine use
- Theoretical rationale based on low density of helium and airflow characteristics

EMPIRIC ANTIBIOTICS

- Not recommended as most infectious triggers are viral

AMINOPHYLLINE

- Generally not recommended as bronchodilator in acute asthma
- No additional benefits over B₂ agonists and substantial toxic effects
- Evidence is against routine use

ASSISTED VENTILATION

- May be required in patients with severe, potentially fatal asthma or those deteriorating despite aggressive first line therapy (decreased LOC, rising pCO₂ and fatigue, inability to maintain O₂ sat)
- Severe airway obstruction and hyperinflation make mechanical ventilation difficult and high

- intrathoracic pressures can lead to barotrauma and cardiovascular collapse
- Use of non-invasive positive pressure ventilation (NIPPV) is still controversial as scant evidence – not routinely recommended at this time
- Consider Ketamine (1.5mg/kg IV) as induction agent as it has bronchodilating effects

Disposition

Response to initial treatment is a better predictor of safety of discharge from the ED than the apparent severity of the attack at presentation.

- Pre-treatment FEV1 or PEF <25-30% predicted (PEF<100L/min), or post-treatment <40% predicted (PEF<200L/min) - admission usually necessary
- Post-treatment FEV1 or PEF 40-60% predicted (PEF 200-300L/min) - discharge possible based on risk factors for recurrence of severe attack
- FEV1 or PEF >60% predicted (PEF>300L/min) – discharge usually safe

PATIENTS AT RISK FOR RELAPSE

- Previous near death episode
- Frequent ED visits
- Frequent hospitalization
- Steroid dependence or recent use
- “Flash” attacks
- Prolonged duration of recent exacerbation
- Poor compliance or understanding of self-management
- Prolonged use of high dose beta-agonists

DISCHARGE INSTRUCTIONS

- Continue short acting beta-agonist - salbutamol 2-4 puffs q4h, then prn once symptoms controlled and return if use required more than q1-2h
- Oral corticosteroids for patients with moderate to severe attack - prednisone 30-60 mg/day for 7-14 days (no need to taper)
- Begin inhaled steroid – Beclomethasone or Budesonide (500-1000 ug/d) or continue inhaled steroid (if patient already on inhaled steroid) even if taking prednisone
- Review written asthma plan to prevent and manage future exacerbations
- Review use and role of different medications and avoidance of triggers
- Ensure follow-up with primary care physician or asthma specialist

COPD

Chronic obstructive pulmonary disease (COPD) is an illness involving progressive airway obstruction. It is characterized by enhanced airway inflammation and edema, with increased airflow limitation, and gas exchange defects. Abnormalities in lung mechanics and mismatch between alveolar ventilation and pulmonary blood flow can lead to respiratory failure. Risk factors for development of COPD include: tobacco smoking (major risk factor), occupational dust, and chemical exposure. The diagnosis of COPD relies on the clinical presentation of a patient with an acute increase in cardinal symptoms beyond normal day-to-day variation, including an increase in:

- Dyspnea
- Cough frequency/severity
- Sputum volume/purulence

Assessment

HISTORY

- History of premorbid functional status
- Severity of airflow limitation
- Duration of symptoms
- Number of previous episodes and hospitalizations
- Co-morbidities
- Current treatment regimen
- Previous need for mechanical ventilation

CLINICAL SIGNS

- Rapid, shallow, purse lip breathing
- Wheezing, decreased breath sounds
- Accessory muscle use
- Paradoxical chest wall movements
- Central cyanosis
- Peripheral edema, right heart failure
- Hemodynamic instability
- Decreased O₂ sat
- Decreased LOC, confusion

Investigations should include CXR, ECG, ABG, CBC, and routine biochemistry. Spirometry is not useful in managing acute exacerbations. Oximetry should be used in all patients, but cardiac monitoring only in specific situations (e.g. dysrhythmia, chest pain, moribund patient)

Treatment

OXYGEN

- Guided by pulse oximetry to treat hypoxia
- Target saturation 88-92%

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- Goal PaO₂ 60-65mmHg
- Venturi mask preferred as delivers more precise O₂ concentrations
- Beware chronic CO₂ retainers whose respiratory drive can be suppressed by higher oxygen concentrations leading to CO₂ narcosis, academia, and eventually respiratory arrest

BRONCHODILATORS

- Inhaled B₂ agonist: Salbutamol – 2.5-5 mg via nebulizer q15min x3 prn or 4-8 puffs via MDI with spacer q15min x3 prn
- +/- anticholinergic: Ipratropium – 500ug via nebulizer q15min x3 prn or 4-8 puffs q15min x3 prn
- No difference between MDI and nebulizer
- No role for long-acting B₂ agonists in acute setting
- Methylxanthines (theophylline, aminophylline) are rarely used as side effects are significant and benefits inconsistent

CORTICOSTEROIDS

- Systemic steroids shorten recovery time, improve lung function and arterial hypoxemia, and reduce risk of early relapse and length of hospital stay
- Oral equal to IV in most exacerbations
- IV given in severe exacerbations, in those not responding to oral steroids, or to those unable to take oral medication
- IV methylprednisolone 125 mg BID-QID
- Oral prednisone 40-60 mg for 7-14 days (taper only if on chronic oral steroids)
- No good evidence for inhaled steroids

ANTIBIOTICS

- Usually given to patients with all 3 cardinal symptoms, 2 cardinal symptoms if increased sputum purulence is present, or if mechanical ventilation is required
- Choice of therapy based on local resistance patterns and usual length is 5-10 days
- Simple (no risk factors)
 - 2nd generation macrolide (e.g. azithromycin), 2nd or 3rd generation cephalosporin (e.g. cefuroxime, cefixime), doxycycline, or TMP/SMX
- Acute exacerbation with risk factors (i.e. >4 exacerbations/yr, cardiac disease, home O₂, antibiotic use in past 3 months, poor underlying lung function)
- Fluoroquinolone, amoxicillin-clavulanic acid

VENTILATION

Non-Invasive Positive Pressure Ventilation (NIPPV): CPAP or BiPAP

- Good evidence for reduction of mortality and intubation rates
- Consider if significant respiratory acidosis/CO₂ retention or severe dyspnea with signs of fatigue

INTUBATION

- Unable to tolerate or fails NIPPV
- Altered LOC, severe agitation, unable to cooperate
- High aspiration risk
- Life threatening hypoxemia
- Cardiovascular instability
- Respiratory or cardiac arrest

Admission Criteria

- No good guidelines exist
- Use clinical judgment regarding ability of patient to cope in most cases
- Lower threshold for admission with pneumonia and other co-morbid illnesses (diabetes, CHF, CAD, alcohol abuse)

WHAT'S NEW

The last ten years has seen a sharp increase in the use of NIPPV for severe COPD exacerbations. NIPPV significantly reduces the risk of intubation and in-hospital mortality, especially in those who demonstrate respiratory acidosis.

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17: GASTROINTESTINAL BLEEDING

Rajani Vairavanathan

OBJECTIVES

1. To recognize patients presenting with gastrointestinal bleeding
2. To differentiate clinically between upper and lower GI bleeds
3. To develop an approach to the management of GI bleeds
4. To develop an awareness of the adjunctive medical treatments available
5. To understand the role of endoscopy and diagnostic imaging in the management of GI bleeds

INTRODUCTION

Gastrointestinal (GI) bleed is a common presenting problem in the ER. The overall mortality rate is approximately 10%. The approach to GI bleeds depends on the patient's age and whether the bleeding is from a proximal ('upper') or distal ('lower') part of the bowel. This division occurs at the ligament of Treitz. In all patients who present with GI bleeding, there is no source identified in 10% of cases.

UPPER GI BLEED (UGIB)

The common causes of UGIB in adults are (in order of frequency):

1. Peptic Ulcer Disease (PUD): H.pylori or NSAIDS
2. Varices (esophageal or gastric)
3. Mallory-Weiss tears
4. Gastritis
5. Esophagitis

In adults, 75% of UGIB are due to PUD, gastric erosions and varices. Patients with PUD should be tested for H.Pylori and treated if positive. Untreated H.Pylori ulcers have higher chance of rebleeding. In patients with UGIB and those with a history of AAA repair, surgical evaluation of the graft is needed to rule out an aortoenteric fistula as the source of the UGIB.

In children, esophagitis, gastritis, and PUD are the most common causes of UGIB.

Common Causes of UGIB in Children

Neonates

1. Esophagitis
2. Gastritis
3. Gastroduodenal ulcers
4. Coagulopathy
5. Cow's milk allergy
6. Congenital malformation
7. Mallory-Weiss tear

Infants + Toddlers

1. Ingestion of foreign body
2. Same causes as in neonates

Older Children and Adolescents

1. Similar causes to Adults

LOWER GI BLEED (LGIB)

The common causes of LGIB in adults are (in order of frequency):

1. Diverticulosis
2. Angiodysplasia (dilated tortuous submucosal vessels)
3. Neoplasm
4. Inflammatory Bowel Disease
5. Anorectal disease (proctitis, hemorrhoids, fissures)

In adults, 80% of LGIB are due to diverticulosis and angiodysplasia. An important point is that the presence of hemorrhoids or fissures in a patient presenting with a LGIB doesn't eliminate the need to search for other causes of bleeding and for colonoscopy.

In children, LGIB are commonly due to infective colitis and IBD. In children less than 2 years old, LGIB are commonly caused by meckel's diverticulum and intussusception.

Common Causes of LGIB in Children

Neonates

1. Swallowed maternal blood
2. Anorectal fissures
3. Necrotizing enterocolitis
4. Malrotation with midgut volvulus
5. Hirschsprung
6. Coagulopathy

Infants + Toddlers

1. Anorectal fissures
2. Milk or soy-induced enterocolitis
3. Intussusception
4. Meckel's diverticulum
5. Hemolytic Uremic Syndrome/ Henoch-Schonlein Purpura
6. Lymphonodular hyperplasia
7. Gastrointestinal duplication

Older Children and Adolescents

1. Infections
2. Polyps
3. Inflammatory Bowel Disease

PRESSENTATION

GI bleeds can present with a wide spectrum of symptoms. If slow and chronic, patients may present with non-specific findings of anemia, chest pain, shortness of breath, or fatigue. In more acute GI bleeds, patients can present with hemodynamic instability. Older patients with GI bleeds may present with symptoms of weakness, dizziness, shortness of breath or loss of consciousness.

Initial evaluation includes assessment of ABCs then a focused history and physical exam to determine the cause and location (upper or lower) of the bleed. UGIB can present as hematemesis, “coffee ground” emesis and/or melena. In UGIB, melena (black tarry stools) is the most common presentation. To confirm melena, the stool should be tested for the presence of occult blood with a guaiac test. It will test positive (blue) in the presence of occult blood (OB). Recent ingestion of bismuth (Pepto-Bismol) or iron can cause stools to be dark and to be mistaken for melena. However, when the stool is tested for the presence of occult blood with the guaiac test, it will test negative in these cases. Hematemesis (bright red or coffee-ground) and hematochezia can signify the presence of UGIB. However, hematochezia is most commonly associated with a LGIB.

HISTORY

History has limited value in predicting the site or quantity of GI bleeding. However, when interviewing a patient it is important to inquire about the following:

- symptoms or risks for GI bleeding
- protracted wretching or vomiting
- features of malignancy

- previous GI bleeds and their cause
- presence of liver disease or alcohol use
- surgical history (AAA repair or peptic ulcer surgery)
- medication review (especially anticoagulants such as coumadin, ASA, clopidogrel (Plavix), dabigatran (Pradax), NSAIDS)

PHYSICAL EXAM

Physical exam should start with a full set of vital signs including mental status (alert, drowsy, comatose) as part of your ABCs. The minimum physical exam should include an abdominal and digital rectal exam (DRE) to detect any masses and observe the colour of stool (melena vs. hematochezia). Any stool obtained from the DRE without the presence of gross blood should be tested for occult blood. Guaiac testing of stool for occult blood (OB) has a few false positives such as ingestion of red meat, red fruits, peroxidase containing foods (broccoli, cauliflower, rhubarb) and methylene blue. Consumption of vitamin C can yield a false negative result.

CLINICAL PEARL

Recent ingestion of bismuth (Pepto-Bismol) or iron can cause stools to be dark and to be mistaken for melena.

It is important to look for signs of coagulopathy (ecchymosis, telangiectasia) and stigmata of liver disease.

INVESTIGATIONS

Any patient suspected of having a GI bleed should have the following laboratory investigations: CBC, PTT/INR, group + screen (consider cross and type), BUN/Cr, (BUN/creatinine ratio > 10:1 is suggestive of UGIB) and LFT.

It is important to repeat lab values in 4-6 hours to follow the clinical course. The hemoglobin and hematocrit may initially be normal if performed immediately after an episode of GI bleed since volume redistribution takes time to occur.

Diagnostic Imaging

Role of abdominal x-rays is limited in UGIB. An upright abdominal film is useful to rule out the presence of free air from a perforated ulcer.

CT abdomen can be useful as the initial test in the management of LGIB. It can diagnosis a pathologic cause of the LGIB and provide info regarding arterial anatomy. For obscure nonlocalized recurrent bleeding with previous negative OGD and colonoscopy, CT may be useful.

MANAGEMENT

The goal of therapy with a patient experiencing a GI bleed is to stabilize the patient, correct shock and correct any coagulation abnormalities.

Patients should be placed on a cardiac monitor. In the emergency department, whether the patient should be monitored in the acute room or resuscitation/trauma room depends on their hemodynamic status.

The initial approach, as in any emergency patient involves the ABCs. Consider intubation for airway protection and risk of aspiration in patients with decreased level of consciousness or significant hematemesis. Two large bore IV (16 or 18G) should be placed in the antecubital fossa. Start immediate fluid resuscitation with IV crystalloid solutions such as normal saline (NS) or Ringer's lactate (RL).

Keep patients NPO in case of need for urgent endoscopy or intubation in the event of a massive rebleed.

Prepare for potential transfusion of blood products for either resuscitation or correction of coagulation abnormalities. Transfuse PRBCs to young, healthy patients if they are hemodynamically unstable after aggressive fluid resuscitation ($> 2L$), hematocrit $< 20\%$ or hemoglobin $< 70 \text{ g/L}$. In elderly patients and CAD patients, PRBCs are transfused when hematocrit $< 30\%$ or hemoglobin < 100 . Transfuse platelets if platelet count $< 50,000$. In the presence of active GI bleed, give vitamin K and fresh frozen plasma (FFP) if INR > 1.5 .

Unactivated prothrombin-complex concentrates (PCC), for example Octaplex, consists of vitamin K dependent coagulation factors and can be used to correct INR. PCC normalizes the INR more rapidly and with less volume load than FFP. Vitamin K

must be given along with PCC, otherwise a rebound rise in INR will occur.

Role of NG aspirate is controversial. A positive NG aspirate leads to shorter time to endoscopy but no change in mortality or length of stay.

The most important clinical predictors of increased risk of rebleeding or mortality from a nonvariceal UGIB are age > 65 , hemodynamic instability, comorbid illness, and fresh blood on rectal exam, in the vomitus or in the nasogastric lavage.

Consider early consultation with GI service, general surgery and ICU if clinically unstable.

Medical Therapy in GI Bleeds

Proton pump inhibitors (PPI) are often used in the treatment of UGIB due to PUD. If an UGIB is suspected, PPI should be initiated. In patients with documented PUD, PPIs have been shown to decrease need for endoscopy in patients with active bleeds and decrease the incidence of a rebleeding post-ulcer bleed in the following 24h. Pantoloc is given initially as a bolus of 80mg IV then an 8mg/h infusion.

Octreotide (Sandostatin) and somatostatin have been shown to have benefit in patients with documented esophageal varices. They decrease splanchnic blood flow and reduce portal pressure. Octreotide is given initially as a bolus of 25-50ug IV then a 50ug/h infusion for 24h.

Vasopressin can be used with caution in patients with severe GI variceal bleeds. Vasopressin is given as 20 units IV over 20min then 0.2 – 0.4 units/min infusion.

Promotility agents (metoclopramide, erythromycin) or gastric lavage can be useful prior to endoscopy in selected patients suspected of having large amounts of blood or food in the stomach.

Endoscopy and Angiography

Endoscopy is the gold standard in the diagnosis and treatment of UGIB. The goals of endoscopy are to document the source of bleeding, help determine the risk of rebleeding and offer treatment. If performed within 12-24h of patient presentation, a lesion can be identified in 85-90% of the cases. Lesions can be biopsied and treated with electrocautery, banding, or injection. The risks associated with endoscopy include aspiration, perforation and increased risk of rebleeding during the intervention.

Colonoscopy is the diagnostic modality of choice in stable patients presenting with acute LGIB. Referral for colonoscopy should be made for all patients presenting with LGIB. Endoscopy should be considered if colonoscopy does not identify a source, especially in the presence of UGIB symptoms or anemia.

Radionuclide imaging and angiography may be used when urgent colonoscopy cannot identify a bleeding source or when the bleeding is too fast to allow a colonoscopic exam.

Radionuclide imaging (tagged red blood cell scan) can detect active bleeding >0.1 cc/min. It is more sensitive and less specific than angiography or colonoscopy. It identifies an area of a bleed but not the precise location.

Angiography requires faster rates of bleeding (1 cc/min) to identify a bleeding source. It has 100% specificity and 30-45% sensitivity. Angiography allows treatment once a bleeding site is identified with intra-arterial infusion of vasopressin or arterial embolization.

Surgery

Surgery consultation is crucial in unstable patients not responding to intravascular volume replacement and/or endoscopy is unable to control bleeding. There is at least a 25% mortality rate in this group of patients. Primary surgical management is indicated in patients with an UGIB associated with a perforated viscus (ulcer) or Boerhaave syndrome (ruptured esophagus from repeated vomiting). In LGIB, preoperative localization of a lesion is necessary to direct surgical intervention.

SUMMARY

- GI bleed is a common presentation in the ED
- Initial management involves ensuring ABCs and volume resuscitation
- Correct any coagulopathy and consider transfusion of PRBCs
- Consider PPI in all suspected UGIB, especially in previously document PUD

- Consider octreotide in suspected variceal UGIB
- Endoscopy is the diagnostic modality of choice in UGIB
- Colonoscopy is the diagnostic modality of choice in LGIB
- Radionuclide imaging and angiography are used in unstable patients or actively bleeding patients in which colonoscopy cannot identify a source of bleeding
- Consider consultations with ICU, GI and general surgery services early for unstable patients

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18: STROKE & TIA

Walter Himmel, Jennifer Devon

OBJECTIVES

1. To define TIA and stroke
2. To rapidly identify patients with TIA and stroke in the emergency department
3. To know the common ‘stroke mimics’
4. To understand the antiplatelet and anticoagulant options for TIA and stroke
5. To know the workup required for TIA and stroke
6. To summarize the management of TIA and stroke in the emergency department
7. To understand the use of thrombolytics for acute ischemic stroke

INTRODUCTION

Stroke is a sudden loss of brain function resulting from an interference with the blood supply to the brain with persistence of symptoms of more than 24 hours, or with evidence of infarction on neuroimaging. Strokes are the third most common cause of death in North America after cardiovascular disease and cancer. They are the most common cause of adult disability. The availability of early CT scans, anti-platelet agents and thrombolytics have led to the replacement of the pessimistic attitudes of the past with a more aggressive and enthusiastic view of medical interventions. The etiology of strokes is about 80% ischemic and 20% hemorrhagic.

A transient ischemic attack (TIA) has been defined traditionally as an acute episode of temporary and focal loss of cerebral function of vascular (occlusive) origin lasting less than 24 hours. More recently the American Stroke Association definition has removed the time component of the definition to focus more on a tissue-based definition. With modern technology, up to 1/3 of patients with symptoms <24 hours will have evidence of infarction on imaging. Their new definition is “a transient episode of neurological dysfunction caused by focal brain, spinal cord, or retinal ischemia, without acute infarction”. Most TIAs last less than 1 hour. An Initial TIA lasting more than 10 minutes is a risk factor for an early stroke. TIAs have the same etiology as strokes and many of the preventive

recommendations are applicable to both. The following discussion will be limited to ischemic strokes and TIAs in adults.

Symptoms

Patients and the general public should be educated to look for sudden onset of any of these symptoms and if present, seek medical attention immediately because “time is brain”.

- Weakness or numbness in the arm, leg, or face
- Speech disturbance such as loss of speech or difficulty understanding speech
- Visual disturbance especially in one eye or diplopia
- Severe headache (usually more concerning for subarachnoid hemorrhage or intracerebral hemorrhage)
- Vertigo or loss of balance especially with any of the above

Is it a stroke?

Some of the key features that help predict the diagnosis of stroke are:

- **Level of consciousness:** normal or slightly decreased in stroke (significant decrease in level of consciousness usually suggests hemorrhage, a stroke mimic, or a severe brain stem stroke)
- **Onset:** the most typical feature is abrupt onset (usually maximal at onset)
- **Focal symptoms:** usually fit the distribution of a single vascular territory
- **Headache:** no headache or mild headache in ischemic stroke (pain more suggestive of hemorrhage or dissection)

Suspect an alternative diagnosis if:

- Decreased level of consciousness
- Gradual onset
- Fever
- Fluctuating signs
- No focal signs

Stroke Mimics

- Seizure: aura, focal deficits, post-ictal phase, often decreased level of consciousness, tend to have positive rather than negative symptoms (e.g. paresthesias rather than numbness)
- Migraine: more gradual onset over many minutes, headache, tend to have positive rather than negative symptoms
- Transient global amnesia: pure amnesia, no focal signs, usually lasts 24 hours
- Syncope: sudden loss of consciousness with rapid recovery
- Hypoglycemia: usually more generalized features BUT may mimic a stroke
- Metabolic encephalopathy/overdose/sepsis: non focal, predominant confusion, slurred speech, decreased level of consciousness without other focal findings
- Tumour: usually not sudden onset, but may mimic TIA/stroke
- Encephalitis: confusion and fever are typical
- Subdural hematoma: less sudden onset
- Bell's palsy: weak frontalis muscle due to peripheral nerve lesion
- Peripheral vertigo: isolated vertigo is usually not a TIA/stroke
- Conversion disorders: inconsistent examination, neurologic findings not in a vascular distribution

EVALUATION

- Stabilize ABC's
- Brief neurological exam
 - Identify possible stroke
 - Exclude stroke mimics as above
- History
 - Most important question is time of onset of symptoms
- Physical Exam
 - Vitals, oxygen saturation, temperature, capillary blood glucose measurement
 - Head and neck – look for signs of carotid bruit or seizure
 - Cardiovascular exam – look for signs of aortic dissection, arrhythmias, valvular conditions
 - Respiratory and abdominal examination – look for co-morbidities
 - Skin and extremities – look for signs of systemic illness such as petechiae, purpura, jaundice
- Neurologic Exam
 - Thorough neurological exam

- Use a standardized scale such as National Institute of Health Stroke Scale (see appendix)

INITIAL INVESTIGATIONS

- Basic blood work (hematology, chemistry, coagulation, glucose)
- ECG +/- telemetry (to identify atrial fibrillation)
 - 1 out of every 6 stroke patients is found to have atrial fibrillation
- Neuroimaging (to rule out intracranial bleed and stroke mimics)
 - Noncontrast CT head or MRI
- Vascular imaging of carotid and vertebral arteries within 24 hours
 - Doppler ultrasound
 - CT angiography or MR angiography (often done at the time of initial neuroimaging)

Why get very early vascular imaging?

TIA or stroke patients who have carotid artery stenosis are considered to be symptomatic and generally will benefit from carotid endarterectomy. Endarterectomy outcomes are far better when surgery is performed within 2 weeks of the event. In patients with severe (>70%) symptomatic stenosis, surgery with endarterectomy is highly beneficial with a number needed to treat (NNT) of 6 to prevent one stroke over 5 years. The NNT for symptomatic moderate (50-69%) stenosis is 22 to prevent one stroke over 5 years.

Therefore, vascular imaging of the neck arteries should be done within 24 hours of the event and if there is an ipsilateral stenosis of 50-99%, the patient should be referred to an experienced neurosurgeon or vascular surgeon as soon as possible for consideration of endarterectomy.

EARLY MANAGEMENT OF STROKE

- **ABCs:** O₂, IV, cardiac monitor; consider intubation for severe strokes
- **Position:** use minimal elevation of head necessary to decrease aspiration (keeping the head of bed flat is the best, if possible)
- **NPO:** until swallowing has been formally assessed
- **Fluids:** normal saline only to keep patient euvolemic
- **Cardiac monitoring:** 24 – 72 hrs of cardiac monitoring to look for atrial fibrillation
- **O₂:** keep O₂ saturation at or above 94%
- **Antiplatelet therapy:** see below

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- **Hypertension:**
 - Do not treat aggressively in first 24 hours due to the concern that immediate lowering of BP may lead to under-perfusion of the brain area around the ischemic segment (penumbra) and further brain ischemia.
 - Exceptions:
 - BP > 220/120 (if so, consider lowering BP by 15%-20% in first 24 hrs)
 - BP > 185/110 if patient is a candidate to receive t-PA
- **Hypotension:** rare, but if present, look for underlying cause (e.g. aortic dissection,) and treat with fluids
- **Hyperthermia:** treat with cooling and antipyretics and search for cause
- **Anticoagulation:**
 - DVT prophylaxis for immobile patients: heparin or low molecular weight heparin (LMWH) subcutaneously
- **Hyperglycemia:** associated with worse outcomes, so target glucose levels at 5-10 mmol
- **Cardiovascular risk factors:** should be evaluated and aggressively managed. Smokers should be advised to quit and provided support to do so.
- **Admission:** evidence supports admission to dedicated stroke unit if possible

Antiplatelet Therapy

Studies have found antiplatelet therapy after stroke or TIA reduces the risk of further vascular events by about 25%. All of the following agents are acceptable first line choices for secondary stroke prevention and the decision about which agent to use should be determined based on the clinical scenario.

Aspirin

- Loading dose of 160 mg -325 mg as soon as possible after CT excludes hemorrhage
- Followed by long term dose of 81 mg per day
- Those who have failed on ASA should use ASA/dipyridamole or clopidogrel

Clopidogrel (Plavix)

- Loading dose of 300mg
- Clopidogrel 75 mg/day alone is as effective as ASA in TIA and stroke
 - Use in patients with ASA allergy or patients who have failed on ASA

ASA + Dipyridamole (Aggrenox)

- ASA/dipyridamole is an option for ASA failures
- Warn patients regarding headaches and

consider acetaminophen initially as headache prophylaxis

THROMBOLYTICS

- Use of thrombolytics has become standard of care in many centres. Successful implementation requires great coordination of many services; therefore most large urban centers are developing centralized Stroke Centers.
- Intravenous Tissue Plasminogen Activator (alteplase), the most commonly used thrombolytic for stroke, is currently approved in Canada if given within 4.5 hours of onset of symptoms, but the benefit is likely better if given sooner ideally within 90 minutes.
- Eligibility determined based on NINDS and ECASS III Stroke Studies
 - Consult neurologist and/or Stroke Team
 - Inclusion Criteria
 - Adults with onset of measurable deficit up to 4.5 hours prior to alteplase administration
 - Exclusion Criteria
 - History of intracranial hemorrhage at any time
 - Stroke or serious head/spine trauma in past 3 months
 - Major surgery in the past 2 weeks
 - Non-compressible arterial puncture in past 7 days
 - Elevated PTT or INR, or platelets <100,000
 - Any other condition that could increase bleeding
 - Symptoms suggestive of subarachnoid hemorrhage
 - Symptoms due to another nonischemic condition e.g. Todd's paralysis
 - Blood glucose <2.7 or >22.2
 - Patient on dabigatran (Pradax) and compliant with medication
 - Any hemorrhage on brain CT
 - Persistently elevated BP > 185/110
 - Evidence of infarction of >33% of the MCA territory
 - Mild or rapidly-improving symptoms
 - Very severe symptoms (NIHSS >22)
 - Brain tumour
 - Metastatic cancer diagnosis
 - Pregnancy (relative contraindication)
 - Seizure at onset (relative contraindication)

TREATMENT OF TIA & MINOR STROKE

A TIA is a warning sign of atherosclerotic disease in general and of an impending stroke specifically. Approximately 10% of patients will have a stroke within 1 week after a TIA.

ABCD² Score

Age > 60 = 1 point
Blood Pressure > 140/90 = 1 point
Clinical Features
Unilateral weakness = 2 points
Speech deficit = 1 point
Other symptoms = 0 points
Duration of symptoms
< 10 min = 0 points
10 -59 minutes = 1 point
>59 minutes = 2 points
Diabetes = 1 point

ABCD² Score:

A patient with TIA who has a higher score may have a higher risk for a stroke; however, a recent study has failed to validate this score. Nonetheless, consider admission to hospital for patients with an ABCD2 score of 5 or 6.

CHA₂ DS₂ Score

CHF = 1 point
HTN = 1 point
Age > 75 = 2 points
Age 65-74 = 1 point
Diabetes = 1 point
Stroke or TIA previously = 2 points
Vascular disease (MI, peripheral arterial disease) = 1 point
Sex category (female) = 1 point

Anticoagulation in the Presence of Atrial Fibrillation

Primary Stroke Prevention

- Patients diagnosed with atrial fibrillation should be risk-stratified for risk of stroke using a standardized tool such as CHA₂ DS₂ - VASc
 - If score 0, patients should receive daily ASA
 - If score 1, patients may receive either warfarin or dabigatran or ASA
 - If score 2 or greater, patients should receive either warfarin or dabigatran
- Warfarin: Target INR of 2.5 (range 2.0-3.0)
- Dabigatran: 150 mg twice daily for most individuals; 110 mg twice daily for patients aged 80 or more years and for patients at risk of bleeding; contra-indicated in renal failure

Secondary Stroke Prevention

- Atrial fibrillation + TIA or minor stroke: start oral anticoagulation (warfarin or dabigatran) immediately
- Atrial fibrillation + acute ischemic stroke
 - Should be on oral anticoagulation but timing for starting is unclear. It is common to wait 2-14 days to repeat CT to exclude intracranial hemorrhage.
 - Heparin or LMWH not recommended
- Consult your neurologist

SUMMARY

- A TIA is a brief episode of neurological dysfunction caused by brain or retinal ischemia with symptoms typically lasting < 1 hour and without evidence of acute infarction on brain imaging, whereas a stroke has symptoms that last for >24 hours or radiologic evidence of infarction
- Be careful to rule out common ‘stroke mimics’ in the emergency department
- Patients with TIAs or stroke require workup with CBC, glucose, ECG, CT head and early Doppler ultrasound of the carotid arteries at minimum.
- Patients should be rapidly evaluated with a careful history and physical examination at a Stroke Centre ideally if they are potential candidates for thrombolytics
- Thrombolytics should be considered if they can be administered within 4.5 hours of the onset of stroke
- Careful attention should be paid to patient position, fluid balance, blood pressure, glucose, fever and oxygenation in those requiring admission to hospital for stroke
- Secondary stroke prevention for most patients should include an antiplatelet agent such as

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- aspirin, clopidigrel or ASA/dipyridamole
- Anticoagulation should be initiated in most patients with atrial fibrillation who have had a stroke for secondary stroke prevention and in selected patients with atrial fibrillation who have not yet had a stroke for primary stroke prevention

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Appendix: National Institutes of Health Stroke Scale

Title	Responses and Scores	Title	Responses and Scores
1.A) Level of consciousness	0—alert 1—drowsy 2—obtunded 3—coma/unresponsive	6. Motor function (leg)	0—no drift 1—drift before 5 seconds 2—falls before 5 seconds 3—no effort against gravity 4—no movement
1.B) Orientation questions	0—answers both correctly 1—answers one correctly 2—answers neither correctly	7. Limb ataxia	0—no ataxia 1—ataxia in 1 limb 2—ataxia in 2 limbs
1.C) Response to commands	0—performs both tasks correctly 1—performs one task correctly 2—performs neither	8. Sensory	0—no sensory loss 1—mild sensory loss 2—severe sensory loss
2. Gaze	0—normal horizontal movements 1—partial gaze palsy 2—complete gaze palsy	9. Language	0—normal 1—mild aphasia 2—severe aphasia 3—mute or global aphasia
3. Visual fields	0—no visual field defect 1—partial hemianopia 2—complete hemianopia 3—bilateral hemianopia	10. Articulation	0—normal 1—mild dysarthria 2—severe dysarthria
4. Facial movement	0—normal 1—minor facial weakness 2—partial facial weakness 3—complete unilateral palsy	11. Extinction or inattention	0—absent 1—mild (loss 1 sensory modality) 2—severe (loss 2 modalities)
5. Motor function (arm)	0—no drift a. Left b. Right 1—drift before 5 seconds 2—falls before 10 seconds 3—no effort against gravity 4—no movement		

19: STATUS EPILEPTICUS

Heather Sues-McKay, Cheryl Hunchak

OBJECTIVES

1. To recognize status epilepticus as a medical emergency
2. To understand and be able to identify the causes of status epilepticus
3. To have a structured approach to the diagnosis and management of status epilepticus

INTRODUCTION

A seizure is a clinical manifestation of abnormal neurologic function caused by aberrant electrical firing of neurons in the brain. Seizures fall into three main categories: 1) generalized, 2) partial, and 3) complex partial.

Status Epilepticus (SE) is defined as **continuous or intermittent seizure activity for more than 5 minutes without regaining consciousness**. This may appear in several forms: continuous generalized seizure activity; recurrent generalized seizures whereby between episodes the patient has persistent depressed level of consciousness; recurrent partial seizures; or rarely, non-convulsive seizures. Non-convulsive seizures are often very difficult to identify and may only manifest as subtle blinking, eye deviation or twitching. Occasionally depressed level of consciousness is the only symptom. This diagnosis is often only confirmed by EEG monitoring.

When a continuously seizing patient is brought to the Emergency Department (ED), they have likely already experienced enough seizure activity to qualify for the diagnosis of SE. Sometimes the patient will begin seizing in the ED and the diagnosis can then evolve from seizure to SE. Aggressive treatment of SE is important due to the high associated morbidity and mortality. It is generally accepted that SE should be terminated within 30 minutes to optimize the chance of avoiding permanent neurological sequelae.

Generalized Status Epilepticus

Generalised SE is the most common form and, of the sub-types, 'generalized tonic-clonic' SE is the most prevalent. The fact that a seizure is generalized implies simultaneous electrical discharge of the entire cerebral cortex with concomitant loss of consciousness. The seizure activity may originate globally or spread from a focal source.

ETIOLOGY

Approximately 30% of all episodes of SE occur in patients who have a known seizure disorder (epilepsy). The remainder are due to varying etiologies that fall into the following categories:

Withdrawal

- Anti-convulsants (non-compliance)
- Alcohols
- Benzodiazepines
- Barbituates

Toxins (drugs that lower seizure threshold)

- Anticholinergics
- Sympathomimetics
- ASA, salicylates
- Lithium
- Lidocaine
- Isoniazid
- And many others....

Acute Structural Injury

- Brain tumor/ mets
- Intracranial hemorrhage(s)
- Stroke

Cerebral Injury

- Infection (abscess, meningitis, encephalitis)
- Trauma
- Hypoxia

Chronic Structural Injury

- Prior traumatic brain injury
- Prior intracranial surgery
- Cerebral palsy
- AVMs

Metabolic Abnormalities

- Hypoglycemia
- Hypo/hypernatremia
- Hypocalcemia
- Lactic acidosis (severe)
- Uremia
- Hypomagnesemia

Chronic Epilepsy

Pregnancy/Postpartum (eclampsia)

Twenty percent of patients with new onset of generalized seizures will have an episode of SE within 5 years. Up to half of SE occurs in patients without a prior history of seizures.

MORBIDITY AND MORTALITY

- Mortality for first episode SE up to 20-30%
- Many deaths in patients treated for SE occur in the weeks following treatment, and in up to 89% of cases, relate to the underlying cause of the SE
- Prolonged SE (>30 min) can lead to permanent neuronal damage

- Acute symptomatic SE is associated with six times higher risk of death than SE related to chronic epilepsy
- Patients with generalised convulsive status (GCSE) respond better to treatment and have a better prognosis than those with non-convulsive status epilepticus (NCSE)
- NCSE is common in patients with extensive brain damage, e.g. large intracranial haemorrhage, hypoxic CNS injury

CLINICAL PRESENTATION

History

The patient who presents in SE cannot provide any history. Information should be sought from family, bystanders and EMS personnel. Important historical features include:

- Time of onset of seizure
- History of prior seizures
- Ingestion of toxins/ETOH
- Associated symptoms – headache, fever
- Past medical history (epilepsy, cancer, HIV, TB, substance abuse, pregnancy or recent delivery)

It is important to differentiate tonic-clonic seizures from syncope and other conditions that cause altered mental status. Some clues that help to differentiate a tonic-clonic seizure from other conditions are: abrupt onset, ‘foaming at the mouth’, tongue biting, urine or fecal incontinence, and a post-ictal state of decreased level of consciousness typically lasting 20-30 minutes.

Physical Examination

SE is a true medical emergency. Physical examination should always begin with the ABC approach. Many patients are unable to protect their airway and/or hypoxic and require intubation. Patients not requiring immediate intubation often become profoundly sedated from the administration of anticonvulsant medications and ultimately require definitive airway management. Supplemental O₂ should be administered. Breathing and circulatory assessment follow, although this may not indicate a source for seizure. A brief neurological exam might reveal evidence of intracranial pathology as the cause for seizure. Always assess a patient’s pupils and observe the movement of their extremities. Look at their skin, pupils and vital signs for evidence of toxidromes.

INVESTIGATIONS

****Treatment and investigations should take place simultaneously in patients with SE****

- Immediate bedside glucose level
- CBC, electrolytes, anticonvulsant drug levels, venous blood gas, calcium, Mg, ETOH level, serum salicylates, urine toxicology screen
- ECG, LP, CT head and other imaging must be deferred until seizure activity has stopped

Once hypoglycemia has been ruled out and/or treated, treatment for generalized seizures should commence.

Consider continuous EEG monitoring (rarely) if:

- Patient does not awaken promptly (suspect non-convulsive SE)
- Large doses of barbiturates used
- Use of longer acting neuromuscular paralysis agents (rocuronium, vecuronium, pancuronium)

TREATMENT

Initial treatment, as with all emergent conditions starts with the ABCs: airway management, oxygen, establishing large bore intravenous lines, cardiac monitoring and IV fluids. Check for hypoglycemia and give 1 ampule of IV D50W immediately if needed. While securing the ABCs and checking the serum glucose at the bedside, prepare to administer a benzodiazepine to terminate seizure activity, followed by a loading dose of phenytoin. If these measures fail to control the seizure activity the patient should be intubated (if not already) and have phenobarbital administered. If IV access is not possible, establish an intraosseous (IO) line for medication administration.

Doses are for normal size adult patients (~70kg):

Benzodiazepines:

- **Lorazepam:** 2-4mg IV q2min, up to 0.1 mg/kg IV, maximum 8–10 mg
- **Diazepam:** 5-10 mg IV, up to 0.15 mg/kg IV, maximum 20-30 mg
- **Midazolam:** 10 mg IM (for patients >40kg) – if IV route unavailable

New evidence suggests that **IM Midazolam is equivalent to IV Lorazepam** for treatment of seizure in the pre-hospital setting if IV access cannot be established. **IV Lorazepam is preferred** to IV Diazepam, if available, due to its longer duration of action (12-24hr vs. 15-60 mins). The onset of action is comparable (~2 mins). If seizure activity fails to

resolve within 5-10 minutes start a second IV line and add:

- Phenytoin : 20 mg/kg IV administered at a rate of 25-50 mg/min (may repeat 10mg/kg dose once to total dose 30 mg/kg) *must be run in separate IV line
- or
- Fosphenytoin: 20 PE/kg IV or IM (PE = phenytoin equivalents) at rate of 100-150 mg PE/min

If above medications are maximally dosed and SE continues, obtain ICU consultation and start one or more of the following infusions:

- Phenobarbital 20 mg/kg IV at 50 mg/min
- Midazolam: 0.2 mg/kg IV loading dose and then infusion of 0.05-0.5 mg/kg/hr
- Propofol: 2-5 mg/kg IV loading dose and then 2-10 mg/kg/hr

CNS and respiratory depression are associated with the benzodiazepines and barbiturates, and careful airway monitoring and management is mandatory.

Non-CNS Complications of SE

- Cardiovascular stress (may be increased by anticonvulsant drugs) → MI, arrhythmias, cardiac arrest
- Hypotension, either as a consequence of prolonged seizures or drug therapy
- Respiratory failure, either due to seizures or drug therapy
- Repeated muscle contractions → rhabdomyolysis, hyperthermia
- Increased parasympathetic activity → sweating, salivary and bronchial hypersecretion
- Non-cardiogenic pulmonary edema

POST TREATMENT STABILIZATION

Many patients will experience up to 2 hours of post-ictal coma or lethargy. This may be markedly extended in those treated with phenobarbital. In patients that do not regain full consciousness over a typical length of time (20-30 mins), look for other causes of prolonged loss of consciousness (e.g. drug ingestions, subarachnoid haemorrhage, meningitis, NCSE). All patients should undergo brain imaging with CT head, if available. When the possibility of generalised NCSE exists, obtain an urgent EEG.

Insert a foley catheter, if not already done, and monitor urine output, obtain a urine tox screen and monitor for rhabdomyolysis. Administer IV fluids to combat the hypotensive effects of the drugs given, prevent rhabdomyolysis, maintain cerebral perfusion pressure, and help cool the patient if they are hyperthermic.

PSEUDOSEIZURES PRESENTING AS SE

Pseudoseizures can be extremely difficult to differentiate from SE, and can occur concomitantly in patients with a documented history of seizures. Suspect pseudoseizures in patients not responding to usual treatments. Patients with SE will develop a transient physiologic leucocytosis, elevation of serum lactate and prolactin levels. These markers will not be elevated in patients with pseudoseizures. (Note: prolactin levels are not routinely drawn in the ED). Pseudoseizures tend to occur in the presence of others and in response to emotional upset. Patients tend to be able to protect themselves from injury during the events. Head thrashing, pelvic thrusting and non-symmetric, clonic extremity movements are more typical of pseudoseizures.

SUMMARY

- SE is defined as more than 5 minutes of continuous seizure activity or a series of seizures between which the patient does not regain complete consciousness
- Withdrawal of anticonvulsants, cerebrovascular disease and alcohol withdrawal account for the majority of cases of SE
- Immediate bedside glucose level is imperative in the initial assessment of SE
- After securing the ABCs and checking a capillary glucose level, administer an IV benzodiazepine to terminate seizure activity, followed by a loading dose of IV phenytoin. If these measures fail to control seizure activity, the patient should be intubated (if not already) and IV phenobarbital, midazolam, or propofol administered
- Anticipate the need for early definitive airway management either due to ongoing SE or apnea secondary to drug therapies
- If no IV access is immediately available the best initial medication choice is Midazolam IM followed by interosseous line insertion
- Suspect pseudoseizures or NCSE in patients not responding to usual treatments

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20: THE AGITATED & VIOLENT PATIENT

Matthew Hodge

OBJECTIVES

1. To understand the “dual safety approach” ensuring safety of the patient and the ED staff
2. To identify common causes of agitation and violent behaviour in the ED patient and understand their management
3. To describe pharmacologic and non-pharmacologic restraints and their appropriate use

INTRODUCTION

Agitated patients in the ED pose a threat to both themselves and to those around them. In order to ensure the safety of both the patients and the ED staff, it is important that agitated patients be treated quickly, effectively, and safely.

Even in the busiest ED, the proportion of patients who present with agitation is low. The lack of agreement about the definition of the “agitated” or “violent” patient coupled with the reality that a person who arrives calm may later become violent or agitated means that certain key principles should be followed whether you see one or dozens of violent/agitated patients each week. These principles include safety measures, use of verbal intervention, use of non-pharmacological restraint and/or pharmacological restraint and identification of possible underlying causes for the agitated behaviour.

Morbidity from agitation and violence in the ED affects both patients and staff, particularly nursing staff. Surveys of ED staff typically report that a majority of those working in the ED have been struck or otherwise physically contacted by a patient. Even if infrequent, such occurrences can have significant adverse impact on working conditions and morale in the ED.

What is agitation?

Agitation describes a range of behaviours including: increased motor activity, (e.g. pacing), verbal outbursts, excessive talking, and irritability. Patients who arrive at the ED already agitated may be different from those who become agitated in the ED. Understanding the circumstances that led up to the agitation is critical to managing it effectively. For example, people who are brought to the ED under

arrest may be agitated simply because they are upset with the police, or they may be agitated because of an underlying medical condition, or recent use of drugs or alcohol in combination with being arrested. It is important to recognize and manage the agitated patient promptly both for the health of the patient and in order to prevent the situation from escalating to violence.

Safety is job one

Before an effective doctor-patient relationship can be established, safety is the first priority. Safety means thinking about two related but distinct concepts:

- the safety of ED staff and other patients – protecting yourself and others from the agitated or violent patient
- the safety of the patient – to the extent possible, protecting him/her from harming him/herself

An approach to safety management is described below. This must include an acute awareness of the surrounding environment and the potential risks involved.

PROTECTING YOURSELF

An important part of Emergency Medicine training is learning how to manage new and unpredictable situations. As a trainee in the ED, if you don’t feel safe, ask a more senior person to assist or accompany you, particularly when seeing a patient who has already been violent in the ED. Other key strategies to keep in mind:

- Avoid turning your back on the patient and stay close to the open door of the room.
- Once you are confident that the patient has no life-threatening ABC issues, use time to your advantage by waiting for the patient to de-escalate.
- When examining patients, avoid wearing any necklace or other jewelry, which can be grabbed or pulled by a patient. If you have a white lab coat, leave it outside the room and bring only essential equipment into the room

Verbal Communication

The first line of intervention when approaching the agitated patient should be an attempt at verbal communication. This will be most effective with mild to moderate agitation.

- introduce yourself to the patient
- use a quiet, reassuring, but firm tone
- provide information and explain expectations
- avoid threats or confrontation
- offering something to eat or drink can be helpful

Choosing & Using Restraints

When verbal communication is unsuccessful or when patients are significantly agitated or violent, restraints may be necessary. Restraints can be divided into non-pharmacologic and pharmacologic restraints. Patients may be physically restrained (non-pharmacologic) so that pharmacologic restraint can be administered.

Non-pharmacologic Restraints

Non-pharmacologic (or physical) restraints typically involve securing the patient to a fixed object, usually a bed or chair. Most hospitals have replaced older leather-with-metal-buckle restraints with soft four-point restraints to be attached to ankles and wrists. There is good evidence that two-point restraints are associated with a higher incidence of injury to the patient than four-point restraints, and should therefore be avoided. As soon as four-point restraints are applied, strong consideration should be given to pharmacologic restraints so as to minimize injury.

Persons in police custody may be restrained by some combination of handcuffs, zip ties, and leg shackles. When patients are brought to the ED restrained in this manner, police should remain with the patient. You may need to ask them to unlock the equipment in order to complete your examination. Explaining to the police and the patient what needs to be done can facilitate the exam and improve compliance.

Older patients may be restrained using a ‘posey jacket’, which typically confines them to the bed. Although used most commonly to manage wandering patients, some EDs will use these on elderly patients.

The basic principles for any use of non-pharmacologic restraints in the ED include:

- goal should be as little restraint as possible for as short a time as possible
- document when restraints are applied
- review assessments of restrained patients regularly
- when/if family and friends are present, explain why restraints are needed and what conditions will enable their removal

Pharmacologic Restraints

Pharmacologic restraint is often preferable since it carries a lower risk of physical injury to the patient

and often facilitates physical examination of the patient and the general functioning of the ED.

All agitated patients should be offered oral medication. If they are unwilling or unable to cooperate, intramuscular or intravenous injection, often following physical restraint, is typically required.

The basic principles for any use of pharmacologic restraints in the ED include:

- Offer oral medication as a first step to all patients with whom you are able to communicate. Be aware of your environment if you are offering the patient tablets so that you are not putting yourself at undue risk.
- Establish a safe environment, including non-pharmacologic restraints if necessary, before administering intramuscular (IM) medication.
- Be mindful of the rare but serious complication of neuroleptic malignant syndrome (NMS) by ensuring vital signs are regularly monitored and recorded by nursing staff and that you are informed of any sign of fever or change in pulse/blood pressure.

The most commonly used pharmacologic agents are benzodiazepines and neuroleptics. The route of administration will depend on the particular situation. If a patient is unable or unwilling to take oral medication, the intravenous route (IV) is preferred because of better absorption and faster onset of action of the drug. Often, an IV is not available and the medication must be given IM.

The particular agents chosen to treat an agitated/violent patient will depend on the patient's past medical history, the circumstances of this ED visit and possible substances already ingested.

Benzodiazepines produce rapid and effective sedation. They are particularly effective in cases of agitation secondary to substance abuse or withdrawal. The most commonly used agents are diazepam, lorazepam, and midazolam. The most common side effects are respiratory depression and confusion.

Haloperidol is the most common neuroleptic drug used in the ED. The optimal strategy is to combine a benzodiazepine with a neuroleptic when managing agitated patients. This is more effective than administering each drug separately and will help to decrease the incidence of extrapyramidal symptoms associated with neuroleptics.

CAUSES OF AGITATION

Agitation in the ED can be classified into 3 broad causes:

1. Medical conditions (e.g. sepsis in an elderly person from a long-term care facility, hypoglycemia, hypertensive encephalopathy, pain from abdominal trauma or an acute abdomen).
2. Substance use (intoxication or withdrawal).
3. Primary psychiatric conditions (typically primary psychotic disorders but many Axis I disorders may have psychotic features and some persons with Axis II conditions may become agitated in response to crises of various types).

Identifying the cause of agitation is important as a guide to treatment and prevention of future episodes. Even if you are unable to examine an agitated patient, it is possible to make observations that can guide your decision-making.

- Age & gender: elderly persons are far more likely to be agitated secondary to delirium arising from an acute medical condition ('organic cause') than they are to be substance users or to be agitated due to a primary psychiatric condition; a person who has been in the ED and becomes agitated may have had progression of the acute medical condition that prompted their ED visit.
- Substance use: ED patients who are substance users may be accompanied by police or companions who can confirm details of their substance use - both name(s) of actual substance(s) used and timing of ingestion are important. An important caveat is that substance users can develop agitation due to medical conditions.
- With the exception of first-episode psychosis, many persons with primary psychotic disorders will be well known to ED staff from previous visits. Review the patient's previous visits (if available), and ask nursing and other ED staff if they recognize the patient. Again, be sure not to forget that people with schizophrenia can also develop acute medical conditions.

PHYSICAL EXAMINATION

The initial examination of the agitated patient is the observation that they are agitated. Remember – safety first. Once they are less agitated or sedated, you may complete your physical examination.

It is also important to recognize signs of imminent violent behaviour such as higher voice tone, increased foul language, and increased muscular tension or uneasiness.

Physical examination should focus on vital signs, signs suggestive of medical conditions, and for the unconscious patient, respectful observation of limbs and torso for evidence of injury. Respectful in this case means that if the patient is to be undressed, provide privacy and if at all possible, have female ED staff remove clothing from and gown female patients. Be sure to document which staff were present when the patient's clothes and belongings were removed/changed.

INVESTIGATIONS

Investigations should be guided by your sense of likely causes of agitation in each particular patient. For all patients in whom you suspect a medical condition as the cause of their agitation, make a point of gathering corroborating information from family or friends or staff (for people from institutions such as long term care facilities). One of the most common causes of agitation among the elderly in the ED setting is delirium, which by definition is an acute change in mental state. While delirium can occur in demented people, one of the most common errors is to assume that the agitated elderly person is agitated because of dementia. A person who was living alone, volunteering at a community centre and doing his own banking 72 hours ago has not suddenly become demented as an explanation for agitation!

Laboratory investigations should be guided by your differential diagnosis in light of the person's demographics, medical history and clinical presentation. When a patient states that they have been assaulted, and particularly if the police are involved, it is often the practice to order radiographs even if clinical judgment suggests that no bony injury will be found.

Drug screens are rarely useful as a guide to management since the management of most intoxication is supportive. If you order a drug screen, be sure to inform the patient of the results before or at discharge, since it will be part of the medical record. Depending upon the circumstances and applicable laws, results may also be subject to subpoena by law enforcement authorities.

DISPOSITION

The disposition of the violent or agitated patient is determined largely by the diagnosis identified through your physical examination and investigations. Patients admitted to hospital with delirium or psychiatric illness may linger in the ED awaiting a bed, and for these patients, simply asking the admitting service to write orders for the care of the patient will provide clear direction to nursing and other staff while the patient is in the ED. When patients are discharged in the care of police, whether under arrest or not, document the badge numbers of the police officers and the time of discharge on the patient chart. Patients whose agitation is due to substance use can be provided information about treatment and counseling resources and should not be discharged if still obviously impaired.

SUMMARY

- Safety first – always consider the safety of ED staff, other patients, and the safety of the patient
- The causes of agitation can be classified into that due to a medical condition, substance use or primary psychiatric condition. Remember that a person known to be in one category may also have another cause of agitation
- Restraints, non-pharmacologic and pharmacologic, should be used sparingly but

effectively. Monitor all persons who have been restrained and document regular reassessments.

CLINICAL PEARL

Two-point restraints are associated with a higher incidence of injury to the patient than four-point restraints, and should therefore be avoided.

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Table 1. Management of the Violent/Agitated Patient in the Emergency Department

	Protecting Staff & Other Patients	Protecting the Patient from him/herself
Environment	<ul style="list-style-type: none"> -place agitated patient in a room with no other patients -if possible, use a room which can be secured to reduce noise transmission to the rest of the ED -bar all visitors until the situation has calmed and their identity can be confirmed; in situations where domestic disputes may have occurred, secure the patient's consent for any visitor 	<ul style="list-style-type: none"> -ensure that room is free of potential weapons or equipment which can be thrown or pushed (furniture should be bolted to floor, use a room with a door rather than one with a curtain) -remove potential weapons (e.g. pocket knives) from the patient
Agents	<ul style="list-style-type: none"> -consider restraints as a last resort rather than a first line agent -if non-pharmacologic restraints are to be applied, ensure that adequate numbers of people are present, that the equipment is in place and that there is a clear plan for restraining the patient -if non-pharmacologic agents are to be applied, offer oral medication if possible but beware of spitting risk; as with physical restraints, ensure that the equipment and personnel are in place 	<ul style="list-style-type: none"> -all persons in physical restraints should be monitored regularly (different hospitals have different policies); document when the restraints were applied and by whom -if pharmacologic restraints have been used and the patient is unconscious (with or without physical restraints) ensure that the patient is positioned to reduce risks of choking if aspiration occurs (e.g. not lying flat on his/her back)
Organization & Leadership	<ul style="list-style-type: none"> -someone needs to be in charge, ideally an experienced physician or nurse -when patients are brought to the ED by police, ensure that police, hospital security, and ED staff are clear about who does what; police often accompany agitated and violent people to the ED but will not be arresting them; reduce the number of security personnel involved to the minimum needed to ensure safety 	<ul style="list-style-type: none"> -if the agitated patient appears capable of communication, use a level, measured voice; make a conscious effort not to scream or yell if you are being screamed or yelled at -work to find common ground; offer food or water if the patient indicates an interest -once the patient is in a safe space, consider leaving and returning after 5-10 minutes as a way to de-escalate a situation

Table 2. Medications for Pharmacological Restraints

CLASS	NAME	DOSE
Benzodiazepines	lorazepam (Ativan)	0.5 – 2.0 mg -can be given sublingually if patient willing & able to cooperate -can be mixed with haloperidol for intramuscular injection -preferred agent from this class due to longer half life (means less frequent dosing needed)
	diazepam (Valium)	5-10 mg every 3-4 hours -alternative to lorazepam -may have faster onset
Typical antipsychotics	haloperidol (Haldol)	2-10 mg -given intramuscularly, often with lorazepam in same syringe
Atypical antipsychotics	olanzapine (Zyprexa)	2.5 – 15 mg -favoured as oral alternative to haloperidol in cooperative patients -can also be given as intramuscular injection and as a sublingual preparation (trade name: zydis)
	Loxapine	25mg, IM -given intramuscularly, onset of sedative effect occurs in 15 to 30 min with peak effect occurring within 1 – 3 hrs, the duration of sedative effect is approximately 12 hrs.

21: Diabetic Emergencies

David Carr

OBJECTIVES

1. To recognize the common diabetic emergencies
2. To understand the pathophysiology of diabetic ketoacidosis and hyperosmolar hyperglycemic state so as to guide management
3. To realize the importance of appropriate electrolyte correction in the management of diabetic emergencies
4. To identify and treat precipitants of diabetic emergencies
5. To make appropriate decisions about disposition of patients with diabetic emergencies

INTRODUCTION

Not a day goes by in the Emergency Department without the treatment of patients with complications related to diabetes. Diabetes and its various complications represent an enormous burden on the population and the health care system. Diabetes affects approximately 2 million Canadians. Aboriginal Canadians are three to five times more likely to develop type 2 diabetes compared to non-Aboriginal Canadians. With an aging population, there is no doubt that the number of patients affected with diabetes will continue to rise and our encounters in the ED will become even more frequent.

One challenge facing ER physicians is staying current with respect to evolving treatments of diabetic complications, new medications, and methods of prevention. This chapter will focus on three important direct complications of diabetes and diabetic care: diabetic ketoacidosis (DKA), hyperosmolar hyperglycemic state (HHS), and hypoglycemia.

DIABETIC KETOACIDOSIS (DKA)

DKA is characterized by the triad of hyperglycemia, ketosis, and acidosis, but there is no clear-cut definition of DKA. One of the more widely accepted criteria of DKA is: Serum Glucose > 16, pH < 7.30, and Bicarbonate < 18.

Epidemiology

DKA is seen primarily in patients with type 1 diabetes. The incidence of DKA is between 4.6 and 8 per 1000 person-years among diabetics. DKA accounts for the initial presentation of diabetes in approximately 25% of patients, and the risk of DKA in children is approximately 1-10% per year. Since the advent of insulin preparations in 1921, the mortality rate of DKA has gone from 100% to approximately 2-8%; however, it remains as the most common cause of diabetic related deaths in children.

Pathophysiology

The pathophysiology of DKA comprises two main concepts:

1. Relative insulin deficiency
2. Counter-regulatory hormone excess

Normally, as plasma glucose increases after a meal, the pancreatic beta cells secrete insulin to counter this rise in glucose. Insulin helps restore the glucose to euglycemic levels by inhibiting gluconeogenesis and glycogenolysis, and by allowing for the uptake of glucose by skeletal and adipose tissues.

In DKA, a precipitating stress causes these homeostatic mechanisms to fail. The most common events that trigger DKA are usually infections or a lack of exogenous insulin. These precipitating diagnoses should be actively sought after in the patient presenting with DKA.

As a result of these stressful precipitants, the body responds by producing counter-regulatory hormones. These hormones include glucagon, catecholamines, cortisol, and growth hormone. The counter regulatory hormones in the face of relative insulin deficiency, lead to an increase in glucose production and an underutilization of glucose metabolism (mainly via glucagon's effect of increasing gluconeogenesis and glycogenolysis). As blood glucose rises, the renal glucose threshold is reached and glucose is excreted into the urine, leading to an osmotic diuresis. This diuresis in turn, leads to losses of glucose, electrolytes, and water, potentially causing dehydration, poor tissue perfusion and lactic acid production.

CAUSES OF DKA

1. Lack of insulin (initial presentation of diabetes, non compliance, under dosing)
2. Infection
3. Myocardial infarction
4. Pulmonary embolism
5. Stroke
6. Pancreatitis
7. Substance abuse
8. Medications (steroids)
9. Trauma
10. Surgery

Additionally, the relative insulin deficiency and counter regulatory excess cause an imbalance leading to an increased state of lipolysis. This leads to the oxidation of free fatty acids and aids in the generation of ketone bodies such as beta hydroxybutyrate, acetoacetate, and acetone. Beta hydroxybutyrate is the predominant ketone produced in DKA (this is relevant as urine dipsticks only measure acetoacetate levels). If these ketones are unused (secondary to the insulin deficiency and glucagon excess), there is an accumulation that leads to a metabolic ketoacidosis.

Clinical Presentation

Typically, patients with DKA will initially complain of polyuria and polydypsia. As the ketosis ensues, they develop nausea, vomiting, and abdominal pain, which further affects their ability to restore their volume losses. The ketosis also contributes to the ‘fruity’ odor on their breaths, which is described but seldom observed. As their serum osmolality (generally above >320) increases, patients can become confused. On physical examination, patients will show some classical signs of volume contraction such as tachycardia, hypotension, poor skin turgor, flat JVP, poor capillary refill and confusion. To compensate for their metabolic acidosis, patients will often be tachypneic and this will often serve as the first clue of their acid-base derangement.

The work up for these patients is variable, depending on the suspected precipitating cause for the DKA. In general, all patients should receive a CBC, electrolytes, creatinine, glucose, serum and urine ketones, serum osmolality, liver function tests, amylase, calcium, magnesium, phosphate, troponin and an EKG and a blood gas. Although it is common to draw arterial blood gases on all suspected DKA patients, venous blood gases are an appropriate alternative. Studies have shown that VBG pH levels

are approximately 0.03 units lower than ABG pH levels, a difference that is not clinically significant. Some physicians still recommend an initial ABG, but all serial blood gases should be drawn from a venous sample. If the clinical situation warrants further tests, one might also consider blood/urine/stool cultures or a chest X-ray.

Treatment

The treatment of DKA is based on restoring the patient’s intravascular volume, correcting their electrolyte imbalance, and replenishing their insulin stores.

Fluid replacement is paramount to the success of the treatment of DKA. Volume restoration essentially turns off the counter regulatory hormone excess, helping to restore euglycemia and correct dehydration. The degree of dehydration varies from patient to patient. Total body water loss has been estimated to be approximately 5-8L or ~100ml/kg in a typical adult male with DKA. Generally speaking, half of the fluid deficit should be replaced in the first 8 hours while the remainder is infused over the next 16 hours. Initially the resuscitation is carried out with isotonic normal saline, administering 1-2L in the first hour. If the serum sodium is high or normal after the initial boluses, the replacement should proceed with 0.45NS at 4-14 ml/kg/hr (depending on the patient’s hydration status). If the patient’s sodium is low, the infusion should be at the same rate, but with normal saline instead. Of note, when the serum glucose falls to approximately 16 mmol/L, the intravenous fluid should be changed to a dextrose containing solution (i.e. D5/0.45 NS).

The administration of insulin will improve hyperglycemia, ketosis and acidosis. However, because of its effect on lowering serum potassium (and potentially blood pressure in dehydrated patients), insulin should not be given until potassium levels are greater than 3.3mmol/L and the BP is adequately restored with fluids. Insulin should be given intravenously as a short acting insulin R drip at 0.1 U/kg/hr. Some experts also recommend an initial bolus of 0.10-0.15 U/kg of insulin R for adults, in addition to the infusion. The specific goal of insulin therapy is to lower the blood glucose by approximately 3-5 mmol/L per hour. Therefore, blood glucose monitoring needs to be repeated often (i.e. every 30-60 minutes). Once the patient’s blood glucose falls below 14mmol/L, the patient should receive their regular subcutaneous injection of insulin. The insulin infusion should be stopped in the following one hour in order to have an overlap of insulin action to prevent a rebound hyperglycemia.

Variations in serum potassium are the most important electrolyte disturbance in DKA. Because of urinary potassium (K) losses, these patients are depleted in their overall K stores. However, in the acidotic environment there are shifts that lead to increased K in the ECF. A patient with DKA may present with a normal serum K value, but the true value of his/her K is actually much lower. Since the administration of insulin will further shift K into the cell, it is very important to replace the K deficits prior to and during insulin administration. Before replacing the patient's K, it is also critical to ensure adequate urine output in order to prevent the potential of precipitating hyperkalemia in the setting of acute renal failure. If the K is between 3.3-5.0mmol/L, administer 20-30 mmol/L of KCl with each liter of fluid. If the K is > 5.0, no K is given and it should be rechecked in 2 hours. If the K is <3.3, hold the insulin and give 40 mmol/L KCL in the 1st hr and then 20-30 mmol/L in an effort to keep the K between 4-5.

The role of bicarbonate therapy in DKA is often questioned. Generally speaking, unless the patient's pH is <6.9, there is no role for bicarbonate therapy. Insulin therapy can also lead to abnormally low serum phosphate and magnesium levels. Although rarely needed, if the patient has hypophosphatemia and is symptomatic, one might consider using a potassium solution containing phosphate when replacing the patient's potassium. Patients with DKA also have sodium losses that are restored with the administration of IV fluids. However, it is very important to be aware of the 'pseudohyponatremia' that exists in patients with hyperglycemia. For every increase in serum glucose by 10 mmol/L (above normal ~ 6 mmol/L), the true serum sodium is actually about 3 mmol/L higher than the measured value. Knowledge and application of this fact can prevent unnecessary concern and inappropriate treatment with apparent low serum sodium concentrations.

CLINICAL PEARLS

The 'Big 3' Aspects of DKA Treatment:

1. Fluid resuscitation
2. Glucose control with Insulin
3. Correction of potassium

Complications

The majority of complications in DKA are related to the fluid and electrolyte replacements administered to these patients. These include hypoglycemia, recurrent hyperglycemia, hyperchloremia, and hypokalemia (which can lead to arrhythmias). Less common complications include ARDS, fluid overload and

thromboembolic phenomenon such as DVT or PE. The most dreaded complication is cerebral edema, which is very rare but often discussed. This complication is seen much more commonly in pediatric DKA patients, with 95% of cases occurring in patients less than 20 years old. It is generally caused by overzealous administration of IV fluids. It is for this reason that the treatment of pediatric DKA is mildly different than that of adult DKA. The maximum estimate of the fluid deficit in children is 100 ml/kg corresponding to ~ 10% dehydration. We replace those fluid deficits over 48 hours, not 24 hours. In addition most experts recommend omitting the initial insulin bolus in children to prevent fluid shifts.

HYPEROSMOLAR HYPERGLYCEMIC STATE (HHS)

Over the years, HHS has been referred to by many names and acronyms. However, it appears that the term HHS has caught on most recently, as it omits the condition of a coma that appeared in previous descriptions. HHS is seen predominantly in type 2 diabetics and is generally defined by the presence of: serum glucose > 33.3 mmol/L, pH>7.3, bicarbonate >15, anion gap <12, serum osmolality >320mOsm/kg, and the absence of serum ketones. The key distinguishing feature between HHS and DKA is the absence of serum ketones.

Pathophysiology

The pathophysiology of HHS, is very similar to that of DKA. However, the presence of a relatively small amount of endogenous insulin in HHS seems to be a key factor in differentiating it from DKA.

Clinical Presentation

HHS typically evolves over several days to weeks, unlike DKA, which usually begins within a day. Compared to patients with DKA, HHS patients tend to be older, suffer from larger fluid losses, and have more co-morbid illnesses. The mortality has been estimated to approach 15% (nearly 3 times that of DKA). They will usually have less GI symptoms and more recognizable neurological deficits than those patients with DKA. In dealing with these types of patients, it is also important to consider other causes of hyperglycemia, ketosis, and acidosis. In your differential diagnosis, you might also want to consider the possibility of starvation ketosis, alcohol ketosis, DKA, lactic acidosis, and toxic ingestions (i.e. salicylate poisoning, toxic alcohols).

Treatment

The treatment goals of HHS start with correcting the volume and electrolyte losses, and identifying the precipitating causes. Treatment is almost identical to that of DKA. One difference is that there is no role for the use of bicarbonate in HSS. This is intuitive as HHS patients are not likely to be as severely acidotic as their DKA counterparts.

Complications

Patients with HHS usually suffer from multiple comorbidities and therefore are more susceptible to various vascular and infectious complications. Similar to their DKA cohorts, most of the complications are iatrogenically related to their fluid and electrolyte replacements. Cerebral edema, ARDS, and coagulopathies remain amongst the most devastating complications.

HYPOLYCEMIA

Hypoglycemia is a frequent complication of diabetes, especially in patients requiring insulin. Clinically, hypoglycemia has traditionally been characterized by Whipple's Triad:

1. Low plasma glucose
3. Symptoms suggestive of hypoglycemia
4. Prompt resolution of symptoms with administration of glucose or related product.

Pathophysiology

Normally, as blood glucose falls, insulin secretion is inhibited. In addition, there is an increase in counter regulatory hormones, mainly glucagon and epinephrine. These hormones stimulate glucose production via gluconeogenesis and glycogenolysis. In order for glucagon to have an effect, the patient must have glycogen stores to mobilize. Patients who are malnourished or alcoholic will likely not have adequate glycogen stores and therefore will not respond to glucagon.

Causes

Most cases of hypoglycemia seen in the ED can be attributed to excessive insulin use in the setting of inadequate oral intake or increased metabolic demands. However, you may also encounter hypoglycemia in the setting of alcohol intoxication, sepsis, liver disease, or the use of oral anti-hyperglycemics (i.e. sulfonylureas). Rarely, patients will present to the ED with hypoglycemia secondary to insulinomas, hypopituitarism, adrenal insufficiency, or medication side effects. The key is to suspect the individual cause and treat appropriately.

Symptoms

Symptoms of hypoglycemia can generally be classified as either autonomic or neuroglycopenic. Examples of the latter include headaches, confusion, seizures, and coma. Autonomic or adrenergic symptoms include diaphoresis, nausea, hunger, tachycardia and palpitations. Patients with long standing diabetes occasionally develop a resistance to the signs or symptoms of hypoglycemia with time. This phenomenon is referred to as 'hypoglycemic unawareness'. Another important consideration is that patients on beta-blockers will have their adrenergic hypoglycemic symptoms blunted in the setting of hypoglycemia.

Treatment

In the pre-hospital setting, hypoglycemia is often treated by the patients themselves with oral glucose-containing fluids or gels. In the ED, the preferred treatment for adults is IV D50 (dextrose 50%) at 1g per kg, typically starting with 1 ampoule (50g). If the patient does not have an IV, the administration of glucagon 1-2mg sc would be a second line treatment. The downside of glucagon is it takes approximately 10 minutes to work and as mentioned previously, will not be effective in patients without adequate glycogen stores.

In the pediatric population, the treatment of hypoglycemia varies by age (neonates D10 IV at 5-10ml/kg, infants and children D25 IV at 2-4 ml/kg, adolescents D50 IV at 1-2 ml/kg). It is important to repeat accucheks every 30 minutes for 2 hours to watch for rebound hypoglycemia. In some situations, a continuous infusion of a dextrose containing fluid will be required.

Disposition

It is important to determine the cause of the hypoglycemia prior to establishing the disposition plan. If hypoglycemia is seen in the setting of a non-diabetic, a thorough workup might need to be pursued. Diabetic patients treated for hypoglycemia who have a quick resolution of symptoms can usually be safely discharged after observation for a few hours. Good diabetic education, assessment /adjustment of their insulin regimen, and adequate follow up with their family doctor or endocrinologist should be arranged prior to discharge. Patients who become hypoglycemic while taking long acting oral hypoglycemics will need a longer observation period.

SUMMARY

- DKA is a potentially life threatening complication of diabetes. It requires a thorough understanding of physiology in order to provide effective and safe treatment.
- HHS is similar to DKA except that it typically evolves over several days to weeks, there is an absence of serum ketones, and there is a relatively small amount of endogenous insulin. HHS patients tend to be older, suffer from larger fluid losses and other co-morbid illnesses. The mortality is higher with HHS, and there is no role for the use of bicarbonate.
- Patients who present with hypoglycemia require very specific educational discharge instructions.

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22: ENT Emergencies

Bryan Au

OBJECTIVES

1. To understand the causes, treatment and ongoing care of epistaxis
2. To recognize pharyngitis and understand its treatment options
3. To recognize epiglottitis and appropriately manage it
4. To differentiate between the various deep-space neck infections and appropriately manage them
5. To familiarize yourself with different methods of foreign body removal from the ears and nose

EPISTAXIS

Introduction

Anterior epistaxis accounts for 90% of all epistaxis. Bleeding most commonly is from Kiesselbach's plexus (Little's area) on the anterior-inferior nasal septum. Posterior epistaxis, which is more severe, usually originates from the posterior branch of the sphenopalatine artery. The diagnosis of posterior epistaxis is suspected when bleeding is not controlled with anterior packing. Common etiologies of epistaxis include nasal or facial trauma, digital trauma, low humidity conditions, atherosclerosis, coagulopathy, foreign body, and idiopathic. A careful history will reveal any contributing conditions such as bleeding disorders.

Treatment

The ABCs, of course, are paramount. Patients are at risk for aspiration, and should also have a hemodynamic/volume assessment. Blood work (CBC, INR/PTT, cross & type) and hemodynamic resuscitation should be done in more severe cases.

Any clots should be cleared by blowing the nose or suctioning. The first intervention is to institute proper first aid measures – most people do not know how to stop a nosebleed! Compress the soft, cartilaginous part of the nose for 10-15 minutes (most people incorrectly apply pressure to the nasal bridge). If this fails twice, place an anterior pack. Apply topical anesthesia/vasoconstrictors such as cocaine or lidocaine with epinephrine on soaked pledgets. One may use the traditional layered Vaseline gauze pack, or a commercial nasal tampon or balloon. Once the bleeding has stopped, the patient may be discharged home with the pack in place with instructions to

follow-up in 48-72 hours for re-assessment and pack removal. Anti-staphylococcal antibiotics such as cloxacillin or cephalexin are usually prescribed to prevent sinusitis and toxic-shock syndrome. If the bleeding ceases with anterior pressure, then silver nitrate cauterity may be applied to the mucosal bleeding site if it can be identified.

If anterior packing does not control the epistaxis, or if posterior epistaxis is suspected, then obtain ENT consultation. Posterior epistaxis requires admission to hospital for posterior packing with monitoring, and possibly embolization by interventional radiology or arterial ligation.

PHARYNGITIS

Introduction

Pharyngitis is an inflammation/infection of the pharynx and tonsils. Viruses cause the majority of cases (e.g. rhinovirus, adenovirus). The most common bacterial cause is group A streptococcus (GAS). Risk factors for GAS include: winter or early spring, age 5-15 years, and recent exposure to someone with GAS pharyngitis. Strep pharyngitis may rarely result in acute rheumatic fever or post-streptococcal glomerulonephritis.

Signs and Symptoms

The patient will typically complain of odynophagia, dysphagia, and fever. Viral infections usually produce other symptoms such as cough, rhinorrhea, myalgia, headache, conjunctivitis, or rash. On physical exam, one may find pharyngeal erythema, tonsillar enlargement, pharyngeal or tonsillar exudate, or tender cervical lymphadenopathy. It is often difficult to clinically differentiate GAS from other viral or bacterial etiologies.

Diagnosis and Treatment

First, assess the ABC's. Although rare, there is a risk of airway obstruction, and patients are often volume-depleted from decreased oral intake. Patients also may require analgesia, antipyretics, and fluids. A major decision is whether or not to treat with antibiotics for presumed GAS pharyngitis. Antibiotics prevent acute rheumatic fever (but not post-strep glomerulonephritis), decrease the risk of transmission, and may reduce symptoms by approximately one day. Most clinicians use the

Centor criteria – the greater the number of criteria present, the greater the likelihood of GAS pharyngitis.

Centor Criteria for Diagnosis of Strep Pharyngitis

1. Tonsillar exudate
2. Anterior cervical adenopathy
3. History of fever >38 C
4. Absence of cough

Patients with 0 or 1 criteria do not require testing or antibiotic treatment. There are a number of different approaches for those patients with 2 or more criteria, depending on the availability of rapid strep antigen testing, reliability of patient follow-up, etc.

Treatment options include:

- obtain a throat culture, and treat only those with positive cultures
 - do a rapid strep antigen test, and treat if positive
 - treat patients with all 4 criteria, with no testing
- There is no one ‘correct’ approach. If antibiotics are prescribed, penicillin VK 300 mg po tid x 10 days is the drug of choice for strep pharyngitis.

There is also evidence that steroids can reduce the pain of severe exudative pharyngitis. Common regimens are a single dose of dexamethasone in the ED, or two days of prednisone.

EPIGLOTTITIS

Introduction

Epiglottitis, or supraglottitis, has now become an adult disease with the advent of the pediatric H. influenzae vaccine. It is a localized cellulitis of the supraglottic tissues. The predominant causative organism is H. influenzae type B, although staphylococcal and streptococcal species may also be involved. The danger in adults, as in children, is obstruction of the airway.

Signs and Symptoms

There is usually a prodrome of typical upper respiratory infection symptoms, followed by a severe sore throat. Severe odynophagia, dysphagia, and a muffled voice are common. There is often tenderness to palpation of the hyoid bone and the larynx. Vital signs may show a tachycardia out of proportion to the patient’s fever. When visualization of the posterior pharynx is relatively normal, but the patient appears to have a great deal of pharyngeal discomfort, or is toxic, suspect the diagnosis of epiglottitis.

If the patient is stable, then a soft-tissue lateral neck x-ray may be obtained. Findings include a thickened, enlarged epiglottis (‘thumbprint sign’), obliteration of the vallecula space, and ‘ballooning’ of the hypopharynx. Visualization of the supraglottic region is the definitive diagnostic test, however. This may be done with a mirror exam, fibreoptic laryngoscopy, or direct laryngoscopy.

Treatment

Patients with suspected epiglottitis must be managed with great vigilance, as airway obstruction may be imminent. You should involve consultants such as ENT, ICU, and anesthesia early. Intubation is often difficult, and should be done in a controlled setting if at all possible. Antibiotics, such as ceftriaxone 1 g IV, should be started in the ED. Some experts recommend IV steroids or nebulized epinephrine to help reduce inflammation/edema and prevent airway obstruction. Disposition is typically to an ICU setting.

DEEP-SPACE NECK INFECTIONS

PERITONSILLAR ABSCESS

Introduction

Peritonsillar abscess (PTA) is the most common deep space infection of the head and neck in adults. It occurs most commonly in older adolescents and young adults. It is usually caused by the extension of a tonsillitis. Most are polymicrobial in origin, with a mixture of aerobes and anaerobes.

Signs and Symptoms

Patients are often febrile, and their vital signs and physical examination may show signs of dehydration from decreased oral intake. Patients will complain of severe odynophagia and dysphagia, often with drooling and trismus. The voice is often a muffled ‘hot potato’ voice. The involved tonsil is enlarged, erythematous, often with an exudate. The tonsillar enlargement may push the uvula to the contralateral side. Examination of the neck reveals tender anterior cervical adenopathy.

Treatment

As always, ensure that the airway is secure. Supportive therapy should be instituted as necessary – fluids, analgesia, and antipyretics. Antibiotics should be given. Clindamycin 600 mg IV is a good option. Drainage of the abscess under local anesthesia may be done by the ENT consultant or the emergency physician, if he/she is comfortable with the procedure. Described techniques include an open

incision and drainage or a simple needle aspiration. It is important to keep in mind the proximity of the carotid artery to the tonsil when performing drainage.

RETROPHARYNGEAL ABSCESS

Introduction

The majority of retropharyngeal abscesses occur in children. Under the age of 4, there are lymph nodes in the retropharyngeal space. These may become infected secondary to respiratory tract or pharyngeal infections, leading to an abscess. After the age of 4 to 6, these lymph nodes atrophy. The pathophysiology of retropharyngeal abscesses in adults is different. They may be caused by extension of a pharyngeal cellulitis, pharyngeal trauma (e.g. fish bones), instrumentation, hematogenous spread, or even local spread from a cervical vertebral infection. Retropharyngeal abscesses are typically polymicrobial in origin, with a mixture of aerobes and anaerobes.

Signs and Symptoms

In addition to the typical odynophagia, dysphagia and fever, patients will often complain of neck pain and stiffness. The symptoms and signs may mimic meningitis in a young child. Children have mistakenly undergone a lumbar puncture because they were febrile, looked toxic, and had a stiff neck. Examination of the posterior pharynx may reveal erythema or diffuse edema.

If the patient is stable, a portable lateral neck x-ray may be performed to assess for swelling of the retropharyngeal/pre-vertebral tissues. If the pre-vertebral space exceeds 7 mm at the C2 level in adults (or $\frac{1}{2}$ the width of the vertebral body in children), or 21 mm at the C7 level in adults (or the full width of the vertebral body in children), this is suspicious for a retropharyngeal abscess. One may also see gas in the retropharyngeal space. CT scan is more definitive but should only be performed in the stable patient, often in consultation with ENT.

Treatment

Like supraglottitis, the airway is of primary concern. ENT consultation should be obtained and IV antibiotics should be started in the ED. High-dose clindamycin is a good choice.

LUDWIG'S ANGINA

Introduction

Ludwig's angina is a serious infection of the floor of the mouth and submandibular space. Dental infection is the most common etiology. It is usually caused by a

mixture of aerobes and anaerobes of the mouth, such as *Staphylococci*, *Streptococci*, and *Bacteroides* species. The most common cause of death is asphyxiation due to airway obstruction.

Signs and Symptoms

Patients may complain of dysphagia, sore throat, neck pain and swelling, and fever. Physical examination usually reveals tender submandibular swelling and erythema. Late signs of impending airway obstruction include dysphonia, drooling, and elevation or posterior displacement of the tongue. Once the airway is secured, CT scan may be obtained to delineate the extent of the infection.

Treatment

Similar to other deep space neck infections, the main concern is with the potential for airway obstruction. Intubation is usually necessary, and can be extremely difficult. Consultations should be obtained from ENT, anesthesia, and critical care. High dose broad-spectrum IV antibiotics should be administered, such as clindamycin, cefoxitin, or piperacillin-tazobactam.

FOREIGN BODIES OF THE EAR & NOSE

Introduction

Foreign bodies of the nose are common amongst pediatric patients. Commonly inserted objects include beads, pebbles, toys, and other small round objects. Foreign bodies in the ear can be seen in both adult and pediatric patients. Common objects include cotton swab tips, insects, as well as small round objects.

Signs and Symptoms

Usually the presentation is straightforward: the patient will have a history of foreign body in the nose or the ear. Nasal foreign bodies are occasionally unnoticed by the parent until some time has elapsed. The most common symptom that brings the patient to medical attention in a delayed fashion is a unilateral foul-smelling nasal discharge. Insects in the ear, which are still alive, can cause significant distress to the patient. The insect should be killed with lidocaine or mineral oil prior to removal.

Diagnosis and Treatment

There are many described techniques for the removal of foreign bodies from the nose and ears. These include alligator forceps, suction catheters, balloon-tipped catheters, and positive pressure applied to a child's mouth in order to expel a nasal foreign body. Gentle irrigation may be successful in removing some ear foreign bodies, but this method is contraindicated

if tympanic membrane perforation is suspected, and for foreign bodies which may swell, such as vegetable matter. Some principles apply to all removal techniques, however. It is vital to obtain maximum patient co-operation. You want your first attempt at foreign body removal to be the best attempt.

Generally speaking, ear foreign bodies are more challenging, as the external auditory canal is extremely sensitive, and most patients (especially young ones) will be less than co-operative. Strongly consider removing ear foreign bodies in children under procedural sedation. It is also vital to obtain adequate illumination when removing a foreign body under direct visualization. After the successful removal of one foreign body, search for others in other similar orifices. Finally, it is important to warn patients and parents beforehand that removal attempts may cause some self-limited bleeding of the ear canal, or epistaxis, so that they are not overly alarmed. After an unsuccessful attempt at removal, it is prudent to refer to an ENT specialist.

SUMMARY

- 90% of epistaxis is anterior, and most can be controlled with simple manual pressure
- posterior epistaxis usually requires ENT involvement and admission to hospital
- most cases of pharyngitis do not require antibiotics; the Centor criteria can help clinically

determine which cases are more likely to be due to Group A strep

- consider epiglottitis when the patient has a severe sore throat or looks toxic, and the pharyngeal exam is unimpressive
- patients with deep space neck abscesses or epiglottitis may suddenly obstruct their airway; monitor them closely
- a lateral neck x-ray in the stable patient may help in the diagnosis of epiglottitis or retropharyngeal abscess
- epiglottitis is now an adult disease; peritonsillar abscess occurs in adolescents and young adults; retropharyngeal abscess usually occurs in young children and babies
- with foreign body removal, make your first attempt your best attempt

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23: EYE EMERGENCIES

George Porfiris

OBJECTIVES

1. To develop an anatomical approach to traumatic eye injuries
2. To categorize visual loss into painful and painless
3. To understand the classic presentations of common traumatic eye injuries
4. To understand the classic presentations of the causes of visual loss
5. To know which patients need urgent referral to an ophthalmologist

INTRODUCTION

Eye problems are commonly seen in the ED. The role of the emergency physician is to make an accurate diagnosis, start the required treatment, and arrange ophthalmological referral if required. A visual acuity must be documented for every patient presenting with an eye or vision complaint; consider it ‘the sixth vital sign’. An approach to two common complaints will be addressed: trauma to the eye and sudden visual loss.

TRAUMA TO THE EYE

An organized, anatomical approach is helpful when approaching a patient who has suffered trauma to the eye (like the commonly seen punch to the eye). Orbital fractures, eyelid lacerations, corneal injuries, hyphemas, globe rupture, lens dislocation, retinal detachment and retrobulbar hemorrhage should all be ruled out before the patient with the traumatized eye leaves the emergency department. These will be discussed in turn.

Starting from the outside and working in, the first significant injury to assess for is an **orbital fracture**. The infraorbital bone is the one most commonly fractured. Physical examination clues would include tenderness and bruising of the infraorbital area, subcutaneous crepitus (air from the maxillary sinus), and loss of sensation under the affected eye (disruption of infraorbital nerve). Diplopia on upward gaze or inability to move the affected eye implies trapping of the inferior oblique or inferior rectus muscles and warrants an urgent referral. The diagnosis is usually made with plain X-rays (facial views) and confirmed with CT.

Most **lacerations** around the eye can be repaired by the emergency physician. There are however certain injuries that should be referred on to an ophthalmologist. These injuries include lacerations through the tarsal plate or the lacrimal apparatus and lacerations that involve the lid margin.

Working our way into the eye, **corneal abrasions** are common and can be detected with fluorescein staining and a cobalt blue filter. Symptoms include a painful red eye, foreign body sensation and mild photophobia. Treatment involves topical antibiotics (such as polymyxin B/trimethoprim drops or fusithalamic ointment) and pain relief with either topical NSAID drops (like diclofenac or ketorolac) or oral narcotics. If the patient wears contact lenses or if the abrasion was the result of contact with vegetal matter (tree branch) an antipseudomonal antibiotic such as ciprofloxacin should be used. Contact lenses should not be worn until the abrasion is healed. Topical anaesthetic drops like tetracaine should not be prescribed, as they are associated with delayed wound healing. There is no need to patch the eye since multiple randomized trials have shown that patching does not affect healing times or improve patient comfort. Follow-up within 48 hours should be arranged to ensure complete resolution.

The next anatomical structure to consider is the anterior chamber. An injury that causes bleeding into the anterior chamber is called a **hyphema**. The blood will usually layer out inferiorly if the patient is upright. The initial treatment involves protecting the eye with an eye shield and referral to an ophthalmologist. They will be followed on a daily basis until resolution to detect complications such as secondary glaucoma, corneal staining, or secondary rebleeding. Admission to hospital may be required with a hyphema that is greater than 50%. Treatment with antifibrinolytic agents such as tranexamic acid should be considered by the ophthalmologist. The patient should sleep with the head of the bed elevated to an angle of 30 degrees.

Globe rupture is the most feared injury following trauma to the eye. Most are clinically obvious presenting with scleral rupture, extrusion of the lens and intra-ocular contents, and loss of vision. There are, however, some cases that are more subtle that must be diagnosed to prevent total loss of the affected eye. Three clues are a flat or shallow anterior chamber, uveal prolapse with a slit like iris, and a positive Seidel test. The Seidel test is also known as

the fluorescein waterfall test because the fluorescein is washed away like a waterfall by the leaking aqueous humor. Treatment is surgical and involves rapid exploration and repair by an ophthalmologist in the operating room. While waiting for surgery, an eye shield should be put on to protect the eye from further injury, tetanus status should be updated, and a dose of prophylactic intravenous antibiotics should be given.

A lens dislocation may occur without globe perforation. The patient usually presents with decreased vision from the affected eye as well as ocular pain. On inspection of the eye the diagnosis is usually obvious as the lens can usually be seen in an abnormal orientation (usually edge-on). This needs to be surgically corrected.

A post traumatic glaucoma can occur following blunt trauma to the eye. The cardinal symptoms are intense eye pain or headache, nausea and vomiting, mild photophobia, and decreased vision. Signs include a fixed mid-dilated pupil, injected conjunctiva, a cloudy cornea, decreased visual acuity, and an increase in intraocular pressure (usually >40). Treatment includes topical pilocarpine drops to shrink the pupil, topical timolol drops to decrease the production of aqueous humour, and intravenous acetazolamide and mannitol to decrease the intraocular pressure. This is done while waiting for the definitive surgical treatment: laser iridotomy.

A post traumatic iritis can also develop and usually occurs between 12-48 hours post injury. The cardinal symptoms are intense photophobia, ocular pain, and decreased vision. Signs include a small pupil (can be irregular if posterior synechiae have developed), decreased visual acuity, and a ciliary flush (redness concentrated around the limbus). Using a slit lamp, one would appreciate a ‘cells and flare’ phenomenon which is described as what you see when you drive with your lights on, in fog. One additional finding is that the patient experiences pain when you shine light in the unaffected eye (due to the simultaneous constriction of both eyes). Treatment involves cycloplegic and vasodilator drops such as homatropine, which prevents ciliary spasm and avoids the development of posterior synechiae. Topical steroids such as predforte are also used to decrease the inflammation, but only under the direction of an ophthalmologist who will be providing daily follow-up. Workup for autoimmune disorders (such as lupus and rheumatoid arthritis) should be considered if it is a recurrent condition. Intraocular pressure should also be measured, as there can be an associated secondary glaucoma.

A **retinal detachment** should be suspected if the patient complains of new floaters and flashes of light or a visual deficit often described as a curtain falling over the visual field of the affected eye. Some detachments can be subtle and may not be seen without a dilated retinal exam by an ophthalmologist using a direct ophthalmoscope. If in doubt, call the ophthalmologist because visual loss can become permanent if the detachment is not repaired in time. The use of ED ultrasound can help in the diagnosis but should not be used if a globe rupture is suspected.

A **retrobulbar hemorrhage** should be considered as a ‘compartment syndrome of the eye’ and is a true eye emergency. After trauma to the eye, bleeding may develop behind the globe. The retrobulbar space is a fixed space with the globe anteriorly and the skull posteriorly, therefore the expanding hematoma begins to push the eyeball forward. The patient complains of intense pain and decreased vision as the hematoma presses on the optic nerve. Proptosis and decreased movement of the eye follow. The definitive diagnosis is by CT scan. The immediate surgical procedure of choice is a lateral canthotomy, which involves cutting of the lateral rectus muscle to allow the expanding hematoma to decompress. This should be done before CT if there is severe proptosis or vision loss. Definitive repair is then carried out in the operating room.

CLINICAL PEARLS

Treatment of Acute Angle Glaucoma

1. Topical pilocarpine
2. Topical timolol
3. IV acetazolamide
4. IV mannitol
5. Laser iridotomy.

SUDDEN VISUAL LOSS

Sudden visual loss can be a very anxiety-producing situation for the patient. Prompt diagnosis, treatment, and referral may be vision saving. The first decision point in the differential diagnosis is whether the visual loss is associated with pain or not.

Painful visual loss

The conditions that cause painful visual loss partially overlap with the ones already discussed in the trauma section. These include acute angle closure glaucoma, acute iritis, corneal abrasion, globe rupture, lens dislocation, and retrobulbar hemorrhage. Optic

neuritis, temporal arteritis, endophthalmitis, and keratitis complete the list.

Optic neuritis usually presents in young women between the ages of 20-40 who present with a decrease in their vision as well as retroocular pain and pain with eye movement. Half the cases have a swollen optic disc on physical exam while the other half have a normal appearing fundus (retrobulbar optic neuritis). There is a high association with multiple sclerosis and it may be the presenting complaint in up to fifty percent of cases. Referral to an ophthalmologist and a neurologist is required. They will arrange for an MRI of the brain (to screen for MS) and consider using intravenous pulse steroids.

Temporal arteritis usually affects patients over the age of fifty. The presenting complaint is usually that of a severe temporal headache. Approximately fifty percent have some degree of acute visual loss in the affected eye. An ESR greater than 50 is usually found. Treatment with oral prednisone is started in the emergency department while waiting for the temporal artery biopsy, which is the definitive test. This can be done as an outpatient by a surgeon or an ophthalmologist. Steroids have been shown to prevent further visual loss if started promptly.

Endophthalmitis (an infection of the globe interior) typically occurs a few days after eye surgery, most commonly cataract removal. The patient usually presents with severe pain, photophobia and decreased vision. Physical examination reveals conjunctival erythema, corneal edema, and a hypopyon (a layer of pus cells in the anterior chamber). Emergent ophthalmologic consultation is required to save the eye. Administration of intravenous and intravitreal antibiotics and partial vitrectomy are the mainstay of treatment.

Keratitis can be traumatic or infectious. Traumatic keratitis can be from physical trauma such as a punch to the eye, or chemical injury such as an alkali burn or ultraviolet burns such as “welders eye” or “snow blindness”. Infectious causes include bacterial (pseudomonas in contact lens wearers), viral (herpes simplex or zoster), or fungal. Treatment depends on the cause but generally involves some sort of antibiotic, antiviral or anti fungal drops and close ophthalmologic follow-up.

Painless visual loss

Painless visual loss can be caused by a retinal detachment, central retinal artery occlusion, central retinal vein occlusion, amaurosis fugax or occipital stroke.

A **central retinal artery occlusion** (CRAO) usually occurs in patients with vascular risk factors such as diabetes, hypertension, hyperlipidemia or vasculitis. The classic presentation is sudden severe painless visual loss. The classic physical exam finding is a pale retina with a cherry red spot. Prognosis is poor and most treatments (paracentesis, intra-arterial TPA) are ineffective. A central retinal vein occlusion is identical to CRAO except that on physical examination, the fundus shows diffuse hemorrhages and a swollen optic disc ('blood and thunder' retina).

Amaurosis fugax is considered to be a transient ischemic attack (TIA) of the retinal artery. Patients usually complain of a 'curtain coming down' over the affected eye. It usually resolves spontaneously within minutes to hours. It should be worked up like a TIA. An ECG is done to rule out atrial fibrillation, carotid dopplers to rule out carotid artery stenosis and a CT of the head to look for old or new strokes. Treatment with low dose ASA should be started in the ED after CT scan has ruled out a bleed. An occipital stroke can cause bilateral visual loss if it affects the visual cortex.

SUMMARY

- An organized, anatomical approach is helpful when treating a patient who has suffered trauma to the eye
- In the patient with trauma to the eye, an orbital fracture, eyelid laceration, corneal injury, hyphema, globe rupture, lens dislocation, retinal detachment and retrobulbar hemorrhage should all be ruled out
- The first decision point in the differential diagnosis of sudden visual loss is whether the visual loss is associated with pain or not
- In the patient with painful visual loss, acute angle closure glaucoma, acute iritis, corneal abrasion, globe rupture, lens dislocation, retrobulbar hemorrhage, optic neuritis, temporal arteritis, endophthalmitis, and keratitis all need to be considered
- In the patient with painless visual loss, retinal detachment, central retinal artery occlusion, central retinal vein occlusion, amaurosis fugax or occipital stroke need to be considered

WHAT'S NEW

Ocular ultrasound performed by trained physicians can expedite the diagnosis and management of several ocular emergencies including globe perforation, retrobulbar hematoma, retinal detachment, lens subluxation, vitreous hemorrhage, and intraocular foreign body.

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24: HYPERKALEMIA

Santosh Kanjeeval

OBJECTIVES

1. To identify the common etiologies of, and those patients at risk for, hyperkalemia
2. To identify the electrocardiogram changes associated with hyperkalemia
3. To manage the acutely hyperkalemic patient

INTRODUCTION

Severe hyperkalemia is a potentially rapidly lethal condition. As such, the initial recognition of patients at risk followed by appropriate management is of paramount importance.

CAUSES OF HYPERKALEMIA

The first question you should ask yourself when you receive an unexpected high potassium result is: Is it really hyperkalemia? Remember, falsely elevated potassium results are common, often due to hemolysis of the sample. Falsely elevated potassium levels can also occur in the setting of severe thrombocytosis or leukocytosis.

True hyperkalemia in the emergency department is most often related to decreased potassium excretion. The kidneys are the major route of potassium excretion. Renal failure, medications that affect kidney potassium regulation (e.g. ACE inhibitors, non-steroidal anti-inflammatories, potassium sparing diuretics), and acute acidosis are the most common culprits resulting in decreased excretion of potassium.

Two further significant causes of hyperkalemia, particularly in the emergency department, are due to the release of potassium as a result of cell death and extra-cellular shifts of potassium. The vast majority of potassium (98%) is found intracellularly. In the setting of trauma, burns, crush injuries and hemolysis, cellular death results in the extra-cellular release of potassium. This effect is often compounded by concomitant acute renal injury associated with these conditions. Several medications (e.g. succinylcholine and digoxin) can cause extra-cellular shifts of potassium by disrupting normal cellular regulation of potassium resulting in hyperkalemia.

Hyperkalemia can be caused by increased potassium intake, typically related to potassium containing

supplements or medications, especially if there is underlying renal impairment.

CLINICAL FEATURES

Recognizing patients at risk for hyperkalemia coupled with accurate assessment of the ECG are key to early identification of hyperkalemia.

Patients with hyperkalemia are often asymptomatic until they develop life threatening cardiac arrhythmias. Some patients may present with neuromuscular complaints such as muscle weakness or cramps and occasionally they may have focal neurologic symptoms. However, generally the symptoms are very non-specific.

The electrocardiogram is of utmost importance in the early recognition of hyperkalemia. There are characteristic changes that can be seen. The first changes include tall peaked T waves and ST segment depression. These are often seen at potassium levels starting at 5.5mEq/L. Beyond this, there is progressive change of the ECG with loss of P waves followed by widening of the QRS complex, prolongation of the PR interval, eventually development of a Sine wave pattern, and finally asystole or ventricular fibrillation. The early changes should be identified and treated as the patient may be otherwise asymptomatic. Of course, these changes are not always present and the potassium levels at which they occur may vary (especially if the patient has underlying chronic renal impairment). As such, a serum potassium level should always be sent if hyperkalemia is suspected.

Although these are the classic ECG findings associated with hyperkalemia, remember that patients may present with a variety of dysrhythmias including second or third degree heart block, ventricular arrhythmias, or asystole.

CLINICAL PEARL

Patients with known renal failure who present to the emergency department in extremis and have ECG findings consistent with hyperkalemia, should be treated on spec for hyperkalemia.

MANAGEMENT

When hyperkalemia is suspected, the patient should be placed on a cardiac monitor, an immediate ECG obtained, and 'stat' electrolyte levels and renal function tests sent.

The goals of management of hyperkalemia are threefold:

1. Protect the heart
 2. Immediately decrease serum potassium by driving potassium intracellularly
 3. Increase excretion of potassium
1. **Protect the heart** by antagonizing potassium directly at the cardiac membrane using calcium. This is accomplished by giving intravenous calcium (usually calcium chloride). This is indicated if there is evidence of QRS widening on the ECG or the patient is hypotensive - a true hyperkalemic emergency. Onset of action is within 5 minutes and lasts approximately 1 hour.
2. **Decrease serum potassium.** Serum potassium can be decreased acutely by shifting potassium intracellularly. Insulin (10units) shifts potassium resulting in a decrease of serum potassium by ~1mEq. Remember to give 1-2 amps of D50W prior to insulin administration to avoid hypoglycemia. A Beta agonist (salbutamol via continuous nebulization) also effectively shifts potassium intracellularly. Bicarbonate, by countering acidosis, will result in an intracellular shift of potassium. Of course this is only effective if the patient is acidotic in the first place. If the patient is clinically dehydrated, a normal saline bolus can stimulate potassium shifting intracellularly. However, as many of these patients have impaired renal function, fluid balance must be carefully managed.
3. **To definitively manage hyperkalemia, potassium must be excreted.** This may be accomplished using exchange resins such as

sodium polystyrene sulfonate (Kayexalate) and sorbitol which results in increased gastrointestinal excretion of potassium. If the kidneys are functioning, hyperkalemia may be reduced via enhanced urinary excretion using a diuretic such as furosemide. If renal function is impaired, dialysis may be emergently necessary to eliminate excess potassium.

SUMMARY

- Hyperkalemia is a potentially rapidly life threatening condition. Recognition of patients at risk and correct ECG interpretation are essential.
- Serum potassium levels may be falsely elevated particularly if hemolysis of the sample has occurred.
- Risk factors for hyperkalemia include renal impairment, acidosis, trauma, burn or crush injuries and certain medications.
- The ECG is key to early diagnosis -characteristic changes include tall peaked T waves, QRS widening, PR prolongation and eventually, progression to a Sine wave.
- Treatment requires: cardioprotection, the acute shift of potassium intracellularly, and the excretion of potassium.

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25: SEPSIS

Kate Lazier

OBJECTIVES

1. To understand the differences between bacteremia, SIRS, sepsis, severe sepsis, and septic shock
2. To understand how sepsis develops
3. To identify the most likely cause and the severity of sepsis based on history and physical examination
4. To know the typical work-up required for sepsis
5. To know key treatment principles for severe sepsis and septic shock

INTRODUCTION

Sepsis is a common emergency department presentation often associated with severe illness and mortality rates approaching 50%. Evidence shows that early recognition and treatment in severe cases can reduce mortality rates.

Definitions

Bacteremia - bacteria in the blood

Systemic Inflammatory Response Syndrome (SIRS)

More than 2 of:

- $>38^{\circ}\text{C}$ or $<36^{\circ}\text{C}$
- Pulse >90 beats/min
- RR >20 breaths/min (or $\text{PaCO}_2 < 30\text{ mmHg}$)
- WBC $>12 \times 10^9 \text{ cells/l}^9$ or $< 4 \times 10^9 \text{ cells/l}^9$ or $>10\%$ bands

Sepsis – SIRS in response to infection

Severe Sepsis - sepsis and signs of organ dysfunction (e.g. acute lung injury, coagulopathy, altered mental status, renal failure, decreased urine output, liver failure, lactic acidosis etc.)

Septic Shock – severe sepsis and hypotension ($\text{sBP} < 90$) unresponsive to IV crystalloid fluid bolus.

PATHOPHYSIOLOGY

A simple infection can become sepsis or even septic shock when the infectious organism causes a systemic response, triggering a complex release of

inflammatory mediators and activating the coagulation cascade. These processes cause diffuse microvascular injury, thrombosis, and endothelial disruption, which result in distributive shock and reduced oxygen delivery to tissues. This, in turn, causes organ dysfunction and lactic acidosis.

ETIOLOGY

The most common sites of infection in severe sepsis are lung (35%), abdomen (21%), urinary tract (13%), skin and soft tissue (7%) and other or unknown sites. In patients over 65 years old, the urinary tract is the most common source of infection. The most common bacteria involved are E. Coli, Strep. Pneumoniae and Staph. Aureus. MRSA (Methicillin-Resistant Staph Aureus) should be considered in patients with recent hospitalizations or from nursing homes. Anaerobes may be involved in intrabdominal and soft tissue infections.

DIAGNOSIS & ASSESSMENT

Everything starts, of course, with the history and physical exam. On history, ask about all systems and note symptoms that point to the source of the infection and severity of the illness. Are there symptoms like dizziness and fainting that might suggest hemodynamic instability? Note also key relevant issues on past medical history such as immunocompromised states (e.g. steroids, HIV/AIDS, chemotherapy/cancer), recent procedures (e.g. indwelling line or catheter, surgery), or other risk factors such as IV drug use.

On examination, pay close attention to vital signs. The classic pattern in severe sepsis/septic shock is tachycardia accompanied by hypotension and fever. Very sick patients are often tachypneic from either metabolic acidosis (blowing off CO_2) or lung disease (or both). There are times when the tachycardia may be masked by beta blockade. Remember that septic patients can also present with bradycardia or hypothermia ($\text{temp} < 36$). Note the overall appearance of the patient and the level of consciousness. Mental status changes are often a sign of severe sepsis or septic shock. Do your ABCs. Is there respiratory distress with a high respiratory rate and increased accessory muscle use? Is the skin warm and flushed (common in early sepsis), or cool because of

vasoconstriction? In addition to respiratory, cardiovascular, and abdominal exams, do a thorough head-to-toe survey looking for more unusual sources of infection. Look in the throat for pharyngitis. Is there a hot swollen joint suggesting a septic joint? Check for neck stiffness and purpura/petechia associated with meningitis. Look for skin ulcers and other skin infections. Is there a new murmur or other features typical of endocarditis? And don't forget the genitals!

INVESTIGATIONS

In order to detect the effect of sepsis systemically and to pinpoint the source of infection, investigations should be broad. In addition to an abnormal **WBC** (usually elevated but sometimes low), patients often develop **thrombocytopenia** and **electrolyte** abnormalities. **Hemoglobin** levels may initially be high from hemoconcentration. It is common for **renal, liver, and coagulation** parameters to be elevated reflecting multisystem organ failure. **Cardiac enzymes** may also be elevated reflecting either sepsis-related myocardial depression or secondary ischemia. Hypoperfusion and the resulting cellular hypoxia means cells use anaerobic metabolism producing increased **lactate** levels and an associated metabolic acidosis (usually with an increased anion gap). The **central venous oxygen saturation** (ScvO_2), measured either with a special probe placed in the SVC or drawn as a gas sample, reflects abnormal peripheral oxygen delivery and metabolism and is often abnormal (i.e. $<70\%$). In addition to these studies, **chest x-ray, urine R+M, C+S, ECG, and blood cultures** should be routine in all suspected cases of severe sepsis/septic shock. Other investigations may include throat swabs, sputum cultures, LP for cell count and culture, stool cultures, and genital swabs. CT abdo-pelvis, echocardiogram, abdominal or pelvic ultrasound may also be indicated to locate the source of infection.

TREATMENT

Treatment of severe sepsis and septic shock proceeds in tandem with assessment as follows:

- ABCs, monitors, and lines:** Place cardiac, BP and O₂ saturation monitors. Give oxygen. Start 2 large bore IVs. Some septic patients will need early intubation because of respiratory distress or decreased level of consciousness compromising airway reflexes. As described below, large volumes of IV fluids and often vasopressors are required to normalize blood pressure and

perfusion. An arterial line is sometimes needed to accurately measure BP. Central lines are often needed for fluid, pressors, and for monitoring CVP and ScvO_2 .

- Early and aggressive IV fluids:** crystalloid (NS) through 2 large bore IVs is mandatory in patients with severe sepsis or septic shock with an initial bolus of 20-40 ml/kg over 15-30 minutes followed by 500 ml every 15-30 minutes. Fluids should be given aggressively to achieve MAP and CVP targets outlined below. Some patients may require up to 6-8 litres of fluid resuscitation. Studies have shown crystalloid (Ringers and NS) to be equivalent to colloid (albumin/ pentastarch) in the fluid resuscitation of septic patients, and since the former is cheaper, it is most often used.
- Early and appropriate antibiotics:** If the source of infection is obvious on initial assessment, then targeted antibiotics may be appropriate. If not, broad-spectrum IV antibiotics should be given. One broad-spectrum approach would be to give piperacillin-tazobactam 4.5 g IV with vancomycin 1g IV if MRSA is suspected and azithromycin 500mg IV if pneumonia is suspected. Your hospital may have its own protocol that you can follow.
- Appropriate surgical intervention:** Some causes of severe sepsis and septic shock require a procedure to control the focus of infection. This may be as simple as removing an infected foreign body like a urinary catheter or draining an abscess. But other patients, such as those with ischemic gut or necrotizing fasciitis, need immediate surgery.
- 'Early Goal Directed Therapy'** describes a target-based resuscitation protocol for cases of severe sepsis and septic shock. Central Venous Pressure (CVP) is measured through a central line (internal jugular or subclavian) and a target of 8-12 mmHg is achieved through frequent fluid boluses. A target Mean Arterial Pressure (MAP) of 65-90 mmHg is achieved by giving vasopressors (dopamine or norepinephrine). Finally, in order to improve oxygen delivery to the tissues, packed red blood cells are transfused to achieve a hemoglobin of 90 and dobutamine, which has inotropic effects, is titrated to achieve a central venous oxygen saturation of $\geq 70\%$.
- Other interventions to consider include:
 - Steroids:** such as IV hydrocortisone may be considered when patients have severe septic

- shock and remain hypotensive despite maximal fluids and vasopressors.
- b. **Intubation, sedation, paralysis and ventilation** may help achieve a ScvO₂>70%.

CLINICAL PEARLS

Treatment Priorities in Severe Sepsis/Septic Shock

1. Aggressive fluid administration
2. Early appropriate antibiotic administration
3. Surgical intervention in some cases
4. Blood pressure control with vasopressors as required

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26: ANAPHYLAXIS

Leeor Sommer

OBJECTIVES

1. To recognize anaphylaxis
2. To consider the differential diagnosis of anaphylaxis
3. To understand the basic pathophysiology of anaphylaxis
4. To review the management of anaphylaxis
5. To provide the appropriate discharge instructions for patients with anaphylaxis

INTRODUCTION

Anaphylaxis is a common and potentially life-threatening emergency. Anaphylactic reactions vary dramatically, from simple cutaneous reactions, to respiratory and cardiovascular compromise and death. While only 500-1500 fatal anaphylactic reactions occur annually in the United States, a significant proportion of the population is at risk for these reactions. With appropriate, early treatment and effective prevention strategies, the risk of morbidity and mortality from anaphylaxis can be diminished.

PRESENTATION

Due to the highly variable nature of anaphylaxis, a clear definition is difficult to attain. All definitions are flawed in some respect. One recent article uses the following useful definition: "Anaphylaxis is a severe allergic reaction to any stimulus, (usually) having sudden onset and generally lasting less than 24 hours, involving one or more body systems and producing one or more symptoms such as hives, flushing, itching, angioedema, stridor, wheezing, shortness of breath, vomiting, diarrhea, or shock".

Symptom onset varies widely, but commonly occurs seconds to minutes after exposure. Occasionally, symptom onset may take several hours.

Differential Diagnosis

The majority of cases of anaphylaxis are quite straightforward and obvious to diagnose. A history of prior allergy and exposure to an offending agent followed by the development of mucocutaneous signs often assist in the diagnosis of anaphylaxis. However, in patients with no previous history of allergy, or those who present with only respiratory, gastrointestinal, or cardiovascular compromise, the diagnosis of anaphylaxis becomes clinically

challenging. There are no laboratory tests that aid in the diagnosis of anaphylaxis in the acute setting. A differential diagnosis depends on the predominant presenting symptom.

Patients presenting with hypotension or shock should be examined to exclude septic shock, cardiogenic shock, hypovolemic shock, pulmonary embolism, tension pneumothorax, cardiac tamponade and vasovagal reactions. The differential diagnosis for respiratory distress includes: asthma, COPD, foreign body aspiration, and vocal cord dysfunction. Patients presenting with anaphylaxis have been misdiagnosed as being anxious or having a panic attack, leading to delays in treatment and morbidity.

Frequency of Signs and symptoms

Signs and symptoms	Frequency (%)
urticaria, angioedema	88
dyspnea, wheeze	47
dizziness, syncope, hypotension	33
nausea, vomiting, diarrhea, cramping	30
abdominal pain	
flush	46
upper airway edema	56
headache	15
rhinitis	16
substernal pain	6
pruritis without rash	4.5
seizure	1.5

Epidemiology and Causative Agents

Limited data is available on the incidence of anaphylaxis in Canada or elsewhere. It is widely considered to be under reported and under recognized. Approximately 1 in every 1000-3000 patient visits to an emergency department is due to an anaphylactic reaction. Anaphylaxis leads to approximately 500-1500 deaths annually in the

United States, with a peak incidence in summer months.

Food allergy is the most common cause of anaphylaxis in North America and Western Europe. 1-2% of Americans are reported to have a food allergy, with 100 food related anaphylactic fatalities reported annually. The most common allergens are peanuts, tree nuts, shellfish, fish, milk, eggs, soy and wheat. Peanuts and tree nuts (cashew, hazelnut, walnut) account for well over half of all food allergies in the United States. There has been a dramatic increase in the prevalence of food related allergy in the United States over the past 2-3 decades. It is hypothesized that the widespread use of peanuts in processed food supplies, leading to earlier and more extensive exposure to these allergens, may account for some of the rise in prevalence of these allergies. A history of asthma or atopy is a risk factor for development of severe food related anaphylactic reactions.

Iatrogenically induced anaphylactic reactions are the second most common cause of anaphylaxis and the leading cause of anaphylactic fatalities. 2-3% of hospitalized patients will have an allergic reaction from a drug given in hospital. 56% of iatrogenic anaphylaxis will occur while in the operating room. Fatal iatrogenic anaphylactic reactions tend to occur very rapidly, with cardiac arrest occurring in less than 5 minutes in the majority of cases. It is imperative that all health care professionals be familiar with the prompt diagnosis and treatment of anaphylactic reactions. Parenteral drugs tend to lead to more severe and more rapidly developing reactions. The most common drugs causing anaphylactic reactions are antibiotics, with penicillin alone accounting for 75% of all fatal anaphylactic reactions. Other β -lactam antibiotics, radiocontrast media, ASA, IV anaesthetics, NSAIDS and opioids are also common causes of iatrogenic anaphylaxis.

Insect stings are another common cause of anaphylactic reactions. 0.5-2% of the American population have a sting allergy, with sting induced anaphylaxis accounting for 40-100 deaths annually in the United States. The risk of recurrence after an episode of sting-induced anaphylaxis is 30-60%.

Latex has recently become a significant allergen causing anaphylaxis. It is estimated that while only 1-2% of the general public has a latex sensitivity, 8-17% of health care workers are latex sensitive.

Pathophysiology

Acute anaphylaxis is an IgE-mediated allergic phenomenon. Allergic anaphylaxis requires prior exposure and sensitization to an allergen. Upon

subsequent exposure, the allergen is recognized and a reaction is triggered. A recognized allergen will bind IgE on the surface of mast cells and plasma basophils, causing them to degranulate and release histamine and other bioactive mediators. Closely related to anaphylaxis, anaphylactoid reactions similarly cause degranulation of mast cells and basophils in a non-IgE mediated cascade. Anaphylactoid reactions are clinically indistinguishable from anaphylaxis and are treated in an identical manner.

Histamine activates H1 and H2 receptors. Pruritis, rhinorrhea, tachycardia, and bronchospasm are caused by activation of H1 receptors. H1 and H2 receptors both mediate headache, flushing and hypotension. Many of histamine's effects can be traced to increased capillary permeability and vasodilation. In the skin, this leads to urticaria. In subcutaneous tissue, this causes angioedema. System wide vasodilation causes hypotension and shock, while increased inflammation in the respiratory and gastrointestinal systems lead to dyspnea, wheezing, vomiting, diarrhea, and crampy abdominal pain.

MANAGEMENT

As with all potential life threatening emergencies, special focus should initially be placed on management of the ABC's. All patients should be placed in a closely-observed setting with continuous O2 saturation and cardiac monitoring.

Emergency airway management in severe anaphylaxis can pose significant problems. In cases of significant airway edema, or intractable wheezing, orotracheal intubation should be initiated. If difficulty is expected due to obstructive airway edema, one can attempt awake fiberoptic intubation, but the patient (and physician) should be prepared for an emergency surgical airway.

Breathing should be augmented with 100% O2 via nonbreathing mask. An inhaled β 2 agonist should be given to wheezing patients. Intravenous access should be obtained in all patients. In cases of hypotension and shock, rapid infusions of 1-2 L of crystalloid (20cc/kg in children) should be started.

Due to the rarity and incidental nature of anaphylaxis, little high grade evidence regarding treatment is available. The majority of treatment recommendations are based on consensus opinion.

The mainstay of pharmacologic treatment of anaphylaxis is epinephrine. Because subcutaneous administration leads to localized vasoconstriction and decreased plasma uptake of epinephrine, an

intramuscular route is the preferred method of administration. The dose of **epinephrine** in anaphylaxis is **0.3–0.5 mg IM** (0.01 mg/kg in children) of 1:1000. Epinephrine acts on α receptors, causing vasoconstriction, leading to an increase in blood pressure and a decrease in angioedema. Its effects on bronchial β_2 receptors causes bronchodilation, while excitation of β_2 receptors on mast cell and basophils lead to a decrease in the release of inflammatory mediators. Patients with a history of cardiac ischemia should be closely watched, but there is no absolute contraindication to the use of epinephrine in anaphylaxis. Medications that may increase the effect of epinephrine include MAOIs and some TCAs.

Patients on β -blockers may not respond to epinephrine. If hypotension persists in these patients, one should administer glucagon 1 mg IV, in repeat doses every 5 minutes (max 10 doses). If bolus administration is successful, a glucagon infusion should be started at 1–5 mg/hr.

As histamine is the prime mediator of anaphylaxis, antihistamines should be administered. An H1 receptor blocker such as diphenhydramine 50 mg IV (1 mg/kg) and an H2 receptor blocker such as ranitidine 50mg (1 mg/kg) IV should both be given.

Although not effective in the acute phase of an anaphylactic reaction, corticosteroids may play a role in minimizing the chance of a second phase reaction. Some patients may improve dramatically after initial therapy, only to have a recurrence of severe symptoms 4–12 hours later. A dose of steroid (e.g. Methylprednisolone 1 mg/kg IV) is thought to reduce the possibility of a second phase reaction.

In cases of severe refractory hypotension, IV epinephrine should be carefully administered in a monitored setting. Doses should be titrated up from 0.01 μ g/kg/min up to 1 μ g/kg/min. The addition of other vasopressors may be required.

CLINICAL PEARLS

Some patients with anaphylaxis may improve dramatically after initial therapy, only to have a recurrence of severe symptoms 4–12 hours later. A dose of steroid is thought to reduce the possibility of a second phase reaction.

DISPOSITION

Instructions and Follow-Up

All patients should be monitored in the emergency department for a minimum of 4–6 hours due to the risk of a second phase, or rebound reaction. Second phase reactions have been reported up to 24 hours after initial exposure. All patients discharged from hospital should be supervised and have ready access to emergency services (911).

There is no clear evidence guiding ongoing treatment with medications after hospital discharge. It is common practice to treat most patients with a non-sedating H1 antagonist antihistamine (e.g. Cetirizine 10mg po od), an H2 antagonist (e.g. Ranitidine 150mg po bid) and a corticosteroid (e.g. Prednisone 50mg po od) each for a 3 day course.

All patients who have had a significant anaphylactic reaction should be prescribed an automated self-injectable epinephrine device (e.g. EpiPen) and instructed on its use. Patients should be instructed to carry the device on their person at all times.

Discharge

An EpiPen contains 0.3mg of 1:1000 epinephrine that should be delivered IM into the lateral aspect of the thigh (vastus lateralis), through clothing if necessary. EpiPen Jr. (0.15mg of epinephrine) should be prescribed to children weighing 10–25 kg and parents should be instructed on its use. Children weighing less than 10 kg pose a special risk, as no automated administration device is available in low doses. Parents should be instructed on how to draw up and administer 1:1000 epinephrine at an appropriate dose.

In addition to epinephrine, the patient may be instructed on self administration of an oral antihistamine (e.g. diphenhydramine 50 mg) in liquid form in the event of a recurrent exposure. All patients should be instructed to seek immediate medical attention in an ER during an allergic reaction, even when treatment has been self initiated.

In cases when an offending allergen is identified, patients should be instructed on strict avoidance of the allergen. Avoidance is the only way to prevent future reactions, although accidental exposures occur in some instances.

A medic-alert or similar identification bracelet should be suggested in case of future exposure. Some physicians suggest that an anaphylaxis emergency plan, containing a photo ID and list of triggers, be given to school offices or workplaces for patients

with severe anaphylaxis. Education of family and close friends may play a role in the early management of severe cases. A referral to an allergy specialist for further education and skin-prick testing in patients without an identifiable cause has been proven to be beneficial.

Patients with insect sting allergy should be referred to an allergy specialist for immunotherapy. Immunotherapy in these reactions can reduce recurrence rates of severe reactions to insect stings by 97%. Desensitization protocols are also available for some antibiotics including penicillin.

SUMMARY

- Anaphylaxis is a multisystem disease
- Although anaphylaxis is common, fatal anaphylaxis is very uncommon
- Prompt recognition and treatment is vital
- Epinephrine (0.01 mg/kg IM) is the mainstay of

treatment

- Discharge instructions should be reviewed to reduce the chances of recurrent severe anaphylaxis

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Pharmacologic Treatment for Acute Anaphylaxis

Drug	Dose / Route	Indication
epinephrine	0.3 – 0.5 mg IM (0.01 mg/kg (1:1000) IM)	hypotension, angioedema, wheezing
IV Crystallloid	1 – 2 L IV (10 – 20 cc/kg)	hypotension
diphenhydramine	50 mg IV (1 mg/kg IV)	pruritis, urticaria, wheezing, angioedema
ranitidine	50 mg IV (1 mg/kg IV)	pruritis, urticaria, wheezing, angioedema
methylprednisolone	80 – 125 mg IV (1 – 2 mg/kg IV)	hypotension, angioedema, wheezing
salbutamol	5 mg in 3 cc NS inhaled (0.15 mg/kg inhaled)	bronchospasm
glucagon	1 mg IV bolus 1 – 5 mg/hr IV infusion	refractory hypotension with β -blockers
aminophylline	5 mg/kg bolus over 20 min then 0.8 mg/kg/hr	refractory bronchospasm

27: ADVERSE DRUG REACTIONS & INTERACTIONS

Walter Himmel, Caroline Thompson

OBJECTIVES

1. To define adverse drug reactions and adverse drug events.
2. To describe important physician behavior to minimize ADR and ADE.
3. To understand Serotonin Syndrome.
4. To understand Drug-induced QT Prolongation.
5. To understand Antibiotic Associated Diarrhea.

INTRODUCTION

Definitions

Terminology recommended by the WHO is preferred by most national adverse drug reaction monitoring centres, including Canada Vigilance of Health Canada, and the FDA of the USA.

Adverse Drug Reaction (ADR): A noxious and unintended response to a drug that occurs during appropriate use of the drug, at doses normally used in humans.

Side Effect: Any unintended (but **not noxious**) effect of a pharmaceutical product, that is **predictable** by pharmacological properties of the drug (may be desirable or undesirable).

Adverse Drug Event (ADE): Any undesirable event, including ADRs as well as preventable adverse outcomes such as human error, noncompliance, overdose, therapeutic failure.

An important clinical feature of ADRs is that they may be difficult to diagnose with absolute confidence. Under-diagnosis leads to under-reporting. Therefore “**Suspected Adverse Drug Reaction**” is an encouraged and important term in clinical use.

Epidemiology

Adverse drug reactions and events are common. Overall incidence is unknown but studies have shown:

- 8% of emergency department visits are due to an ADR

- 3% - 8% of hospitalized patients were admitted because of an ADR
- 7% of admitted patients will experience a serious ADR during their stay
- 0.3% hospitalized patients die as a result of an ADR

CLINICAL PRESENTATION

Be vigilant! In patients who are using medications, **all clinical presentations** can potentially represent adverse drug events. Emergency Physicians often include ADR/ADE as part of “toxic-metabolic” in the differential diagnosis. Think of ADR when patients present with the following:

- Angioedema/ anaphylaxis/cough/wheeze
- Altered mental status/ delirium/drowsiness
- Coagulopathy/ hemorrhage
- Dyspepsia/ GI bleed
- Diarrhea/ C. difficile colitis
- Hypoglycemia/ electrolyte disturbance
- Orthostasis/weak & dizzy/falls
- Neutropenia
- Rash
- Syncope/sudden cardiac death

CLINICAL PEARL

The most common presentations of ADR are: hypoglycemia, hypotension, coagulopathy or hemorrhage, rash, mental status change, and GI symptoms.

Etiology of Adverse Drug Reactions (ABCDE)

Type A: “Augmented” –Dose-dependent and predictable from the known pharmacology of the medication. Often related to drug interactions. Requires dose adjustment or discontinuation. 75% of all ADR are this type.

Type B: “Bizarre” –Idiosyncratic or immunologic. Unrelated to pharmacology –

anaphylaxis, anaphylactoid, malignant hyperthermia. Withhold and avoid future use.

Type C: “Chronic” – Cumulative dose-related. (adrenal insufficiency secondary to corticosteroids). Prolong withdrawal, reduce dose, or avoid use.

Type D: “Delayed” - Late effects – teratogenesis, carcinogenesis, tardive dyskinesia.

Type E: “End of Use” – Withdrawal effects (e.g. opioids). Withdraw slowly and with clinical supervision.

PATIENT SAFETY: PHYSICIAN BEHAVIOURS THAT REDUCE THE RISK OF ADRS

30-50% of adverse drug events are preventable.

1. Ensure proper documentation of current medications, herbal remedies, and drug allergies.
2. Does this patient have important risk factors for an ADR?
 - Allergies

- Pregnancy
- Breastfeeding
- Renal impairment
- Hepatic impairment
- Cognitive/behavioral/compliance issues
- Advanced age is a risk factor due to subclinical hepatic and renal function resulting in slower metabolism

3. Does this medication have drug-drug interactions? Don't rely on your memory. Use a computerized drug interaction system to minimize human error such as:

- Lexi-Comp on UpToDate
- Epocrates
- Electronic Medical Record software
- Computerized physician order entry

4. Dose adjustment for renal impairment or age:
 - What is the creatinine?
 - Certain drugs require modification in renal insufficiency – digoxin, lithium, ACE inhibitors, diuretics, NSAIDS (avoid), some beta-blockers, and many antibiotics
 - Calculate the creatinine clearance before starting a new drug

$$eC_{Cr} = \frac{(140 - \text{Age}) \times \text{Mass (in kilograms)} \times \text{Constant}}{\text{Serum Creatinine (in } \mu\text{mol/L)}}$$

Where Constant is 1.23 for men and 1.04 for women.

1. Explain the drug and its side effects/risks to the patient.
 - Warn patients about important side effects to expect and how to avoid them. (orthostasis, esophagitis, gastritis)
 - Warn patients about the dangers of mixing prescribed medication with self-medication and/or street-drugs.
2. Long-term management of polypharmacy:
 - Review medications and discontinue unnecessary drugs
 - Consider drugs as cause of new symptoms
 - Avoid high-risk drugs (opioids, NSAIDs)
 - Avoid treating side effect of one drug with another drug

IMPORTANT EXAMPLES OF ADVERSE DRUG REACTIONS

The examples given feature some important clinical scenarios but this list is by no means intended to be exhaustive.

Most Common Adverse Drug Reactions

Aspirin, Warfarin, NSAIDs, and diuretics are the most common drugs implicated in adverse drug reactions among patients being admitted to hospital. These drugs have very well proven efficacy but are also in very wide use with well-known risks, including a significant mortality risk. The 2 tables below are taken from BMJ 2004, ‘Adverse Drug Reactions as cause of admission to hospital: prospective analysis of 18,820 patients.’

Most Common Drugs causing Adverse Drug Reactions:

NSAIDs (30%)	GI bleeding, peptic ulceration, hemorrhagic stroke, renal impairment
Diuretics (27%)	Renal impairment, hypotension, electrolyte disturbances, gout
Warfarin (10%)	GI bleeding, hematuria, high INR, hematoma
ACE inhibitors	Renal impairment, hypotension, electrolyte disturbance, angioedema
Antidepressants	Confusion, hypotension, constipation, GI bleed, hyponatremia
B-blockers	Bradycardia, heart block, hypotension, wheezing
Opiates	Constipation, vomiting, confusion, urinary retention
Digoxin	Symptomatic toxic digoxin levels
Prednisolone	Gastritis, GI bleeding, hyperglycemia, osteoporotic fracture
Clopidogrel	GI bleeding

Most Common Fatal Adverse Drug Reactions:

- | | |
|----------------------------|---------------------------------------|
| 1. GI Bleeding | Aspirin, NSAIDs, prednisone, warfarin |
| 2. Perforated PUD | Aspirin/NSAIDs |
| 3. Intracranial Hemorrhage | Aspirin/warfarin |
| 4. Renal failure | ACEI, diuretic |

Serotonin Syndrome

Serotonin Syndrome is a Type A ADR (dose-dependent and predictable) with significant morbidity and mortality. It occurs during concurrent use of two or more drugs that enhance central nervous system serotonin activity, typically an SSRI plus another serotonergic drug.

- Antidepressants: SSRI, SNRI, MAOI, lithium
- Antibiotic: linezolid (MAOI activity)
- Stimulants: cocaine, amphetamine, ecstasy
- Migraines: sumatriptans
- Opioids: meperidine, dextromethorphan, tramadol
- Herbal remedies: St. John's Wort

The clinical triad of Serotonin Syndrome:

1. Altered mental status (confusion, agitation, coma)
2. Autonomic nervous system dysfunction (fever, shivering, sweating, diarrhea, tachycardia, tachypnea, high or low BP)

3. Neuromuscular abnormalities (ataxia, hyperreflexia, myoclonus, tremor, rigidity, akathisia)

Management:

- Avoidance and early recognition
- Discontinuation of serotonergic drug
- Resuscitation: sedation, ventilation, cooling, anticonvulsants, and antihypertensives
- Rule out infectious, metabolic, endocrine, or other toxic etiology
- Antiserotonergic agent such as cyproheptadine

Drug Induced QT Prolongation

Myocyte repolarization is driven by the outward movement of K⁺ ions through K⁺ channels. Blockade of these K⁺ currents leads to QT interval prolongation, which is associated with torsades de pointes, ventricular fibrillation, and sudden cardiac death. This is a Type A ADR (dose-dependent and

predictable) with significant risk of sudden cardiac death.

Many drugs are implicated, and drug-drug interactions are the main concern:

- Antiarrhythmic Class III (potassium channel repolarization)
 - amiodarone, sotalol,
 - procainamide (its metabolite has Class III side effects)
- Antipsychotics - haloperidol, chlorpromazine, clozapine
- Antibiotics - fluoroquinolones, macrolides
- Antimalarials - quinine, quinidine
- Antifungal - fluconazole
- Antihistamines

The risk of torsades de pointes during usage of these drugs is increased by patient metabolic factors such as inborn K⁺ channel abnormalities and electrolyte disturbances (hypomagnesemia, hypokalemia).

Remember that antiarrhythmic drugs are pro-arrhythmic. It will remind you to look up drug-drug interactions causing QT prolongation in patients taking antiarrhythmic drugs.

Antibiotic-Associated Diarrhea and Pseudomembranous Colitis

Antibiotic-associated diarrhea is a common and serious adverse event following antibiotic use. It's generally considered non-preventable (Type A ADR) although antibiotic overuse is widely recognized, and thus many patients are suffering a preventable illness (ADE).

Diarrhea is **commonly** associated with antibiotic use:

- 5 to 10% of patients treated with ampicillin
- 10 to 25% of patients treated with amoxicillin-clavulanate
- 15 to 20% of those who receive cefixime

Pseudomembranous colitis is an important type of antibiotic-associated diarrhea, usually caused by **C. difficile**. It is an opportunistic infection, most frequently associated with elderly, co-morbid, hospitalized patients, on broad-spectrum antibiotics. All antibiotics, however, have been linked to **C. difficile** colitis.

C. difficile colitis presents with pain, fever, and foul-smelling, watery diarrhea, and can be complicated by toxic megacolon that requires colectomy. It has high morbidity and mortality, and high health care costs. Treatment is complicated by relapse in up to 25% of

cases. Enteric outbreaks of **C. difficile** are difficult to contain because the bacteria are spore forming and difficult to kill on hospital surfaces.

SUMMARY

Medications are often beneficial, but can also be harmful. Their use is complicated by side effects, adverse drug reactions, and other adverse events. Before starting a new drug, please be vigilant in considering patient characteristics, verifying current medication lists, and looking up potential drug-drug interactions.

Consider adverse drug reactions in all clinical presentations. Recognize serotonin syndrome with altered mental status, autonomic dysfunction, and neuromuscular findings. Look for QT prolongation on ECG and beware of drug-drug interactions. Expect to see adverse drug reactions frequently.

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28: VAGINAL BLEEDING

Sev Perelman

OBJECTIVES

1. To recognize life-threatening causes of vaginal bleeding
2. To develop an approach to the causes of vaginal bleeding according to age
3. To review the treatment of dysfunctional uterine bleeding
4. To diagnose and manage vaginal bleeding in pregnant patients

INTRODUCTION

In any patient with vaginal bleeding, the first priority is to establish the level of clinical stability. The ABCD approach is essential. Ask about symptoms of lightheadedness (pre-syncope), SOB and chest pain. All vital signs must be taken in all patients. Orthostatic changes in heart rate and blood pressure should be assessed; early signs of hypovolemia may be subtle, especially in younger patients.

For hemodynamically unstable patients, provide O₂, obtain IV (at least two 18 gauge) or intraosseous access immediately, bolus 1-2 litres of normal saline, and transfer to an acute care monitored bed. Order blood for CBC, electrolytes, urea, creatinine, INR, PTT, blood group type ('group and screen') and bHCG. Ask that urine output be monitored.

The assessment of vaginal bleeding begins with a focused history and physical examination. Age is an important factor in determining the etiology of vaginal bleeding. In practical terms, vaginal bleedings can be divided into two groups: related or unrelated to pregnancy. The latter can be further classified as non-uterine, ovulatory, and anovulatory bleeding. Any vaginal bleeding occurring outside cyclical menses is abnormal. Vaginal bleeding secondary to trauma may be caused by sexual abuse or assault (although, most sexual abuse victims suffer minimal trauma).

Definitions

The normal menstrual cycle lasts 28 ± 7 days, the flow lasts 4 ± 2 days, and the average blood loss is 40 ± 20 ml.

Menorrhagia (hypermenorrhea) - menses >7 days or >80mL (>4 fully soaked pads), or <21 day recurrence

from any cause. Loss of >60ml of blood per month may result in anemia.

Metrorrhagia - irregular vaginal bleeding outside the normal cycle

Menometrorrhagia - excessive irregular bleeding

Dysfunctional uterine bleeding - abnormal vaginal bleeding due to anovulation

Postcoital bleeding - bleeding after intercourse

Life Threatening Causes of Vaginal Bleeding

- Ectopic pregnancy
- Abruptio placentae
- Placenta previa
- Uterine rupture
- Post-partum hemorrhage
- Post partum or post procedural endometritis
- Trauma

Origins of Vaginal Bleeding

- Extra-uterine (PID, endometriosis)
- Uterine (fibroids, mucosal hyperplasia, IUD)
- Cervical (cervicitis, hyperplasia, cancer)
- Vaginal (vaginitis, trauma, FB)
- Genito-urinary (urethral, UTI)
- Intra-abdominal hemorrhage
- Fistula (IBD)

VAGINAL BLEEDING UNRELATED TO PREGNANCY

Children & Neonates

Uterine bleeding may be normal in the first 6 weeks of life and is related to estrogen withdrawal. Any bleeding thereafter is pathological. Common causes of vaginal bleeding in pre-pubertal girls include: vaginitis (10%), precocious puberty (21%), GU tumors (10%), vulvar pathology (8%), and trauma (including sexual abuse) (8%).

Most cases of perineal trauma are the result of bicycle injuries. In penetrating trauma, a rectal exam should be done in order not miss a rectal injury. This often needs to be done under procedural sedation or occasionally, under general anesthesia. In every case of pelvic trauma in children, you must assess their

ability to void prior to discharge due to the high incidence of urethral spasm and urinary retention.

Another cause of bloody discharge is vaginal foreign bodies. This is actually not bleeding, but rather infectious or inflammatory exudate with a sero-sanguinous component and is often foul smelling.

A less common cause of perineal bleeding is urethral prolapse. This is mostly present in people of African descent between the ages of 2 and 10 years. This 1-2 cm purple-red mucosal evisceration of the urethra presents as vaginal bleeding in 90% of cases. Treatment with estrogen cream is very effective.

During the examination, make children and parents comfortable. Try to examine the child on their parent's lap. Avoid using a speculum in pre-pubertal girls; one may do a rectal examination if really necessary, to access pelvic pathology.

Child abuse needs to be excluded. Look for evidence of external/internal trauma or behavioral changes. Children with seborrhea and psoriasis may present with bleeding after a relatively minor trauma.

Young Women

Onset of menarche in North American girls is 12.5 years of age (mean) with the lower limit of normal at 10 years of age. During the first 2 to 5 years after the onset of menarche, menses may be irregular and are often anovulatory.

All women of childbearing age are pregnant until proven otherwise. Do not omit a pregnancy test in women who deny being pregnant or in those with previous tubal ligations. Always try to interview patients without parents present. Be patient. If so requested, try to find a female physician and always ensure presence of a female nurse during the exam.

Always ask the patient about age of menarche, date of last period (whether it was normal or delayed), whether menses are usually regular or not (predictor of normal ovulation), and method of contraception (regularity, compliance, barrier protection, IUD). Ask about risk factors for HIV, hepatitis, STD, PID and ectopic pregnancy. Assess for the possibility of sexual abuse and domestic violence.

Cyclical vaginal bleeding should be distinguished from menorrhagia. Vaginal bleeding can be painful or painless. If painful, enquire about onset, duration, location, radiation, severity, quality, aggravating/relieving factors, previous episodes and associated features. Associated features may include

GU, GI symptoms, weight loss, history of bleeding disorders, and family history.

Anovulatory bleeding is often irregular (prolonged amenorrhea with periodic menorrhagia). This is due to fluctuating estrogen levels, which at times fall below the level supporting the endometrium.

Typically, there is minimal or no cramping.

Anovulatory vaginal bleeding in this age group may be related to eating disorders, weight loss, exercise, and stress. One in a 100 women develops premature ovarian failure and may suffer from atrophic mucosal changes resulting in bleeding. Obesity and polycystic ovaries may result in higher levels of estrogen and anovulation. Oral contraceptive use is the most common cause of mid-cycle bleeding and may require adjustment of the oral contraceptive pill to one with different (higher) estrogen content. Pre-menstrual spotting, however, is usually the result of an inadequate luteal phase and may require a higher progesterone level in the pill.

Bleeding disorders may present as vaginal bleeding. Up to 70% of women with Von Willebrand's disease and 50% of women with hemophilia A and B have vaginal bleeding as their first presentation. The differential should also include cervical, vaginal and lower GU tumors, and bleeding urethral caruncles.

Older Women

In North America, menopause occurs on average at 51 years of age. Estrogen levels are very variable. To assist with the diagnosis, a history of peri-menopausal symptoms is important. The most common etiologies of vaginal bleeding in older women are exogenous estrogens, including phytoestrogens – e.g. soya based natural remedies (30%), atrophic changes (30%), and endometrial cancer (15%). The first two causes are referred to as dysfunctional uterine bleeding (DUB).

In the Emergency Department, most of the definitive tests (FSH, Estrogen level, TSH, etc) are of little value. These tests and a pelvic ultrasound should be arranged by the family physician to rule out malignancy, premature ovarian failure, or other pathology.

Treatment of Dysfunctional Uterine Bleeding

- Acute cessation of bleeding with IV estrogens (conjugated estrogen 25 mg IV over 15 min q 2-6 hrs prn) +/- progesterone (100 mg IM) followed by supportive estrogen regimen (usually by means of higher estrogen BCP (e.g. Triphasil, Minovral). You may use a slow taper technique: e.g. 4 tabs

for 2 days, then 3 tabs for 2 days, 2 tabs for 2 days, and then 1 tab daily to finish the package.

- Tranexamic acid (Cyklokapron) is also effective for cessation of acute bleeding. It exerts an antifibrinolytic effect through reversible blockade on plasminogen. The drug has no effect on blood coagulation parameters or dysmenorrhea. 30% of patients will experience nausea and leg cramps. Tranexamic acid 500 -1000 mg po q6h for the first four days of the cycle reduces menstrual blood loss by up to 40%, based on 10 randomized placebo-controlled trials.
- NSAIDS decrease menstrual blood loss by 20 to 50 percent by decreasing endometrial prostaglandin levels which are elevated in women with heavy menstrual bleeding. Therapy should start on the first day of menses and be continued for five days, or until cessation of menstruation.
- Other regimens (e.g. Danasol, Progestins, Progesterone impregnated intrauterine devices – IUDs, and GnRH agonists) should be left for the gynecologists. Dilatation and curettage (D&C) may be considered when other methods have failed.

VAGINAL BLEEDING RELATED TO PREGNANCY

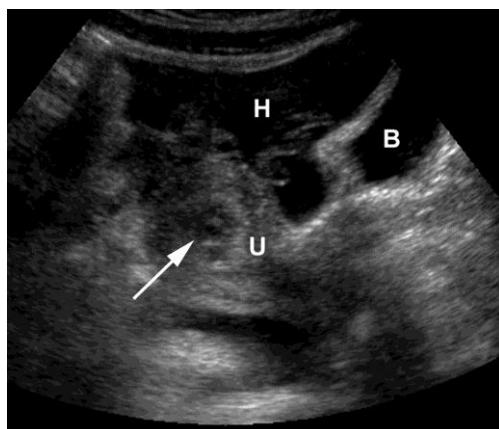
Always exclude ectopic pregnancy during the first 20 weeks of gestation. Always exclude placenta previa or abruptio placentae later in pregnancy.

Early Pregnancy (1st trimester)

Up to 40% of women have cramping and spotting during the first 20 weeks of gestation. Half of these women (20%) undergo first trimester abortion and 60% of those are related to chromosomal abnormalities. Bleeding and expulsion of uterine contents usually starts a week after the death of the fetus.

Traditionally, abortions are classified as threatened, inevitable, missed, complete, incomplete, spontaneous, and induced. In reality, if on pelvic examination, the cervical os is open, it is an inevitable miscarriage. If the cervical os is closed, one does not know until the pelvic or trans-vaginal ultrasound results are available whether it is a threatened or a missed abortion. In many women, especially multigravidae or after cervical surgery (cryotherapy), it is very easy to misinterpret a wide external cervical os as an ‘open cervix’. The use of fenestrated forceps

can help determine the patency of the cervical os. Apply gentle pressure with the forceps, and if they “fall into the uterus”, the cervix is truly open. However, the need for a bimanual pelvic exam under these circumstances is diminishing. In the modern ED, most physicians are certified as independent ED Echo practitioners and are skilled in ED ultrasonography which can identify both the presence of an IUP and intra-abdominal fluid (blood).



Levine D Radiology 2007;245:385-397
Hemoperitoneum in patient with ruptured ectopic pregnancy. Sagittal view of the pelvis shows the uterus (U) surrounded by free fluid and hematoma (H). This amount of blood around the uterus is highly suspicious for a ruptured ectopic pregnancy. B = bladder. Fluid looks black.



Levine D Radiology 2007;245:385-397
Sagittal view of the right flank shows free fluid (FF) adjacent to the kidney consistent with a large amount of hemoperitoneum in a patient with ruptured ectopic pregnancy.

In a pregnant patient with vaginal bleeding, the immediately relevant questions are:

- Is the woman hemodynamically stable?
- Is she febrile? Is it a septic abortion (endometritis)?
- If the cervical os is open, is there any tissue that can be removed to ease the woman’s pain and decrease bleeding?
- Is it an ectopic pregnancy?

- If bleeding is prolonged, are there any retained products?
- Does the woman have a bleeding disorder?
- What is the patient's Rh status? Anti-Rh (D) immune globulin (RhoGAM 300ug) should be offered to Rh negative women within 72 hours after any vaginal bleeding in pregnancy
- What is the patient's psychological reaction to the event?

Inevitable abortions as well as incomplete abortions require pain control and follow-up with serial beta HCG and possibly serial ultrasounds. The current approach to incomplete abortion is more reliant on expectant management rather than on immediate dilatation and curettage. Patients should be instructed to return to the ED if bleeding exceeds 4 pads per hour, if they become lightheaded, or if they become febrile.

Misoprostol is indicated for treatment of incomplete abortion for women with uterine size less than or equal to 12 weeks at presentation (unless large fibroids are present). Women with suspected ectopic pregnancy or hemodynamic instability should not be treated with misoprostol. Use of misoprostol for incomplete abortion has a success rate of 66 -93%. Women eligible for misoprostol, but with an IUD in place, should have the IUD removed before drug administration. Misoprostol is given as 400-800mg intra-vaginal tablets that may be repeated in 12hrs. Close follow up is required. Due to the high success rate, surgical intervention is not recommended prior to 7 days after treatment (unless medically necessary). Make sure you supply women with strong analgesics, as they will have painful cramping, especially if the cervix was closed at presentation.

In women with hyperemesis, vaginal bleeding, large-for-dates uterus, and pregnancy-induced hypertension before 24 weeks gestation, suspect gestational trophoblastic disease. A high beta HCG combined with an abnormal ultrasound is diagnostic.

Ectopic Pregnancy

Approximately 1% of diagnosed pregnancies are ectopic. Risks include PID, IUD, infertility treatment, pelvic surgeries, tubal ligations, and endometritis. Mortality is 1 in 850 women if treated, but nearly 100% if untreated. Median time for rupture of a tubal pregnancy is 8 weeks; however, interstitial (corneal) ectopic pregnancies can rupture later in pregnancy (12-16 weeks) with catastrophic results.

Historical risk factors are absent in 50% of cases. Pain is absent in 10% of cases. Vaginal bleeding is absent in 20% of cases. Shoulder pain may be present (phrenic and liver capsule irritation with blood). The abdomen may be tender, but without signs of peritoneal irritation in 10-15% of cases. The pelvic exam is unreliable to rule out ectopic pregnancy. Beta HCG levels tend to be lower in ectopic pregnancy but are of little help due to poor sensitivity. A progesterone level < 17 ng/mL has 73% specificity and 70% sensitivity for ectopic pregnancy.

The Emergency Physicians performed ultrasound has a pooled sensitivity estimate of 99.3% (95% confidence interval [CI] 96.6% to 100%), with negative predictive value of 99.96% (95% CI 99.6% to 100%), and negative likelihood ratio of 0.08 (95% CI 0.025 to 0.25) on a recent meta-analysis.

Visualization of an intrauterine pregnancy by an emergency physician is generally sufficient to rule out ectopic pregnancy. [Ann Emerg Med. 2010;56:674-683.]

Serum bhcg In Ectopic Pregnancy

- May be positive within 7-10 days of conception
- Doubles approximately every 48 hours in 85% of normal intrauterine pregnancies of 4-6 weeks
- In more than 80% of ectopic pregnancies the rise in serum HCG is <66% in 48 hrs (in about 13% of ectopics, the doubling time is normal - false negatives)
- About 15% of normal pregnancies have subnormal doubling time
- At levels of 500-1500 IU/L, an intrauterine sac should be seen on trans-vaginal ultrasound
- Less than 10% of ectopic pregnancies have a pseudo-gestational sac
- There is a report of 2 patients with beta-hCG levels < 10 IU/L presenting with ruptured ectopic pregnancy and hemoperitoneum(J Reprod Med. 2007 Jun;52(6):541-2).

If there is no prior reliable ultrasound result demonstrating an intrauterine conception as well as the absence of an adnexal mass, an ultrasound needs to be done. An intrauterine conception should be visible with a trans-vaginal ultrasound if the beta HCG is more than 1500 IU/L. Beware of the possibility of co-existing intra- and extra-uterine pregnancies, especially in women undergoing in-vitro fertilization treatments.

There is no single test that rules out ectopic pregnancy. Familiarize yourself with the protocol used in your hospital. If your clinical suspicion is high, obtain consultation.

Management Of Ectopic Pregnancy

Many of the bleeding ectopic pregnancies are treated surgically, often laparoscopically. However, it is becoming more common to treat stable ectopic pregnancies that present with only minor spotting with oral methotrexate (MTX). A single dose of MTX has demonstrated high success rates of 86 to 94%. The optimal candidates for MTX treatment are hemodynamically stable, willing and able to comply with post-treatment follow-up, have a bHCG level ≤ 5000 mIU/mL, and no fetal cardiac activity. Ectopic mass size less than 3 to 4 cm is also commonly used as a patient selection criterion.

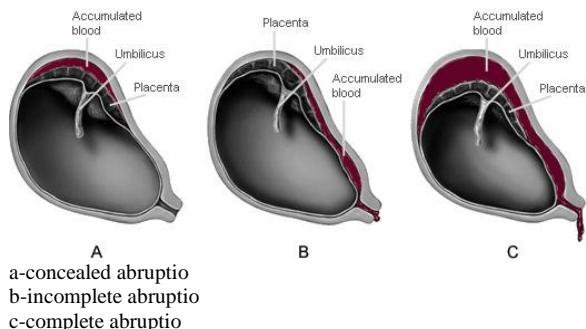
Contraindications: hemodynamic instability, signs of impending or ongoing ectopic mass rupture (i.e. severe or persistent abdominal pain or >300 mL of free peritoneal fluid outside the pelvic cavity), clinically important abnormalities in baseline hematologic, renal, or hepatic laboratory values, immunodeficiency, active pulmonary disease, peptic ulcer disease, hypersensitivity to MTX, coexistent viable intrauterine pregnancy, or breastfeeding.

Relative contraindications: women with a high baseline hCG concentration (greater than 5000 mIU/mL) are more likely to require multiple courses of medical therapy or experience treatment failure. The presence of fetal cardiac activity is significantly associated with treatment failure (OR 9.1, 95% CI 3.8-22.0). Ectopic size ≥ 3.5 cm and presence of free peritoneal fluid on US. Check with your local Gynecology team for a protocol they use.

Late Pregnancy (3rd trimester)

The diagnostic possibilities for vaginal bleeding in late pregnancy range from trivial to life threatening and include placenta previa, abruptio placentae, and uterine rupture.

Abruptio placentae is a premature separation of the normally implanted placenta from the uterus (0.4-3.5% of all deliveries). It is associated with various hypertensive, cardiovascular, and connective tissue autoimmune disorders. Often, it is associated with various degree of trauma. Abruptio placentae classically presents with a tender contracted uterus, signs of hemorrhage (remember that in concealed abruptio, there may not be any external bleeding), and fetal distress. Fetal heart rate can be heard by 16 weeks and normally is 120-160 beats per minute. Abruptio placentae is often misdiagnosed as preterm labor.



Dark shadow within the placenta (indicated by "Xs") is a bleeding within the placenta, resulting in abruptio.

Placenta previa is the result of implantation and placenta development over the internal cervical os. It occurs in 1 in 200 deliveries with a mortality of 0.03%. Risks include multiparity, abnormal uterus, fibroids, and prior surgeries. Women older than 30 years are 3 times more likely to have placenta previa than women younger than 20 years. Classically, placenta previa presents with painless bleeding, often preceded by trauma or intercourse. Do not attempt a pelvic examination because it may worsen the hemorrhage in placenta previa. Any patient presenting with vaginal bleeding after the first trimester should have an ultrasound prior to any pelvic exam.



Placenta previa(plc) covers internal os(os) on a sagittal trans-abdominal ultrasound.

Uterine rupture occurs in 0.05% of pregnancies. Maternal mortality is 8% while fetal mortality is 50%. It is usually seen during labor, so you may encounter it in the ED when a patient presents following an attempted home delivery.

Non-life-threatening causes of third trimester bleeding include ‘bloody show’, cervical traumatic hemorrhage, and marginal sinus rupture.

Postpartum Hemorrhage

Postpartum hemorrhage is seen in varying degrees in 28% of patients.

Causes of Postpartum Hemorrhage:

- Uterine atony
- Uterine rupture
- Lacerations/tears
- Retained placental tissue
- Uterine inversion
- Coagulopathy

Careful assessment of uterine tone and the birth passage is necessary. If atony is present, Oxytocin 30 units in 1 litre of normal saline at 200 cc/hr can be helpful. Ultrasound will assist in ruling out retained products. Make sure that the patient is afebrile. Postpartum endometritis may either complicate bleeding or in fact be the contributing factor.

DISPOSITION

In severe vaginal bleeding and in all unstable patients, notify the specialty services promptly and get them involved early. Most patients in late pregnancy are assessed in the Labor and Delivery unit directly and bypass the Emergency Department. If

they are triaged, ensure they are stable before they are sent upstairs.

Stable patients with suspected ectopic pregnancies ideally should not be sent home without an ultrasound assessment.

Patients Discharged from the Emergency Department

- Instruct them to return if bleeding, pain, or other symptoms get worse. Lightheadedness and pre-syncope should be explicitly described and explained.
- For patients who are returning for ultrasound to rule out an ectopic pregnancy, consider clear fluids for 24 hours in case they require surgery.
- Ensure appropriate and timely follow-up (reassessment in 24 hours).
- Remember to support the patient and her family psychologically during these challenging events. Get social workers involved if possible.

SUMMARY

- Take any vaginal bleeding seriously. It is easy to miss a serious condition in the midst of ‘same again’ miscarriages and dysfunctional uterine bleedings. Broaden your differential diagnosis.
- Always check vital signs.
- You won’t be faulted for doing a pregnancy test, but you might be for not doing it.
- Imaging should not delay definitive management. Unstable patients should not be sent for imaging.
- Try to give patients written discharge instructions. Make sure there is a follow up plan.

CLINICAL PEARLS

1. Every woman is suspected to be pregnant
2. Pelvic examination requires a chaperone being present
3. The beta HCG level is irrelevant if you suspect ectopic pregnancy
4. Externally bleeding abruptio placentae and placenta previa are often clinically indistinguishable.
5. Try to do a bedside ultrasound before you do a bimanual exam
6. Consider a diagnosis of placental abruption for every patient in premature labour.
7. Absence of vaginal bleeding does not exclude placental abruption.
8. Normal ultrasound findings do not exclude placental abruption.

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29: THE PREGNANT PATIENT IN THE ED

Shauna Martiniuk

OBJECTIVES

1. To identify unique concerns in pregnant patients treated for other medical conditions
2. To understand the differences in radiation exposure for different modalities
3. To review the work-up of pregnant patients with suspected thromboembolic disease
4. To understand the specific complications related to IVF treatment
5. To identify the patient with a hypertensive emergency
6. To review the management of major trauma in the pregnant patient

INTRODUCTION

Emergencies related directly to pregnancy such as abortion, placenta previa, or postpartum hemorrhage are covered elsewhere in this manual. This chapter will cover imaging issues, medical conditions, IVF emergencies, and trauma in the pregnant patient presenting to the Emergency Department.

PHYSIOLOGY OF PREGNANCY

- Increased WBC with a left shift
- Increased coagulation factors and increased risk of thromboembolism
- Increased cardiac output by 30-40%
- Systolic murmur of pregnancy
- ST wave changes on ECG
- No change in peak flow rates
- Increased GFR with decreasing normal creatinine levels
- Cholestasis

DIAGNOSTIC IMAGING

Diagnostic imaging studies are often required to evaluate conditions unrelated to pregnancy. Ultrasound and MRI are the preferred imaging tests in pregnancy although contrast agents can still confer some risks. Side effects of strong magnetic fields on the fetus are unknown. Nuclear medicine studies confer risk to the fetus as certain substances will concentrate in specific tissues and the fetus will be

exposed to substances that are then excreted from the maternal bladder.

Despite risks to the fetus, imaging studies must still be performed if required for the health of the mother. The estimated incidence of childhood malignancy after in utero radiation exposure is about one in 16,000 per mGy (1Gy=100 rads)

Estimated Fetal Radiation Doses during Radiodiagnostic Procedures

Examination	Fetal Dose (mrad)
CXR	<1
Dental XR	<1
Abdominal XR	250
Hip and femur XR	300
Lumbar spine XR	400
Head CT	<10
Chest CT	16
Abdomen CT	3000
Ventilation lung scan	1-19
Perfusion lung scan	6-12
Pulmonary Angiography	<50

- Background fetal radiation exposure during pregnancy is 100mrad

Radiation Risks to Fetus

<5,000 mrad	minimal risk
>10,000 mrad	minor IQ reduction increased childhood cancer
10,000-20,000mrad	malformations

DVT AND PULMONARY EMBOLISM (PE)

- The risk of PE is 15 times higher postpartum than in pregnancy
- The relative risk of thromboembolism among pregnant or postpartum women is 4.29
- Undiagnosed PE has a mortality of up to 30%. This risk decreases to 2-8% if diagnosed and treated
- Physiologic changes in pregnancy can mimic the signs and symptoms of PE, for example chest pain and shortness of breath
- D-dimer increases in uncomplicated healthy pregnancies and therefore has limited use in the work up of a pregnant patient in whom you suspect thromboembolic disease

- Diagnosis of DVT made using doppler ultrasound

Diagnosis

For acute, seriously ill patients, portable echocardiogram is the modality of choice (to look for a dilated and hypokinetic right ventricle amongst other findings).

For all other patients, a chest x-ray should be done to rule out infection or pneumothorax. A doppler ultrasound should be performed as it confers no risk to the fetus. If the doppler ultrasound is positive for DVT and there is a clinical suspicion of PE, then a presumptive diagnosis of PE can be made and no further testing is required. If the Doppler and CXR are negative and the patient has no lung disease, a half-dose VQ scan should be performed. The patient should be hydrated well and empty the bladder frequently to clear radionuclides from the bladder. If the patient has prior lung disease or an abnormal CXR, then CT pulmonary angiogram should be done. Overall, a VQ scan is more accurate in pregnancy and delivers less radiation to the maternal breast and lung as compared to a pulmonary CTA.

Discuss imaging options with your radiologist. Informed consent should be obtained from the patient.

Treatment

- Heparin – usually low molecular weight SC
- warfarin is a teratogen
- Life-threatening PE (persistent and severe hypotension) should still receive thrombolytic therapy

CLINICAL PEARL

For acute, seriously ill pregnant patients in whom you suspect pulmonary embolism, portable echocardiogram is the imaging modality of choice

PHYSICAL ABUSE DURING PREGNANCY

The rates of physical abuse during pregnancy have been reported to be between 5.7 and 6.6%. Physical abuse is associated with adverse maternal and fetal outcomes. Of women physically abused, 18% suffered a miscarriage or other internal injury as a result. The most commonly injured area is the abdomen. Severe blunt trauma to the abdomen could result in spontaneous abortion, fetal death, placental abruption, preterm labour and delivery, and fetal injuries. Risk factors for abuse include prior abuse, low income, unmarried, alcohol abuse in the partner,

and unplanned pregnancy. It is important to consider the possibility of abuse when evaluating any pregnant patient in the emergency department, especially for those patients receiving little or no prenatal care.

MEDICATION USE IN PREGNANCY

Vigilance must be maintained when prescribing medications to a pregnant patient. Both the type of medication and the term of the pregnancy must be taken into consideration, as well as the risks and benefits to both parties. Useful resources include Motherisk and drug databases. Herbal medications are not without risk, and their safety should be assessed before use.

IVF EMERGENCIES

Ectopic and Heterotopic Pregnancies

- Ectopic risk is 1.9% in the general population
- IVF ectopic risk is 2.1%

Heterotopic Pregnancies

- Defined as the coexistence of two pregnancies at two different implantation sites (usually an intrauterine and extra-uterine gestation)
- 1% incidence after IVF
- 40-84% are diagnosed on transvaginal ultrasound on initial presentation
- Be aware of this possibility when evaluating anyone receiving IVF

Ovarian Hyperstimulation Syndrome (OHSS)

- Encountered in patients undergoing controlled ovarian hyperstimulation cycles (patients usually receiving exogenous gonadotropins)
- Pathophysiology – increased capillary permeability with depletion of the intravascular volume and third space accumulation (abdomen, pleural space, pericardial space)

SIGNS / SYMPTOMS ACCORDING TO SEVERITY

- Mild – abdominal distension/discomfort, nausea and vomiting and/or diarrhea, ovaries enlarged 5-12 cm
- Moderate – abdominal ascites on ultrasound
- Severe – clinical evidence of ascites, hydrothorax or difficulty in breathing, hemoconcentration, hypercoagulability, thromboembolic phenomenon, decreased renal perfusion and function, ↓Na, ↑K

HISTORY

- Presence of risk factors, abdominal pain and bloating, increased abdominal girth, early satiety, shortness of breath, chest pain, nausea and vomiting, weight gain, decreased urine output

PHYSICAL EXAMINATION

- Signs of hypotension, hemodynamic instability, dehydration
- Pleural effusions, congestive heart failure
- Abdominal ascites, peritoneal signs
- Pitting edema
- Do not do a pelvic examination as enlarged ovaries can be fragile

INVESTIGATIONS

- All vitals including oxygen saturation
- If the patient has mild abdominal bloating or discomfort that is not accompanied by vomiting or diarrhea or other signs of advanced disease, no investigations are required
- If the patient describes pain, has nausea or any other symptoms of more advanced disease, investigations should be done
- CBC, INR, PTT, electrolytes, liver function tests, creatinine, urea
- CXR only if symptomatic
- Transvaginal ultrasound to view ascites and the size of ovaries

TREATMENT

- Self-resolving condition
- Correct and maintain intravascular volume - no diuretics. Usually 1-2L of NS in 1st hour to restore tissue perfusion
- Support renal and respiratory function. May need to drain hydrothorax and ascites
- Prevent thrombotic events – may need LMWH
- Laparotomy for ovarian torsion/rupture/internal hemorrhage

ADMISSION CRITERIA

- Not tolerating oral food or liquid
- Hemodynamic instability
- Respiratory compromise
- Peritoneal signs (possible ovarian torsion, hemorrhage, rupture of enlarged ovarian cysts, abscess)
- Tense ascites
- Hemoconcentration, leukocytosis, sodium <135 mEq/L, potassium > 5.0 mEq/L, abnormal liver or renal function tests
- Decreased oxygen saturation

OUTPATIENT MANAGEMENT

- Limit activity (no sexual intercourse or impact-type activities), but not bedrest due to increased risk of thromboembolic events
- Weigh daily, if >2lbs/d increase, see MD
- Monitor fluid intake (at least 1L/d of mostly electrolyte balanced fluid) and output
- Daily follow-up (call or visit)
- See MD if worse
- Oral analgesics, antiemetics

HYPERTENSIVE EMERGENCIES

- 16% of maternal deaths in developed countries are from pregnancy related hypertension (HTN)
- Edema is no longer used in the definition of eclampsia as it occurs in too many women to be discriminant
- Proteinuria fluctuates over 24 hours, therefore it may not be picked up randomly. Diagnosis is in a 24 hour urine specimen.

Gestational Hypertension

- Systolic BP >140mmHg or diastolic BP >90 developing after the 20th week of gestation
- No proteinuria
- BP returns to normal <12 weeks postpartum
- Final diagnosis made only postpartum

Preeclampsia

- 5% incidence in pregnancy
- Systolic BP >140mmHg or diastolic BP >90 after 20 weeks gestation
- Proteinuria >300mg/24hr (>1+ dipstick is suggestive)
- May also have: increased creatinine, low platelets, microangiopathic hemolysis, increased LDH, raised ALT or AST, persistent headache or other cerebral/visual disturbances, persistent epigastric pain, oliguria

Eclampsia

- Grand mal seizures that cannot be attributed to other causes in women with preeclampsia
- Unrelenting severe headaches or visual disturbances are ominous signs
- 10% of seizures will develop before the onset of proteinuria

Chronic Hypertension

- BP>140/90 before pregnancy or diagnosed before 20 weeks gestation OR

- Diagnosed after 20 weeks and lasts beyond 12 weeks postpartum

Superimposed Preeclampsia (on chronic hypertension)

- New onset proteinuria >300mg/24 hr in hypertensive women without proteinuria <20 weeks
- A sudden increase in proteinuria or BP or low platelets in women with hypertension and proteinuria before 20 weeks gestation

HELLP Syndrome

- Hemolysis, elevated liver enzymes, low platelets
- Found in 20% of women with severe preeclampsia or eclampsia
- Decreased organ perfusion secondary to vasospasm and endothelial activation in pre-eclamptics
- Increased perinatal mortality and morbidity is caused by large placental infarcts, small placental size, abruptio placentae
- Epigastric or right upper quadrant pain is likely from hepatocellular necrosis, ischemia and edema
- Thrombocytopenia results from platelet activation, aggregation and microangiopathic hemolysis caused by severe vasospasm
- May also result in bleeding, pulmonary edema, renal insufficiency, infection, CNS morbidity
- Treat by delivery of fetus if >34 weeks or if severe maternal disease

Risk factors

Preeclampsia - multiple pregnancy, history of chronic hypertension, age > 35 years, obesity, African-American ethnicity, possibly nulliparity

Eclampsia - most commonly occurs in the third trimester, but can also occur intra and postpartum. Be vigilant in detecting preeclampsia in postpartum patients as 5-17% of eclampsia occurs in the 48 hour to 4 week postpartum period.

Management

PREECLAMPSIA

- Admit new hypertensives if persistent/worsening BP or development of proteinuria, order CBC, creatinine, liver enzymes, proteinuria measurements, daily weights
- Decrease physical activity
- Delivery is the cure since most cases persist until after delivery
- Antihypertensive medications for early mild preeclampsia do not show improved outcomes, and some medications have resulted in growth-restricted infants

SEVERE PREECLAMPSIA AND ECLAMPSIA

- Consider a differential diagnosis including epilepsy, encephalitis, meningitis, cerebral tumor, cysticercosis, ruptured cerebral aneurysm
- Deliver fetus if >32 weeks gestation, may consider medical management in select patients at <32 weeks
- Treat severe preeclampsia with IV magnesium sulfate (4-6g loading then 2g/hour to maintain levels 4.8-8.4 mg/dL) to prevent seizures. Also used to prevent recurrent seizures.
- Watch for magnesium toxicity with decreased patellar reflexes, respiratory depression, respiratory paralysis and arrest
- If toxic, reverse effects with calcium gluconate 1g IV and hold magnesium sulfate
- Antihypertensives: **Hydralazine** 5mg doses in 15 minute intervals when diastolic BP >105mmHg or systolic >160 until diastolic 90-100 mmHg or IV **Labetolol** 20 mg IV followed by repeat doses every 10 minutes, maximum dose 300mg.
- Fluid: 60-125 cc/hr maximum even if oliguric to avoid pulmonary and cerebral edema.

TRAUMA

Physiologic Changes in Pregnancy

- Decreased gastric motility, increased gastric reflux
- Increased maternal blood volume and cardiac output - blood loss of 30% before a patient becomes hypotensive
- Increased respiratory rate and oxygen consumption
- Enlarged uterus – needs to be in left lateral decubitus position to decrease inferior vena cava compression

Etiology

- MVA (55-70%)
- Assaults (11-21%)
- Falls (9-22%)

Physical and sexual abuse occurs in 17-32% of pregnancies. Look for multiple injuries at different stages of healing, inconsistent explanations of injuries, delays in seeking health care.

Blunt Abdominal Trauma

- **Placental abruption, uterine rupture, fetal death or distress, preterm labour**
- Abruptio incidence is up to 40% in severe blunt abdominal trauma

Penetrating Abdominal Trauma

- Gravid uterus is protective of other abdominal contents, but there is a high rate of fetal demise
- Low threshold for exploratory laparotomy after penetrating trauma

Management

- Maternal resuscitation is always the first priority; fetal outcome is directly related to maternal outcome
- ABC's - oxygen, IV, monitor
- Place mother in **left lateral decubitus** position
- Monitor baby and mom - blood loss affects baby first and may be the first sign of major injury
- Check skin colour, temperature, urine output, mental status,
- Displace the gravid uterus for CPR, initiate caesarean section within 4 minutes of maternal arrest if fetus over 24 weeks.
- IV fluids first, vasopressors if needed, but will decrease oxygen delivery to fetus by decreasing blood flow
- Tocolytic therapy is controversial
- Secondary survey - pelvic exam for trauma and cervix changes; check for amniotic fluid with nitrazine paper (blue, pH over 7).
- Fetal monitoring - doppler ultrasound and cardiotocographic monitoring (monitor for a minimum of 4 hours if over 20 weeks gestation to rule out abruption)
- Monitor for >24 hours if regular contractions, bleeding, abnormal fetal heart tracing, abdominal pain, low platelets/fibrinogen
- Give Rh-IG to Rh-negative mothers with trauma

CYSTITIS & PYELONEPHRITIS

- Urinary tract infections are common complications of pregnancy. Upper tract infections in particular may lead to significant morbidity for mother and fetus – preterm labour, transient renal failure, ARDS, hematologic abnormalities, sepsis, and shock.
- Bacteriuria is an important risk factor for pyelonephritis in pregnant women. It should be screened for in the first trimester.
- Treat asymptomatic bacteriuria with a 3-day course of antimicrobials as it is associated with preterm delivery and low birth weight.
- Treat cystitis for 7 to 10 days (usually a cephalosporin such as cephalexin or nitrofurantoin). Amoxicillin is safe but has high resistance.

- Follow-up with a urine culture one week after therapy for asymptomatic patients or those with cystitis as one third will experience a recurrence.
- Pyelonephritis requires admission and IV antibiotics

ASTHMA

- Asthma worsens in pregnancy in one third of patients, one third get better, and one third stay the same
- Treat patients with significant symptoms – also monitor fetus if over 24wks
- Deliver fetus if pO_2 below 60mmHg and the mother is intubated
- Patients may have oral steroids if required to treat disease, consult with OB. Side effects include preterm birth, low birth weight
- Otherwise, treat the same as a nonpregnant patient, including inhaled corticosteroids. Early evidence with montelukast is reassuring; larger studies still required to verify the safety of other leukotriene modifiers.

PNEUMONIA

- Preterm labour has been reported
- Consult OB before any discharge home, monitor fetus
- Treat with antibiotics, maintain oxygenation >95%

HYPEREMESIS GRAVIDARUM

- Vomiting is intractable, associated with dehydration, lab abnormalities (hypokalemia, increased LFTs), weight loss, mild hyperthyroidism
- Check CBC, electrolytes, renal function, urine, ketones, LFTs, TSH, serum free T4
- Ultrasound to rule out a multiple gestation, trophoblastic disease
- Treatment: rehydrate, IV medications (dimenhydrinate), electrolyte balance – often need up to 5 litres IV fluid

POSTPARTUM ENDOMETRITIS

- Polymicrobial infection
- Caesarean section is the most important risk factor, along with prolonged labour, prolonged rupture of membranes, internal monitoring,

maternal diabetes, bacterial vaginosis, colonization with Group B streptococcus

Diagnosis

- Fever, uterine tenderness, foul lochia, chills, lower abdominal pain, headache, malaise, sepsis
- Usually occurs in the first week after delivery but may occur up to six weeks postpartum
- Lab: CBC, vaginal C&S, cervical swabs for GC & Chlamydia, blood C&S
- No characteristic ultrasound findings

Treatment

- Mild cases may be treated as an outpatient with oral amoxicillin-clavulanate for 7 days
- Broad spectrum antibiotics (e.g. IV clindamycin plus gentamicin) and admission for more severe cases

SUMMARY

- Ultrasound and MRI are the preferred imaging tests in pregnancy, although contrast agents can still confer risks
- Radiation effects of CT scanning to the fetus as well as the mother must be weighed against the likelihood and seriousness of the disease in question
- Pregnancy is a major risk factor for thromboembolic disease. Doppler ultrasound is used to work up suspected DVT. It can also be used as the initial test in PE.
- Physical abuse is common in pregnancy with the majority involving the abdomen
- Vigilance must be maintained when prescribing medications to a pregnant patient
- Pregnant patients having undergone IVF are at higher risk than the general population for heterotopic and ectopic pregnancies
- Ovarian Hyperstimulation Syndrome (OHSS) is encountered in patients undergoing controlled ovarian hyperstimulation cycles, and presents with abdominal discomfort/distension and can progress to hydrothorax, hemoconcentration, hypercoagulability and decreased renal function
- Severe pre-eclampsia and eclampsia are treated with large dose IV Magnesium as well as antihypertensives (Hydralazine and/or Labetolol)

- When managing pregnant patients with major trauma, maternal resuscitation is always the first priority; fetal outcome is directly related to maternal outcome
- Placental abruption, uterine rupture, fetal distress and preterm labour are the major concerns in pregnant patients with blunt abdominal trauma

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30: UROLOGICAL EMERGENCIES

Shirley Lee
updated by Natalie Wolpert

OBJECTIVES

1. To describe the causes and appropriate management of acute hematuria.
2. To describe the diagnosis and management of urinary tract infection
3. To describe the diagnosis and management of testicular torsion
4. To appropriately manage urinary retention

INTRODUCTION

Urologic emergencies are a common presentation to the Emergency Department (ED). Some urologic emergencies require urgent diagnosis and intervention and are potentially life-threatening if the diagnosis is missed. The goal of this chapter is to outline an approach to common urologic conditions that can present to the ED. Nephrolithiasis will be covered in the ‘Renal Colic’ chapter.

ACUTE HEMATURIA

The initial approach to hematuria in the ED is to differentiate it into painful and painless causes.

Painful Causes

- Obstruction
- Ureteral or bladder stone
- Urinary Tract Infection (UTI)
- Trauma
 - Iatrogenic (e.g. foley)
 - Urethral trauma (may be seen in trauma with pelvic fracture)

Painless Causes

- **Urinary tract cancer**
 - Renal
 - Ureter
 - Bladder
- Anticoagulation
- Benign Prostatic Hypertrophy

Note that *painless gross hematuria is bladder cancer until proven otherwise*. It is essential that the ED management of these patients include urgent Urology follow up for cystoscopy.

Initially assess the ABCs. Hypotension and tachycardia can indicate early hypovolemic shock. This may be secondary to blood loss (from prolonged or severe hematuria) or urosepsis in the setting of a

UTI. Lab investigations should include: Urine R&M +/- C&S, CBC + diff, group & screen (if there is evidence of significant acute blood loss), renal function, and electrolytes. Diagnostic imaging may be required depending on the clinical scenario.

Continuous Bladder Irrigation (CBI)

CBI is used in the ED to irrigate blood clots in the bladder. It is important to use a large catheter (20-24 French), as smaller catheters are inadequate for clot irrigation. The first choice catheter is a Silastic with coude tip.

As the urine clears, the rate of the CBI flow can be slowed down, with the irrigation stopped if the catheter has been clear for 1 hour. Remember to re-check the vital signs in the patient, as they are at risk for hypovolemia, and to repeat their hemoglobin if they have had a significant amount of bleeding.

Management of Clots

Catheter Not Clearing

The most common pitfall is due to the catheter remaining bloody and not clearing. This is usually due to active bleeding or residual clots in the bladder. In this situation, irrigation of the bladder is needed to remove the clots. This is done at the bedside using a large Toomey syringe and a catheter irrigation tray.

Catheter Blocked

Irrigation of the catheter may be required if it appears to be obstructed by clot.

If CBI is ineffective, Urology consultation should be considered.

URINARY TRACT INFECTION & PYELONEPHRITIS

Urinary tract infections and pyelonephritis are clinical diagnoses (with overlap in spectrum between the two). Classic presentations are:

- UTI – frequency, urgency, internal dysuria, suprapubic pain, hematuria
- Pyelonephritis – fever, chills, flank/back pain, nausea/vomiting, urinary symptoms, CVA tenderness

The probability of a UTI in a woman with typical symptoms is 50%. The absence of symptoms

suggesting vaginitis or cervicitis (vaginal irritation, bleeding, discharge) raises the probability to 90%. However, the presence of such symptoms reduces the likelihood to 30% and should mandate further investigations to rule out sexually transmitted infections/PID.

Note that **elderly patients can have atypical presentations** of UTI/pyelonephritis. They do not always have a fever and may present with; altered mental status or delirium, non-specific complaints such as vomiting or abdominal pain, or in extreme cases, SIRS, sepsis, or septic shock.

Urinary Tract Infection Investigations

The urine dipstick is used to detect leukocyte esterase (neutrophil enzyme) and nitrite (which is sensitive for Enterobacteriaceae only and NOT for staphylococcus, enterococcus, or pseudomonas infections). The urine dip is only 75% specific and 82% sensitive. Urine microscopy (R&M) and gram stain (C&S) can increase the detection of UTI to >90%. Culture is not necessary to make the diagnosis in patients with uncomplicated UTI; a positive dipstick or microscopy, combined with suggestive clinical symptoms, is adequate. Further investigations of UTI in the following patient groups should be considered: children <5 yr old, males, recurrent UTI, and fever > 3 days.

Lower UTI

Common pathogens include: E.coli (75-90%), staphylococcus saprophyticus (5-15%), and less commonly proteus, klebsiella, enterobacter, pseudomonas, and enterococcus.

Treatment

A minimum of 3 days of antibiotics works in uncomplicated UTI. This decreases side effects from the antibiotic and enhances cure rates. Specific antibiotic choices should follow local resistance patterns. Current recommended first line agents include TMP-SMX DS PO BID for 3 days (if resistance rates <20%) or Nitrofurantoin 100mg PO BID for 5 days. Fluoroquinolones should be reserved for more complex cases or upper tract infections due to increasing resistance patterns. It is important to note that Cephalosporins (except Cefixime), B-lactams, and nitrofurantoin are not effective for short-course therapy. Cephalosporins, (in particular 3rd generation) may be an option but are not considered first line. Longer duration treatment (7 days) is recommended for children, pregnant women (where nitrofurantoin or cephalosporins are first line), and patients with comorbid neurologic or anatomic abnormalities. If it is a complicated UTI, consider a

parenteral first dose of antibiotics, followed by 10-14 days of oral therapy to eradicate bacteriuria.

Pyelonephritis

Treatment options include: antibiotics (IV or oral), anti-pyretics for fever, and IV fluids, and anti-emetics for vomiting/dehydration. Analgesia may be required. Certain patients may be managed as outpatients provided the following criteria are met:

- Normal vital signs
- Normal renal function
- No evidence of GU tract obstruction
- Adequate pain control
- Adequate hydration
- Able to tolerate oral antibiotic
- Patient reliable for follow-up

Antibiotic choices include ciprofloxacin for 7-10d, TMP-SMX for 14 d (these choices are appropriate only if low resistance patterns). An IV dose of Ceftriaxone is recommended if high fluoroquinolone resistance rates are present.

Investigations

Urine R&M, C&S, CBC + diff, renal function and electrolytes, and consider blood culture in complex or septic patients.

Diagnostic Imaging

Consider if suspect stone or obstruction, or if the diagnosis is unclear.

TESTICULAR TORSION

Testicular torsion is a classic cause of medico-legal malpractice. If a male patient presents with abdominal pain, always ask if it is radiating to their testicle (as they may be too embarrassed to mention it at triage). The **GU exam** is a **mandatory** part of the physical exam in a male patient presenting with abdominal pain. Torsion results from twisting of the spermatic chord, resulting in a lack of blood supply to the testicle and subsequent ischemia. Patients are typically under 25 years of age, but torsion can occur at any age.

Classically, torsion presents with sudden onset of severe pain in the testicle, which may radiate to the abdomen and flank. The typical signs are a swollen, exquisitely tender testicle with a horizontal lie and an absent cremasteric reflex. However, classic signs may not always be present. Torsion is a urological emergency, as '*time is testes*'. Because torsion effectively causes acute ischemia of the testicle, it requires urgent intervention for testes viability **within 6 hours** from time of pain onset. Although ultrasound

is the diagnostic test of choice, if you are suspicious of testicular torsion, consult Urology immediately. Do not order the ultrasound first, as this may cause significant delay to surgery beyond the window of testicular viability. If the urologist agrees with your assessment, they will immediately take the patient to the operating room to detort the testicle and perform a bilateral orchiopexy to prevent future torsion from occurring.

URINARY RETENTION

Urinary retention is the inability to pass urine due to an obstruction of flow. The patient with urinary retention often presents to the ED with severe suprapubic pain. They may have impressive distention of their lower abdomen on physical examination due to a massively enlarged bladder.

Female

Retention is frequently associated with infection in female patients, therefore a urine R&M should be sent. Urinary catheterization can be challenging in a large patient, so always get assistance. As well, remember to place a urine basin under the pelvis before you insert the foley, as there can be a very large urinary volume output once the catheter is inserted.

Male

The usual cause is benign prostatic hypertrophy (BPH). Catheterization may be difficult, especially in patients over 60 years old. Do not use a smaller catheter than 16 French. Always document total drainage (ml) accurately, as this helps determine the treatment plan for the patient by the urologist in follow-up. Again, send the urine for R&M +/- C&S. The patient should be started on an alpha-antagonist (e.g. Flomax CR 0.4 mg OD). The foley should be removed for trial of void in one weeks follow-up.

Post Urinary Retention Care

Monitor the urine output post retention every 2 hours, as the patient can develop post obstructive diuresis. This is defined as >200 cc/hr urine output. Post obstructive diuresis may result in dehydration and hypokalemia. The causes are both physiological (fluid retention post-op), and/or pathological (renal failure preventing the patient from concentrating their urine).

If the urine output is >400 cc after 2 hours, the patient requires a minimum maintenance IV of normal saline at 50 cc/hr. The following blood work should be drawn: electrolytes, creatinine, BUN every 6 hrs, then every 12 hrs x 2. The main goals of treatment are to replace half the fluid loss with a 0.45% normal saline

bolus, increase the maintenance IV as needed, and let the patient orally hydrate as much as possible.

SUMMARY

- Differentiate hematuria in the ED into painless and painful causes
- Painless hematuria is bladder cancer until proven otherwise, ensure appropriate Urology follow-up
- CBI is used in the ED to irrigate blood clots in the bladder
- Urinary tract infections and pyelonephritis are clinical diagnoses
- Testicular torsion classically presents in a young patient with severe onset of testicular pain, swelling, a horizontal lie, and an absent cremasteric reflex
- Testicular torsion is a surgical emergency and necessitates immediate Urologic consultation
- Urinary retention in men is most commonly caused by benign prostatic hypertrophy (BPH), and is treated with insertion of a urinary catheter and initiation of an alpha antagonist
- Post urinary retention care involves monitoring the urine output in order to detect post obstructive diuresis, which results in dehydration and hypokalemia

CLINICAL PEARLS

Examination of the scrotum is a mandatory part of the abdominal examination in males.
Testicular torsion is a surgical emergency and requires urgent Urology consultation. Diagnostic evaluation must not delay this.

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31: RENAL COLIC

Peter Glazer

OBJECTIVES

1. To recognize the patient with renal colic
2. To know the appropriate lab studies and imaging studies for renal colic
3. To develop a differential diagnosis and understand when further investigations are required
4. To review the treatment of renal colic
5. To know which patients with renal colic require consultation and admission
6. To understand how size and location of the calculus predicts prognosis

INTRODUCTION

Renal colic refers to the severe pain caused by the impaction or passage of a calculus (stone) in the ureter. It is amongst the most painful conditions a patient will ever endure. Unfortunately, it is a common condition with a lifetime incidence of approximately 8%. Age is an important determinant in making the diagnosis of nephrolithiasis, which most commonly occurs between the ages of 20 and 50. It is also three times more common in males. Initial ED management is focused on providing adequate pain relief and excluding other serious diagnoses.

PATHOPHYSIOLOGY

Most stones arise in the kidney when urine becomes supersaturated with a salt that is capable of forming solid crystals. Calculi in the kidney are generally asymptomatic. Symptoms arise when a calculus becomes impacted in the ureter as it passes towards the bladder. Once in the bladder, symptoms subside almost immediately.

CLINICAL PRESENTATION

History

Renal colic typically presents as sudden onset of unilateral flank pain that radiates anteriorly and inferiorly toward the groin. Patients are commonly described as writhing in pain, and are often unable to find a comfortable position to relieve their symptoms. This is an important feature, as with most other

serious abdominal emergencies, patients prefer to minimize movement and lie still. Patients with renal colic often have nausea and/or vomiting.

Physical Exam

The physical exam is particularly important, as it is helpful in making the diagnosis of renal colic and excluding potential catastrophes that mimic renal colic. In renal colic, the vital signs are typically normal or there may be mild tachycardia or hypertension due to pain. Patients are generally afebrile. The presence of a fever can suggest an infected ureteral stone (a urological emergency), or an alternate diagnosis such as pyelonephritis, appendicitis, or diverticulitis. The main and often only physical exam finding in renal colic is unilateral CVA tenderness. In general, there should not be any significant anterior abdominal tenderness. The presence of anterior abdominal tenderness and especially peritoneal findings should lead one to consider other diagnoses. The presence of a pulsatile mass, abdominal bruit, or diminished/absent pulses in the distal extremities suggests abdominal aortic aneurysm (AAA).

CLINICAL PEARL

The main and often only physical exam finding in renal colic is unilateral CVA tenderness. In general, there should not be any significant anterior abdominal tenderness. The presence of anterior abdominal tenderness and especially peritoneal findings should lead one to consider other diagnoses.

DIFFERENTIAL DIAGNOSIS

There is a long list of diagnoses that can mimic renal colic. Some of the most important ones to consider when the diagnosis is in doubt include: AAA, aortic dissection, renal artery thrombosis, appendicitis, and pyelonephritis.

Renal colic is unusual in the elderly. Patients over the age of 50 that present with presumed renal colic need a thorough history and physical exam to rule out life threatening emergencies such as ruptured AAA. The elderly should usually be imaged as renal colic is rare in this age group and life-threatening emergencies are

more common. When an AAA is missed, it is most commonly misdiagnosed as renal colic.

INVESTIGATIONS

Lab Studies

The most important and cost effective test in presumed renal colic is a **urinalysis**. Classically, the urine dipstick in patients with renal colic reveals microscopic hematuria, no white blood cells, and no nitrites. The presence of hematuria on urine dipstick is approximately 90% sensitive in detecting nephrolithiasis. The leukocyte esterase and nitrites should be negative. A urine dipstick positive for leukocyte esterase and/or nitrates is suggestive of infection. Likely diagnoses can include pyelonephritis or an obstructed infected stone, which is a urologic emergency that requires urgent decompression.

A urine or serum beta HCG test should be performed on all women of reproductive age. A positive result suggests the diagnosis of ectopic pregnancy.

To image or not to image?

There is some controversy regarding which patients to image when the presumed diagnosis is renal colic. A non-contrast CT scan for renal colic exposes the patient to a significant amount of radiation and not every patient presenting with renal colic needs imaging. In general, patients who present with a presumed first presentation of renal colic should be imaged. There should also be a low threshold to image elderly patients. In addition, whenever the diagnosis is in doubt, imaging is indicated. On the other hand, a patient with a previous history of renal colic (confirmed with imaging or stone analysis) whose history, physical, and urinalysis all suggest renal colic and whose symptoms resolve in the ED with appropriate analgesia, does not need emergent ED imaging. Appropriate follow-up should be ensured.

Imaging Modalities

In determining the imaging modality of choice, consideration must be given to the tests' accuracy in identifying ureteral stones, the presence of obstruction, as well as the tests' ability to identify diseases that mimic renal colic. In addition, consideration must be given to the side effects of each test. Five radiographic modalities can be used: CT, ultrasound, IVP, MR, and a plain abdominal x-ray (KUB).

CT

In most centres, a non-contrast helical CT from the top of the kidneys to the bottom of the bladder is the preferred imaging modality. CT is highly sensitive and specific for the detection of renal stones. It is also helpful in determining signs of ureteral obstruction such as ureteral dilatation, perinephric stranding, dilatation of the collecting system, and renal enlargement. The additional advantage is its superior ability to detect mimickers of renal colic. However, since contrast is not used, it can miss some cases of AAA and appendicitis and will miss most cases of renal infarction or dissection.

The main risk of CT is exposure to a significant amount of ionizing radiation. It should therefore be used judiciously in young patients, and especially in patients with multiple previous presentations of renal colic that have had prior CT studies. It should generally be avoided in pregnancy.

Ultrasound

In contrast to CT, ultrasound has no known side effects. However, it has only modest sensitivity for detecting ureteral stones. Ultrasound is most helpful in diagnosing stones in the proximal and distal ureter but is not sensitive for midureteral stones. It is also less sensitive than CT at detecting mimickers. It is, however, 98% sensitive for detecting hydronephrosis.

MR

MR Urography has poor sensitivity for detecting ureteral stones yet excellent sensitivity in detecting ureteric obstruction. It does not emit any ionizing radiation. However due to its cost, time required, lack of patient monitoring, and limited availability, it is currently rarely used for renal colic.

KUB

Although 90% of urinary calculi are radiopaque, plain abdominal radiographs are neither sensitive nor specific for the detection of nephrolithiasis and should not be used to rule in or rule out a stone. Once a stone has been detected however, the KUB can be used in follow-up to follow its progression.

TREATMENT

Renal Colic is usually an exquisitely painful condition, and the top management priority should be rapid and adequate analgesia. Parenteral opioid analgesia such as intravenous morphine is the mainstay of treatment. Multiple doses are often necessary. In addition, NSAIDs such as Ibuprofen or

Naproxen (po), or Toradol (IV) should be given concurrently unless there are contraindications such as peptic ulcer disease, renal impairment, or diabetes. NSAIDs should also usually be avoided in the elderly. To help facilitate passing of the stone, an alpha-blocker such as Tamsulosin (Flomax) 0.4mg once daily for 7-10 days should be prescribed for almost all patients with distal ureteric stones.

Patients who are clinically dehydrated should be given an IV of normal saline. Excessive hydration of euvolemic patients should be avoided, as it has not been conclusively shown to aid in stone passage.

DISPOSITION

Absolute indications for urological consultation and admission are:

- Intractable pain or vomiting
- Obstructed stone with infection
- Single kidney
- Transplanted kidney with obstruction

The majority of patients can be discharged from the ED. They should be prescribed an oral opiate as well as an NSAID, and told to return if they develop fever, uncontrolled pain, or vomiting. Appropriate follow-up should be arranged with a urologist or their primary care physician. Urine straining and stone analysis will help with urological follow up in planning prophylactic treatment strategies.

WHAT'S NEW

Multiple recent studies have demonstrated that alpha blockers such as Tamsulosin (Flomax) help facilitate the passage of distal ureteric stones. The presumed mechanism of action is related to ureteric smooth muscle relaxation. Based on the mounting evidence, Tamsulosin 0.4 mg per day for approximately 7 – 10 days should be prescribed for virtually all patients with distal ureteric stones.

PROGNOSIS

Over 80% of stones will pass spontaneously. In general, smaller stones are most likely to pass spontaneously. If the stone is 4 mm or smaller, they will eventually be passed 90% of the time. Stones 5-7 mm have a 50% chance of passing spontaneously. Stones larger than 7mm are unlikely to pass spontaneously, and should be referred for consideration of lithotripsy. More than half of patients will have a recurrent presentation of renal colic in less than 10 years.

SUMMARY

- Renal colic typically presents with severe flank pain radiating to the groin causing the patient to be ‘writhing’ in pain
- The most important and cost effective test in presumed renal colic is a urinalysis
- Non-contrast helical CT is the preferred imaging modality for most presentations of renal colic
- Morphine, NSAIDs, alpha-blockers, and fluid rehydration are the mainstays of renal colic treatment
- Absolute indications for urological consultation and admission are intractable pain or vomiting, obstructed stone with infection, single kidney, and transplanted kidney with obstruction
- Most patients with renal colic can be discharged from the emergency department with specific instructions and follow-up

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32: PEDIATRIC CARDIOPULMONARY RESUSCITATION

Suzan Schneeweiss

OBJECTIVES

1. Identify differences between the pediatric and adult airway
2. List ‘normal’ vital signs for pediatric patients
3. Review the ABCs of resuscitation for pediatric patients
4. Understand the various methods of vascular access in the pediatric population
5. Become familiar with some of the resuscitation medications

INTRODUCTION

Unlike adults, pediatric cardiac arrest is usually caused by an underlying respiratory abnormality. In general, the myocardium in children is normal and the final common pathway leading to cardiac arrest is usually hypoxia. The second most common cause of cardiac arrest is circulatory failure from hypovolemia (loss of fluid or blood, sepsis). Although survival from in-hospital arrest has improved up to 27% with better neurologic outcome, survival from out-of-hospital arrests is only 6% with poor neurologic outcome. Pulseless arrests (PEA/asystole) have the worst outcome, whereas infants and children with a pulse but poor perfusion and bradycardia have the best survival (64%) to discharge. Ventricular fibrillation occurs in 5–15% of cardiac arrests with survival from 0–30%. It is more likely to be associated with survival with good neurologic outcome when it is the initial arrest rhythm.

AIRWAY

Airway control is the initial step in pediatric resuscitation. One must consider the differences in the pediatric vs. adult airway to optimally manage a pediatric airway.

- Large tongue relative to the oral cavity
- Higher and more anterior larynx - C3-4 vs. C5-6 in adults
- Long floppy epiglottis
- Subglottic area is the narrowest portion of the infant larynx
- Large head - when lying supine on a backboard, the neck is relatively flexed due to the large occiput, and may obstruct the airway. In infants and toddlers, the head should be in a neutral position (“sniffing position”) with avoidance of

hyperextension of the neck as this may obstruct the airway.

Airway adjuncts are used to facilitate an open airway in children by displacing the tongue or soft palate from pharyngeal passages. An **oropharyngeal airway** is used to bypass the tongue and should only be used in an unresponsive child without a gag reflex. **Nasopharyngeal airways** are generally better tolerated, but should not be used if there is suspicion of skull base trauma in order to avoid potential infection or penetration of the anterior cranial fossa with insertion. Note that when bag-mask ventilation is unsuccessful, and endotracheal intubation is not possible, insertion of a **laryngeal mask airway (LMA)** by an experienced provider is acceptable to provide a patent airway and support ventilation.

Tracheal Intubation

- Tracheal intubation is the best method for establishing and maintaining a patent airway in children who are comatose or in those with respiratory or cardiac arrest.
- Both cuffed and uncuffed endotracheal tubes may be used. Consider a cuffed endotracheal tube if there is a large glottic air leak, poor lung compliance, or high airway resistance.
- Do not use cricoid pressure for intubation if it interferes with ventilation, or the speed or ease of intubation.

Estimation of endotracheal tube (ETT) size in children

- Infants to 1 year = 3.5 ETT uncuffed
- 1 – 2 yrs = 4.0 uncuffed ETT
- Child > 2 yr. = (Age/4) + 4 uncuffed ETT
- Child >2 yr = (age/4) + 3.5 cuffed ETT
- Internal diameter of ETT = size of the child's little finger

Verification of endotracheal tube placement

- Bilateral chest movement, listen for equal breath sounds over both lung fields
- Listen for gastric insufflation over stomach
- Check exhaled CO₂ (end-tidal CO₂)
- Check oxygen saturation if perfusing rhythm
- If uncertain, direct laryngoscopy
- Chest X-ray in hospital setting

If patient deteriorates consider following (**DOPE**):

- Displacement of tube
- Obstruction of tube
- Pneumothorax
- Equipment failure

BREATHING

An infant or child with respiratory distress may initially present with an increased respiratory rate and effort, or decreased breath sounds. They may progress to a decreased level of consciousness or decreased response to pain or stimulation. They may also exhibit poor skeletal muscle control and finally cyanosis. The increased work of breathing often produces nasal flaring and use of accessory muscles of breathing. Head bobbing, grunting, stridor, and prolonged expiration are signs of significant respiratory distress. An acutely ill child with slow or irregular respirations is an ominous sign of impending respiratory arrest.

Tachypnea without other signs of respiratory distress ('quiet tachypnea') is often an attempt at maintaining a normal pH (compensatory respiratory alkalosis) in response to a metabolic acidosis.

Normal respiratory rates for age are shown in table 1 below. A normal respiratory rate alone should never determine if breathing is adequate. The respiratory rate must be interpreted with appearance, work of breathing, and air movement. A child with increased work of breathing and poor air entry may be in impending respiratory failure.

Table 1. Normal Respiratory Rate for Age

Age	Respiratory Rate (breaths/minute)
Infant	30 - 60
Toddler	24 - 40
Preschooler	22 - 34
School-age child	18 - 30
Adolescent	12 - 16

Bag-Mask Ventilation

A self-inflating bag-mask device should be used when assisting ventilation. The mask should fit over the mouth and nose to provide a tight seal and avoid any air leakage. In infants and children, the ventilation bag should have a minimum volume of 450-500ml. Anesthesia bags are more difficult to use, require experienced personnel, and should always have an attached pressure manometer. Always use oxygen with a flow rate of a least 10-15 L/min and a reservoir bag attached to the self-inflating bag to deliver high

oxygen concentration (60% to 95%). Use only enough force and tidal volume to make chest rise visibly. Remember to insert an NG tube if bagging for more than a few minutes, as gaseous distention can quickly lead to impaired ventilation.

CIRCULATION

Evaluation of circulatory status involves measurement of heart rate and blood pressure. Both heart rate and blood pressure vary with age. Heart rate may be influenced by other factors such as temperature, anxiety, and pain. Blood pressure may be difficult to measure in children because of lack of patient cooperation and confusion about proper cuff size (2/3 length of upper arm or thigh). Signs of circulation to the skin (skin temperature, capillary refill time, and pulse quality) are also used to assess circulatory status. Note that capillary refill time may be affected by ambient temperature, site, and age, and lighting can influence its interpretation.

Table 2. Hypotension by Age

Age	Systolic Blood Pressure (mm Hg)
Term neonates (0 – 28 days)	<60
Infants 1 to 12 months	<70
Children > 1 year	70+(2X age in years)
Children > 10 years	90

Table 3. Normal Heart Rate for Age

Age	Heart Rate /min
Infant	100 - 160
Toddler	90 - 115
Preschooler	80 - 140
School-age child	70 - 120
Adolescent	60 - 100

Shock results from inadequate tissue oxygen delivery. In children, this is most often due to hypovolemia from fluid loss or sepsis. Initially, compensatory mechanisms such as tachycardia and increased systemic vascular resistance maintain cardiac output and perfusion pressures. Decompensation occurs when compensatory mechanisms fail and result in hypotensive shock. Sustained tachycardia in the absence of known causes such as fever and pain may be an early sign of cardiovascular compromise. Bradycardia may be a pre-terminal cardiac rhythm indicative of advanced shock and is often associated with hypotension. Hypotension is a late sign of shock and is often associated with high morbidity and mortality.

Compensated Shock:

- Tachycardia
- Cool and pale distal extremities
- Prolonged (>2 seconds) capillary refill
- Weak peripheral pulses compared to central pulses
- Normal systolic blood pressure

Decompensated Shock (inadequate organ perfusion)

- Depressed mental status
- Decreased urine output
- Metabolic acidosis
- Tachypnea
- Weak central pulses
- Deterioration in colour (e.g. mottling)

Cardiac compressions should be immediately initiated in children when there is no pulse palpable, or heart rate is <60 bpm and there is evidence of poor systemic perfusion. Carotid pulse is often difficult to palpate in infants (<1 year) due to their short necks. The brachial artery or femoral artery should be used for palpation of a pulse in infants. Good cardiac compressions require a **chest compression rate of 100/min and a depth of compression of one third to one half of the anterior-posterior diameter of the chest ('push hard' and 'push fast')**. Rotate compressor every 2 min to avoid fatigue and deterioration in quality of cardiac compressions.

Table 4. Ventilation / Compression Schedule for Pediatric Resuscitation

	Compression to ventilation ratio
No advanced airway, one rescuer	30:2
No advanced airway, two rescuers	15:2
Advanced airway (e.g. ETT, LMA)	8 – 10 breaths/min without pausing chest compression

Volume Expansion

Fluid administration during resuscitation is used to restore effective circulating volume in hypovolemic shock states, restore oxygen-carrying capacity in hemorrhagic shock states, and correct metabolic imbalances.

A fluid bolus consists of **20 ml/kg of an isotonic solution** such as normal saline (NS) administered as quickly as possible. Repeated boluses may be necessary to restore the circulating blood volume particularly for hypovolemic and septic shock.

Blood replacement is indicated in children with severe acute hemorrhage if a child remains in shock after 40 -60 mL/kg of crystalloid. It is extremely important to reassess the child's response to each bolus of fluid. No clear recommendations for use of colloid fluid have been made.

Vascular Access

Vascular access is a crucial step in pediatric resuscitation; however peripheral venous catheterization is frequently unobtainable under these circumstances. Central venous catheter placement requires training, experience, and may be a time-consuming procedure; therefore, it is not recommended as the initial route of vascular access in an emergency.

Intraosseus (IO) access is a rapid, safe, and effective route for vascular access in children. It can be used to deliver any type of fluid (including blood) and medication. Drug onset of action and drug levels are comparable to peripheral venous access. A rigid needle is inserted through the cortex of the bone into the medullary cavity preferably at the anterior medial aspect of the tibia 1-3 cm below the proximal tibial tuberosity. Other sites of insertion include the distal tibia, distal femur, and iliac crest. Complications include infection, fracture, and compartment syndrome. Once an alternate site of vascular access has been established, the intraosseus line should be removed.

Endotracheal Drug Administration

- If IO or IV access is not possible
- Lipid-soluble drugs such as lidocaine, epinephrine, atropine, and naloxone ("LEAN")
- Flush medications with at least 5 mL of NS
- May double or triple dose of lidocaine, atropine, or naloxone via ETT. For epinephrine 10 times the dose is recommended.

MEDICATIONS USED FOR CARDIAC ARREST AND RESUSCITATION

Medication for cardiopulmonary resuscitation should be calculated based on actual weight. If weight is not known then length-based measurements can be used (e.g. Broselow tape®). Drug administration should not exceed adult recommended dosages.

Epinephrine

- Drug of choice for cardiopulmonary arrest
- Has both alpha and beta adrenergic properties, however, it is the alpha adrenergic mediated

- vasoconstriction which is the most important property acting to restore aortic diastolic pressure
- Catecholamines are not as effective when the patient is acidotic and hypoxic so attention to ventilation, oxygenation, and circulation is essential

Sodium Bicarbonate

- Routine administration of bicarbonate is not recommended for cardiac arrest
- Note excessive bicarbonate may impair tissue oxygen delivery, cause hypokalemia, hypocalcemia, hypernatremia, hyperosmolality, decrease ventricular fibrillation threshold, and impair cardiac function
- May be indicated for some toxicological emergencies and special situations such as hyperkalemic cardiac arrest

Glucose

- Infants have high glucose requirements and low glycogen stores
- Hypoglycemia is common during periods of high stress and energy needs
- Glucose monitoring is essential during resuscitation and can be done quickly with a bedside glucose test strip

Atropine

- Parasympatholytic drug used to treat symptomatic bradycardia and vagally mediated bradycardia that can occur with intubation attempts
- Sinus bradycardia usually results from hypoxemia and is initially treated with oxygenation and ventilation
- When atropine is administered, the dose must be sufficient to produce vagolytic effects and avoid paradoxical bradycardia

Calcium

- Not routinely given during resuscitation and may be harmful.
- Calcium may enter the cells with organ reperfusion following an ischemic event and produces toxic effects
- Indicated for the treatment of documented hypocalcemia, hyperkalemia, hypermagnesemia, and calcium channel blocker overdose

Dopamine

- Used for the treatment of circulatory shock following resuscitation, or when shock is unresponsive to fluid administration

- It is an endogenous catecholamine with complex cardiovascular effects
- With higher doses, it stimulates beta receptors and alpha adrenergic vasoconstriction

Adenosine

- Used for treatment of supraventricular tachycardia
- Causes a temporary atrioventricular (AV) nodal conduction block and interrupts reentry circuits that involve the AV node

CARDIOPULMONARY ARREST

Pulseless Cardiac Arrest

- Child unresponsive, no breathing
- Call for help and send for defibrillator (manual or AED)
- Health care provider check for pulse (< 10 s – brachial in infant or carotid/femoral in child)
- Start CPR (with supplementary oxygen if available)
- High quality CPR = chest compressions of adequate rate and depth, with complete recoil after each compression, avoid interruption and excessive ventilation
- Attach ECG monitor or AED pads to determine if “shockable” rhythm [ventricular fibrillation (VF) or rapid ventricular tachycardia (VT)] or “non-shockable” rhythm [asystole or pulseless electrical activity (PEA)]

“Nonshockable Rhythm:” Asystole/Pulseless Electrical Activity (PEA)

- Continue CPR
- Second rescuer obtains vascular access and delivers epinephrine 0.01 mg/kg (0.1 mL/kg of 1:10,000 solution) while CPR continues; repeat dose every 3 – 5 minutes
- Check rhythm every 2 minutes with minimal interruptions in chest compressions
- Search for reversible causes: Hypothermia, Hypoxia, Hydrogen ion (acidosis), Hypoglycemia, Hypovolemia, Tension Pneumothorax, Tamponade (cardiac), Toxins, Thrombosis (pulmonary and coronary)

“Shockable Rhythm”: VF/pulses VT

Defibrillation with an automatic (AED) or manual defibrillator is indicated for ventricular fibrillation and pulseless ventricular tachycardia. AEDs can detect VF in children and can differentiate “shockable” from “non-shockable” rhythm. AEDs may be equipped with a pediatric attenuator for defibrillation in children < 8yrs. If a pediatric attenuator is not available, then an AED with standard

electrodes may be used. In infants < 1 yr, a manual defibrillator is preferable.

- At any time if rhythm becomes **shockable**, give a shock and immediately resume chest compressions for 2 minutes before rechecking rhythm
- Initial dose 2 J/kg as quickly as possible; resume CPR X 2 min; if shockable rhythm persists, give 1 shock 4 J/kg then give epinephrine (repeat sequence every 3 – 5 min); continue CPR X 2 minutes, if shockable rhythm persists give 1 shock at 4 J/kg and continue CPR X 2 min; if shockable rhythm persists, continue CPR and give amiodarone or lidocaine if amiodarone is not available

TACHYARRHYTHMIA

Supraventricular Tachycardia (SVT)

A 12-lead ECG and clinical presentation should help distinguish sinus tachycardia from supraventricular tachycardia. SVT is a narrow QRS complex tachycardia with little beat-to-beat variability and abnormal or absent P waves. In infants, SVT is associated with a HR of > 220bpm and in children, HR is > 180 bpm. It is the most common tachyarrhythmia in children.

- If hemodynamically stable, begin with vagal maneuvers (e.g. ice to face in an infant without occluding airway, blowing through narrow straw in older child); if unsuccessful, adenosine is the drug of choice
- If hemodynamically unstable, then consider synchronized cardioversion (0.5 to 1 J/kg; increase to 2J/kg for subsequent attempts)
- **Synchronized cardioversion** is used to treat symptomatic patients with supraventricular tachycardia or ventricular tachycardia (with pulse) accompanied by poor perfusion, hypotension or heart failure

SUMMARY

- Pediatric cardiac arrest is most often caused by respiratory failure or hypovolemia

- Anatomic differences between the adult and pediatric airway must be considered when securing the airway of a pediatric patient
- An acutely ill child with slow or irregular respirations is an ominous sign of impending respiratory arrest
- A fluid bolus in pediatric patients consists of 20ml/kg of an isotonic solution such as normal saline administered as quickly as possible
- Intraosseous access can be obtained rapidly and is a good alternative or adjunct to intravenous cannulation in the initial resuscitation of the pediatric patient
- Good quality CPR: 100 compressions per minute, 1/3 to 1/2 depth of the chest, allow for full recoil of chest after compression, minimize interruptions, and avoid excessive ventilation
- Recognize specific indications for resuscitation medications
- Defibrillation is indicated for ventricular fibrillation (VF) or pulseless ventricular tachycardia both of which are uncommon in children unless there is an underlying cardiac condition

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33: PEDIATRIC RESPIRATORY EMERGENCIES

Suzanne Schuh

OBJECTIVES

1. To review the presentation and management of asthma
2. To review the presentation and management of bronchiolitis
3. To review the presentation and management of croup

INTRODUCTION

Pediatric respiratory emergencies are often very anxiety provoking for both parents and physicians. These emergencies account for a large percentage of ED visits. While the specific management of these illnesses has changed substantially in the past decade, the basic principles have remained the same.

ASTHMA

Asthma is the most common chronic illness of childhood, affecting approximately 10% of children. It is also the most common pediatric medical emergency.

Both bronchospasm and extensive airway inflammation constitute an integral part of acute exacerbations. This inflammation persists for weeks after an acute episode; therefore therapy needs to be guided accordingly.

The most common precipitating factor is a viral upper respiratory tract infection. Other triggers include: specific allergens (minority), cold exposure and humidity changes, airway irritants such as fumes, dust and air pollution, exercise, emotional stress, and certain medications (e.g. aspirin or beta blockers).

A focused history must include: the duration of symptoms, recent precipitating factors, current medications (doses frequency), past ICU admissions, frequency of previous exacerbations and hospitalizations, and associated medical illnesses. Tachypnea, suprasternal retractions, nasal flaring, poor air entry (often with minimal wheezing), lethargy, difficulty speaking in sentences and feeding problems signify severe disease.

Management

- Frequent re-assessment and monitoring of vital signs and respiratory status is critical.
- Oxygen - children with oxygen saturation below 90% require supplemental oxygen. Initial pre-treatment O₂ sat at or below 90% (room air) usually indicates need for hospitalization.
- Beta agonists - Ventolin (salbutamol) via Metered Dose Inhaler (MDI)/spacer. Nebulizer route of treatment is minimized since MDI is as effective, less expensive, and has a lower risk of viral transmission. The dose of salbutamol is 400 mcg per dose for children <15 kg and 800mcg per dose for bigger children. Treat q20 min q1h depending on severity. Supplemental K+ may be required if intensive therapy is needed for long periods.
- Anticholinergics - Atrovent (ipratropium) 80 mcg/dose with Ventolin, for the first 3-6 doses is indicated in severe disease only.
- Corticosteroids - indicated for all except mild exacerbations, give within one hour of ED triage.
 - Prednisone 2 mg/kg (max 60 mg)
 - Dexamethasone 0.3 mg/kg orally
 - Prednisolone (Pediapred) 2mg/kg
 - Dexamethasone 0.3 mg/kg orally in the ED, then Predniso(lo)ne 1mg per kg x 4 days orally.
 - Intravenous hydrocortisone 5 mg/kg /dose q 6h if critically ill, repeated vomiting, or deteriorating status
 - Do not substitute inhaled for systemic corticosteroids during acute management
- For refractory/severe cases: Magnesium sulfate 40 mg/kg IV over 30 minutes. Monitor for hypotension.
- IV salbutamol is used in patients not responsive or deteriorating on above regimen, usually in ICU setting.
- Intubation is rare (moribund, exhausted, hypoxic, or hypercarbic children)
- Patients with significant respiratory distress 4 hours after corticosteroids and/or persistent need for frequent inhaled bronchodilators, need to be hospitalized.
- Poor previous asthma control, poor social situation, poor parental knowledge of the disease, and multiple visits for the same episode constitute additional risk factors.

CLINICAL PEARL

Metered dose inhaler is as good as nebulizer, produces fewer side effects, is less expensive, and has a lower risk of viral transmission.

Discharge Therapy

- Salbutamol 4–8 puffs/dose q4h prn via MDI and spacer (with a mask < 6 years, with a mouthpiece 7 years or older) for 24–48 hours, then 2 puffs qid prn x 1 week. Educate on use of spacer.
- Pediapred / Prednisone 1mg/kg daily x 4–5 days orally. No tapering necessary.
- Inhaled corticosteroids - fluticasone 50 mcg bid for 4–8 weeks after discharge
- Mandatory follow up at 24–48 hours
- Return if increased work of breathing requiring bronchodilators <q4h.

BRONCHIOLITIS

The first episode usually presents as respiratory distress with wheezing/crackles and cough in an infant under one year of age. It has a viral etiology (usually RSV), and usually presents December to April. Severe bronchiolitis and atopy in first degree relatives is an important risk factor for subsequent development of asthma. Crepitations are common and not an indication for X-ray or antibiotics. Predictors for future deterioration include: age under 7 weeks, weight under 4 kg, prematurity, HR>180/min, RR≥80/min, saturation≤88%, and co-morbidities. Use a low threshold for admission.

Management

- Oxygenation - supplemental O₂ if O₂ saturation below 90%
- Hydration - frequent, small feeds or IV if poor intake/dehydration
- Pharmacotherapy is not usually effective and not routinely recommended. Neither bronchodilators nor corticosteroids alone change the course of the disease. Although there is preliminary evidence that a combination of inhaled epinephrine (1:1000 3 mlx2 doses q 30 min) and oral dexamethasone (1 mg/kg) may lower hospitalization, this regime is currently not a standard of care.
- Criteria for admission are: severe retractions/grunting, inability to hydrate orally, O₂sat < 90% on room air, very poor feeding, < 2 months of age, associated respiratory/cardiac disease, RR>70-80/min, and repeated visits.

- CXR is not indicated in typical bronchiolitis - only in atypical cases to rule out other diagnoses (congenital lung anomalies, cardiac disease). Approximately 20% of cases have associated pneumonia, which is viral.

Discharge Therapy

- Frequent small feeds and return if markedly increasing retractions, cannot feed, or inappropriate lethargy.
- Stress that symptoms often last 2–3 weeks
- Pharmacotherapy not indicated
- Follow-up in 24–48 hours

WHAT'S NEW

Pharmacotherapy is not usually effective and not routinely recommended in bronchiolitis.

CROUP (LARYNGOTRACHEOBRONCHITIS)

Croup is a viral syndrome (usually Parainfluenza) most common in infants 6–36 months of age, and presents with a ‘barking’ cough, hoarse voice, inspiratory stridor +/- respiratory distress. Onset is usually at night. Infants with a history of prolonged ventilation may have tracheal stenosis with a fixed airway obstruction and a greater risk for severe croup.

Persistent stridor at rest, subcostal retractions, tachypnea, poor air entry, and anxiety all herald severe disease. Fever is common but toxicity rare.

Management

- Humidity is ineffective.
- Corticosteroids - indicated in all degrees of severity. Dexamethasone 0.6 - 1 mg/kg orally. Only 1 dose necessary in most cases (long half-life). Takes 4 hours for full effect.
- Nebulized epinephrine 1:1000 (2.5 - 5 ml) in severe respiratory distress. It is effective immediately but has a short half-life of 30min-1 hour (improvement may be transient). Mandatory observation for 2 hours after use. Repeat in 1 h if incomplete response.
- Criteria for admission - poor response to oral dexamethasone (after 4 hours) or repeated doses of inhaled epinephrine required.

Consider an alternate/co-existent diagnosis if there is no response to nebulized epinephrine, toxic appearance, or past history of frequent severe croup and/or chronic feeding difficulties.

CLINICAL PEARL

All infants with croup require a single dose of oral dexamethasone.

SUMMARY

- The MDI route of bronchodilator delivery is preferred in virtually all asthmatics with all degrees of disease severity.
- Give corticosteroids within one hour of ED triage.
- Consider IV Mg in severe/refractory cases.
- The red flags for airway deterioration in bronchiolitis include: young age, low weight, highly abnormal vitals, significant hypoxia on arrival, and co-morbidities.
- Supplemental oxygen and hydration is the mainstay of bronchiolitis emergency treatment. There is little evidence for the role of pharmacotherapy in bronchiolitis.
- All patients with croup require a single dose of oral dexamethasone, regardless of severity.

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34: FEVER IN INFANTS & CHILDREN

Leah Harrington

OBJECTIVES

1. To review the Rochester Criteria for determining whether febrile infants are unlikely to have a serious bacterial illness
2. To know age-specific work-ups for infants and children with fever
3. To know treatment options for infants and children of different ages with fever

INTRODUCTION

The approach to the management of fever in infants and children is formulated based on the infant's/child's age, their clinical status, and the presence or absence of a focus.

Definitions

Fever - temperature $> 38.0^{\circ}\text{C}$ rectal (tympanic measurements have been shown to be unreliable in children < 3 years).

Fever without source - an acute febrile illness in which the etiology of the fever is not apparent after a careful history and physical examination.

Serious bacterial infection (SBI) - includes meningitis, sepsis, bone and joint infections, urinary tract infections, pneumonia, and enteritis.

Lethargy - a level of consciousness characterized by poor or absent eye contact; or the failure of a child to recognize parents; or failure to interact with persons or objects in the environment.

Toxicity – a clinical picture consistent with the sepsis syndrome (i.e. lethargy, signs of poor perfusion, marked hypoventilation, hyperventilation or cyanosis).

THE ROCHESTER CRITERIA

- Criteria used for determining febrile infants unlikely ($\leq 1\%$) to have serious bacterial illness
- Age: < 60 days
- General appearance: appears well

Past medical history

- Born at ≥ 37 weeks gestation
- No perinatal antimicrobial therapy
- No past or present antimicrobial therapy
- No unexplained hyperbilirubinemia
- No previous hospitalization
- No chronic or underlying illness
- Not hospitalized longer than mother at birth

Physical exam

- No evidence of skin, soft tissue, bone, joint, or ear infection

Laboratory values

- Peripheral WBC $5-15 \times 10^9$ cells/L
- Absolute band count $\leq 1.5 \times 10^9$ cells/L
- ≤ 10 WBC/ HPF on spun urine sediment
- ≤ 5 WBC/ HPF of stool smear (only if diarrhea present)

Boston and Philadelphia Protocols are also used to assess risk of SBI in infants with fever with no focus.

MANAGEMENT APPROACH BY AGE

Management of the febrile infant 0-1 months

months of age: clinical evaluation is inadequate to exclude serious bacterial infection

INVESTIGATIONS

Febrile infants 0-1 months require a full sepsis work-up:

- CBC and differential (consider also venous blood gas, lytes, glucose, urea, creatinine)
- Urinalysis
- Cultures: blood, urine (catheter or suprapubic), and CSF (lumbar puncture)
- Chest X-ray (required only if history and/or physical suspicious for respiratory focus)

DISPOSITION

- Hospitalization with parenteral antibiotics (ampicillin and cefotaxime *or* ampicillin and gentamicin)

- Hospitalization without initial antimicrobial coverage may be considered for the low risk infant

WHAT'S NEW

There is debate with the management of the neonate age 1-2 months. A full sepsis work-up is routinely recommended for infants less than 1 month of age. Generally, infants 1-3 months are assessed using the Rochester Criteria with only high risk infants, receiving a full sepsis work-up. This is literature that promotes a more conservative approach in the 1-2 month age group recommending a full sepsis work-up for these infants. There may be variation in observed practice in the 1-2 month age group.

Management of the febrile infant 1-3 months of age:

Lethargic/toxic infant with no focus of infection:

INVESTIGATION

- Full sepsis work-up

DISPOSITION

- Hospitalization with parenteral antibiotics (ampicillin and cefotaxime *or* ampicillin and gentamicin)

Nonlethargic/nontoxic infant with no focus of infection:

INVESTIGATION

- CBC and differential, urinalysis
- Blood and urine cultures

RISK OF SBI

Rochester Criteria may be used as guideline to determine if infant is 'high risk' or 'low risk'

DISPOSITION

'High risk' - full sepsis work-up, parenteral antibiotics, hospitalization

'Low risk' – discharge (no antibiotics, 24 hour follow-up) or admission (observation, no antibiotics)

Management of the febrile infant 3-36 months of age:

no single approach is supported in the literature to date

Lethargic/toxic infant with no focus of infection:

INVESTIGATION

- Full sepsis work-up

DISPOSITION

- Hospitalization with parenteral antibiotics (cefuroxime for r/o sepsis or ceftriaxone for r/o meningitis)

Nonlethargic/nontoxic infant with no focus of infection:

Temp < 39.5 °C

- Observation, clinical decision, 24 hour follow-up

Temp > 39.5 °C

- Observation, clinical decision, 24 hour follow-up
or
- CBC and differential, urinalysis
- Blood culture, urine culture (if findings on urinalysis)
- 'Low risk' - no antibiotics, 24 hour follow-up
- 'High risk' - amoxicillin may be given until culture results available, 24 hour follow-up

SUMMARY

If a focus of infection is present, antibiotics must be chosen with consideration for the likely etiologic agent and the age of the child. The child's clinical status must be determined either through observation or laboratory evaluation and disposition should reflect the assessed risk level of the child (i.e. admission versus close follow-up).

In a well appearing child > 36 months with a fever and no focus, consideration must be given to the need for urinalysis. Urine specimens collected via a bag in the non-toilet trained child must never be sent for culture, as results are generally contaminated by more than one organism. If there are positive findings in the urine dip or on microscopy, a catheter or suprapubic specimen must be obtained for evaluation. The toilet trained child may provide a midstream urine specimen for evaluation.

Children who are immunocompromised on the basis of underlying pathology (e.g. sickle cell disease) or medications (e.g. steroids, immunosuppressants) must be carefully assessed and managed. These children generally should be managed in conjunction with a pediatrician.

Clinical assessment of children for streptococcal vs. viral pharyngitis has been shown in the literature to be extremely insensitive. Prior to the initiation of antibiotic treatment a

throat swab must be sent. Treatment can safely be deferred until the result of the swab is available, thereby avoiding unnecessary use of antibiotics.

Viruses cause the majority of fevers in children of all ages. Excessive use of antibiotics not only serves to increase the risk of resistance within the community but within the individual child as well. Common childhood infections such as otitis media have now been found to be viral in origin in up to 80% of cases. If antibiotics are required review the hierarchy of antimicrobial therapy for a particular condition and start at the bottom, not at the top (eg. initiate therapy for otitis media with Amoxacillin, not a cephalosporin).

A child's condition can deteriorate rapidly. Assured follow-up is the most important part of the management plan. If you are unsure of the parents understanding of the need for or the ability to ensure follow-up for the child, hospital observation or Home Care Nursing visitation may be appropriate.

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WHAT'S NEW

Common childhood infections such as otitis media have been found to be viral in origin in up to 80% of cases.

35: ABDOMINAL PAIN IN INFANTS & CHILDREN

Leah Harrington

OBJECTIVES

1. To review some common and some life-threatening causes of abdominal pain in infants and children
2. To understand when to suspect serious causes of abdominal pain in infants and children
3. To review the management of pediatric constipation
4. To review the assessment and management of pediatric gastroenteritis

INTRODUCTION

The assessment of abdominal pain in infants and children is challenging. As opposed to most adults and older children, infants and young children are either verbally unable to communicate their symptoms, or if able to verbally communicate, they are unable to localize their abdominal pain well. Nonetheless, there are important clues in the history, physical, and investigations that help the emergency physician narrow the differential diagnosis and facilitate appropriate management.

While the majority of pediatric patients presenting to the emergency room have relatively benign diagnoses, there are several important life-threatening diagnoses, which must always be considered.

The approach to abdominal pain in infants and children is based on the infant's/child's age, acuity of presentation, and general clinical status. The following is a case presentation format of some common and/or life-threatening pediatric illnesses that cause abdominal pain. Although appendicitis is not discussed here, it must always be on the differential of any child presenting to the emergency room with abdominal pain.

CASE 1

A one week old infant presents to the ED with a history of bilious vomiting and distress. The parents state that the infant is pulling his legs up to the abdomen and appears to be in pain.

History

Historical inquiry should be directed by the differential diagnosis generated from the presenting information. Acuity of presentation as well as the duration and progression of symptoms must be established. History of the pregnancy, and the perinatal period is important to determine if factors supporting a 'septic set-up' are present.

MALROTATION AND VOLVULUS

- Sudden onset of abdominal pain and bilious vomiting in previously well infant, *or*
- Sudden onset of abdominal pain in infant with transient episodes of bilious vomiting or past 'feeding intolerance'

Differential Diagnoses

Malrotation: Congenital nonrotation of the midgut loop with associated abnormal fixation of the bowel mesentery resulting in the small intestines lying on the right and the large on the left

Malrotation with midgut volvulus: The above with consequent twisting of the midgut around the superior mesenteric artery resulting in ischemia and subsequent necrosis of the bowel

Sepsis: Must be considered in infants presenting with vomiting, distress, and lethargic/toxic appearance. Sepsis may precipitate paralytic ileus or be associated with ischemic/necrotic gut.

Other congenital/acquired obstructive processes: duodenal atresia, intussusception, Hirschsprung's disease, and necrotizing enterocolitis.

Physical exam

General appearance:

- May appear lethargic/toxic and progress to shock.
- Abdominal exam:
- Mild distention associated with a high obstruction, diffuse tenderness, a dilated loop of bowel may be palpable.

Rectal exam:

- Gross blood associated with bowel ischemia/necrosis.

Initial management

- Assess ABC's
- Stabilize the infant: consider intubation if infant toxic or in shock - fluid resuscitation 20ml/kg normal saline bolus (repeat boluses prn to correct fluid/electrolyte imbalance)
- NPO + nasogastric drainage
- Urgent pediatric surgical consult

Investigations

Bloodwork:

- CBC & differential, lytes, urea, creatinine, VBG, blood and urine culture, type and cross

Abdominal X-ray:

- Supine and upright views
- Loops of small bowel overriding the liver shadow
- Dilated loops of bowel with multiple air-fluid levels
- Markedly dilated stomach and duodenum

Upper GI series:

- Absent ligament of Treitz and C-loop of the duodenum
- Duodenum lies to the right of the spine
- Jejunum coiled spring appearance in right upper quadrant

Important Points

- Bilious vomiting in an infant is malrotation with volvulus until proven otherwise
- Stabilization is key - never send an unstable infant for investigation (e.g. xray, upper GI)
- Surgical consultation/intervention is urgent as bowel ischemia can progress rapidly if volvulus is present

CASE 2

An 18 month old male infant presents to the ED with a 12 hour history of intermittent colicky abdominal pain.

History

The history should detail the nature of the abdominal pain and the associated symptoms. The differential diagnosis for abdominal pain in this age group will guide the focus of the history. Always inquire about the child's birth and past medical/surgical history (e.g. born at 28 wk GA suffered necrotizing enterocolitis).

INTUSSUSCEPTION

- Previously well child or prior gastroenteritis/viral infection
- 3-12 months of age (peak 10 months) and predominant in males
- Intermittent colicky abdominal pain lasting several minutes
- Vomiting may be bilious
- Blood per rectum /currant jelly stools is a late sign

Differential Diagnoses

Intussusception: Telescoping of the bowel at the ileo-cecal junction leading to ischemia, subsequent necrosis, and death if undiagnosed and untreated. The triad of intermittent abdominal pain, vomiting, and RUQ abdominal mass plus occult/gross blood per rectum has a positive predictive value of 100%.

Constipation: Presents with vague to severe recurrent abdominal pain. Hard stool may be palpated on abdominal exam and rectal.

Gastroenteritis: Presents with crampy intermittent abdominal pain, vomiting, and diarrhea (may be blood streaked due to viral /bacterial agents). Family history of siblings/contacts similarly affected may be present.

Urinary tract infection: May present with intermittent suprapubic pain, fever, vomiting, dysuria, and foul smelling urine. Urinalysis may demonstrate leukocytes, nitrates, and bacteria.

Physical Exam

General appearance:

- During painful episodes lasting 4-5 minutes, the child will cry inconsolably and may draw the legs up to the abdomen. With cessation of pain, the child will stop crying and resume quiet activity. The child may appear lethargic.

Abdominal exam:

- Diffuse tenderness, distention, RUQ sausage shaped mass

Rectal exam:

- Mass may be palpated occasionally, gross blood with mucus (currant jelly stool) is a later finding.

Initial Management

- Assess ABC's
- Stabilize the child - IV normal saline bolus (20ml/kg and repeat boluses prn)
- NPO + surgical consult

Investigations

Abdominal X-rays: Low specificity for condition may show signs of obstruction.

Abdominal ultrasound: Negative predictive value of 97% in pediatric centre.

Air/barium enema: Gold standard for diagnosis and treatment, contraindicated if perforation, complete obstruction, or hemodynamic instability present.

Important Points

- Classic triad of intermittent abdominal pain, vomiting, and currant jelly stool appears in only 10 to 20% of cases of intussusception - index of suspicion must be high to avoid missing the diagnosis.
- Intussusception may follow an interval of gastroenteritis, with enlarged mesenteric nodes thought to be acting as lead points.
- Older children may present with intussusception, especially if lead points are present (e.g. intestinal lymphosarcoma) or in association with Henoch Schonlein purpura.

CASE 3

A 3 year old child presents to the ED with a complaint of intermittent crampy abdominal pain. Parents state that the child has been soiling her underwear for the past 2 days with streaks of brown loose stool.

CONSTIPATION

Constipation can affect all age groups from infancy forward and is one of the most common causes of pediatric abdominal pain. Differential diagnoses for the infant/child presenting with suspected constipation will be formulated depending on the age, acuity of presentation, associated symptoms, and past medical history. Important questions include passage of meconium within the first 24 hours of life, description of stools, dietary habits, medications taken, and recent viral illnesses.

Differential Diagnoses

Constipation: Stool frequency of <3 BM/week, painful passage of hard/large stools

Acute: 1-4 weeks, most common etiologies - viral illness or dietary change

Chronic: > 4 weeks, etiologies - functional, organic/mechanical obstruction (Hirschsprung's),

drug induced (iron supplementation), endocrine (hypothyroidism), neuromuscular disorders (cerebral palsy)

Functional: Not due to organic, anatomic, or pharmaceutical causes

Fecal soiling: Stool deposited on underwear regardless of presence of organic/anatomic lesion

Encopresis: Fecal soiling in presence of functional constipation in child \geq 4 years of age

Other conditions to consider: gastroenteritis, urinary tract infection, intussusception, appendicitis, chronic recurrent abdominal pain

Physical Exam

General appearance:

The acutely constipated child will appear generally well hydrated and nourished, but may have significant pain causing the child to draw the legs into the abdomen. The chronically constipated child may appear as above if functional etiology or may present with failure to thrive or appear unwell or toxic if secondary to organic/obstructive processes.

Abdominal exam:

- Stool may be palpated, abdomen is generally soft, diffusely tender with lack of peritoneal signs
- If organic/obstructive process is present, exam of the abdomen may appear acutely surgical

Rectal exam:

- Hard stool may be felt in the rectum, overflow loose stool may also be present.

Investigations

Acute functional constipation requires minimal work-up (i.e. urinalysis). Radiologic studies are not indicated in uncomplicated constipation. Chronic constipation with a suspected organic origin should be investigated as per findings of history and physical exam in an outpatient setting.

Management

Evacuation of stool for the infant/child experiencing minimal discomfort may be achieved using oral preparations:

Infants

Corn syrup:

- Breastfed: 5-10 ml in 2-4 oz of water/fruit juice daily
- Bottlefed: 7.5-30ml/total formula/day or 5-10 ml every second feed

- Prune juice/infant prunes for infants > 2 months- 30 ml/month of age/day
- Mineral oil for infants > 1 year - 1-3 ml/kg/day

Toddlers/preschoolers/school age

- Mineral oil/Lansoyl 1-3 ml/kg/day divided into 1-2 doses (double dose every 3 days until results, may take up to 5 days to work)
- Lactulose 5-10 ml/day (double the dose daily until stool produced)
- Senna (Senokot) syrup: 1-5 yr-5ml daily; 5-10 yr -10 ml daily

Evacuation of stool for greater than minimal discomfort may include:

- Disimpaction (consider premedication of child prior to procedure)
- Infant to 2 yr: glycerin/dulcolax suppositories
- Children > 2 yr: hypertonic phosphate solution (Fleet® enema) - approx. 2 ml/kg body weight and/or normal saline enema 20ml/kg body weight

Prevention of re-accumulation:

- Infants: see above management recommendations
- Children > 1 yr: Mineral oil/Lansoyl 1-3 ml/kg/day for 3-6 weeks

Fiber recommendations:

- Fibre supplements/purified fibre not suitable for children <4 years
- Natural food fibre is suitable for infants <1yr (i.e. pureed fruits, vegetables, and infant cereal)
- Older children may have several servings daily of fiber-rich foods
- Eliminate constipating foods (i.e. rice, cheese, bananas)

Important Points

- Acute constipation can generally be treated using the oral route (i.e. stool softeners and dietary adjustments); enemas and suppositories can be traumatic for children so use sparingly.
- Fleet® enemas and other hyperphosphate solutions can be life threatening if used improperly; children <5 yr with congenital/acquired GI or GU abnormalities may be predisposed to metabolic derangement from these solutions.

CASE 4

A 9 year old girl is brought to the ED with complaints of intermittent abdominal cramping. She has had 3 episodes of non bilious vomiting and watery diarrhea.

GASTROENTERITIS

Gastroenteritis is one of the most common causes of pediatric abdominal pain. A careful history must be obtained to ensure other conditions presenting in a similar manner are not missed. Determine the amount of vomiting and diarrheal losses in addition to the amount and type of fluid tolerated to assist in your assessment of the child's hydration status.

Differential Diagnoses

Gastroenteritis: Pediatric diarrhea is viral in up to 80% of cases. Bacterial etiologies may more commonly present with bloody diarrhea and some require antibiotic treatment.

Rotavirus: 3-15 months, lasts 5-7 days, fever and vomiting precede non-bloody diarrhea.

Other common viruses: Norwalk, Adenovirus, Torovirus

Other conditions to consider: appendicitis, intussusception, urinary tract infection, constipation, malrotation with volvulus, hemolytic uremic syndrome

Physical Exam

General appearance: may range from mild to severe dehydration/shock

Abdominal exam: soft, generally not markedly distended, no signs of peritonitis, hyperactive bowel sounds

Rectal exam: soft to liquid stool, may be blood streaked to grossly bloody depending on etiology

Initial Management

Assess ABC's and stabilize as required.

Severely dehydrated:

- Fluid resuscitation 20ml/kg normal saline IV bolus (repeat boluses prn)
- Consider a 2-4 hour NPO period for bowel rest
- Continue IV rehydration with a glucose and saline solution (1.5 x maintenance) until oral rehydration established

Mild to moderate dehydration and tolerating fluids:

- attempt oral rehydration with an oral rehydration solution (i.e. Pedialyte, Lytren, Gastrolyte)
- start with 30 ml solution every 15-20 minutes, if fluid not tolerated institute IV rehydration

Investigations

Bloodwork: Consider CBC & differential, lytes, urea, creatinine, VBG

Urinalysis: Dipstick and microscopy to determine presence of infection + quantify specific gravity

Management

Infants: Continue breastfeeding and supplement with oral rehydration solution

	Mild dehydration (0-5%)	Moderate dehydration (5-10%)
1st hr	20ml/kg/hr ORS	20ml/kg/hr ORS
next 6-8hrs	10ml/kg/hr ORS	15-20ml/kg/hr ORS
8-24hr	ORS ad lib early refeeding	ORS ad lib early refeeding
> 24hrs	Delay refeeding only if severe vomiting	Delay refeeding only if severe vomiting

Refeeding: bland diet (i.e. rice, noodles, soups, bananas, rice cereals).

Important Points

- Infants <3 months of age must be carefully assessed for hydration status and should be rehydrated under medical observation. Follow-up within a 24 hour period is essential. If you are unsure of the parent/caregiver's ability to ensure follow-up, hospital observation or Home Nurse visitation may be appropriate.
- There is no role for anti-diarrheal agents in the treatment of pediatric gastroenteritis. Antibiotics should not be instituted until a bacterial etiology requiring treatment has been identified. The use of 'home-made' oral rehydration solutions is strongly discouraged based on the potential for serious error in preparation and subsequent metabolic derangement.
- Caution must be taken in labeling the child presenting with vomiting in the absence of diarrhea as 'gastroenteritis'. A thorough assessment must be carried out to rule out other possible diagnoses.

SUMMARY

- Bilious vomiting in an infant is malrotation with volvulus until proven otherwise
- The classic triad of intermittent abdominal pain, vomiting and currant jelly stool appears in only 10 to 20% of cases of intussusception - index of suspicion must be high to avoid missing the diagnosis
- Acute constipation can generally be treated using the oral route (i.e. stool softeners and dietary adjustments); enemas and suppositories can be traumatic for children so use sparingly
- Caution must be taken in labeling the child presenting with vomiting in the absence of diarrhea as 'gastroenteritis'. A thorough assessment must be carried out to rule out other possible diagnoses.

WHAT'S NEW

Ondansetron (Zofran) is an anti-emetic that has been used for many years in adults undergoing chemotherapy. Recently, data supports the use of this drug in mild to moderate pediatric gastroenteritis with vomiting in the emergency department.

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36: ANIMAL BITES

Tim Rutledge

OBJECTIVES

1. To understand the special issues regarding the management of mammalian bites, including prevention of infection, tetanus and rabies
2. To understand the indications for suturing
3. To understand the indications for antibiotics
4. To be aware of the special circumstance of the human bite clenched fist injury
5. To go about the appropriate steps required for patients with potential rabies exposure

INTRODUCTION

Animal or human bites account for up to 1% of emergency department visits. Over 80% of reported mammalian bite wounds are due to dogs, cat bites comprise 5-10% and approximately 3% are human bites. About 1 in 2 people will be bitten by a mammal during their lifetime.

The management of bite wounds differs from that of other wounds in a few important ways, most of which relate to the prevention and/or treatment of bite-related infections. With a multitude of pathogens in the mouths of every mammal, the most common complication of bite wounds is infection. These infections can be local (e.g. cellulitis, abscess) or systemic (e.g. hepatitis, encephalitis). Thus the assessment and treatment of bites wounds must include components of care that address the risk of bite-related infections.

CLINICAL ASSESSMENT

History

- time and circumstances of bite
- symptoms
- information about the biting mammal regarding the risk of transmission of systemic infections (e.g. rabies, hepatitis, tuberculosis, HIV)
- co-morbid conditions (immunocompromised state)
- allergies
- tetanus status

Physical Examination

- assess wound for direct tissue damage

- skin, tendon, neurovascular structures, bone (a dog's jaws can exert a force of up to 450 psi)
- note the type of wound as they differ in their risk of infection
- Types of wounds in order of increasing risk of infection: abrasions, lacerations, puncture wounds, crush injuries

X-rays

- when possibility of bony injury or to rule out gas-producing infections
- always get skull films in children with scalp bite wounds (+/- CT head) to rule out cranial perforation

Wound Cultures

- only indicated when there is clinical evidence of a wound infection (e.g. progressive erythema, purulent discharge, signs of sepsis)
- otherwise, the results can be misleading
- always let the lab know that the source is a bite wound (and which mammal)

WOUND MANAGEMENT

Good early initial wound care is the single most important factor in preventing bite-related infections.

Bite wounds should be thoroughly cleansed as soon as possible with copious irrigation. An 18 - 20 gauge angiocath on a 20 cc syringe will provide adequate irrigation pressure for most wounds. Irrigate only exposed tissue in puncture wounds, avoiding injection of fluid into tissues as this can result in hydrodissection along tissue planes, potentially spreading infection. There is no proven value in opening or widening puncture wounds for irrigation.

Crushed and devitalized tissues should be debrided. Debridement significantly decreases the risk of infection and optimizes the cosmetic and functional repair of crush wounds.

To suture or not to suture?

Certain bite wounds are at higher risk of infection if they are sutured. The risk of wound infection is related to the vascularity of the tissue. Vascular tissues such as the face, scalp and perineum are much less likely to get infected than less vascular areas such

as pretibial regions, hands and feet.

The decision to suture a bite wound is based on the risk of infection as well as cosmetic and functional considerations. Uncomplicated lacerations not at high risk for infection (see Table 1) can be closed primarily if treated early with copious irrigation. Wounds at high risk for infection or those already infected should not be sutured. A sterile dressing should be applied and the patient should be followed up closely. There is the option of a "secondary" or "delayed" repair, which refers to wound closure after 3 - 5 days of successful treatment that usually includes antibiotics.

BITE WOUND INFECTIONS

The most common complication of mammalian bite is infection.

Table 1. High Risk Criteria for Infection

Wound Factors	Patient Factors
<ul style="list-style-type: none"> puncture wounds crush injuries wounds greater than 12 hours old hand or foot wounds wounds near joints 	<ul style="list-style-type: none"> immunocompromised patient: diabetes, splenectomy, chronic alcoholism, AIDS, on immunosuppressive meds patient age greater than 50 years prosthetic joints or valves

DOG BITES

Reported bite wound infection rate: 10-30% (compare to < 5% for clean, non-bite lacerations). Which dogs bite?

- more aggressive breeds i.e. German shepherds, Pit Bull terriers, Doberman pinschers
- unneutered young male dogs

CAT BITES

- reported bite wound infection rate: 10 - 50%
- wounds can look relatively innocuous *but* long slender teeth cause deep puncture wounds
- full thickness puncture wounds are 3 times more likely to get infected than partial thickness wounds
- because cats often lick their paws, similar infections can result from scratches

HUMAN BITES

- reported bite wound infection rate: up to 50%
- high rate of infection is likely because there is often a delay in seeking treatment
- history of human bite often denied - be suspicious
- common sites: scalp (particularly in children), nose, ear, hand, forearm, breast and genitalia
- if portion of tissue bitten off, instruct ambulance crew or police to bring all tissue to hospital
- human bites may also transmit hepatitis B, tuberculosis, or syphilis
- transmission of HIV through a human bite is rare but has been reported

Human Bite Clenched Fist Injury

Mechanism

- tooth meets fist and penetrates the thin MCP joint capsule > may lacerate extensor tendon
- hand opens, extensor tendon retracts, sealing puncture site creating an anaerobic environment
- tell-tale laceration may be proximally retracted
- inoculation may extend widely along fascial planes of the hand
- high potential for serious complications: septic arthritis, osteomyelitis
- X-rays: skyline view of the MCP joint more sensitive for joint damage

CLINICAL PEARL

Any penetrating injury in the vicinity of a MCP joint should be considered a Human Bite Clenched Fist Injury until proven otherwise.

Treatment

These injuries require aggressive therapy. This begins with early copious irrigation +/- debridement. Sutures should be avoided. If seen within 24 hours and there are no overt signs infection, outpatient management with antibiotics may be appropriate. In these cases the patient's hand should be immobilized in a bulky gauze hand dressing. The patient should be instructed to soak the hand QID and keep it elevated as much as is practical. Close follow-up is essential. If there are signs of infection at presentation, start iv antibiotics. A surgical consultation should be arranged for consideration of formal debridement of the joint in the OR. Formal debridement is recommended for all patients exhibiting pus who present later than 24 hours after the bite.

MICROBIOLOGY OF BITE WOUNDS

- bite wounds involve many different species of bacteria and are often polymicrobial
- Staph & Strep are often involved
- gram negatives and anaerobic organisms are also common

Bacteria Worthy of Special Mention

Pasteurella multocida

- a nonmotile pleomorphic gram negative rod
- present in flora of virtually all animals
- causative organism in up to 80% of cat bite infections and up to 50% of dog bite infections
- infections usually become clinically evident within 24 hours (other bite wound infections tend to manifest after 24 hours)
- propensity to cause metastatic infections: septicemia, osteomyelitis, tenosynovitis, meningitis
- penicillin is drug of 1st choice
- also sensitive to doxycycline, TMP-Sulfa (Septra), 2nd generation cephalosporins, ciprofloxacin and fluoroquinolones
- not covered by cloxacillin, clindamycin, erythromycin, cephalexin, or aminoglycosides

Capnocytophaga canimorsus

- gram-negative rod, opportunistic
- normal flora in dogs and cats
- can cause an overwhelming sepsis, multisystem failure, and DIC
- case fatality rate 25% - fatalities usually have predisposing (immunocompromising) conditions (e.g. splenectomy)
- special culture requirements
- sensitive to: penicillin (1st choice), tetracycline, erythromycin, clindamycin, 2nd & 3rd generation cephalosporins, and ciprofloxacin

Eikenella corrodens

- anaerobic gram-negative rod - requires special medium for culture
- often involved in human bite wound infections
- covered well by penicillin, ciprofloxacin, clavulin, and septa
- not covered by erythromycin, cloxacillin, clindamycin or keflex

ANTIBIOTIC THERAPY

Prophylactic Antibiotics for Mammalian

Bite Wounds

The value of prophylactic antibiotics for fresh, uninjected bite wounds has the subject of much debate in the literature over the past 30 years. The current weight of evidence does not support the routine use of prophylactic antibiotics for all bite wounds. However, prophylactic antibiotics are indicated in high-risk situations (see Table 1).

Prophylactic regimes usually consist of full doses for 3 to 5 days. To optimize the effect of antibiotics used to prevent bite wound infections, an appropriate agent must be selected and it should be administered as soon as possible. In very high-risk situations, the initial dose is most effective when given intravenously.

Antibiotic Choice

The recommendations below are appropriate for bite wound prophylaxis or treatment of established infections. They are good choices for dog, cat and human bites.

1st line:

- amoxacillin-clavulanic acid (Clavulin)

2nd line:

- 2nd generation cephalosporins
(e.g. cefuroxime, cefaclor, or cefoxitin iv)

3rd line

combination therapy:

clindamycin PLUS
fluoroquinolone* or doxycycline* or Septra

* avoid in pregnant women and children

- Do not use erythromycin, cloxacillin or cephalexin

ADMISSION CRITERIA

- severe injuries (e.g. cranial perforation, penetrating injuries of tendons, joints, CNS, open fractures)
- severe infections (e.g. osteomyelitis, meningitis, septicemia)
- infections in high risk patients (e.g. immunocompromised)
- not responding to oral therapy
- Patients with bite wound infections that are ill enough to require hospital admission warrant an infectious disease (ID) consultation, as these infections can be complex

TETANUS

Do not forget to administer tetanus prophylaxis as per the tetanus guidelines. Tetanus infections do complicate mammalian bite wounds (there are twice as many cases of human tetanus from animal bites as there are rabies). The spores of Clostridium tetani are ubiquitous in soil, and on the teeth and in the saliva of animals, so there is a risk of tetanus from any animal bite that penetrates the skin.

Remember that all wounds other than clean, uncontaminated are “tetanus prone,” so most mammalian bites are indeed tetanus prone [definition: deep (>1 cm), more than 6 hrs old, crushed, contaminated, or infected wounds]. This means most bite victims should receive tetanus vaccine (Td) if they have not had a booster in the last 5 years, and if they have not had their primary series, they should get tetanus immune globulin (TIG). Note that the risk of inadequate prior immunization is particularly high for elders or persons raised in underdeveloped countries.

RABIES

Rabies is a rhabdovirus infection of the CNS that is almost always fatal. In the U.S. and Canada, wild animals are by far the most important vectors of rabies, accounting for over 80% of cases. Since 1980, over 80% of cases in humans have been caused by bat bites. Human rabies is rare in Canada, with only 3 cases reported since the year 2000. All 3 of these cases were due to bat rabies, and there was no clear history of a bat bite in any of these cases.

Worldwide, particularly in less well-developed countries, domestic animals account for over 95% of all human rabies; most of these are from dogs.

The rabies virus is transmitted via the saliva or CNS material of infected animals. It enters unmyelinated sensory nerves at point of inoculation, travels via retrograde axoplasmic flow at 8 to 20 mm/day along the nerves to the posterior columns of the spinal cord, then enters the brain where it causes a fatal encephalitis.

Animals can be carriers for several days before manifesting signs of the disease (e.g. drooling, aggressive behavior). The average incubation period in humans is 30 to 60 days (range: 10 days to 1 year). Common wildlife sources in North America: foxes, skunks, bats and raccoons. Common domesticated animal sources in North America: cattle, dogs, cats, sheep and horses.

Additional Points:

- Rabies is rare in rodents (rats and squirrels), rabbits, birds, and reptiles.
- Travelers to areas where canine rabies is endemic are at significantly increased risk (e.g.: Thailand, parts of India, Africa, Central and South America).
- A bite exposure is defined as any penetration of the skin by the teeth of an infected animal.
- Bat bites may not be felt and may leave no visible bite marks.
- Non-bite exposures can occur when there is contamination of open wounds or mucous membranes with the saliva (e.g. lick of an open wound) or other potentially infectious material (such as CSF or brain tissue, as may occur in dressing out carcasses) of a rabid animal.
- A few cases of rabies have been attributed to aerosol exposures (2 in lab setting, a few from within caves containing millions of bats).
- Petting or handling of an infected animal (other than a bat), and exposure to low-risk body fluids (e.g. blood, urine, stool) are not significant exposures and do not require immunoprophylaxis.

Rabies Postexposure Immunoprophylaxis

Consult with Public Health/Animal Control if there is *any* concern about rabies. They will be able to give you up-to-date information on the possibility of rabies infection in the biting animal, and assure close follow-up to rule out rabies when the animal can be identified. They will arrange for observation of the animal when necessary. Since 1990 in Ontario, it has been mandatory to report any animal bite or other animal contact that may result in rabies.*

(* REGULATION 557 of Health Protection and Promotion Act)

Postexposure Immunoprophylaxis is most effective when provided within 48 hours.

Persons not previously immunized:

- Passive immunization with Rabies Immune Globulin (RIG): 20 IU/kg
- Active immunization with rabies vaccine (e.g. HDCV): 5 injections of 1 ml IM over 28 days, given on days 0, 3, 7, 14, and 28
- Always administer rabies vaccine into deltoid. Treatment failures have been documented when it has been administered elsewhere.
- The WHO recommends that as much as possible of the dose of RIG should be infiltrated into, and around the wound(s) as is anatomically feasible. Any remaining RIG should be given IM. If there

are extensive wounds, it is advisable to dilute the RIG in saline to make up an adequate volume for wound infiltration.

Persons previously immunized:

- 2 doses of HDCV 1 ml IM (into deltoid) on days 0 and 3. RIG should not be administered.

Rabies Treatment Notes

Low risk domestic animal:

- observe animal x 10 d
- treat at first sign of rabies

Wild animal:

- treat as rabies exposure unless animal available for testing
- Fluorescent Antibody Test (FAT) results can usually be obtained within 24 hours

Livestock & small animals (e.g. squirrels, rodents):

- consult with public health
- err on the safe side if in doubt, especially if the animal's behaviour was unusual
- if available, can sacrifice the animal for FAT testing

Prompt postexposure prophylaxis is indicated in the following situations:

- head or neck bites, particularly in a child
- corticosteroid use or other immunosuppressed state
- victim bitten in high risk area for dog or cat rabies

If rabies is a strong concern, do not suture the wound as this promotes viral replication.

HDCV adverse reactions occur in about 25% of patients. They are usually minor local or systemic reactions. Anaphylaxis and neuroparalytic reactions are rare. Corticosteroids should be avoided if at all possible in treating reactions as they may inhibit the development of active immunity to rabies.

RIG & HDCV can be used in pregnancy when indicated.

Special Note Regarding Bat Exposure

Bat bites may be tiny and go unrecognized. In the past, postexposure prophylaxis has been recommended in all situations in which there is a reasonable possibility that contact with a bat has occurred, including when a bat is found in the same room as a sleeping person, an unattended child or disabled person.

With more recent information on the low incidence of

rabies in bats and the cost/benefits of the approach above, the current recommendation in Ontario is that postexposure prophylaxis should be administered for bat exposures only when:

- A bat bite or scratch has occurred
OR
- When there is direct contact with a bat (the bat should be observed to touch the person) *AND* the following cannot be eliminated: a bat bite or scratch, or saliva from the bat entered an open wound or mucous membrane.

Because of the subtleties of bat exposures, it is highly advisable to consult with Public Health in all cases of bat exposures.

SUMMARY

- Irrigation, debridement, sutures, X-rays, tetanus immunization, rabies prophylaxis and antibiotics are important aspects of management to consider for each patient with a bite wound
- Bites are at high risk for infection
- Clavulin is the 1st line antibiotic for most mammalian bites
- Consult Public Health for all mammalian bites where there is any possible risk of rabies

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37: HEAT EMERGENCIES

Joel Lexchin

OBJECTIVES

1. To understand who is at risk of a heat emergency
2. To diagnose the various types of heat emergencies
3. To differentiate between the two types of heat stroke
4. To understand the approaches to treatment and be able to initiate treatment protocols

INTRODUCTION

Heat related injuries are more common than one might expect in a country more known for its winters than its summers, but heat emergencies are not just the product of high environmental temperatures. They are influenced by the drugs that patients are taking, by their psychiatric states and by the type of activity that they are undertaking, among other factors. The purpose of this chapter is to present the mechanisms by which heat emergencies take place and discuss general methods of treatment for the various levels of heat emergencies that physicians in an emergency department will encounter.

MEANS OF HEAT TRANSFER

There are four different methods by which heat is exchanged between the body and the environment:

1. Conduction
transfer of heat by direct physical contact
2. Convection
heat loss to air and water vapour molecules circulation around the body
3. Radiation
heat transfer by electromagnetic waves
4. Evaporation
conversion of liquid (sweat) to a gas

Evaporation is the major mode of cooling in hot conditions.

WHO IS AT RISK?

There are four different populations at risk for heat related emergencies:

1. Athletes and military recruits engaged in strenuous activities
2. Elderly
3. People at risk for dehydration

- infants because of their high surface to mass ratio
- individuals taking diuretics
- individuals with poor fluid intake e.g. elderly, those with nausea and vomiting, etc.
- 4. People on certain medications that interfere with sweating or that alter the response of the hypothalamus
 - anticholinergics
 - phenothiazines
 - tricyclic antidepressants
 - MAO inhibitors

HEAT ILLNESSES

Heat related illness are divided into three categories – minor, heat exhaustion and heat stroke.

Minor

Heat Cramps

These are brief intermittent and often severe muscular cramps occurring in muscles that are fatigued by heavy exertion. Heat cramps tend to occur after exercise when the victim has stopped working and is relaxing. They are caused by copious sweating during exertion and excessive hypotonic fluid replacement during exertion leading to salt deficiency.

The treatment for heat cramps is oral salt solution made by dissolving 1/4 to 1/2 teaspoon salt in a quart of water and rest in a cool environment

Heat Edema

Heat edema is usually characterized by minimal edema of the feet and ankles. The main reason for diagnosing this condition is to avoid costly and invasive investigations of other causes of edema. Diuretics are not indicated and the usual treatment is simply leg elevation and support stockings as needed.

Heat Syncope

There is a predilection for this type of syncope in the elderly. The cause is a combination of cutaneous vasodilation, pooling of blood in the lower extremities due to prolonged standing and volume loss. The condition is self-limiting once the person becomes horizontal. In examining a patient with heat syncope, be careful to assess for injuries secondary to the fall.

Prickly Heat

Prickly heat is caused by the blockage of sweat gland pores by a macerated stratum corneum. The acute phase presents with intensely pruritic vesicles on an erythematous base and is usually confined to clothed areas of the body.

Heat Exhaustion

The presenting symptoms of heat exhaustion are vague malaise, fatigue, headache, intense thirst, weakness and anxiety. The core temperature is often normal and if it is elevated, the temperature is less than 40°C. Most importantly, mental function is intact.

Signs of heat exhaustion include tachycardia, orthostatic hypotension and clinical dehydration may be present.

No blood work is necessary for mild cases, and for more serious ones the usual blood work includes CBC, electrolytes, creatinine, glucose and BUN.

Since heat exhaustion is primarily a volume depletion problem, rapid recovery generally follows fluid administration. For mild cases an oral salt solution may be used while for more serious cases use intravenous normal saline. In addition the patient should rest in a cool environment. If there is any doubt about whether the diagnosis is heat exhaustion or heat stroke, treat the patient for heat stroke.

Heat Stroke

Heat stroke is a medical emergency with mortality as high as 40%. In 80% the onset is sudden, while in the other 20% there are nonspecific prodromal symptoms lasting minutes to hours that may include weakness, dizziness, nausea, vomiting, anorexia, headache and confusion.

The major feature of the diagnosis includes signs of severe CNS dysfunction—usually coma, seizures or delirium. However CNS symptoms may be subtle, manifesting only as inappropriate behaviour or impaired judgement. Core temperature is typically >41°C, but may be lower. Temperature should be obtained with a rectal, esophageal or bladder probe—the esophageal reading is most accurate. The skin is hot and dry, but sweating may persist.

There are 2 different forms of heat stroke—classic heat stroke (CHS) and exertional heat stroke (EHS). Table 1 highlights the important differences between the two forms.

DIFFERENTIAL DIAGNOSIS

- CNS injury or infection
- cerebral falciparum malaria
- thyroid storm
- drug-induced heat illness (e.g. amphetamines, cocaine)
- infectious diseases such as typhoid fever and typhus
- delirium tremens
- hypothalamic hemorrhage
- pheochromocytoma
- neuroleptic malignant syndrome

TREATMENT (See Table 2)

Assess the ABCs.

If heat stroke is suspected, but the diagnosis is in doubt, treatment should be initiated while considering other diagnoses. Cooling will not harm patients who are hyperthermic from other causes.

Effective heat dissipation depends on the rapid transfer of heat from the core to the skin, and from the skin to the external environment. The transfer of heat from the core to the skin is facilitated by active cutaneous vasodilatation, and therapeutic cooling techniques are therefore aimed at accelerating transfer of heat from the skin to the environment, without compromising flow of blood to skin.

Immediate Cooling (See Table 3) should be initiated with ice-water immersion:

- evaporative cooling using large circulating fans and skin wetting (atomized shower of water at temperature of 15°C)
- ice packs to axillae, groins, neck
- internal cooling methods (cold water irrigation of stomach, rectum, peritoneal lavage) are rarely used

CLINICAL PEARLS

ASA and acetaminophen are not indicated in heat emergencies, as the thermoregulatory control centre has not been reset.

There is no evidence to support a specific temperature end point at which cooling should be stopped. A rectal temperature of 39.5°C has been used in a large series. The goal is to

avoid overshooting as this may trigger shivering and impede ongoing cooling efforts (shivering can be treated with diazepam or chlorpromazine).

ASA and acetaminophen are not indicated as the thermoregulatory control centre has not been reset. Dantrolene was found ineffective in a double-blind randomized study.

Although hypotension is common, initial management is cooling (not fluids), as most patients are relatively normovolemic, peripherally vasodilated and suffering from distributive shock, high output failure and severe dehydration.

The average intravenous fluid need is about 1 litre per hour for 4 hours using either Ringer's lactate or saline. If hypotension persists after the use of cooling techniques and fluids, insert a central line. Vasopressors should be used with caution, as the resultant vasoconstriction may retard heat loss.

Output should be monitored with a foley catheter, and temperature, with a rectal probe.

Blood work consists of: CBC, electrolytes, BUN, glucose (using both the dextrostix and laboratory methods), LFTs, lactate, INR, PT, PTT and blood gases. (It is unclear if blood gases should be corrected for temperature.)

There should be monitoring of: coagulation parameters (to watch for development of coagulopathy), electrolytes, osmolarity, CK and myoglobin (to watch for rhabdomyolysis)

Recovery of central nervous system function during cooling is a favourable prognostic

sign and should be expected in the majority of patients who receive prompt and aggressive treatment. Residual brain damage occurs in about 20% of patients and is associated with a high mortality rate.

SUMMARY

Risk factors

- strenuous activity
- age
- dehydration
- medications

Heat stroke

- medical emergency
- major feature is altered mental status
- obtain core temperature with rectal, esophageal or bladder probe
- classic versus exertional heat stroke
- treat with immediate cooling

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Table 1: Characteristics of Heat Stroke

Characteristic	Classic	Exertional
Age group	Elderly	Men (15-45)
Health status	Chronically ill	Healthy
Activity	Sedentary	Strenuous exercise
Drug use	Anticholinergics, diuretics, anti-psychotics, anti-hypertensives, anti-depressants	Usually none
Sweating	Usually absent	Often present
Lactic acidosis	Usually absent, poor prognosis if present	Common, may be marked
Rhabdomyolysis	Unusual	Frequently severe
Hyperuricemia	Modest	Severe
Acute renal failure	<5%	25-30%
Hypocalcemia	Uncommon	Common
DIC	Mild	Marked
CPK	Midly elevated	Markedly elevated
Hypoglycemia	Uncommon	Common
Mechanism	Poor dissipation of	Excessive endogenous

Table 2: Treatment of Heatstroke

Condition	Intervention	Goal
Hyperthermia	Monitor rectal and skin temperatures; continue cooling	Keep rectal temperature <39.5°C and skin temperature 30°C to 33°C
Seizures	Give benzodiazepines	Control seizures
Respiratory failure	Consider elective intubation (for impaired gag and cough reflexes or deterioration of respiratory function)	Protect airway and augment oxygenation (arterial oxygen saturation >90%)
Hypotension	Administer fluids for volume expansion, consider vasopressors, and consider monitoring central venous pressure	Increase mean arterial pressure to >60 mm Hg and restore organ perfusion and tissue oxygenation
Rhabdomyolysis	Expand volume with normal saline and administer intravenous furosemide, mannitol and sodium bicarbonate	Prevent myoglobin-induced renal injury; promote renal blood flow, diuresis, and alkalization of urine
Electrolytes	Monitor serum potassium and calcium levels and treat hyperkalemia	Prevent life-threatening cardiac arrhythmia

Source: NEJM 2002;346:1978-88

Table 3: Methods of Cooling**Techniques based on conductive cooling**

External*	Internal†
Cold-water immersion Application of cold packs or ice slush over part of the body or the whole body Use of cooling blankets	Iced gastric lavage Iced peritoneal lavage

Techniques based on evaporative or convective cooling

Fanning the undressed patient at room temperature (20°C to 22°C)
Wetting of the body Surface during continuous fanning‡
Use of a body-cooling unit§

*Because external cooling results in cutaneous vasoconstriction, vigorous massage of the skin is recommended

†Internal cooling, which has been investigated in animals, is infrequently used in humans. Gastric or peritoneal lavage with ice water may cause water intoxication.

‡The skin is covered with a fine gauze sheet that has been soaked in water at 20°C while the patient is fanned. The fanning is reduced or stopped if the skin temperature drops to <30°C.

§A body-cooling unit is a special bed that sprays atomized water at 15°C and warm air at 45°C over the whole body surface to keep the temperature of the wet skin between 32°C and 33°C.

Source: NEJM 2002;346:1978-88

38: BURNS

Laura Hans

OBJECTIVES

1. To review the classification of burns
2. To identify high risk patients including inhalation injury, electrical and chemical burns with ocular injury
3. To review initial burn management including fluid resuscitation and wound care
4. To recognize criteria for transfer to a burn centre

INTRODUCTION

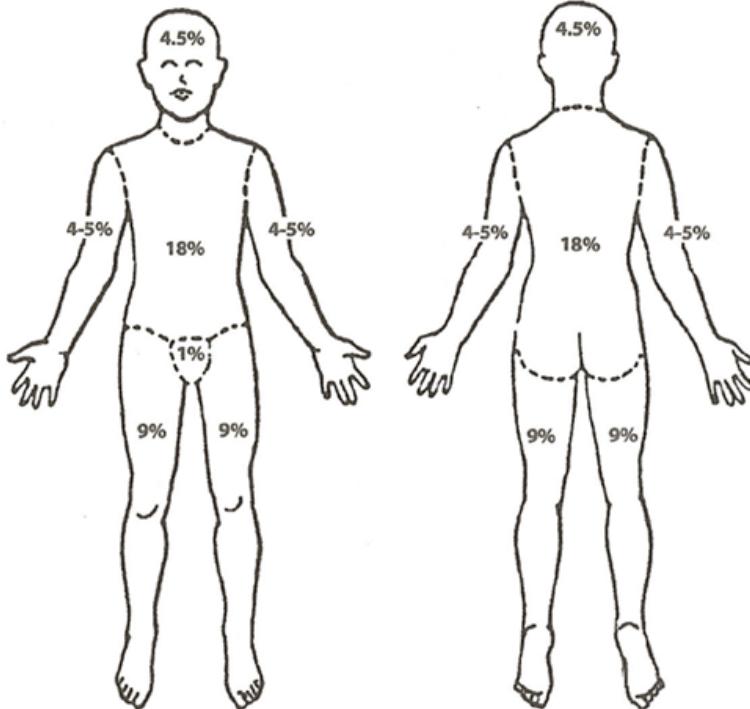
Burns are a common presenting concern in the Emergency Department. Most patients are treated and discharged for follow up with their health care providers. A subset of patients is admitted to burn centres to receive specialized care. Patients can present with seemingly minor injuries and quickly deteriorate with airway compromise and metabolic derangement. Mortality increases with larger burn size, older age with the presence of comorbidities and an inhalational injury. Careful

consideration should be given to children presenting with burns to ensure that it has not resulted from abuse or neglect.

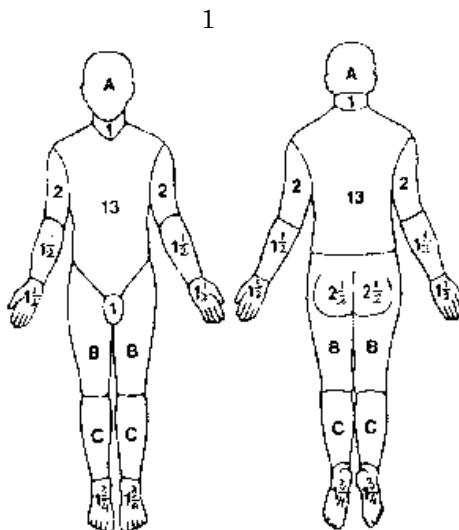
BURN CLASSIFICATION

Burn size is quantified as the percentage of body surface area (BSA) involved. There are 3 main methods to determine the size of a burn:

1. **Rule of nines:** Dividing the body into segments that are approximately 9%, or multiples of 9's. The perineum makes up the last 1%. In infants, the rule of nines must be modified to accommodate for a larger head and smaller legs. This allows for a quick estimation of the BSA affected. (Figure 1)
2. **Patient's hand:** The size of a burn can be estimated by using the back of a patient's hand. The area represents approximately 1% BSA.
3. **Lund and Browder burn diagram:** A more precise determination can be done with this diagram that is also age adjusted. (Figure 2)



Figure



LUND-BROWDER CHART
Relative Percentage of Body Surface Area Affected by Growth

Age in years	0	1	5	10	15	Adult
A-head (back or front)	9½	8½	6½	5½	4½	3½
B-1 thigh (back or front)	2¾	3¼	4	4¼	4½	4¾
C-1 leg (back or front)	2½	2½	2¾	3	3¼	3½

Figure 2

Burn Depth has historically been described as first, second, third or fourth degree. Burns are currently described both with the older terminology along with the classification superficial partial thickness, deep partial thickness and full thickness.

- Superficial (first degree) involves epidermis only i.e. sunburn. Skin is painful and tender with no blister formation. Healing usually occurs within 1 week without scarring.
- Superficial partial thickness (second degree) extends to epidermis and superficial dermis, blister formation occurs and the burns are very painful. Scarring may occur and healing time is approximately 2-3 weeks.
- Deep partial thickness (second degree) involves hair follicles, sebaceous glands. Skin is blistered and exposed dermis is pale white to yellow in colour. Sensation is absent. Healing is prolonged, up to 2 months. Scarring is common. Surgical treatment may be required for grafts or improvement of function depending on the affected site.
- Full thickness burns (third degree) involve the epidermis and all dermal layers. The skin is

pale, insensate and charred or leathery. Surgical treatment is required, spontaneous healing does not occur. Scarring will occur in all cases of full thickness burns.

- Fourth degree burns extend through skin to all underlying layers e.g. fat, muscle, and bone. Amputation or reconstruction is often required.

INHALATIONAL INJURY

Victims exposed to fire in a closed space are at risk for inhalational injury. There is increased mortality associated with this type of injury. Patients are acutely at risk of an upper airway obstruction from mucosal edema and later can develop bronchospasm and acute respiratory distress syndrome (ARDS). Expose to toxic inhalants should be suspected: carbon monoxide (CO) and hydrogen cyanide presenting with altered level of awareness and metabolic acidosis.

Inhalational injury should be assumed in patients presenting with facial burns, singed nasal hair, soot in the mouth, nose or sputum, hoarseness or

stridor, wheezing or dyspnea. 100% oxygen should be started by mask followed by consideration for early intubation.

Arterial blood gases should be drawn and bloodwork to determine CO level. The half life of CO with room air is 240-320 minutes, with 100% O₂, 90 minutes and hyperbaric oxygen, 20 minutes. Transfer to a hyperbaric chamber is indicated in those with levels >25 or 20 if pregnant, amongst other criteria.

BURN MANAGEMENT

The patient is managed first with attention to the “ABC’s”. If multisystem injury is suspected the patient should have spinal precautions in place and a cervical collar on. In cases of inhalational injury prompt intubation should be considered. Additional issues related to burn treatment are: cooling of the affected area, fluid resuscitation, wound care, disposition-transfer to a specialized burn centre.

Cooling should start in the prehospital setting. This may involve immersion in cool water for 30 minutes or application of cool compresses. Ice should not be used. In patients with large BSA burns the patient should be covered in dry wraps or sterile drapes to prevent hypothermia. All adherent and non-adherent clothing should be removed. Constricting jewelry should be removed.

Fluid resuscitation is based on the Parkland formula. It is indicated for burns equal or larger than 20% BSA, partial thickness or greater. The formula is: **4cc x wt (kg) x %BSA during the first 24 hours**. Time zero is taken from the time of the burn, not from the arrival in the ED. Half of the fluid is administered in the first 8 hours and the second half in the following 16 hours. Ringer's lactate is the preferred choice of fluid. In those with inhalational injuries, fluids resuscitation is carefully monitored to limit progression to ARDS. Importantly, the formula is meant as a guide only. The progress of the patient, i.e. urine output, needs to be monitored to resuscitation.

Pain control should start immediately in the ED. Burns are very painful and often IV analgesia is required.

Wound care: After cooling, burns are covered with a gauze dressing and polysporin ointment for superficial partial thickness and flamazine

(silver sulphadiazine) for deep partial thickness burns (except on the face). Blisters can be left intact in the ED setting, but may be debrided at a later time, especially those that are large or occur over joints. If burns are deep and circumferential vascular compromise of distal areas can occur. Escharotomy may be required. Most commonly affected areas are hands and fingers, forearm, chest and neck. A longitudinal incision through the burned area is made to the subcutaneous fat to allow tissue to expand.

Prophylaxis: No role for prophylactic antibiotics. Tetanus should be updated.

Disposition: Guidelines for transfer of patients to a specialized burn centres include (American Burn Association):

- Partial thickness or full thickness burns > 10% ages <10 or >50
- Partial thickness or full thickness burns > 20% in all others
- Partial-thickness and full-thickness burns involving the face, hands, feet, genitalia, or perineum or overlying major joints
- Full-thickness burns greater than 5% BSA in any age group
- High voltage electrical burns, including lightning injury
- Significant chemical burns
- Inhalational injury
- Burn injury in patients with comorbidities that could complicate management, prolong recovery, or affect mortality.
- Burn patient in whom concomitant trauma poses an increased risk or morbidity or mortality may be treated initially in a trauma center until stable before transfer to a burn center
- Children with burns in centres without appropriate personnel or equipment
- Patients requiring social, emotional or long-term rehabilitative support

SPECIAL CONSIDERATIONS

Electrical burns

Electrical injuries are divided into high (>1000 volts) and low (<1000 volts) voltage. Exposure to high voltages can result in specific types of injury:

- Cardiac dysrhythmias: occurs at time of injury asystole or ventricular fibrillation, will not develop in the ED

- Deep muscle injury and necrosis: damage underestimated by the Parkland formula, may lead to fasciotomy and/or amputation
- Rhabdomyolysis: if extensive muscle damage is suspected, aggressive fluid resuscitation should be part of initial management
- Compartment syndrome: presence of myoglobinuria and extensive burns increases the risk for development of compartment syndrome

Chemical burns

Chemical burns can be divided into alkali and acid. Acids cause coagulation necrosis and formation of a leathery eschar. Alkalies cause liquifaction necrosis and deeper burns. Aggressive irrigation is the main treatment for chemical burns. PH paper is used to determine the presence of additional chemical, ideally applied 10-15 minutes after irrigation is stopped. The principal of immediate irrigation is critical in the care of an ocular burn. A strong alkali can penetrate the cornea, anterior chamber and retina causing blindness. Penetration of an alkali can result in globe perforation. The affected eye should be irrigated with 1-2 L of NS continuously for at least 30 minutes. Acids may

not require as much irrigation. Ophthalmology should be consulted for a corneal burn.

SUMMARY

- Burn classification is based on assessment of size and depth
- Increased mortality is seen older patients, especially with comorbidities, in those with inhalation injuries and large burn size
- Intubation should be considered in those with an inhalation injury
- Remember to order carboxyhemoglobin and screen for developing metabolic acidosis
- Fluid resuscitation is based on the Parkland formula
- Specific patients may require transfer to a burn centre

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39: HYPOTHERMIA & COLD INJURIES

Joel Lexchin

OBJECTIVES

1. To understand the risk factors for cold related emergencies
2. To be able to recognize the signs and symptoms of hypothermia
3. To diagnose the four different types of cold related injury
4. To describe the different methods for treating hypothermia
5. To understand the principles behind the treatment of frostbite

INTRODUCTION

Because of the extremes of Canadian weather, cold related emergencies are relatively frequent problems in emergency departments. Individuals may present with a lowered core temperature (hypothermia) and/or peripheral cold injuries. By the end of this chapter the student should be able to:

- Understand who is at risk for cold related emergencies and why
- Be able to explain the mechanisms of heat loss
- Know the pathophysiology behind cold related injuries
- Diagnose the four different types of cold related injury
- Understand the different methods for treating hypothermia and under which circumstances each method should be used
- Understand the principles behind the treatment of frostbite

HYPOTHERMIA

Hypothermia is defined as a core body temperature of less than 35°C, but it is important to recognize that hypothermia does not only occur in cold weather.

Who is at risk?

Patients at the extremes of age

- neonates, because of their large body surface area
- elderly often lose their ability to sense cold and are often malnourished

- both groups have a limited ability to increase heat production and to conserve body heat (decreased capacity for vasoconstriction)

Individuals with altered sensorium

- alcoholics and people using drugs that interfere with central thermoregulation by impairing centrally mediated thermoregulation (for example, barbiturates, phenothiazines, tricyclic antidepressants and benzodiazepines).

Individuals with acute incapacitating illness

- for example, those with severe infections, DKA, immobilizing injuries

Individuals with metabolic abnormalities

- hypothyroidism, hypoadrenalinism, hypopituitarism, hypoglycemia

Immersion in cold water

Individuals with a deficiency in muscle and/or fat

- less body fat decreases tissue insulation, malnutrition decreases fuel available for heat generation

Individuals with peripheral and autonomic neuropathy (e.g. diabetics)

- peripheral neuropathy impairs cold-induced nociception that might trigger retreat from cold
- autonomic neuropathy impairs reflex peripheral vasoconstriction

MECHANISMS OF HEAT LOSS

The degree to which each of following components contribute to heat loss will depend on multiple variables including age, size of victim and elements to which victim exposed.

- Radiation is responsible for up to 55% of heat loss.
- Evaporation is responsible for 30% of heat loss; 25% from the skin and 5% from the airway.
- Conduction is responsible for 15% of heat loss and is especially important in victims of submersion as water has 25-30 times the thermal conductivity compared to air.

- Convection is a relatively minor component of heat loss unless the victim is exposed to a particularly cold and windy environment.

DIAGNOSIS

Be suspicious for hypothermia based on the circumstances of the patient. In order to help assess the patient's temperature, touch the trunk with your bare hands. The core temperature varies depending on the anatomical site at which the temperature is measured—the esophageal temperature gives best non-invasive reading while the rectal and tympanic temperatures lag behind esophageal temperature in nonsteady state conditions. Therefore, they are higher than esophageal temperatures during cooling but lower during warming. The most significant inaccuracy in temperature measurement occurs during transition from cooling to warming; the heart and esophageal temperature may be rising, but rectal temperature will continue to drop for up to 1 hour. When using a rectal probe, make sure it is designed to measure low temperatures.

SIGNS/SYMPOTMS

CNS

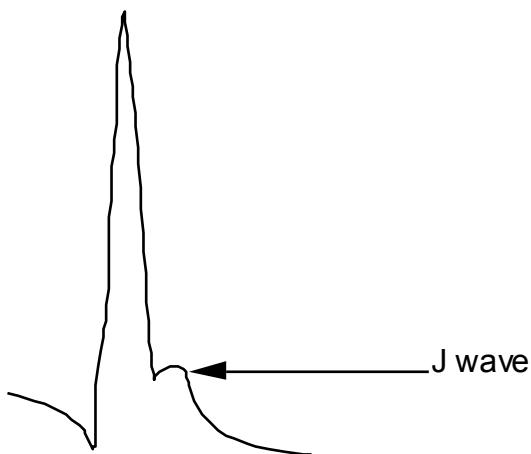
There is a progressive decrease in the level of consciousness proportional to the degree of hypothermia. Elderly patients may be confused, lethargic and uncoordinated.

Shivering ceases when body temperature falls below 30-32°C.

ECG

look for J (Osborn) waves

Figure 1. ECG Changes in Hypothermia



Respiratory

An initial tachypnea is followed by a progressive decrease in the respiratory rate and tidal volume.

Cardiac

Patients are at risk for arrhythmias at temperatures <30°C. The typical progression in rhythm is:

- sinus bradycardia ->
- atrial fibrillation ->
- slow ventricular response ->
- ventricular fibrillation ->
- asystole

The hypothermic myocardium is extremely irritable, and ventricular fibrillation may be induced by a variety of manipulations and interventions that stimulate the heart, including rough handling of the patient.

Peripheral pulses may be very difficult to palpate—allow at least 1 minute to detect pulses. If the patient has a perfusing rhythm, avoid CPR, as this may precipitate ventricular fibrillation.

Behavioural

Paradoxical behavior of burrowing in the snow and undressing are seen in up to half of patients dying from hypothermia. These behaviours seem to occur at core temperatures below 32°C where hypothalamic dysfunction sets in and patients experience a sensation of extreme warmth.

Stages of Systemic Cold Injury

Depending on the presenting signs and symptoms patients are put into three stages (see table below)

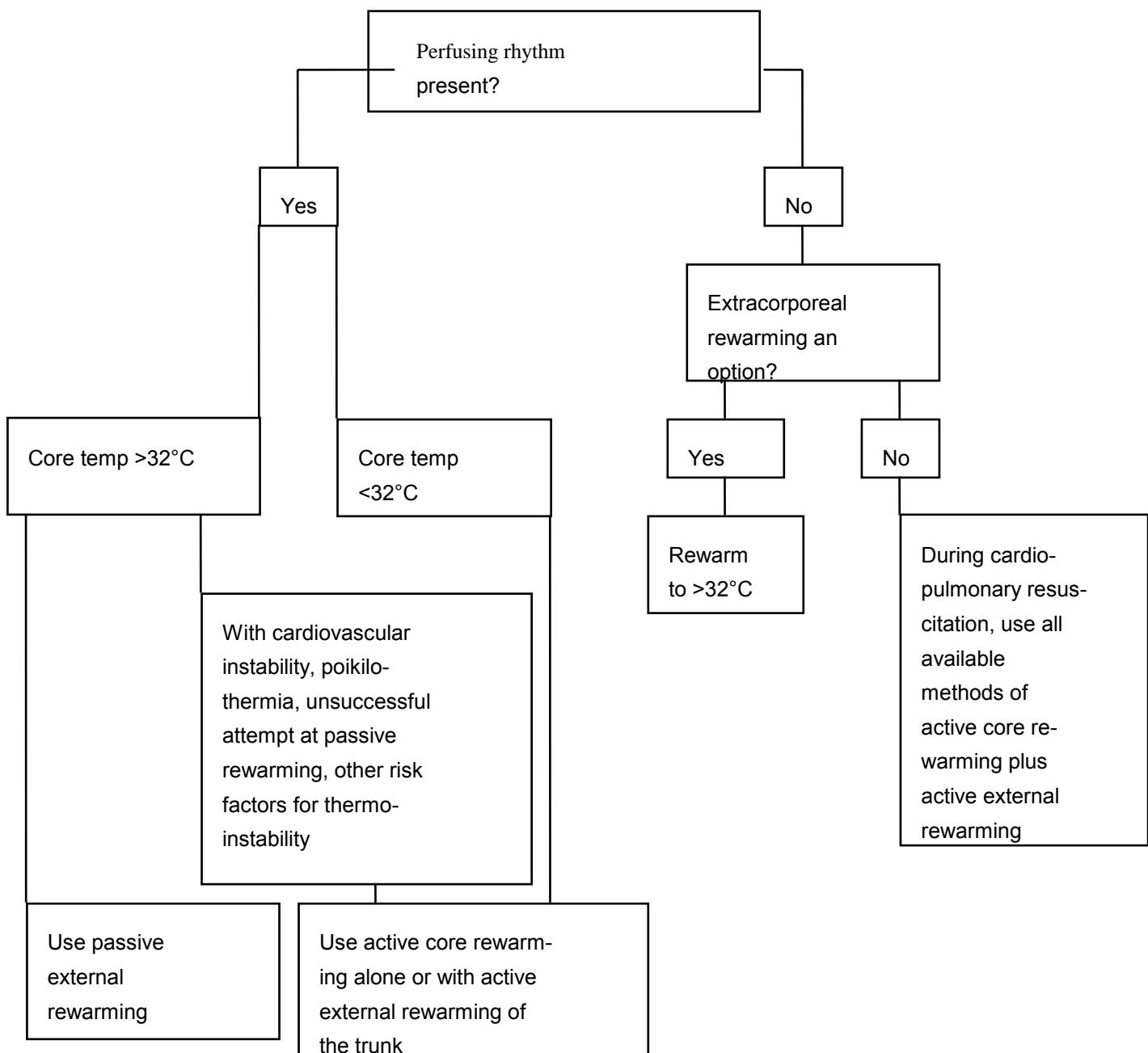
Stage	Features
1	Core temperature 35-37°C Strong shivering and piloerection Poor fine motor coordination; hands become numb Shallow breathing, fatigue, nausea, visual disturbance
2	Core temperature 32-34.9°C Violent shivering, pallor, distal cyanosis Poor gross motor coordination, stumbling Confusion despite alertness
3	Core temperature <32°C Cessation of shivering, reduced level of consciousness progressing to stupor Paradoxical behaviours such as burrowing and undressing Bradycardia and tachyarrhythmias, reduced respiration Cold diuresis, organ failures, death

Sheridan et al. New England Journal of Medicine 2009;361:2654-62

TREATMENT (See Algorithm)

- assess ABCs
- intubate if unable to protect airway
- check glucose (dextrostick) or else give bolus D50W empirically
- blood work: CBC, electrolytes, creatinine, total CK, INR, PT, PTT, blood gases
- maintain continuous temperature and cardiac monitoring

- assume dehydration and give fluid bolus of 250-500 cc normal saline
- hypotension not responding to infusions of normal saline should be treated with dopamine
- if there is no evidence of perfusion, assume the patient is in cardiac arrest and initiate CPR; make one attempt to defibrillate at 200 W-sec (a frozen thorax may make chest compression difficult)
- at temperatures below about 28°C pharmacotherapy (lidocaine, epinephrine) is generally not helpful
- begin rewarming (see algorithm below)

Table 1. Approach to Re-warming

METHODS OF REWARMING

Passive External Rewarming

This method can be used for mild hypothermia. It consists of putting patients in a warm environment and insulating them with warm blankets to minimize the normal processes of heat dissipation.

Active External Rewarming

The indications for this method are controversial. Optimal candidates are previously healthy with acute hypothermia e.g., from immersion in cold water, and they need to be hemodynamically stable.

This method may be associated with fatal arrhythmias due to 'afterdrop', which is a continued decrease in the deep core temperature after rewarming has started due to either the return of cold blood from peripherally vasoconstricted extremities, or conductive heat loss from the core to the cold periphery. To try and avoid afterdrop, heat should be applied only to trunk. The trunk can be heated through various possible methods:

- immersion in warm (40°C) water, hot water bottles, plumbed garments which recirculate warm fluids, heating pads or blankets, radiant heat sources.

Active Core Rewarming

Various methods are available and can be combined:

- treatment of choice for arrested hypothermic patients who have not sustained significant trauma is cardiopulmonary bypass extracorporeal shunt—hemodialysis, arteriovenous or venovenous
- administration of warm humidified O_2
- warm intravenous fluids
- warm saline delivered through nasogastric tubes, foley catheters, rectal tubes
- warm saline peritoneal lavage
- warm saline through closed pleural irrigation via large-bore thoracostomy tubes

CLINICAL PEARL

Active external warming of the hypothermic patient may be associated with fatal arrhythmias due to 'afterdrop', which is a continued decrease in the deep core temperature after rewarming has started due to either the return of cold blood from peripherally vasoconstricted extremities, or conductive heat loss from the core to the cold periphery. To try and avoid afterdrop, heat should be applied only to trunk.

PERIPHERAL COLD RELATED INJURIES (FROSTBITE)

PREDISPOSING FACTORS

At temperatures below -10°C , the wind speed increases the risk of cold-induced injuries. With a wind chill index of -25°C there is a risk of frostbite and with a wind chill index of -45°C exposed skin will freeze in minutes.

There is no data to suggest that age or sex affects an individual's susceptibility to peripheral cold injuries.

The risk of cold related injuries is enhanced by:

- prior cold injury which may produce autonomic dysfunction that sensitizes individuals to recurrent cold injuries
- conditions that impede peripheral circulation e.g., vasoconstrictive medication, nicotine, hypotension, atherosclerosis, arteritis, diabetes, immobility, constrictive clothing, cramped conditions, contact with metal
- conditions affecting judgement e.g., psychiatric disorders, alcohol ingestion, substance abuse

PATHOPHYSIOLOGY

Frostbite occurs when tissue temperature drops below 0°C . The tissue is damaged by both the freeze-thaw insult and subsequent progressive dermal ischemia. The degree of irreversible tissue damage is related to the length of time the tissue remains frozen.

There are two putative mechanisms of injury—architectural cellular damage from ice-crystal formation and microvascular thrombosis and stasis. In the prefreeze phase, the skin temperature drops below 10°C and cutaneous sensation is lost. In the freeze-thaw phase, the timing, location and rate of ice-crystal formation is dependent on the circumstances of the exposure. In addition to ambient temperatures, wind and moisture will increase the freezing rate.

When tissue thaws there is an intense inflammatory reaction that leads to capillary sludging and thrombosis often leading to permanent local vasculitis. Edema forms and slowly resolves and the final demarcation of tissue damage may not be evident for 60-90 days.

PRESENTATION

Symptoms usually reflect the severity of exposure. All patients have some initial sensory deficiency in light touch, pain or temperature and 75% will have numbness. A history of complete acute anesthesia in a painful cold digit suggests a severe injury. Significant pain usually accompanies the reestablishment of perfusion.

There are four different forms of peripheral cold injury.

Chillblains

- mild form of dry-cold injury often following a repetitive exposure to cold
- young females with a history of Raynaud's phenomenon are especially at risk
- usually affects facial areas and dorsum of hands and feet and is accompanied by pruritis, erythema and mild edema
- there is no specific treatment in the emergency department but nifedipine (20-60 mg) can be used daily for prophylaxis

Trench Foot (immersion syndrome)

- produced by prolonged exposure (>12 hours) to wet cold at temperatures above freezing
- initially the feet often appear cyanotic, cold and edematous with numbness and leg cramping
- after warming the skin remains erythematous, dry and very painful to touch
- bullae commonly develop and vesiculation proceeds to ulceration and liquefaction gangrene in severe cases
- protracted symptoms of pain during weight bearing, cold sensitivity and hyperhidrosis often last for years

Frostnip

- superficial cold insult, equivalent to a first degree burn, that is manifested by transient numbness and tingling that resolves after rewarming
- it does not represent true frostbite because there is no tissue destruction

Frostbite

- actual freezing of tissue has occurred and the frozen tissue often appear mottled or violaceous-white, waxy or pale yellow
- favourable presentation
 - normal sensation, warmth and colour
 - soft, pliable subcutaneous tissue
 - early formation of clear large blebs that extend to the tips of digits

- ominous presentation
 - in severe cases it will not be possible to roll the dermis over bony prominences
 - delayed appearance of small hemorrhagic blebs
 - a residual violaceous hue after rewarming is ominous
 - lack of edema may also suggest significant tissue damage (post-thaw edema usually develops in less than 3 hours)
- no prognostic features are entirely predictable to distinguish between a non-serious and serious injury and weeks may pass before a demarcation appears between viable and nonviable skin

Stages of local cold injury

Similar to systemic cold injury local cold injury can be divided into stages depending on the presentation (see table below):

Stage	Features
1.	burning and numbness; pallor warms to erythema
2.	insensate; pallor warms to blistering; perfusion after warming
3.	insensate, frozen; frozen warms to hemorrhagic blisters; variable perfusion or necrosis after warming.

Sheridan et al. New England Journal of Medicine 2009;361:2654-

TREATMENT

Rewarming

- in the prehospital setting, remove nonadherent wet clothing
- immerse the injured area in a warm (40-42°C) water bath, monitoring water temperature with a thermometer and continue rewarming until the part feels pliable and distal erythema is noted - usually requires 10-30 minutes of immersion
- avoid premature termination of rewarming (rewarming at lower temperatures is less beneficial to tissue survival and rewarming at higher temperatures may compound the injury by producing a burn wound)
- avoid thawing and refreezing at all costs
- avoid direct heat to the injured area as it will lead to thermal burns

Adjunctive therapy

- encourage active gentle movement of affected part by patient - avoid direct tissue massage
- use frequent parenteral analgesia to relieve pain associated with rewarming
- after the injured area has thawed, keep it elevated to minimize edema formation, apply sterile dressings and handle the involved areas gently
- there is no good consensus on how to manage frostbite blisters, they can be left intact, debrided or aspirated
- except for minor cases, all patients should be hospitalized—administer ibuprofen 400 mg every 12 hours to inhibit thromboxane A production by injured tissue and benzyl penicillin 600 mg every 6 hours
- give tetanus prophylaxis if necessary

SUMMARY

Hypothermia

- hypothermia does not necessarily require a cold environment
- risk factors include: age, altered sensorium, acute incapacitating illness, metabolic abnormalities, immersion in cold water, deficiency in muscle and/or fat, peripheral and autonomic neuropathy
- be suspicious for hypothermia depending on the condition – touch the patient with your bare hands, use a rectal temperature probe specifically designed to measure low temperatures
- cold myocardium is extremely irritable and ventricular fibrillation is easily induced
- for patients with a core temperature below 32°C use active core rewarming

Peripheral Cold Related Injuries

- the risk of these injuries is enhanced by prior cold injuries, conditions that impede peripheral circulation, conditions that impede judgement
- frostbite occurs when tissue temperature drops below 0°C and the tissue is damaged by both the freeze-thaw insult and subsequent progressive dermal ischemia
- there are four forms of injury: chillblains, trench foot, frostnip and frostbite
- the underlying principle behind the treatment of frostbite is continuous rewarming in a warm (40-42°C) water bath for between 10-30 minutes avoiding thawing and refreezing and direct heat to the area

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40: NEAR DROWNING

Andrew McDonald

OBJECTIVES

1. To understand who is at risk for near-drowning
2. To understand how near-drowning first affects the lungs and then secondarily other body systems
3. To understand how supportive care is used after a submersion event
4. To understand who needs to be observed in hospital and who can be discharged from the emergency department

INTRODUCTION

Drowning, defined as death by suffocation in a liquid or ‘the process of experiencing respiratory impairment from submersion/immersion in liquid’, occurs approximately 8,000 times per year in the USA and 450,000 times per year worldwide. There are approximately two cases of near drowning for every drowning death. Drowning is the third leading cause of accidental death in many countries; toddlers and teenagers having the highest incidence, highest at risk group age 1-4.

Certain factors have been identified which contribute to the risk of drowning. Use of alcohol or drugs around bodies of water, overestimation of swimming skill, risk-taking, hyperventilation before swimming (causing hypoxia), trauma while in the water (e.g. diving injuries), seizures while swimming, and child abuse or neglect (inadequate supervision) all contribute to drowning risk. Other high risk groups include participants in open water aquatic activities, boat refugees, and new immigrants from regions where swimming skills are underemphasized.

Preventative measures that reduce drowning incidents include:

- having appropriate fencing of pools and other appropriate barriers to water access
- properly supervising swimmers
- training of children and adults in swimming skills and water safety
- enforcing a reduction of consumption of alcohol during water sports
- training in bystander CPR

PATHOPHYSIOLOGY

The most important concept in the pathophysiology is that near drowning causes lung damage and hypoxemia. Hypoxic encephalopathy can result and this represents the second main organ system injury.

Pulmonary

Aspiration of fluid leads to:

- loss of surfactant causing atelectasis
- injury to the alveolar-capillary membrane leading to interstitial and alveolar edema
- leakage of protein-rich fluid into the alveoli (most pronounced after salt water aspiration)
- obstruction of small airways by water and particulate matter
- bronchospasm
- chemical injury if stomach contents aspirated

Ventilation-perfusion mismatching and shunting, caused by the above factors, result in hypoxemia. Subsequent infection can worsen the primary pulmonary injury, particularly if contaminated fluid is aspirated. Atelectasis and alveolar edema contribute to increased work of breathing through decreasing lung compliance and this can contribute to respiratory failure. All of these factors can result in the primary derangement in near drowning: respiratory failure and hypoxemia.

While some of these changes can start soon after the submersion event, the onset of symptoms can be more insidious, and develop over hours and even possibly days. In particular, pulmonary edema and infections can develop hours to days after the initial event. So called ‘secondary drowning’ (delayed presentation of symptoms) probably represents this slow progression over hours or even days, where the initial findings are not initially impressive.

A small cohort of patients (10-15%) who are submersed is reported to suffer from severe laryngospasm, glottic closure and hypoxemia instead of aspiration. This is often referred to as ‘dry drowning’.

Neurological

The neurological insult is that of hypoxic encephalopathy. Any cerebral edema that ensues (as with the lung, this can be delayed and progressive) is due to a period of cerebral hypoxia caused by the

pulmonary insult, and not due to the aspirated fluid directly injuring the brain.

Cardiac

Sudden death immediately after immersion in cold water occurs occasionally, and is thought to be secondary to arrhythmias due to catecholamine surge. In some patients, during a prolonged submersion, the hypoxemia and acidosis eventually lead to a decline in cardiac output, bradycardia and eventually electromechanical dissociation as the terminal event. Ventricular fibrillation is uncommon unless triggered by hypothermia. In many patients who are rescued before this cardiovascular collapse, the cardiovascular system remains relatively stable. Hypotension without a dysrhythmia must therefore prompt a search for other yet unexplained sources.

Remember that the patient may have suffered a traumatic injury concomitantly, and this needs to be considered in the assessment. Diving, particularly in shallow water, can cause injuries such as cervical spine fractures; however, these injuries are uncommon and should not unduly distract from, nor delay, managing the primary problems, and hypothermia may be a co-existent problem (see chapter on Cold Related Emergencies).

CLINICAL PEARL

'Secondary drowning' where the onset of symptoms, as a result of pulmonary edema and infections, can be insidious, developing over hours, and even possibly days, must be anticipated in the near drowning patient.

CLINICAL FEATURES

These features are predictable based upon the pathophysiological changes described above. Respiratory insufficiency may develop in the field, in the Emergency Department or over hours to days. All of the typical signs of respiratory failure, including hypoxia, must be watched for. A chest X-ray should be performed, and findings can range from a normal X-ray to peri-hilar (interstitial) pulmonary edema to florid alveolar (non-cardiogenic) pulmonary edema. Blood gases may demonstrate hypoxia as well as respiratory acidosis and metabolic acidosis (through impaired tissue perfusion if there is circulatory collapse). In most cases, some evidence of respiratory compromise will be seen within the first 4 hours after the event, if it is going to occur.

Level of consciousness may be depressed even to the point of coma, and yet even deeply comatose patients may have excellent recovery.

Baseline blood test to document electrolytes, renal function, hemoglobin and clotting profile should be obtained.

MANAGEMENT

Supporting vital functions and anticipating problems are the mainstays of treatment.

In all cases, supplemental oxygen should be provided until adequate oxygenation is demonstrated. Airway and breathing assessment may indicate a need for intubation. Some patients will need to be intubated to provide airway protection, ventilatory support or pulmonary toilet. If there is any question of a cervical injury, cervical spine protection should be instituted (particularly during intubation). Need for ventilatory support depends on the nature of the lung insult and may include:

- High flow oxygen by face mask,
- non-invasive ventilation [e.g. biphasic positive airway pressure (BiPAP), or continuous positive airway pressure (CPAP)]
- intubation and mechanical ventilation with positive end-expiratory pressure(PEEP)

Cardiac monitoring, intravenous access and oxygen saturation monitoring should be instituted for all but the most minor submersion events. For those with altered level of consciousness, basic principles of brain resuscitation should be adhered to, including:

- adequate oxygenation
- avoidance of hypotension and fluid overload
- correction of hypoglycemia or hyperglycemia
- aggressive treatment of seizures.

The role of steroids and prophylactic antibiotics remains controversial, and they are not indicated routinely at present.

DISPOSITION AND OUTCOME

Early resuscitation brings the highest chance of survival after a near-drowning incident. Cardio-pulmonary collapse not treated until ambulance arrival decreases the survival rate compared to cases with bystander CPR.

Disposition decisions should be based on factors such as:

- age of the patient (a low threshold for admitting young and elderly patients)
- ‘significance’ of the event based on history obtained
- any symptoms of pulmonary compromise or objective signs of ventilatory problems
- appreciation that respiratory distress can appear and progress over time

For the purpose of making disposition decisions, patients can be divided into four groups:

- Patients with no evidence of a significant submersion episode can be discharged after a short period of observation (at least 4 hours)
- Patients who are asymptomatic after a significant immersion episode should be admitted for observation (or have an extended period of observation in the emergency department)
- Patients with evidence of hypoxemia and/or mild CNS symptoms will need admission and supplemental oxygen until the abnormalities resolve
- Critically ill patients will need ICU monitoring (and possible airway and/or ventilatory support) for severe hypoxemia and/or depressed level of consciousness.

Outcome in the first three groups above is typically excellent. Results in severely ill patients are variable, but not entirely dismal. For example, 1/4 of patients who require full CPR and have an initial GCS of 3 in the Emergency Department survive with intact neurological function. There are no decision guidelines with sufficient predictive accuracy to guide physicians when to withdraw or withhold resuscitation after near-drowning, and case reports of full recovery after prolonged submersion have been documented.

A combination of hypothermia along with near drowning appears to have some protective effect, especially in children. This may be due to the decreased metabolic rate at a lower body temperature, and possibly due to the ‘diving reflex’, which causes bradycardia and shunting of blood to vital organs upon immersion of the face in cold water.

SUMMARY

- Near-drowning affects children and adolescents more than other age groups
- Pulmonary effects of aspiration lead to hypoxemia and respiratory failure
- Hypoxic encephalopathy can cause long term morbidity in severe cases
- Supportive care, including ventilatory support if indicated, is the main treatment

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41: TOXICOLOGY

Margaret Thompson

OBJECTIVES

1. To develop an organized approach to the poisoned patient
2. To recognize the common toxidromes
3. To know the indications for various decontamination methods
4. To understand the role of antidotes
5. To understand the utility of laboratory tests in the poisoned patient

INTRODUCTION

Unintentional injuries are the 4th leading cause of death in the Province of Ontario. After motor vehicle accidents and falls, poisonings are the third most common cause of unintentional injuries, although the motivation for the overdose may not strictly be unintentional. In addition, for every person who dies from their injury (as poisonings are categorized), 250 persons are ‘injured’. From the Chief Coroner of the Province of Ontario, there are approximately 500 deaths per year attributable to a poisoning. Thus, in addition, there could be up to an additional 125,000 victims from poisonings per year in the Province of Ontario. Emergency Physicians are the medical community’s experts in toxicology. It is therefore incumbent on them to rapidly identify, stabilize and manage the poisoned patient.

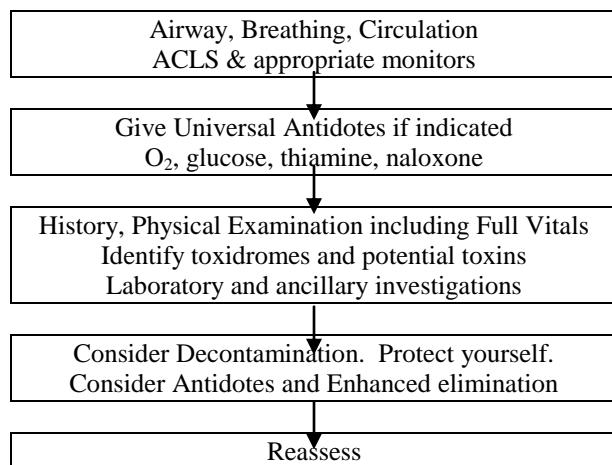
INITIAL APPROACH

As with any critically ill patient, Airway, Breathing and Circulation must be addressed before the nuances of the poisoning can be managed. There are very few poisonings for which standard ACLS protocols would not be appropriate. Remember that a differential diagnosis (including metabolic, infectious and structural causes) must be considered.

First, treat the patient and not the poison.

Second, remember that it is the dose, not the substance that makes the poison.

Third, clinical manifestations of poisoning may be delayed for hours, even days after the exposure. When signs and symptoms do develop, their onset may be precipitous.



HISTORY

- Circumstances leading to the presentation?
- What was available?
- How much was available?
- When was the exposure?
- Why? This speaks to intent and possible prevention.
- What symptoms did the patient have? What are the symptoms now?
- What treatment has been given so far? Results of that treatment?

PHYSICAL EXAMINATION

Following a primary survey, resuscitation and stabilization of any critically ill patient, a detailed secondary survey is conducted to identify unexpected pathology, assess response to therapy and determine etiology of the presentation. Specific elements to be assessed include:

- general appearance
- full vital signs
- mental status
- signs of trauma (include C-spine evaluation)
- signs of seizure
- signs of infection
- signs of chronic alcohol or substance abuse (spider nevi, track marks)

The poisoned patient may present with symptoms secondary to many systems' involvement. Classic presentations could include altered mental status (coma or seizures, hallucinations), dysrhythmias, altered vitals, altered saturations. There is an extensive toxicological differential for every symptom expressed.

Paramount to toxicology is the identification of toxidromes. A toxidrome is a constellation of clinical signs and symptoms associated with a particular class of substances. Identification of a particular toxidrome suggests treatment options, expected course of intoxication and predicts outcome as well as response to therapy. At times, when multiple substances are involved, a classic toxidrome may not emerge.

Table 1: Classic Toxidromes

Toxidrome	Manifestations	Etiology
Anticholinergic Mnemonic 'blind as a bat, dry as a bone, red as a beet, mad as a hatter, and hot as a hare'	Hyperthermia Dilated pupils Dry skin & mucous membranes Agitation Hallucinations Seizures Tachycardia Urinary retention Ileus	Antihistamines Atropine & belladonna Diphenhydramine Tricyclic antidepressants
Cholinergic	Salivation, Seizures Lacrimation Urinary incontinence Diarrhea, Diaphoresis Bronchospasm Bronchorrhea Bradycardia Emesis, Excitation	Carbamates Nerve gases Organophosphates
Sympathomimetic	Hyperthermia Mydriasis Diaphoresis Tachycardia Hypertension Excitation Seizures	Amphetamines ASA Cocaine LSD PCP Theophyllines Sedative & alcohol withdrawal
Narcotic & Sedatives	CNS depression Respiratory depression Hypotension Miosis +/-	Barbituates Benzodiazepines Ethanol GHB Opioids Other sedatives

CLINICAL PEARLS

Clinical manifestations of poisoning may be delayed for hours, even days after the exposure. It is therefore important to start treatment for some poisoned patients *before* they develop symptoms.

LABORATORY & ANCILLARY INVESTIGATIONS

The local Poison Control Centre should be called for every suspected poisoning for assistance in management as well as for their own statistical purposes. Besides identification of a toxicodrome, the Emergency Physician may rely on classic

investigations to identify those substances which may have no clinical manifestations immediately, but which may cause late symptoms and signs. The anion and osmolar gaps are calculated to point to these intoxications. Laboratory parameters must be measured to calculate these gaps.

$$\text{Anion Gap} = \text{Na}^+ - (\text{HCO}_3^- + \text{Cl}^-)$$

Normal AG < 10 mmole/L

$$\text{Osmolal Gap} = (\text{Measured} - \text{Calculated}) \text{ Osmolality}$$

$$\text{Calculated Osmolality} = 2[\text{Na}^+] + \text{BUN} + \text{Glucose} + 1.25 \times \text{Ethanol}$$

Normal Osmolal Gap should range from -14 to +10 mosmoles/L

Table 2: Differential Diagnosis of Increased Gaps

Increased Anion Gap	Increased Osmolar Gap
Methanol	Ethylene Glycol
Uremia	Isopropyl Alcohol
Diabetic Ketoacidosis	Glycerol
Alcoholic Ketoacidosis	Mannitol
Starvation Ketoacidosis	Methanol
Phenformin	Sorbitol
Paraldehyde	
Iron	
INH	
Lactic acidosis	
Ethylene Glycol	
Salicylate	
Carbon Monoxide	
Cyanide	
Toluene	

Arterial or venous blood gases, and measured serum bicarbonate (HCO_3^-) confirm acidemia. Electrolytes, transaminases, amylase and specific toxin levels may be indicated for specific poisonings. Acetaminophen, salicylate and ethanol levels should be measured in all intentional overdoses and in all patients who present with an altered level of consciousness. Generally, a urine drug screen is costly and is not helpful in the emergency management of the poisoned patient.

An electrocardiogram (ECG) gives information regarding sodium channel blockade if the QRS is prolonged. Other cardiac toxins will prolong the QT. CXR can show aspiration pneumonia from a hydrocarbon, ALI (acute lung injury) from heroin or salicylates, for example, or a pneumothorax from

cocaine inhalation. An abdominal film can show radio opaque substances including chlorinated hydrocarbons (eg. chloral hydrate), heavy metals, iron, enteric coated products or drug packaging in the smuggler.

DECONTAMINATION

The amount of drug available to be absorbed into the circulation and thus to target tissues determines the potential toxicity that a patient may develop. Our goal is to limit this amount by decontamination of the patient. Remember that many formulations of substances and many routes of exposure exist. Skin, hair, fingernails, nasal passages, conjunctiva and other orifices can contain residual drug to be

decontaminated. The gastrointestinal tract is the most common route of exposure.

The balance of evidence suggests that the earlier an attempt at decontamination is made, the more toxin, on average, is able to be removed. Ipecac, an emetic, is more harmful than helpful in gastrointestinal decontamination and is not recommended, even in the home. There is inadequate evidence available to suggest that the routine use of lavage (the insertion of a tube and the irrigation of the stomach) is advantageous in the care of the poisoned patient, but might be considered in the rare intoxication. There is no evidence that a cathartic alone will benefit the intoxicated patient and should not be prescribed.

There is inadequate evidence to suggest that the routine use of activated charcoal will decrease morbidity and mortality in all poisoned patients, but it should be considered in patients who present within one hour of their gastrointestinal exposure to a toxin. For substances that slow gastrointestinal motility or gastric emptying and for substances that may form bezoars in the stomach, consideration for the late administration of activated charcoal should be given. It is controversial as to whether a dose of cathartic should accompany single dose activated charcoal, but is generally not recommended.

Whole bowel irrigation is a modality of purging the gastrointestinal tract by delivering, via a nasogastric tube, polyethylene glycol, at rates from 500cc/hr (infants) to 2000cc/hr (adults) until rectal effluent is clear. This method of decontamination is *not* to be used in the routine care of the poisoned patient, but to be considered when the exposure has been to a toxin that is not bound to activated charcoal (eg. heavy metals), slow release medications and drug smugglers who swallow packets containing large amounts of drugs.

ENHANCED ELIMINATION

Once a toxin is absorbed into the circulation, it can circulate as free drug, be bound to plasma proteins, or

cross cell membranes to attach to target organs. Toxins are eventually eliminated from the body as unchanged drug in the urine, feces, respirations or sweat or the substance is metabolized to allow elimination via these routes. There are a very few toxins, which, once absorbed, can actually be eliminated more quickly than natural homeostasis would allow. These are toxins that are small in molecular weight, not highly bound to serum proteins, are weakly acidic or basic, are not widely distributed throughout the body or are secreted directly into the gastrointestinal tract.

Table 3: Selected toxins for which enhanced elimination should be considered

Enhanced Elimination Modality	Toxin
Multiple Dose Activated Charcoal	Carbamazepine, Phenobarb, Quinine, Theophylline
Urine Alkalization	Chlorpropamide, Methotrexate, Phenobarb, Salicylates
Hemodialysis	ASA, Ethylene Glycol, Lithium, Methanol, Valproate

ANTIDOTES

Antidotes are agents that may be used to reverse, minimize or prevent the effects of a poison. As there are limited antidotes available, they are never a substitute for aggressive supportive care. Commonly used antidotes are summarized in Table 4.

Table 4: Selected Antidotes

Toxin	Antidote	Comments
Acetaminophen	n-acetylcysteine	Multiple regimes
B-blocker	Consider high dose insulin /euglycemia	Select fat soluble beta blockers consider intralipid. Select water soluble beta blockers consider dialysis
Calcium antagonist	Calcium	Chloride has 3X elemental calcium as does gluconate
Calcium antagonist	Consider high dose insulin / euglycemia	Consider intralipid
Carbon monoxide	Oxygen	Hyperbarics is controversial
Digoxin & like substances	Digoxin FAB fragments	Dosing calculations
Ethylene glycol	(Ethanol) or Fomepizole	Can use oral ethanol if no other antidote available.
Hydrofluoric Acid	Calcium	May be given intravenously, topically, intraarterially
Iron	Deferoxamine	Slow intravenous infusion
Isoniazid	Pyridoxime	High dose for seizures
Methanol	(Ethanol) or Fomepizole	As for ethylene glycol
Methemoglobinemia	Methylene blue	For symptomatic patients Caution G6PD deficiency
Narcotics	Naloxone	Low dose for chronic abuse
Organophosphates	Atropine plus pralidoxime	Atropine for muscarinic Sx 2-pam to prevent nicotinic
Sodium channel blockers	NaHCO ₃	IV boluses for wide QRS & seizures
Sulfonylureas & related	Octreotide	Q6-12h dosing prn

SUMMARY

- ABCs and ACLS protocols, followed by universal antidotes, identification of toxicodromes, appropriate laboratory investigations, specific antidotes, decontamination and elimination are the fundamental aspects of care of the poisoned patient
- treat the patient and not the poison
- clinical manifestations of poisoning may be delayed for hours, even days after the exposure
- it is the dose, not the substance that makes the poison
- the four classic/common toxicodromes are anticholinergic, cholinergic, sympathomimetic and narcotic/sedative
- the osmolar and anion gaps are an integral part of the workup of the poisoned patient
- activated charcoal, whole bowel irrigation and gastric lavage are methods of GI contamination
- enhanced elimination methods include multiple dose activated charcoal, dialysis and urine alkalinization

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42: ALCOHOL RELATED ILLNESS

Brian Goldman, Simon Kingsley

OBJECTIVES

- To recognize the manifestations of alcohol intoxication
- To know the management of alcohol intoxication and withdrawal
- To be able to recognize selected complications of alcohol abuse
- To develop an approach to ingestion of toxic alcohols

INTRODUCTION

Approximately 3% of all visits to the ED are alcohol related. Ethanol is a CNS depressant that inhibits neuronal activity. It acts by depressing the excitatory neurotransmitter glutamate and increases the inhibitory activity of neurotransmitters GABA and glycine. Complication of alcohol consumption is commonly in the Emergency Department. Patients may present with injury, mental health concerns, social or situational crisis and physical complications related to long term alcohol use.

Approach to the Awake, But Intoxicated Patient

Clinical Features

Symptoms and signs of ethanol intoxication vary based on the time course of consumption. Initial ingestion results in frontal lobe disinhibition, causing transient stimulation and excitatory behaviors. Further consumption results in slurred speech, nystagmus, slowed responses to verbal, visual or physical stimuli and decreased motor coordination/control. Loss of awareness is often more profound in ethanol-naïve patients. Disconjugate Glasgow Coma Scales can occur, with eye and verbal scores staying low despite proportionally higher motor scores. Hypotension with reflex tachycardia and syncope may occur. Hypothermia can occur with substantial intake.

All patients should be thoroughly examined for associated injuries, especially head injury and extremity fractures. Non-ethanol related causes of altered level of awareness should always be entertained, which can co-exist with intoxication.

Laboratory

Ethanol intoxication is accompanied by an elevated serum ethanol level. However, it is important to

remember that because of tolerance, blood ethanol levels correlate poorly to the clinical manifestations of intoxication. Ironically only a negative serum ethanol level is truly helpful in the assessment of the comatose patient, as it rules out ethanol intoxication as a cause.

All patients with a depressed level of awareness should be checked for hypoglycemia. Hypoglycemia, can also accompany ethanol ingestion due to impaired liver functioning and poor nutrition. Consideration should be given to obtaining electrolyte levels, since many patients will have low potassium and/or magnesium.

Alcohol intoxication will result in an elevated osmolar gap. If the patient is noted to have a significant acidosis a cause other than ethanol intoxication should be explored. Laboratory evidence of chronic ethanol abuse includes elevations in liver enzymes (especially GGT), thrombocytopenia and hyperchromic macrocytic anemia.

Management

Intoxicated patients with a presumptive diagnosis of ethanol intoxication should be held in the ED until awake and oriented with a Glasgow Coma Scale (GCS) of 15. A patient needs to be able to respond appropriately to questions and to ambulate normally prior to discharge. Patients with a decreased level of consciousness should be monitored closely and have neurovitals recorded every hour. The frequency of neurovitals may be decreased in stable patients. Mental status that fails to improve should prompt a search for other possible etiologies such as subdural hematoma, polydrug overdose, metabolic diseases or a postictal state.

It is not necessary to order serial ethanol levels as they do not correlate with recovery from ethanol intoxication. Patients with altered mental status should have a saline lock for intravenous access, however, IV fluids are not usually necessary. To prevent Wernicke's encephalopathy, patients with ethanol intoxication should receive thiamine 100 mg po, IM or IV. Activated charcoal is not efficacious.

Severely intoxicated patients, by definition, almost always lack capacity to refuse care. As such, it is prudent for the clinician to prevent departure of eloping patients until a medical emergency has been ruled out and until they regain capacity or an

appropriate substitute decision maker is present. Appropriate physical and chemical restraint (such as haloperidol) may be employed for incapacitated patients under the Health Care Consent Act (not the Mental Health Act) to safely prevent elopement.

Intoxicated patients are often treated in a patronizing and belittling manner. Such attitudes, combined with the patient's disinhibited state, can cause further inappropriate behavior. Always show respect for all patients, no matter how intoxicated they may appear to be.

Careful assessment that a patient is oriented, lucid and appears to comprehend all discharge instructions should be documented on discharge.

Other Suggestions

- If possible, keep intoxicated patients in a quiet and non-stimulating environment where they can be observed.
- Where appropriate, consider offering such patients referral to a detoxification centre
- Never discharge an intoxicated patient to the care of a friend or relative who may also be intoxicated
- Intoxication can mask almost any injury or illness
- Many illnesses mimic intoxication
- Acutely intoxicated patients will require repeated exams over several hours prior to discharge

ALCOHOL RELATED ILLNESSES

Withdrawal Symptoms

Alcohol withdrawal is a syndrome characterized by the stimulation of several neurochemical axes, including the sympathetic, dopaminergic, and serotonergic systems. Withdrawal can occur up to 7 days after acute ingestion, and correlates in severity to amount of alcohol consumed, as well as duration of consumption. Patients exhibit tachycardia, hypertension, and occasional elevated temperature. Patients become agitated, tremulous, diaphoretic. Nausea and vomiting may occur. Seizures are common with substantial withdrawal. In its most extreme form, Delirium Tremens, hallucinations, delirium and confusion can occur often accompanied by profound autonomic dysfunction: hyperthermia, tachycardia, hypertension, and seizures. Delirium tremens is life-threatening and often requires aggressive supportive care, including possible intubation and paralysis, in the ICU setting.

Benzodiazepines constitute the mainstay of treatment of alcohol withdrawal, best exemplified by continuous assessment of the patient's CIWA-Ar

score. This is a validated score that measures ten variables on a 7-point scale (0 – being normal activity, 7 – being severe symptomatology). These variables include agitation, anxiety, auditory disturbances, clouding of consciousness, headache, nausea and vomiting, sweats, tactile disturbances, tremor and visual disturbances. Low scores (< 8) generally do not require medication; higher scores require generous amounts of benzodiazepines such as diazepam. Familiarize yourself with your local Emergency Department's protocol. Supportive care includes assessment of medical illnesses and complications, a quiet environment, nutrition, supplementation with thiamine and multivitamins.

Alcohol Related Seizures

Between 2.5% and 33% of heavy drinkers who binge on alcohol and then stop drinking precipitously will have at least one generalized seizure. While patients who have chronic consumption of ethanol have many reasons for seizure (including electrolyte abnormalities, propensity for head injury, and medication noncompliance), the vast majority are precipitated by withdrawal, occurring between 8–48 hours following the point at which alcohol level begins to fall. These alcohol withdrawal seizures are typically:

- brief (1-5 mins) generalized major motor seizures with a brief postictal phase
- single, but occasionally 2-3 and rarely up to 6 in a 6-hour period

Seizures occurring after 48 hours are not simple alcohol withdrawal seizures, and may be related to delirium tremens, an underlying metabolic disorder, or structural CNS disorder.

Wernicke's Encephalopathy

Wernicke's encephalopathy is a rare neurological disorder arising in alcoholics due to chronic thiamine deficiency. Poor dietary intake coupled with increased GI losses and malabsorption leads to an acute disorder characterized by a triad of oculomotor abnormalities (nystagmus, lateral gaze palsies or disconjugate gaze), cerebellar dysfunction and global confusion. The classic presentation is rare, with the complete triad present in <10% of autopsy proven cases.

The mainstay of management is thiamine. Start with 100mg po, IM or IV. Continue thiamine 50-100 mg/day until normal diet can be resumed. Ocular signs may be reversed in as little as 1-6 hours. Ataxia and confusion may improve over days to months.

Hypokalemia

50% of patients in alcohol withdrawal are hypokalemic. All alcoholics are at risk of hypokalemia. This is due to:

- decreased oral intake
- increased renal losses due to diuretic effect of alcohol and/or decreased magnesium stores
- increased GI losses due to diarrhea and vomiting.

Potassium levels of 1.5 - 2.5 mEq/L are not uncommon and may lead to rhabdomyolysis. The treatment of hypokalemia may require large amounts of potassium (up to 300 mEq in 24 hours) as well as correction of hypomagnesemia. Failure to correct potassium despite adequate supplementation suggests hypomagnesemia.

Alcohol-Associated Cardiovascular Disease

1. Hypertension
2. Cardiomyopathy
3. Alcohol Induced Arrhythmias ("Holiday Heart")

"Holiday Heart" Syndrome

This condition occurs most commonly in patients who have an acute increased consumption superimposed on chronic alcohol intake. Typical arrhythmias include atrial fibrillation, atrial flutter, premature atrial and ventricular complexes, supraventricular as well as occasional ventricular tachycardia. Alcohol-induced arrhythmias are more common in those with pre-existing heart disease.

Alcoholic Ketoacidosis (AKA)

Alcoholic ketoacidosis tends to occur in chronic alcoholics who abruptly decrease their alcohol consumption following a binge. The metabolic pathways that generate ketosis following abrupt cessation are poorly understood, but the clinical manifestations are typical of ketosis: symptoms include abdominal pain and cramping, nausea and vomiting and occur within 48 hours of the decrease in alcohol intake. Tachycardia and hypotension are often present. It can be difficult to separate AKA clinically from other similarly-presenting syndromes in patients that cease alcohol consumption; simple alcohol withdrawal, gastritis, chronic pancreatitis, and alcoholic hepatitis all present initially with similar symptoms. Lab investigations in AKA reveal an increased anion gap metabolic acidosis and the presence of ketones in the urine and blood. Treatment starts with ABCs, thiamine, volume repletion and glucose. Patients who have IV saline and glucose tend to recover more quickly than those given saline alone.

The condition usually resolves quickly with appropriate treatment, often in 8-16 hours.

OTHER ALCOHOLS

Some individuals consume other forms of alcohol through misadventure, when ethanol-containing beverages are unavailable, or cannot be afforded. These include methanol, ethylene glycol and isopropyl alcohol. Depending in part on the quantity consumed, such alcohols are generally considered poisonous to the body and can cause tremendous morbidity, if not death. The presence of one of these variant forms of alcohol is suspected in the following circumstances:

- Suspicious history of unknown ingestant
- Unexplained increased anion gap metabolic acidosis
- Unexplained osmolar gap

Volatile alcohols contribute to the measured serum osmolality. While there is no true "normal" osmolar gap, a gap of more than 10 between the calculated ($2 \times \text{Na} + \text{glucose} + \text{BUN}$) and measured serum osmolality is considered abnormal and suggests the presence of an unmeasured source: typically ethanol, methanol, ethylene glycol and / or isopropanol. A gap of <10 however does NOT imply the absence of such alcohols. Many hospitals measure only ethanol directly, with the other alcohols requiring a formal request for more extensive assays. Adding the serum ethanol level to the calculated serum osmolality should result in a number that is within 10 milliosmols of the measured serum osmolality. If the gap is >10, or if the gap is within normal limits there is a strong suspicion of ingestion of other alcohols, the serum assays for the respective alcohol should be performed.

Isopropyl Alcohol

Isopropyl alcohol is commonly found in products such as rubbing alcohol, skin and hair care products, hand sanitizer, as well as various industrial solvents. Isopropyl alcohol is roughly twice as intoxicating as ethanol and the duration of action roughly 2-4x longer. It is oxidized in the liver by alcohol dehydrogenase into acetone, causing ketonemia and ketonuria. However the acetone does NOT go on to produce acetoacetate or beta-hydroxybutyrate, and hence does NOT lead to metabolic acidosis. The hallmarks of isopropyl alcohol toxicity include:

- ketosis but NO acidosis
- normal blood glucose

- clinical features similar to those of ethanol poisoning but of longer duration
- fruity odor of acetone or smell of rubbing alcohol on the breath

Severe poisoning is marked by early onset of coma, respiratory depression and hypotension.

Complications include gastric hemorrhage, severe hypotension, and rhabdomyolysis. The treatment of isopropyl alcohol poisoning includes attention to the ABCs of resuscitation, as well as management appropriate for ethanol poisoning. As for all alcohols, activated charcoal is not indicated. Hemodialysis is indicated for hypotension refractory to fluids and pressor agents. Patients with lethargy or profound loss of awareness should be admitted to hospital.

Methanol

Methyl alcohol or methanol is found in windshield washer fluid, varnishes and antifreeze, or is created through improper fermentation and distillation of homemade ingestable alcohol products (aka wood alcohol or "moonshine"). Alcohol dehydrogenase converts methanol into the extremely toxic metabolite formic acid. Formic acid is directly toxic to neurologic tissue and induces a severe anion gap metabolic acidosis, as well as renal failure. Symptoms typically occur up to 12-18 hours following ingestion. The cardinal features of methanol poisoning include:

- CNS depression
- Visual disturbances and blindness
- Abdominal pain
- Marked anion gap-type metabolic acidosis
- Osmolar gap
- Possible renal failure

Since time is of the essence, treatment should be instituted as soon as the diagnosis is strongly suspected. The main goal of treatment is to reduce the creation of new formic acid while enhancing the elimination of both methanol and formic acid already present. Administration of ethanol by intravenous drip effectively competes with methanol for alcohol dehydrogenase and thus helps prevent the formation of formic acid. Fomepizole, a strongly competitive inhibitor of alcohol dehydrogenase, is also effective in blocking the synthesis of formic acid. Ethanol should be used if the patient is allergic to fomepizole or if the drug is not available.

Folic acid should also be administered, as it is a cofactor in the metabolism of formic acid and enhances its elimination through conversion to carbon dioxide. Liberal doses of bicarbonate should be administered to correct acidosis.

Hemodialysis indications vary depending on local guidelines, but in general is indicated for patients with the following:

- Visual or CNS disturbances
- Ingestion of more than 30 ml of methanol
- Severe metabolic acidosis
- Renal failure
- Elevated methanol level > 15

Ethylene Glycol

Ethylene glycol is found in antifreeze, cosmetics, detergents, lacquers, preservatives, and glycerine substitutes. It is metabolised in the liver and kidneys into aldehydes, glycolates, and oxalates. While all are toxic, it is oxalic acid and its calcium precipitate calcium oxalate that accounts for the majority of the morbidity in victims. Calcium oxalate precipitates in brain tissue resulting in edema, neurological suppression and coma, and in renal tubules causing renal failure. The initial clinical presentation includes CNS depression, hallucinations, coma and seizures. The second phase 12-24 hours following ingestion, arises from cardiorespiratory depression and includes tachycardia, hypertension, respiratory distress syndrome, congestive heart failure, and circulatory collapse. The third phase, 72 hours following ingestion, is characterized by damage to the kidney. Symptoms in this phase include flank pain, tenderness of the kidneys, oliguria, and renal failure. Calcium oxalate crystals may be observed in urinalysis in up to 50% of patients. Patients may have life-threatening hypocalcemia. The diagnosis is often entertained and treatment instituted before confirmatory drug levels are obtained. Ethylene glycol poisoning should be considered in the following:

- signs of intoxication yet no ethanol smell on the breath
- anion gap-type acidosis
- osmolar gap
- calcium oxalate crystalluria
- renal failure

Treatment is similar to that of methanol. Good supportive care is a foundation of treatment. As is the case with methanol, ethanol or fomepizole are used as inhibitors of alcohol dehydrogenase. Patients with hypocalcemia should receive supplemental calcium by intravenous drip. Pyridoxine and thiamine help facilitate the safe metabolism of ethylene glycol. Hypomagnesemia should also be corrected.

Hemodialysis is indicated in the following instances:

- The history or clinical presentation suggest ethylene glycol poisoning
- Elevated serum concentration of ethylene glycol
- Signs of nephrotoxicity

- Metabolic acidosis
- Neurological or cardiorespiratory compromise

SUMMARY

- Ethanol intoxication is extremely common
- The manifestations of ethanol intoxication do not correlate to the serum ethanol level
- The approach to assessment is to rule out secondary injuries, and other conditions that masquerade as ethanol intoxication
- Patients whose level of awareness fails to improve should be checked for other causes including subdural hematoma
- Isopropyl alcohol intoxication presents similarly to ethanol intoxication but is often of earlier onset and can have more serious complications
- Acute methanol and ethylene glycol poisonings are potentially life-threatening poisonings that may benefit from fomepizole and/or ethanol administration as well as dialysis

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43: TRAUMA RESUSCITATION

Dave MacKinnon

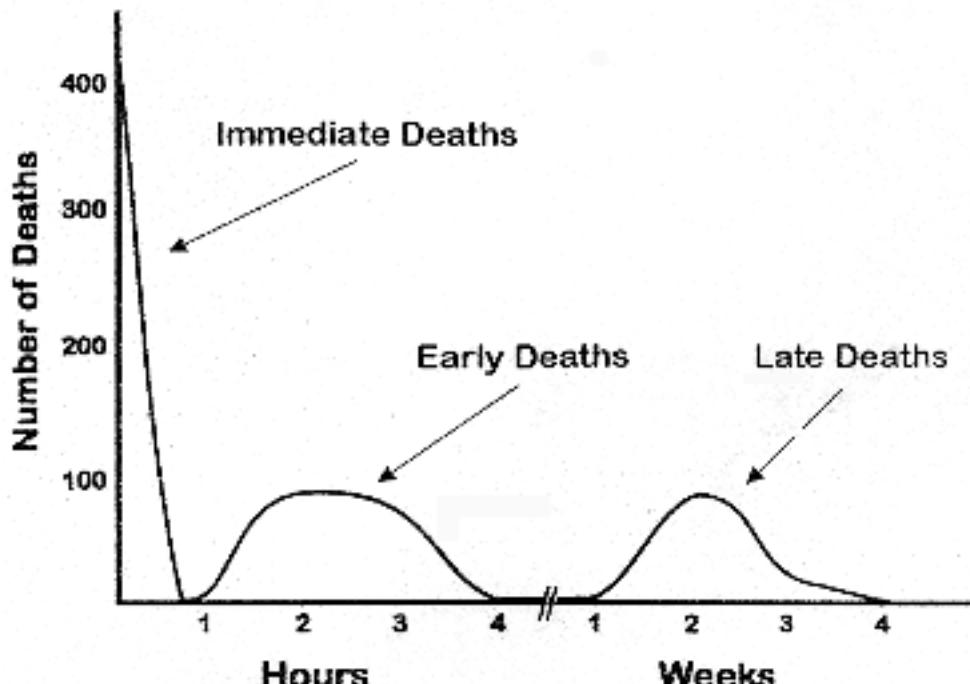
INTRODUCTION

Much has changed in the care of trauma patients over the last several years. While trauma was once considered a “surgical disease”, the emergence of Emergency Medicine as its own specialty has shifted the care. According to recent data from the Ontario Trauma Registry, of the approximately 1,000,000 ED visits each year in Ontario for trauma, only 6% are admitted to hospital. This means that most trauma patients are managed and discharged by the ER

physician. Hence, you will likely be exposed to more trauma during your ER rotation than your surgical rotations.

To understand why our primary survey and resuscitation of trauma patients is important, we need to appreciate the trimodal distribution of deaths following trauma. Deaths can be grouped into the three time periods demonstrated in the oft-cited “Trauma Death by Time of Injury” graph shown here:

Trauma Death by Time of Injury



The **immediate** group of deaths occurs from seconds to minutes following trauma. These are generally “in the field” deaths. They are usually a result of major vascular injuries (e.g. aortic or cardiac rupture), major head injuries, high C-spine injuries or complete airway obstruction. For the most part, these patients are considered unsalvageable. A reduction in these deaths relies on injury prevention through public education, etc.

The third group of “**late deaths**” occurs days to several weeks after the injury. They are usually in-hospital and may be from complications of the injury itself or from hospital-related complications. They

may include but are not limited to sepsis (e.g. from indwelling central lines), pulmonary embolism (e.g. post-fracture or from immobilization) and pneumonia (e.g. post-pulmonary contusion).

The primary survey and resuscitation of trauma patients targets the deaths that occur during the second peak, which occur from minutes to hours post-injury (**early**). Most of these patients’ injuries are considered “salvageable”, but they must be recognized and acted upon in a timely manner. These deaths are usually related to the **ABCs** --- airway, breathing or circulation problems.

The led to the concept of the “**Golden Hour**”; it underlines the need for rapid assessment, resuscitation and management of trauma patients who may have survivable, treatable injuries and thus the prevention of “early deaths”.

Our challenge in the ER is recognition and resuscitation of significantly injured trauma patients. To assist us in this, an organized, methodological approach should be used for each trauma patient. The Advanced Trauma Life Support (ATLS) Course is a reasonable 1st step in developing this systematic approach. It emphasizes:

1. assessing, resuscitating and stabilizing the patient’s condition rapidly, accurately and according to priority
2. determining if the patient’s needs exceed the facility’s capabilities, therefore anticipating arrangement for intra or inter-hospital transfer

To truly become proficient in dealing with the polytraumatized patient, there is no replacement for real life exposure to cases. Our hope is that your ER rotation will provide you with this exposure.

STANDARDIZED APPROACH TO ASSESSMENT AND RESUSCITATION

An effective, useful, standardized approach needs to be simple yet thorough; that’s the main strength of ATLS. The approach to the polytraumatized patient involves:

1. **Primary Survey (ABCDE)**
2. **Resuscitation**
3. **Secondary Survey (Head to Toe)**
4. **Investigations**
5. **Definitive Care**

This chapter deals with

1. **Primary Survey (ABCDE) and**
2. **Resuscitation.**

THE “TEAM” BASED APPROACH

It is important to emphasize the “team” based approach. The trauma team in an ER will include the physicians (may range from one person in a small hospital to a large group in trauma centres), nurses, x-ray technicians and other support staff. Timely, effective care is only realized if the team is working in collaboration. It is important to understand the different roles occurring simultaneously:

Physicians: Primary survey/ resuscitation and secondary survey

Nursing staff: O2, 2 large bore antecubital IVs, cardiorespiratory monitor, vitals, bloodwork, medications

X-ray technicians: radiographs

Support staff: exposure, supplies

There will certainly be overlap of the roles of the “team” members depending on the available manpower, but the breakdown above is a good way to bring order to the often-hectic environment of the trauma room – i.e. “organized chaos”.

PRIMARY SURVEY AND RESUSCITATION

While listed separately, the primary survey and resuscitation are in essence done simultaneously.

A – Airway (with C-Spine protection)

B – Breathing

C – Circulation

D – Disability (Neurologic Evaluation)

E – Exposure/Environmental Control

The concept of the “ABCs” comes from the fact that issues pertaining to airway kill more quickly than those pertaining to breathing, which in turn, kill quicker than blood volume loss. Initial trauma evaluation adds in the “D” and “E” as they are important to address in the initial evaluation.

A – Airway (with cervical spine protection)

Assessing the patient’s airway patency is the first priority for any ER patient. It is often said, there is no point in moving on to “Breathing” and “Circulation” if there is not a patent airway, as B and C require A. As most of our trauma patients are awake, this most often involves simply hearing the patient talk; this indicates a patent airway. Altered voice, moaning, stridor or absence of any sounds may indicate non-patent airway.

A general approach to airway is covered in another chapter, but this section will focus on specific airway issues in trauma. Airway obstruction in this population may result from blood, emesis, teeth, fractures or soft tissue swelling. There may also be a non-patent airway in a patient with decreased level of consciousness from head injury or shock/cardiac arrest.

One unique trauma situation is a patient with a mandible fracture. These patients, if given a paralytic (such as succinylcholine) for the intubation, usually

are an easier intubation as the laryngoscope 'pulls' up the mandible and exposes the cords more easily.

Management of airway issues can range from simple to extremely difficult. Simple loose foreign bodies may be easily removable. Blood or emesis may be suctioned. The chin lift or jaw thrust is a simple initial maneuver to relieve obstructions. ***The head tilt should be avoided in trauma patient as C-spine precautions must be maintained until C-spine injury has been ruled out.***

Some patients will require a more definitive airway due to their injury or altered level of consciousness. The usual method of "securing" the airway is endotracheal intubation.

Rapid Sequence Intubation

Over the past 10-15 years, rapid sequence intubation (RSI) has become the preferred method for endotracheal intubation of ER patients. Studies have demonstrated that a higher success rate is achieved when RSI is used. The main feature of RSI is that a paralytic is given which significantly improves the view on laryngoscopy. The caveat is that the patient is no longer able to breathe on their own, so patients should only undergo RSI if we are confident we can secure their airway. If unable to intubate an airway after a paralytic is given, the options are bag-valve-mask ventilation until the paralytic has worn off, or obtaining a surgical airway.

Surgical Airway

If patients are selected for RSI carefully, a surgical airway is rarely performed in the trauma room. Besides a failed RSI, it may need to be performed when severe facial trauma renders endotracheal intubation impossible. This may include severe facial/neck trauma with massive swelling.

The emergent surgical airway is via cricothyrotomy. An incision is made through the cricothyroid membrane (i.e. ***below*** the thyroid cartilage). A "Cricothyrotomy Kit" may be used which uses the Seldinger technique (introducer needle > guidewire > tracheostomy tube).

An alternative, simple method uses simply a scalpel and a #5 cuffed endotracheal tube (ETT) (the smallest available cuffed ETT) is as follows:

1. Vertical skin incision over the site of the cricothyroid membrane
2. Horizontal incision through the cricothyroid membrane
3. Pass the #5 cuffed ETT through the hole and inflate the cuff (a small Kelly may be used to hold the cricothyroid incision open during the passage)

A tracheostomy (done just superior to the suprasternal notch) involves dissecting through superficial vessels, and is not used for the "emergent" surgical airway; a cricothyrotomy ***avoids*** these vessels.

Other Methods

Other methods include nasotracheal intubation. This method is rarely used for several reasons including the possibility of basal skull/cribiform plate fracture (ETT may enter the brain!) and the spike in intracranial pressure associated with this method. The widespread use of RSI has also made nasotracheal intubation mostly obsolete.

Bronchoscope-guided intubation may be used by skilled providers for significant swelling or other difficult airways.

Protect the C-spine

In the setting of blunt trauma, all seriously injured patients should be assumed to have a cervical spine injury until clinical and radiographic assessment has ruled out an injury. For this reason maneuvers to manage the airway must be performed with the head maintained in a neutral position. This is most easily done with one provider crouching at the head of the bed on the left side. Their hands are placed on the sides of the patient's head to stabilize the C-spine while the C-collar is temporarily removed and the airway is dealt with. This should be their only responsibility during this time to ensure proper protection.

B – Breathing and Ventilation

After airway is assessed, all patients should undergo quick auscultation of their lungs for equal breath sounds. Always rule out a tension pneumothorax. This is immediately life threatening (albeit rare) but very treatable. Absent breath sounds on one side, deviated trachea to the opposite side and distended neck veins are indicative of a tension pneumothorax. The treatment is immediate needle decompression with a large bore needle (14 gauge) through classically the 2nd intercostal space at the mid-clavicular line (although may also be done at the 5th intercostals space, mid to anterior axillary line), followed by chest tube insertion at the 5th intercostal space (i.e. at or just above the level of the nipple), mid to anterior axillary line.

Apart from identifying this critical initial condition, the chest should be exposed, inspected and palpated. Inspection looks for equal rising of the chest, contusions and signs of flail chest segments. Palpation will detect tenderness from rib fractures,

flail segments and subcutaneous emphysema from pneumothoraces or tracheobronchial tree injuries. Repeat auscultation may reveal decreased air entry from a pneumothorax or hemothorax.

During this time, close attention is given to the SaO₂. Supplemental oxygen should be initially provided to all significantly injured trauma patients.

C – Circulation (with hemorrhage control)

Circulation is one of the most complex parts of the trauma resuscitation. It can initially be assessed by simple observation – the awake, talking patient has a good enough circulation to perfuse their brain. There are several parameters that we follow to assess circulation:

1. Level of consciousness
2. Pulse
3. HR
4. BP
5. Skin color
6. Urine output
7. Base deficit (from an ABG or VBG)

Hypotension (“shock”) in the trauma patient is by far most commonly due to hemorrhage. Our task in these patients is to rule out other causes of shock, rapidly identify the site of the hemorrhage and stop the bleeding.

Other causes of shock

The mnemonic “SSHOCK” is used for the causes of shock:

S – septic – rare in trauma, patient would have to have concurrent illness

S – spinal (i.e. neurogenic) – may result from high spinal with loss of sympathetic trunk – these patient present with a low BP and a **low HR**

H – hypovolemic – usually hemorrhagic in trauma; by far the most common cause

O – obstructive – may occur from tension pneumothorax or pericardial tamponade

C – cardiogenic – rare in trauma – possibly from large blunt myocardial injury

K – anaphylac(K)tic – rare in trauma

Keeping those in mind, your differential for the hypotensive trauma patient should look something like:

1. Hemorrhagic
2. Hemorrhagic
3. Hemorrhagic
4. Obstructive (tension PTX or cardiac tamponade, etc.)
5. Neurogenic (from high spinal injury with loss of sympathetic trunk >> clue is hypotension and bradycardia)

6. Cardiogenic (i.e. massive blunt myocardial injury – rare)
7. Other (i.e. Non-traumatic – sepsis, MI, anaphylaxis, etc --- may have “caused” the trauma --- i.e. “medical trauma”)

As these other causes of shock in trauma patient are quite uncommon, once we have considered them, we rapidly search for the source of hemorrhage.

Hypotension by system

The easiest way to think of the hypotension is by system:

Head

An intracranial bleed does not cause shock from blood loss. A severe head injury may cause hypotension from compression of the brainstem; this is usually accompanied by bradycardia as well.

Neck

A penetrating neck injury with major vessel involvement can result in significant blood loss. This is usually evident on inspection. Initial management is direct pressure, followed by rapid transfer to the OR for repair.

Chest

Several injuries in the chest can be the source of the hypotension:

1. **Tension pneumothorax** – must maintain an index of suspicion (despite the fact it's quite rare) as it is immediate life threatening and very treatable. Look for decreased breath sound on the affected side, tracheal deviation to the other side and distended neck veins. Requires immediate needle decompression followed by chest tube insertion.
2. **Hemothorax** – decreased breath sounds may be heard if large enough. Diagnosed on CXR. Treatment is placement of a chest tube, monitor bleeding from the chest tube, and replace the losses.
3. **Aortic arch injury** – a rare injury for patients who survive transfer to the ER. Most aortic injuries die on scene. The aorta usually rips at the ligamentum arteriosum. May see clues on CXR, but need CT to diagnose. Treatment is open or endovascular repair by a cardiac surgeon.
4. **Ruptured Diaphragm** – A rare injury. If large enough to cause hypotension by itself, requires immediate surgical repair in the OR.
5. **Penetrating heart/large vessel injury** – penetrating trauma injuries are approached differently than blunt trauma injuries. A hypotensive penetrating chest trauma patient will usually get bilateral chest tubes due to the high incidence of hemothorax. If pericardial fluid is

seen on the ER U/S, they will require thoracotomy in the OR for presumed cardiac injury. Emergent thoracotomy is performed in the ER if the patient arrests prior to transfer to the OR.

Abdomen

The abdomen is a common cause of hypotension in trauma patients because of the fragile structures within – namely the liver and spleen. Because of the high incidence of injuries to these structures, they are evaluated early in hypotensive trauma patients. The advent of the FAST (Focused Assessment with Sonography for Trauma) exam – essentially an ER U/S – has allowed us a window into intra-abdominal and pericardial bleeding while bypassing critical time we used to spend obtaining a CT scan.

1. **Spleen** (~50% of pts with abdominal injuries) – patient may have tenderness in the LUQ on exam. The FAST exam looks for free fluid in the spleno-renal space. Treatment may range from immediate OR for the unstable patient to embolization of the bleeding splenic vessels via angiography to simple observation for the stable patient.
2. **Liver** (40%) – look for tenderness in the RUQ on exam. The FAST exam looks for free fluid in Morrison's pouch. Management is similar to the spleen, ranging from immediate OR to embolization to observation.
3. **Retroperitoneum** (15%) – most often due to renal laceration. Usually contained and rarely requires immediate OR
4. **Other** – may include injury to the bowel, IVC, pancreas, bladder. With the exception of the IVC, they rarely cause immediate hypotension.

Pelvis

The pelvis is another common source of bleeding in polytraumatized patients. Pelvic bleeding can be loosely divided into arterial vs. venous bleeding. As you would imagine, patients with arterial bleeding can become unstable more quickly. Assessment for pelvic bleeding begins with “loading” the pelvis on physical exam to assess for stability. An AP pelvis x-ray will help determine fractures. An unstable pelvis is much more likely to have significant bleeding associated with it.

Our most useful tool in the trauma room is the ER FAST U/S exam. This should be positive if there is a significant amount of blood in the pelvis. While the “gold standard” is considered a CT scan, in unstable patients, the FAST will promptly determine the likely source of blood loss and will minimize delay in definitive OR management.

Management of pelvic bleeding

Our initial treatment of an unstable pelvis fracture is simple wrapping tight with a bed sheet or pelvic binder, which will help tamponade bleeding vessels. It is therefore considered a resuscitation measure and should be done as soon as an unstable pelvis is discovered. This will help to temporize the bleeding, but if the patient remains unstable, the options are:

1. **Pelvic angiography with embolization** – effective but not readily available. Bleeding arterial vessels in the pelvis can be embolized.
2. **OR for pelvic packing** – more readily available and may be effective but may still require angiography/embolization after. A pelvis external fixation (“ex-fix”) device can be applied by orthopedics in the OR, but is not commonly done early as the pelvic binder is felt to have similar efficacy in temporizing the bleeding.

Long bone fractures

The classic teaching is that femur fractures may have an associated 1.5 L (humerus much less) of blood loss in that compartment; in reality they are rarely the main cause of hypotension in trauma patients. The treatment is splinting of the fracture to help tamponade the bleeding.

External bleeding

These sites are usually obvious if the patient is properly exposed. The most effective initial treatment is **direct pressure** to the area. Avoid the temptation to “clamp” the culprit vessel(s). This results in more bleeding while trying to position the clamp, and may damage a repairable vessel.

Treatment of Shock

The unstable nature of trauma patients requires early active treatment while we are looking for the source of the hypotension. Two large bore, peripheral IV catheters should be started. The most readily accessible peripheral veins are in the antecubital fossa. The standard initial treatment is a 2L bolus with normal saline (NS). There is no evidence in the literature to support the use of ringer's lactate (RL) or colloid (e.g. pentaspan). NS is less expensive, and compatible with most medications/fluids given (e.g. blood).

If a patient is hypotensive after 2 L NS, PRBCs should be started. Type O blood should be immediately available in most centers. O +ve is used for all males and females past childbearing age (>age 50). O -ve is used for females below age 50 to prevent Rh sensitization. Type-specific blood takes about 20-30 minutes, while fully cross-matched blood takes about 45-60 minutes.

On some occasions, PRBCs may be started immediately if the patient is in profound shock and it is clear early that a patient will need blood; delaying blood in these patients can result in “too much” NS and problems with dilutional coagulopathy.

D - Disability

The initial neurological assessment in trauma is intentionally simple and quick; we simply want a general idea of the patient’s level of consciousness. The mnemonic “AVPU” is used:

- Alert
- responds to Verbal stimuli
- responds to Painful stimuli
- Unresponsive

Besides using one of the above general descriptive, the pupils and general muscle function are also quickly examined. If a patient has a dilated pupil and contralateral hemiparesis, they may require urgent operative decompression. Hence the term “moving all 4s” affords us some time for focus on other areas.

The Glasgow Coma Scale (GCS) is another relatively easy way to follow a patient’s level of consciousness. A decreasing GCS requires rapid search (i.e. urgent CT head) for surgically treatable head injuries (epidural or subdural hematomas). ***Even a brief period of hypotension*** with a significant traumatic head injury predicts a ***poor outcome***, so be sure to adequately treat the ABCs.

E - Exposure and Environmental Control

All trauma patients should be fully exposed, which usually involves cutting off their garments in the most urgent cases. Remember that many of our traumas are “well”, and some clothing (i.e. pants) may be simply removed. Once undressed, patients need to be “logrolled” to assess the back using:

1. Inspection for penetrating injuries, contusion, lacerations
2. Palpation for T and L spine tenderness
3. Rectal exam to assess for a high-riding prostate (a contraindication to foley insertion), gross blood (indicative of bowel injury)

The “logroll” is done with one provider stabilizing the neck for C-spine protection. On that provider’s count to “3”, the patient is turned by 2 or 3 others (away from any painful injuries to minimize discomfort). The backboard is removed first before the back is inspected as above. Leaving a patient on a backboard unnecessarily can lead to skin ulcerations in as little as 2 hours.

N.B. - the **logroll** exam is especially important for **penetrating trauma patients**. It is usually done rapidly in these patients as wounds in the posterior cardiac box may require emergent thoracotomy if the patient arrests.

“Environment” is included to remind us to keep the patient warm and dry. The combination of the external environment (especially in winter), undressing, and massive fluid resuscitation can lead to hypothermia, which in turn can worsen an already coagulopathic patient. The room temperature should be turned up, blankets applied and warmed IV fluids used to minimize the hypothermia.

INITIAL INVESTIGATIONS

Most major polytraumatized patients will require:

1. CXR
2. AP pelvis
3. FAST exam

Other radiographs may be done as indicated. The decision to perform CT scans is based on the condition of the patient and investigations already done. While the CT is our best tool to rule in or out injuries, some patients may require urgent OR, i.e. be too “unstable” for the CT scanner.

FURTHER WORKUP / TREATMENT

A sound knowledge of proper resuscitation of the polytraumatized patient provides the basis for trauma care. The ATLS-based “ABCDE” method is a simplified, systematic way to prioritize resuscitation.

Once complete, the provider will move on to secondary assessment, further investigations and definitive care.

REFERENCES

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44: THE SECONDARY TRAUMA SURVEY

Mike Brzozowski
Updated by Laura Hans

OBJECTIVES

1. To understand the non-sequential nature of performing a secondary trauma survey
2. To review the examination and management of the head, neck, chest, abdomen, musculoskeletal and neurologic systems in the traumatized patient

INTRODUCTION

The objective of the secondary survey is to identify the majority of injuries, which may be present in a trauma patient. This is performed after the initial primary survey (identifying life threatening injuries) and the initial resuscitation. The secondary survey is an organized, head to toe assessment of the trauma patient. It has been said that after the survey is finished the patient will have had a complete examination including “fingers and tubes in every orifice”. Also at this time, blood work is sent (CBC, Electrolytes, BUN, Cr, PT and INR, Lipase, Alcohol level, and any other indicated tests), including group and screen or crossmatch for blood as indicated by the estimated blood loss and ongoing blood loss and the urgency for blood product administration.

The following is a systematic approach to the examination. This approach is only a guide, and with experience one will develop ones' own individual approach. It assumes that the primary survey has been completed, that any immediate life threatening injuries have been addressed and that the resuscitation is ongoing as are repeated re-evaluations of the patient's general status.

During the secondary survey numerous activities are taking place simultaneously, including your examination, X-rays and Ultrasound, and the application of splints/dressings. This makes it difficult to write out a sequential set of instructions to follow. However, one should try to prioritize assessment and resuscitation efforts given the patient's ever-changing status.

The evaluation of the trauma patient with significant risk of injury has changed with the advent of extremely fast and accurate CT scanners. Many centers liberally employ the use of CT scan as an evaluative tool. It is not uncommon in the stable

trauma patient, to scan the head, cervical, thoracic and lumbar spines, the chest and abdomen and pelvis if fractured. CT Angiography is also used to evaluate potential vascular injury. Plain XR is usually limited to the chest, pelvis and extremities.

HEAD AND NECK

As with the other anatomic subgroups that will be discussed, much of the exam will depend on the level of consciousness of the patient. Obviously more alert patients will be able to cooperate and tell you what hurts and provide information regarding motor and sensory functions. Regardless, a reasonable place to start when examining the head and neck is the pupils and eyes. Pupillary size and response to light should be noted. Any abnormality in gross visual acuity and anatomic disruption of the eye and or orbit should be assessed. Contact lenses should be removed at this time.

Examination of the face should include palpation of all facial bones, and note should be made of swelling, deformity and lacerations. Remember that injury to the head and face indicates a threat of cervical spine injury, and thus until the C-spines are radiologically and/or clinically cleared, appropriate precautions should be taken. At this point one should consider the placement of a nasogastric tube. Contraindications to this procedure include facial smash, evidence of anterior basal skull fracture (raccoon eyes, nasal cerebral spinal fluid leak), and arguably, penetrating neck trauma with the potential of esophageal penetration. In the blunt trauma population, particularly the obtunded intubated patient, an orogastric tube may be a safe alternative to the NG tube.

Patients may often have significant trauma to the scalp, which may go undetected if not carefully assessed. The entire scalp should be palpated, including the occipital region, looking for lacerations, hematomas and evidence of fractures. The tympanic membranes should also be assessed for the presence of hemotympanum or otorrhea. With in-line stabilization of the C-spine, the cervical collar should be removed to visually inspect the neck for injury. The secondary survey should be done with full spinal precautions in place. Helmets should be removed

with spinal immobilization using as little traction as possible. With the help of a second person, the C-spines should be palpated for tenderness and deformity. One person should hold the head and immobilize the head and neck (not applying traction) while the other removes the collar and palpates the neck. The collar is then reapplied until the spines are cleared.

A general rule with respect to the penetrating trauma victim is to apply compressive dressings to any wounds and resist the temptation to explore them in the trauma room. The exploration is generally best performed in the operating room.

CHEST

Included in the evaluation of the chest should be a quick reassessment of the patient's respiratory status (e.g. respiratory rate, comfort of breathing). If any deterioration is noted, the patient must be re-evaluated for immediate life-threatening chest injuries.

Inspection of the chest should include looking for signs of a flail segment, decreased respiratory expansion, or contusion to the chest wall. Next, palpation of the entire rib cage is necessary, looking for crepitus from either sub-cutaneous air or bony fractures. Auscultation of the lung fields is carried out, paying attention to asymmetric air entry indicating most often pneumothorax, hemothorax, or pulmonary contusion.

The clinical exam alone may provide enough information to indicate the need for a needle thoracostomy or a chest tube. For example the diagnosis of a pneumothorax may be made with the presence of palpable rib fractures and sub-cutaneous air on one side, and thus a chest tube would be indicated even prior to obtaining a chest X-ray. If no clear indications exist for immediate intervention, the CXR should be one of the initial X-ray studies done.

CLINICAL PEARL

Ultrasound can be used during the secondary survey to diagnose small pneumothoraces or hemothoraces that can be missed on plain films. Early detection of these injuries is important especially in those who are to receive positive pressure ventilation or be transported

ABDOMEN

Perhaps one of the most difficult areas to evaluate is the abdomen. The abdominal cavity provides a space that can harbour large amounts of occult blood, before any overt clinical change is appreciated. For this reason, with any significant change in a patients' hemodynamic status, the abdomen should be strongly considered as a source of blood loss. The general principles apply: inspection, palpation and auscultation, and the performance of diagnostic tests to aid in identification of injury. The importance of repeated serial examinations should also be stressed.

Indications for immediate laparotomy include an alert patient with obvious peritonitis, a rapidly distending abdomen, a patient with suspected abdominal trauma and signs of hemodynamic instability, positive FAST or Focused Abdominal Sonography in Trauma examination, or significant CT scan, a positive Diagnostic Peritoneal Lavage (DPL) and certain situations in penetrating trauma.

In patients without indications for laparotomy, and significant risk for intra-abdominal pathology, FAST and/or CT abdomen are done to rule out injury. FAST examination has largely replaced any indications for DPL today. It serves as a "virtual" DPL indicating the presence or absence of free fluid in the abdomen presumed to be blood in the trauma patient. A good quality FAST can reliably detect approximately 200cc of free intraperitoneal fluid. With experience even smaller volumes can be detected. In a stable patient with significant injury or mechanism, a CT is often performed to more definitively rule out injury. One may often see injuries that do not cause a large amount of free fluid yet are present on CT scan.

As part of the secondary survey a rectal exam should be performed. This will yield information about lower gastrointestinal bleeding, prostatic position and integrity of the rectum in the scenario of pelvic fracture, and anal sphincter tone. A Foley catheter should be placed at this time as well.

Contraindications to a Foley catheter include perineal smash or hematoma, blood at the urethral meatus, and a high riding prostate. If a female, consider a speculum and/or bimanual exam if there is evidence of pelvic trauma.

MUSCULOSKELETAL

All extremities are to be evaluated to identify swelling, deformity, contusion, tenderness, and abnormal movement. Any area suspected for fracture should obviously be X-rayed, time permitting with respect to the overall status of the patient. Special

attention should be paid to fractures that may potentially be open. These are treated with gross debridement, lavage, dressing and splint application, in addition to antibiotic administration and addressing the patient's tetanus immunization status.

The patient should be log rolled, and the thoracic and lumbar spines palpated for tenderness and deformity. Remove the long spine board, as prolonged immobilization on this board may cause pressure sores on pressure points. A thin mattress on a rigid stretcher provides adequate support even in the presence of thoraco-lumbar fractures. In the presence of penetrating trauma, the log roll is performed early looking for additional penetrating wounds. Multiple wounds are common and must be carefully searched for especially in areas such as the gluteal cleft and axillae.

Finally, the pelvis requires examination. Just as the peritoneal cavity can hide blood loss, so too can the retroperitoneal space in the form of a retroperitoneal hematoma, most commonly from a pelvic fracture. The iliac crests and pubic symphysis are palpated, and the pelvis should be examined for stability in three planes: lateral compression, antero-posterior compression, and vertical stability.

NEUROLOGICAL ASSESSMENT

A thorough head to toe neurological exam needs to be documented. Early documentation of the Glasgow Coma Scale is useful since it provides an objective neurologic "snapshot" in time. Changes in mental status can then be objectified and patient care may be directed. For example the head-injured patient, may require definitive airway management plus hyperventilation and consideration transfer to a neurosurgical centre.

Extremities should be examined for any motor or sensory deficit suggesting a spinal cord injury.

SUMMARY

- the secondary survey is simply a thorough evaluation of the trauma patient including the head, neck, chest, abdomen, musculoskeletal and neurologic systems
- it includes a focused physical examination as well as the 'fingers and tubes in every orifice' approach
- during the secondary survey numerous things are taking place simultaneously, including your examination, X-rays, and the application of splints/dressings
- injury to the head and face indicates a threat of C-spine injury, and thus until the C-spines are radiologically and/or clinically cleared, appropriate precautions should be taken
- the abdominal cavity provides a space that can harbor large amounts of occult blood, before any overt clinical change is appreciated. For this reason, with any significant change in patient status, the abdomen should be strongly considered as a source of blood loss.
- all extremities are to be evaluated to identify swelling, deformity, contusion, tenderness, and abnormal movement
- at the end of the survey, the clinician should have an accurate estimation of the patient's injuries, and the plans for definitive management can be initiated

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45: HEAD AND CERVICAL SPINE TRAUMA

Nazanin Meshkat

OBJECTIVES

- To develop an approach to system-specific physical examination as part of the primary and secondary trauma resuscitation
- To develop an approach to the classification and management of head and c-spine injuries
- To develop an approach to the radiologic interpretation of c-spine injuries
- To describe the Canadian CT head rules and the Canadian c-spine rules

HEAD INJURIES

ANATOMY

The brain consists of the cerebrum, cerebellum, and brainstem. It is lined by the meninges. The pia is the innermost layer of the meninges, followed by the arachnoid and the outermost layer dura. The brain is bathed by the cerebrospinal fluid, produced by the choroid plexus located in the lateral ventricles.

The internal environment of the brain is finely balanced. The blood-brain barrier maintains the microenvironment of the brain tissue by tightly controlling the materials that cross the capillary brain interface. The normal intracranial pressure (ICP) fluctuates between 5-15 mm Hg. Appropriate cerebral blood flow is maintained through the process of autoregulation and the cerebral perfusion pressure (CPP). CPP = mean arterial pressure – ICP.

PHYSICAL EXAM

As part of the initial workup for head injuries, a physical exam, looking for signs of trauma, and a neurologic exam should be performed.

Signs of Trauma

As part of the primary trauma survey establish the presence of head trauma by looking for obvious signs of trauma, while protecting the c-spine with immobilization. In the secondary survey perform a more detailed examination, looking for bruising, soft tissue swelling, lacerations and fractures. A specific type of fracture, the basilar skull fracture, must be suspected if the following signs are present: blood in the ear canal, hemotympanum, rhinorrhea,

otorrhea, “Battle's sign” (retro-auricular hematoma), “Raccoon eye” (peri-orbital ecchymosis).

Neurologic Exam

During the primary trauma survey, perform a screening neurologic exam, consisting of the **Glasgow Coma Scale (GCS)** and pupillary exam. As part of the secondary trauma survey perform a more detailed neurologic exam. In unconscious patients determine brainstem function by looking at the respiratory pattern, pupil and eye function (the corneal reflex, Doll's eye (oculocephalic reflex), Caloric Test (oculovestibular test).

Glasgow Coma Scale

Assess the patient for each of the 3 following categories. Determine the GCS score. The maximum GCS is 15. The minimum GCS is 3.

EYE OPENING

- 4 Spontaneous
- 3 To verbal command
- 2 To pain
- 1 None

VERBAL

- 5 Alert and oriented
- 4 Disoriented
- 3 Incomprehensible
- 2 Moans and sounds
- 1 None

MOTOR

- 6 Follows commands
- 5 Localizes pain
- 4 Movement or withdrawal to pain
- 3 Decorticate positioning
- 2 Decerebrate positioning
- 1 None

CLASSIFICATION OF HEAD INJURIES (HI)

Head injuries are classified as mild, moderate and severe. Approximately 80% of HI are minor head injuries (Glasgow coma scale = 14-15), 10% moderate (GCS = 9-13), and 10% severe (GCS 8 or less).

MINOR HEAD INJURIES

Most head injuries are minor. Of all minor HI, less than 1% will have a surgically significant lesion. Not all patients with minor HI need further workup (i.e. a CT scan) if their physical exam does not raise any concerns. A number of trials have tried to determine indications for CT scanning in patients with minor HI. The “Canadian CT Head Rules” is a clinical decision rule (CDR) that helps clinicians determine the need for a CT head in patients with minor head injuries.

Canadian CT Head Rule

In this CDR, minor head injuries were defined as the presence of amnesia, loss of consciousness, disorientation, and a GCS 13-15 as a result of a HI in the previous 24 hrs. Patients with penetrating trauma, obvious depressed skull fractures, acute neurologic deficits, seizures, bleeding disorders or on anticoagulation, pregnant patients and those under 16 years were excluded from the decision rule.

The rule consists of 5 high risk and 2 medium risk criteria.

High Risk Criteria

- GCS < 15 two hours after injury
- suspected open or depressed skull fracture
- signs of basilar skull fractures
- vomiting > 2 episodes
- age > 65

Medium Risk Criteria

- amnesia > 30 minutes
- dangerous mechanism (pedestrian struck by motor vehicle, occupant ejected, fall >3 ft or 5 stairs)

Imaging ordered based on high risk criteria had a sensitivity of 100% and a specificity of 69% in detecting significant brain injuries. The sensitivity and specificity of all 7 factors for detecting clinically important HI on CT were 98% and 50%, respectively. If applied to this patient population the clinical rule would eliminate the need for CT in 382 patients (12.2%), although it would have missed 4 brain injuries classified as clinically significant (however

none required surgical intervention or had permanent neurologic sequelae).

There are a number of limitations to this CDR. Depending on baseline utilization rates, at some centers the rule could actually increase number of CTs. The study defines minor head injuries as a GCS of 13-15, whereas current guidelines categorize a GCS of 13 as a moderate HI (due to the increased incidence of significant brain injuries in these patients).

Concussion

A concussion is a type of minor HI after a direct or indirect blow to the head characterized by a transient change in cognitive or neurologic function (with no findings on CT). The change in cognitive or neurologic function may include any of the following - confusion and amnesia, disorientation, loss of consciousness, restlessness, lethargy, irritability, or brief seizure immediately after the insult (known as “impact seizure”). Repeated insults to the brain can cause permanent cognitive damage. The Sport Concussion Assessment Tool 2 (SCAT2) can be used as a guide for concussion assessment (available at [http://www.cces.ca/files/pdfs/SCAT2\[1\].pdf](http://www.cces.ca/files/pdfs/SCAT2[1].pdf)).

There are a number of available guidelines for the management of sustained concussions during sport activities. In recent years there has been a move away from the classification or “grading” of concussions in leading the management of patients when returning to play. The Concussion In Sport Group’s current recommendation is to measure recovery from symptoms to guide return to play decisions in a graduated manner (Please refer to Table 1). The 1997 American Academy of Neurology Return to Sport Guidelines will be updated towards the end of 2012 (<http://www.aan.com/go/practice/concussion>).

Table 1: Graduated Return to Play Protocol*

Rehabilitation Stage	Functional Exercise at Each Stage	Objectives at Each Stage
1. No Activity	Complete physical and cognitive rest	Recovery
2. Light aerobic exercise	Walking, swimming or stationary cycling keeping intensity < 70% MPHR; no resistance training	Increase heart rate
3. Sports-specific exercise	Skating drills in ice hockey, running drills in soccer, no head impact activities	Add movement
4. Non-contact training drills	Progression to more complex training drills, e.g. passing drills in football and ice hockey; may start progressive resistance training	Exercise, coordination and cognitive load
5. Full contact practice	Following medical clearance, participate in normal training activities	Restore confidence and assess functional skills by coaching staff
6. Return to play	Normal game play	

*From: Consensus Statement on Concussion in Sport 3rd International Conference on Concussion in Sport

MODERATE AND SEVERE HEAD INJURIES

In moderate and severe head injuries mechanical forces cause permanent damage to the brain. This mechanical damage (primary brain injury) is irreversible. However, following this initial mechanical injury there are a number of derangements that can cause secondary brain injury. The treatment of head injuries aims to control the factors that can lead to secondary brain injury. These include hypotension, hypoxia, anemia, abnormal glucose control, and hyperthermia.

MANAGEMENT OF MODERATE AND MAJOR HEAD INJURIES

As with any emergency situation begin with the ABC's.

Airway

If the patient is not protecting their airway (GCS < 9) prepare to intubate the patient using rapid sequence intubation (RSI). Consider the following premedication drugs to prevent raising the intracranial pressure - lidocaine (1.5-2 mg/kg IV push) to attenuate cough reflex and vecuronium (0.01 mg/kg IV = defasciculating dose) to prevent fasciculations with succinylcholine. The evidence for these interventions is poor, however they are often used. When choosing an induction agent, an appropriate agent is one that does not raise the intracranial pressure, such as etomidate (0.3 mg/kg IV), midazolam (0.1-0.3 mg/kg IV), propofol (1-2 mg/kg IV), or thiopental (3mg/kg IV – the availability of this drug is currently limited due to a recent halt in production). The latter two drugs can lower the blood pressure, an event that should ideally be prevented in head injuries as it reduces cerebral blood flow and can increase mortality. Always have an alpha agonist such as phenylephrine at hand in the event that hypotension ensues.

Breathing

Maintain oxygen saturations above 90% (or PaO₂ > 60 mmHg)

Circulation

Treat and avoid hypotension by maintaining a systolic BP > 90 mmHg (in children 70 + age in years x 2) to ensure adequate cerebral perfusion.

Other

Provide analgesia and sedation. Maintain normothermia, correct electrolyte abnormalities and hypoglycemia, and maintain a hematocrit greater than 30% (to maintain appropriate oxygenation of brain tissue). Look for and reverse anticoagulation agents. Obtain a CT head and appropriate neurosurgical intervention for evacuation of hematomas and/or ICP monitors. Steroids are not to be used as they increase mortality. The use of induced hypothermia is controversial in the setting of head injuries. Prophylactic use of anti-seizure medications can decrease the incidence of early post traumatic seizure (within 7 days), however are not associated with improved outcomes or prevention of late post-traumatic seizures.

Increased ICP

If there are signs of increased ICP (such as anisocoria, labile BP, Cushing syndrome = progressive hypertension, bradycardia and respiratory depression), treat the increased ICP. If an ICP monitor is present (not usually the case in an emergency setting) aim for an ICP < 20-25 mmHg.

1. Initial Therapy

- Elevate head of bed
- Keep head midline
- Provide sedation
- Avoid unnecessary stimuli
- Provide analgesia
- Mannitol 0.25 - 1g/kg IV if deteriorating despite the measures above (monitor for arterial hypotension)
- Neuromuscular blockade
- Mild hyperventilation aiming for a PaCO₂ of 30-35 mmHg

2. Secondary Therapy (for refractory raised ICP)

- Barbiturates or propofol to control elevated ICP
- Hypertensive therapy
- Moderate hyperventilation (PaCO₂ < 30 mmHg)
- Decompressive craniectomy

CLINICAL PEARL

Hyperventilation reduces PaCO₂ which temporarily reduces the ICP by cerebral vasoconstriction and subsequent reduction of cerebral blood flow. The onset of action is within 30 seconds and it peaks by 8 minutes. The aim of hyperventilation is to reach an arterial PCO₂ of 30-35 mm Hg (avoid PaCO₂ < 25 as it can cause ischemia due to severe vasoconstriction). Hyperventilation should be avoided during the first 24 hours after injury when cerebral blood flow is often critically reduced. Hyperventilation can lower ICP by 25%. *It should not be used prophylactically.*

CERVICAL SPINE INJURIES**ANATOMY**

There are seven cervical vertebra, C1-C7. C7 articulates with the first thoracic vertebra. C1 or atlas is a ring-like structure consisting of an anterior arch, posterior arch and two lateral masses. The lateral masses of C1 articulate with the occipital condyles of the skull. The anterior arch of C1 articulates with the odontoid process (or dens) of C2 or axis. C2 consists of a body, the odontoid, two lateral masses and a spinous process. C3-C7 are similar - they consist of a body, two lateral masses, and spinous processes. The bony vertebra are separated by intervertebral discs. A complex set of ligaments hold the vertebra and intervertebral discs together.

PHYSICAL EXAM

While examining the patient with a suspected c-spine injury, protect the c-spine by immobilizing it and log-rolling the patient. C-spine immobilization is paramount during the primary trauma survey. As part of the secondary survey, examine the c-spine by palpating the spinous processes for tenderness, bony abnormalities and step deformities. Proceed to a neurologic exam by testing motor, sensory and reflex function. Observe the breathing pattern. Test the rectal tone and the bulbocavernosus reflex.

RADIOLOGIC EVALUATION

The radiologic evaluation of C-spine injuries begins with x-rays. The standard trauma x-rays consist of an anteroposterior (AP), lateral, and odontoid views. Sometimes two oblique views are also included. Flexion and extension views may be ordered to rule

out ligamentous injuries once bony injuries are ruled out on the three standard views.

THE LATERAL VIEW

The lateral view is the most useful. An adequate film visualizes C1-T1. If the film is not adequate a swimmers view should be obtained. Use the ABC'S approach to interpret the x-ray.

A for Alignment

Follow the anterior and posterior contour lines. A translation of one vertebra over another of greater than 3.5 mm and an angulation greater than 11 degrees is considered significant. Also follow the spinolaminar line. The diameter between the posterior cortex and the spinolaminar line should be greater than 18 mm.

In children there is a normal phenomenon called pseudosubluxation where there is a normal “translation” of C2 on C3 (less often C3 on C4). The translation can be viewed in the flexed and neutral position and is not associated with soft tissue swelling (see below), and the posterior cervical line (or the line of Swischuk) should not be more than 2mm away from the spinolaminar line drawn between C1 and C3.

B for Bone

Follow the bony contours of the vertebra looking for breaks in the cortex.

C for Cartilage

First, look at the disk spaces to ensure that they are of equal length throughout. The anterior and posterior aspect of the individual discs should be equal. Second, measure the pre-dental space - it should be less than 3 mm. Third, the distance between the lowest part of the occiput base and the dens should be less than 12 mm. Finally, look at the facet joints - they are arranged at a 45 degree angle, stacked on top of each other.

S for Soft Tissue

The retropharyngeal space lies anterior to the c-spine. At about C4 it widens due to the presence of the esophagus. The retropharyngeal space measures less than 7mm at C2 and 21mm at C7 (or alternatively less than the height of the vertebra at C2 and less than the width of the vertebra at C7).

ANTERO-POSTERIOR VIEW

Trace the bony contours looking for breaks in the cortex. Identify the spinous processes - they should align in a straight line. Finally look at the bilateral uncus processes - they should also align.

ODONTOID VIEW

This view provides a more detailed view of the C1-C2 articulation. In an adequate film, the odontoid should be centered, aligning with the gap between the upper incisor teeth. Again, follow the bony contours looking for breaks in the cortex. The lateral masses of C1 and C2 should align. The distance between the lateral processes should be equidistant.

THE CANADIAN C-SPINE RULE

Not all trauma patients require x-rays of the c-spine. The “Canadian C-spine Rules” is a decision rule to aid in the ordering of c-spine x-rays. It is only applicable to alert and stable trauma patients where cervical spine injury is a concern. (See rule at end of chapter).

CT and MRI

If x-rays are normal and there is a strong clinical suspicion for c-spine injuries further investigations should be carried out. CT scan of C-spine can detect fractures. MRI is superior at detecting ligamentous injuries.

MANAGEMENT

If a C-spine injury is suspected, immobilize the spine at all times. Stabilize the airway and circulation. In patients with suspected injuries at or above C5, early intubation may be warranted due to compromise of the phrenic nerve. Perform a quick neurological exam before intubation if able. If there are neurologic deficits, there is some evidence to suggest that steroids may be beneficial if given within 8 hours of injury, however it should be weighed against increased steroid associated complications such as sepsis. Proceed to imaging. If a C-spine injury is established consult neurosurgery or orthopedics depending on the practice pattern of your center.

SUMMARY

Head Injuries

- Perform an assessment of head trauma as part of the primary and secondary trauma survey.
- Establish the presence of mild, moderate or severe head injuries.
- In mild head injury determine the need for imaging by using decision rules such as the CT Head Rules AND your clinical judgment. For concussions, provide follow-up instructions for return to sports/activity to minimize permanent cognitive damage.
- In moderate and severe head injuries, prevent secondary brain injury by preventing hypoxia (may require intubation), hypotension, and other insults. Be alert for signs of high intracranial pressure and treat accordingly. Consult neurosurgery.

C-Spine Injuries

- If a c-spine injury is suspected, immobilize the c-spine, and maintain c-spine precautions by log-rolling the patient.
- Obtain adequate x-rays and systematically assess the x-rays using the ABCS approach. Pay special attention to the lateral view.
- If a c-spine injury is strongly suspected in the case of 'normal' x-rays, perform further imaging using CT or MRI.
- Not all patients require imaging in the first place; use the C-spine rule AND clinical judgment to determine the need for imaging.

- If a C-spine injury is established consult neurosurgery or orthopedics depending on the practice pattern of your center.

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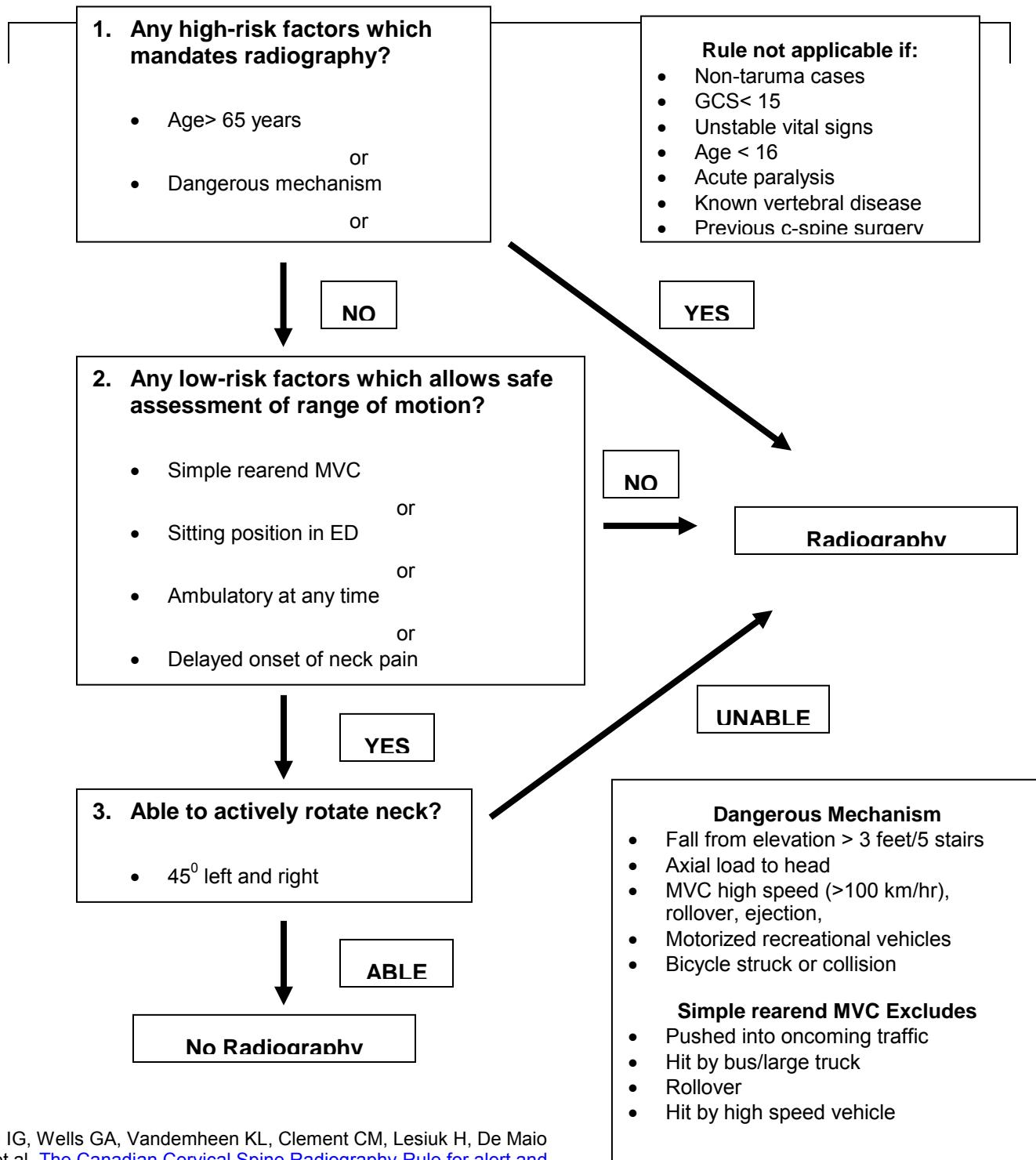
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Normal Lateral C-spine X-ray



Canadian C-Spine Rule

For alert (GCS = 15) and stable patients where cervical spine injury is a concern.



Stiell IG, Wells GA, Vandemheen KL, Clement CM, Lesiuk H, De Maio VJ, et al. [The Canadian Cervical Spine Radiography Rule for alert and stable trauma patients](#). JAMA 2001;286:1841-1848.

46: FACIAL/NECK TRAUMA

Glen Bandiera

OBJECTIVES

1. To recognize, categorize and develop a treatment plan for various common facial injuries
2. To define the classification and treatment of dental injuries
3. To understand the anatomy and treatment of penetrating neck injuries

INTRODUCTION

Facial injuries are often anxiety-provoking for both patients and caregivers due to the heavy personal identity associated with facial appearance. Caregivers in particular often have difficulty in approaching a patient with a significant facial injury. Furthermore, significant morbidity may ensue in the future if treatment is not appropriate early after an injury. Luckily, most facial injuries are straightforward to manage as long as one is attentive to a few guiding principles and specific considerations. This chapter addresses several common types of facial injuries and emphasizes approaches and pitfalls in their management.

FACIAL FRACTURES

The face is designed to protect sensitive structures housed within it yet provide a functional and esthetically appealing shape. For example, the eyes are protected by solid infra- and supra-orbital rims, the zygomatic arch protect the muscles of mastication from direct injury and the more delicate central face, difficulty though it may be to think of it this way, functions as a crumple zone to protect the brain and brain stem from frontal assault. The patterns of bony injury that occur in the facial area are thus quite predictable.

Fractures of the nose

Nasal bones are solid near their base and become thin at the distal edges. Fractured nasal bones can be detected clinically through obvious displacement or instability. Rarely are xrays needed. If the patient is having trouble breathing, the nasal aperture can be opened up by using a tongue depressor or nasal speculum to elevate the fracture fragments outward. Otherwise the patient should be counseled that final alignment will need to wait for 2-3 weeks until the swelling subsides and a plastic surgeon can make an assessment.

All patients with nasal trauma should have an inspection of their nasal septum for hematoma. Any submucosal collection of blood (septal hematoma) should be drained and the nare packed to avoid the creation of an ischemic depression in the septum (saddle nose deformity).

In severe cases of nasal trauma, the bones may be pushed backwards and a fracture of the interorbital rim may occur. In this case, a referral should be made for early surgery to reconstruct the underlying sinus to avoid chronic infection and fistulae. This should be suspected if hypertelorism (widening of the eyes) is noted.

Midface fractures

The bones of the midface are usually injured by direct frontal blows. The fracture can occur in the anterior wall of the maxillary sinus or extend further back to the skull base. If the latter occurs, there are three typical patterns that commonly found, depending on the magnitude of the inciting force (Figure 1). A LeFort I fracture extends down through the upper alveolar ridge to either side of the mouth and the upper teeth and bones will be mobile. A LeFort II fracture extends through (or just inferior to) the infraorbital rim and the nasal aperture and upper teeth will move as a unit separate from the cheekbones (zygoma). A LeFort III fracture extends across through the orbits or just above them and the entire midface (including the zygomas) will be mobile.

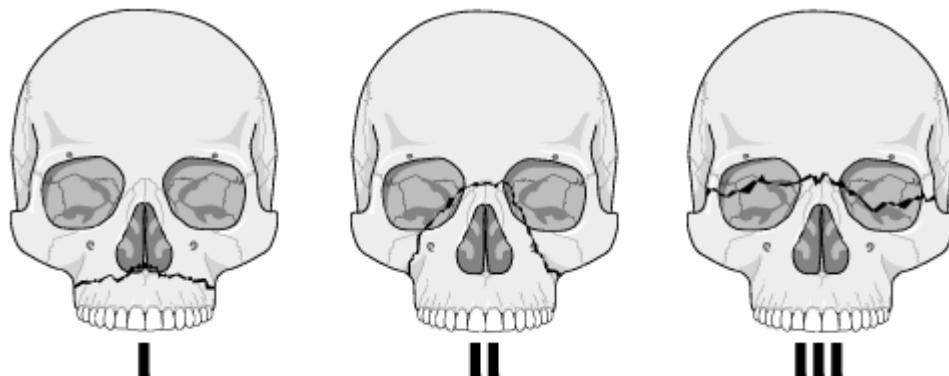


Figure 1: The LeFort Classification of fractures.

The definitive test for most facial fractures is a CT scan of the facial bones, although a series of plain films can be done as a screening test for injury in low risk patients. For all lefort fractures, an early referral to plastics is indicated to plan surgery. Isolated anterior sinus fractures can be seen within a few days. One should always be careful to assess for orbital injury and dental injury in these cases. Antibiotics should be given for all open and sinus fractures.

Fractures of the mandible

The mandible can be fractured at any place but the most common locations are the angle, the paramedian mental area and the condyle. Since the mandible is a three-dimensional structure fixed at either end (in the temporomandibular joints or TMJs) it is highly unusual to have an injury at one location. Usually there are two injury sites and the second can be a subluxation or dislocation of one or both TMJs. Fractures of the mandible should be suspected when the patient: has an irregular bite (malocclusion), feels misalignment in the jaw, has point tenderness of the bone, has loose teeth, feels strong pain when biting down on a tongue depressor you forcefully try to remove from the mouth, or has intraoral bleeding. All teeth should be examined for potential subluxation, fracture or avulsion. The definitive test for mandibular fractures is a panorex plain film. Open fractures (in the mouth or to the outside) should be covered with antibiotics, including anaerobic coverage for intraoral defects. Treatment of mandibular fractures includes antibiotics (if open), analgesia, and early referral to plastic surgery for assessment (within 2-3 days).

Temporomadibular dislocations

If a patient has malocclusion but not obvious bony tenderness or defects, especially after relatively minimal trauma (such as overly aggressive yawning or minor bump on the side of the face) a TMJ

dislocation should be suspected. When this occurs, the powerful muscles of mastication contract and lodge the mandibular condyle anterior to the joint space. Reduction is usually relatively easy, but the patient must be lightly sedated and given analgesia. The caregiver places their thumbs (wrapped in several layers of gauze to protect them from being inadvertently bitten during the reduction and with gloves on) inside the mouth, outside of the teeth along the inferior alveolar ridge with the fingers wrapped underneath the jaw on the outside of the face. Force is then applied downward on the jaw to release the mandible from the maxilla and then the jaw is tilted posteriorly so the mandible rocks back into the joint space. Do not allow your thumbs to get caught between the teeth as the joint slips back in place since the power of the jaw musculature can cause significant injury.

Fractures of the zygoma

The zygomatic arches can be fractured by direct force. If depressed they should be elevated surgically to ensure proper alignment. Treatment involves referral as an outpatient to a plastic surgeon unless there is displacement significant enough to impede movement of the underlying muscles, in which case the patient will not be able to open and close the mouth and urgent surgical referral should be made.

SOFT TISSUE INJURIES

Facial lacerations should be treated as with all other lacerations in terms of general wound assessment and repair. In addition, wounds should be copiously irrigated and repaired with as little tension as possible (see “Special Considerations”, below). When possible, repair wounds with skin glue. Avoid staples and large sutures in the face.

When repairing lacerations, it is important to try and minimize the eventual effects of scarring. Measures to minimize scar development and visibility include:

1. Remove sutures at day 5 if possible.
2. Use the smallest suture possible, usually a 5-0 or 6-0.
3. Be sure to place sutures at regular, closely spaced intervals.
4. Advise patients to avoid sunlight, use strong sunscreen, or cover the wound when in sunlight for up to 12 months post-injury.

There are several unique areas of the face to consider when planning wound management:

1. Lacerations of the eyelid require a careful search under the lid for bleeding or protruding fat, which might indicate a full thickness laceration (and thus potential injury to the eye itself). Always examine the eye for surface lacerations. Full thickness lacerations involving cartilage should be repaired by an expert.
2. Repair of skin over cartilage (eyelid, earlobe, nares) should be done by an experienced provider. Skin should be brought together loosely over the cartilage and sutures should NOT be placed through cartilage.
3. It is particularly important to ensure that all skin edges match up exactly with the opposite side of the wound to avoid notching (ear) or obvious irregularity (such as with the vermillion border of the lips, or for lacerations involving the eyebrow).
4. Lacerations of the lips should be repaired starting with the vermillion border to ensure proper alignment. For full thickness lip lacerations, only repair the mucosal surface if there is a significant soft tissue defect; mucosal surfaces heal very well on their own otherwise.
5. The lips are a common area for burns, especially in pediatrics. It is important to remember that the labial artery can be thrombosed at the time of a burn and there is a risk of rebleeding when the clot dislodges. This can precipitate a significant amount of bleeding at 5-7 days post-injury for which the parents should be vigilant.

DENTAL INJURIES

Dental injuries are common, especially in children, and can be missed if not specifically considered. There are several types of injuries:

1. Avulsion – the tooth has been removed from the socket.
 - a. Rinse socket with Normal saline
 - b. If the patient has the tooth, sterilize it with providine, chlorhexidine, or hydrogen peroxide (5 second rinse in any of these). Cleanse the tooth immediately with sterile normal saline. Place tooth in socket. Secure with bone wax.
 - c. If tooth cannot be replaced or if patient asks for advice about storage, have the tooth preserved in milk.
 - d. If the tooth is not available, seal the socket with gauze or bone wax, especially if there is bleeding. Occasionally, gelfoam or other coagulant may be required to stop bleeding. In pediatric patients, consider a chest or neck xray to search for inhaled/ingested tooth.
 - e. Consider an xray to determine if there is residual tooth root.
 - f. Refer patient to dentistry for definitive treatment.
2. Fracture – the tooth has been broken
 - a. Check to see if the residual tooth is loose and splint if necessary with bone wax or Skin glue.
 - b. Classify tooth fracture according to Ellis Classification (See Figure 2)
 - c. If the fracture is Ellis 3 or 4 (Pulp exposed), consider covering the tooth with bone wax for pain relief.
 - d. Refer patient to dentistry for definitive care and follow-up.
 - e. Warn patient about temperature sensitivity and sharp tooth edges.
3. Subluxation (Loose Tooth) – tooth is still in situ but is loose
 - a. The patient may complain of looseness but may not notice
 - b. ALL front teeth should be palpated and pressed to assess for stability, also blood at the gumline is strong indirect evidence of either a root fracture or a loose tooth.
 - c. ANY movement suggests injury and should mandate a referral to dentistry for splinting
4. Impaction – a tooth may be pushed into the socket
 - a. Patients may complain of visible or palpable deformity of the teeth
 - b. May be indicated by bleeding at the gumline
 - c. Should be referred to dentist for ongoing care.

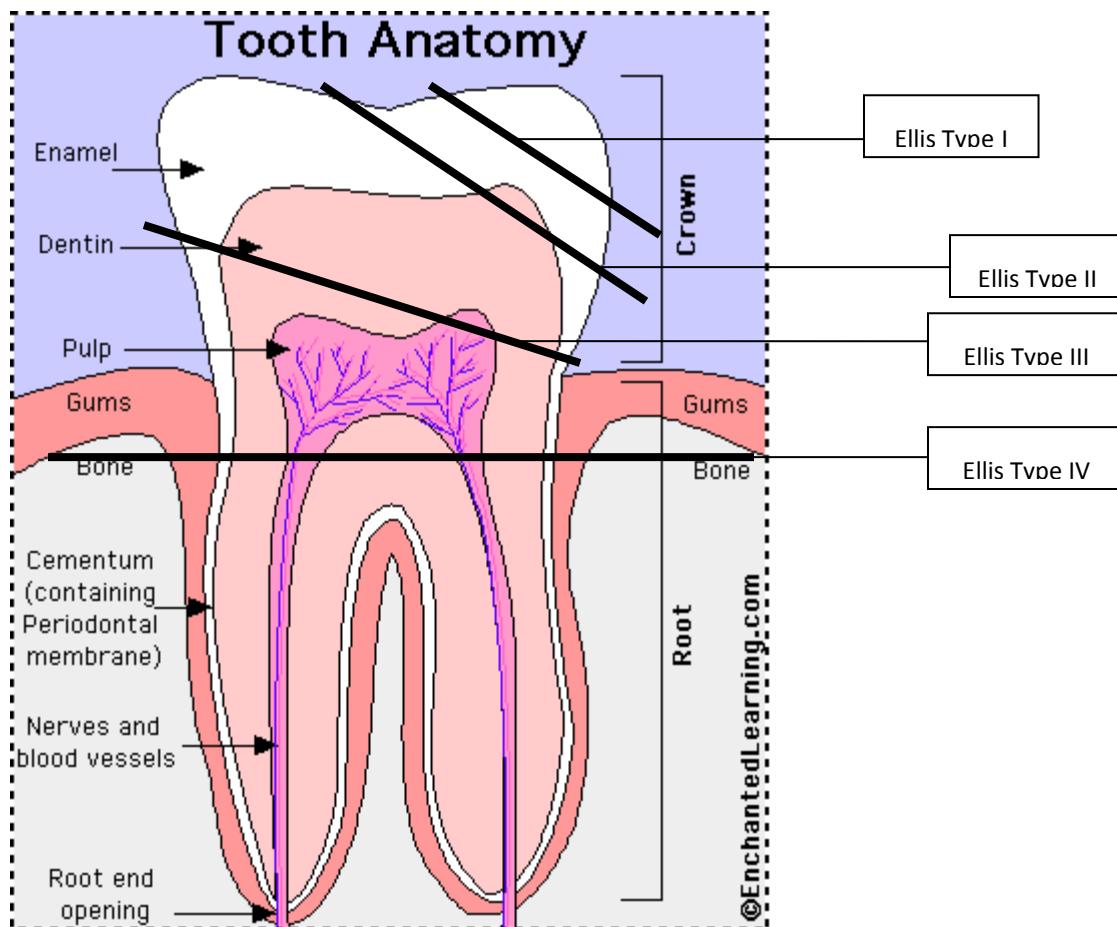


FIGURE 2: Ellis Classification of Tooth Fractures

PENETRATING NECK INJURIES

Neck injuries can be life-threatening. Important structures that can be damaged in the neck and the implications are shown in table below. Patients with neck injuries require urgent assessment using an ABC approach since they are at risk of airway compromise (from swelling, bleeding, direct compression). Signs of upper airway obstruction include: stridor, tachypnea, increased muscle use (sternocleidomastoid, intercostals, trapezius, etc.) abnormal breathing patterns, obvious swelling or masses, external bleeding, bleeding from the mouth. If any of these are present, preparation should be undertaken immediately for intubation by an EXPERIENCED provider. An anatomically disrupted airway is not the time to try your first intubation. One should anticipate any or all of: bleeding, swelling,

displaced airway, altered anatomy, false lumens, or damaged vocal cords. Ideally, patients will be intubated anytime there is concern about the ongoing patency of the airway. This should be done with local anaesthesia and moderate to deep sedation rather than full general anaesthesia. A quick look with a direct laryngoscope, a fiberoptic scope, or a glidescope will help the provider to decide if further sedation or paralysis is safe. If a quick look reveals relatively normal anatomy, then a rapid sequence approach with deep sedation and paralysis can be attempted. If otherwise, an 'awake' intubation should be attempted and a full array of alternative airway interventions should be available, including a cricothyrotomy kit. Even this intervention can be particularly difficult in a patient with a neck injury due to the potential for altered anatomy and hematoma overlying the trachea.

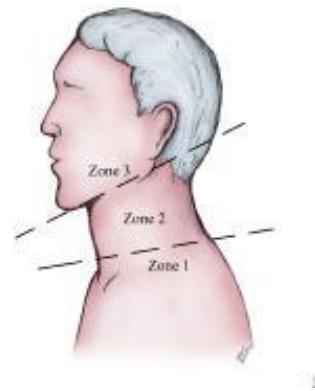
Structure	Implication
Carotid Artery	Hemorrhage, hematoma, stroke, airway compromise
Jugular Vein	Hemorrhage, hematoma, airway compromise
Thyroid Gland	Thyroid dysfunction, hemorrhage, airway compromise
Trachea	Airway compromise
Larynx	Voice abnormalities, airway compromise
Inner Soft Tissues	Airway compromise, pneumomediastinum/thorax, deep space infection
Esophagus	Hemorrhage, deep space infection
Lung	Pneumothorax, hemothorax
Lymphatics	Chylothorax, infection

Important Structures in the neck.

Classically, the neck is divided into zones to help direct investigation of injuries. Zones 1 and 3 are difficult to access surgically and thus an imaging modality should be used to assess for injuries in these areas. Zone 2 is easily explored surgically and a low threshold for surgical exploration should be maintained. Alternatively, with newer imaging options, a good quality scan can rule out significant injury. CT scanning is the modality of choice with newer resolutions able to detect small injuries with good sensitivity. A patient should be assumed to have any or all of: airway, esophageal and vascular injury until proven otherwise. Therefore, imaging using oral and intravascular contrast is required. Since the ability of even modern CT scans to detect microperforations of the esophagus is limited (since they may not bleed or leak air much), many experts advocate doing a rigid (for the upper larynx and esophagus) and flexible endoscopy to rule out injury. The rapid nature and severity of infections after esophageal injury mandate early exploration for these injuries (within 6-12 hours).

Penetrating neck injuries that can be demonstrated by exploration or imaging to be external to the platysma muscle can be repaired in the ED using simple wound repair technique. Any injury that violates the platysma needs to be explored surgically or through a combination of non-invasive and noninvasive visualization as noted above. The latter should be done by an experienced trauma specialist or surgeon.

Figure 3: Zones of the neck for penetrating injury



SUMMARY

- Careful assessment of head and neck injuries is required
- Nasal bone films are rarely indicated in the acute setting
- Remember injury patterns in the midface categorized by leFort 1, 2 and 3
- Suspect a mandible fracture when a patient presents with malocclusion
- Open mandible fractures require antibiotics and a plastic surgery referral
- Lacerations around the eye and ear should be examined carefully and repair should be done by an experienced person

- Neck injuries need to be assessed urgently, airway assessment and intubation should be performed by an expert
- Zone 2 injuries penetrating through the palatysma are often explored in the operating room

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47: COMMON ORTHOPEDIC INJURIES

Laurie Mazurik
Updated By **Fernando Teixeira**

OBJECTIVES

1. To learn a systematic approach to describing fractures
2. To review the initial management of life and limb threatening orthopedic injuries
3. To review the recognition and management of a sample of some commonly encountered orthopedic injuries in the Emergency Department

INTRODUCTION

This chapter is not meant to be a comprehensive review of orthopedic injuries that may present to the Emergency Department (ED). Rather, it is a sampler of the more common orthopedic injuries seen in the ED.

Describing Orthopedic Injuries

It is important to have a systematic approach when describing a fracture to another person (such as the ‘Ortho’ resident on the phone at 3 am). A few important pieces of information are required, and they are the same for every fracture:

- Open vs. closed
- Neurovascular status: intact vs. compromised
- Which bone(s) is/are broken
- Location the fracture: i.e., proximal humerus; metacarpal head
- Type of fracture: i.e. oblique vs. transverse; simple vs. comminuted)
- Alignment of fragments: Angulation, Displacement, Shortening

A few standard points are important here:

- displacement or dislocation is described by the position of the distal fragment relative to the proximal fragment
- angulation is described by the position of the point of the angle (see discussion under ‘Colles’ fracture’ below)

LIFE AND LIMB THREATENING INJURIES

When an orthopedic injury is the cause of an unstable patient it is generally because of blood loss. For example, certain pelvic fractures can cause blood losses of 3L or more, a fractured femoral shaft 1.5L, and a tib-fib fracture 0.75L.

Fractures may result in delayed mortality as well. Delayed threats to life include sepsis (open fractures) and fat embolism.

Limb threatening injuries are caused by compromise to the neurovascular status of the extremity. Orthopedics needs to be consulted for all limb threatening injuries. These include:

- all open fractures
- extensive soft tissue injuries ie. degloving or crush injuries, include Plastic surgery consult
- amputation and some dislocations i.e. knee, consider Vascular surgery consult
- suspicion of compartment syndrome

CLINICAL PEARL

Forearm and lower leg fractures are more likely to be complicated by compartment syndrome. Look for pain out of proportion, parasthesia, pallor, pulselessness and paralysis. The limb may or may not feel cool.

MANAGEMENT

Diagnosis of the fracture is done mainly by plain films but may also include CT and /or MRI imaging. Remember to examine the joint above and below a fracture to rule out associated joint injuries.

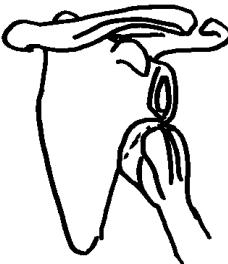
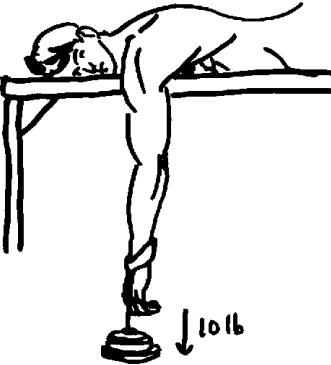
The basic principles of fracture management are the obtaining and maintaining of fracture reduction. Obtaining a reduction may involve manipulating a fracture while maintaining a reduction involves the splinting of a fracture.

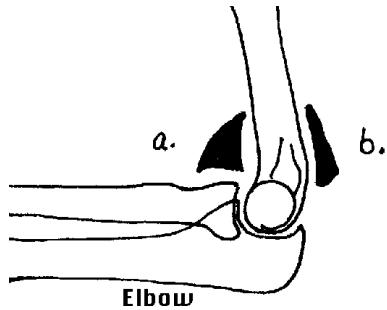
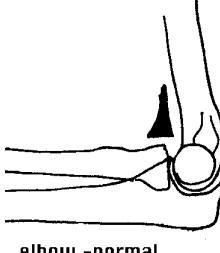
If a reduction is performed, post-reduction films should be obtained in the ED.

Format of the Following Section

Questions are placed on the left side of the page and answers on the right, in 'flash card style'. Cover the right side, attempt to respond, then uncover the correct response as you go.

COMMON UPPER EXTREMITY INJURIES

<p>Q: What injury is shown here?</p> <p>What nerve is at risk of injury?</p> <p>What would you expect to see on the lateral x-ray?</p> 	<p>A: Anterior Shoulder Dislocation.</p> <p>At risk: axillary nerve that supplies sensation to the lateral aspect of the shoulder, and motor to the deltoid.</p> <p>The lateral view would show the humeral head sitting anterior to the glenoid (the glenoid is the centre of the 'Mercedes Benz' sign on the transscapular view)</p> <p>Management: Reduce (see below), immobilize in shoulder immobilizer and re-X-ray.</p> <p>Outpatient Ortho follow-up (unless open joint or unable to reduce = immediate ortho consult).</p> <p>Posterior dislocations are rare; may occur with seizures or following electrical shock.</p>
<p>Q: How is an anterior shoulder dislocation reduced?</p>	<p>A: Many methods have been described. 2 common reduction techniques are illustrated below.</p> <p>Stimson method</p> 

<p>Anterior shoulder dislocations (continued)</p> <p>Like any dislocation, the key is to provide sufficient analgesia and sedation, and to apply firm, constant traction.</p>	<p>The ‘traction-counter-traction’ method</p>  <p>Another one to consider is ‘Scapular Rotation’.</p>
<p>Q: What do ‘a’ and ‘b’ represent on this lateral elbow X-ray?</p> <p>What is their significance?</p> 	<p>A: Both represent radiographic signs of elbow effusion. A tiny anterior fluid collection may be normal (see below); any posterior fluid is abnormal.</p> <p>a. Anterior effusion - called the ‘sail sign’ because it looks like a sail billowed out by the wind (as opposed the normal appearance of lateral view of the elbow):</p>  <p>b. Posterior Fat pad sign Significance: If you see a or b in the absence of an obvious fracture suspect an: occult fracture of the radial head in an adult (treatment = sling) or a nondisplaced supracondylar fracture of the distal humerus in a child (treatment = posterior slab)</p>

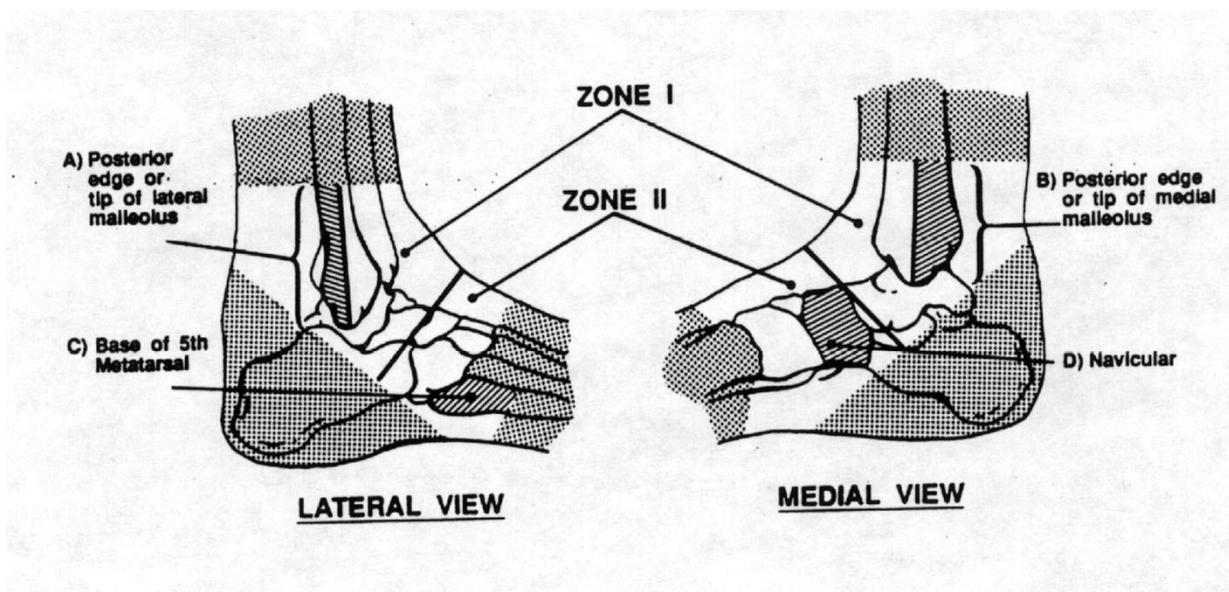
COMMON LOWER EXTREMITY INJURIES

Ankle injuries are very commonly seen in the Emergency Department. It used to be common practice to X-ray everyone with an ankle injury. However, the number of X-rays showing a fracture was low.

Q: What are the Ottawa Ankle Rules, and how are they used?

A: The Ottawa Ankle Rules are a decision tool that were developed in order to reduce the number of ankle and foot X-rays ordered while not missing fractures. The name of the Rules is a bit of a misnomer, since they apply to both ankle and foot X-rays.

They consist of a few simple clinical criteria that are used to determine who may be safely discharged without an ankle or foot X-ray.

**An ankle X-ray series is only required if:**

There is any pain in zone I, AND any of these findings:

- (i) bony tenderness at A OR
- (ii) bony tenderness at B OR
- (iii) inability to bear weight both immediately AND in the ED

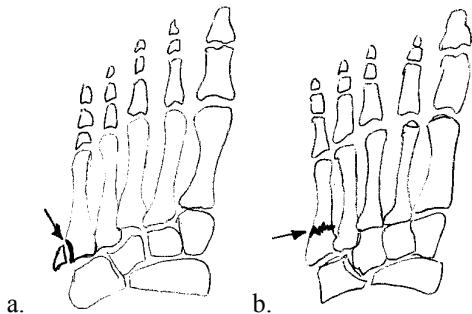
A foot X-ray series is only required if:

There is any pain in zone II AND any of these findings:

- (i) bony tenderness at C OR
- (ii) bony tenderness at D OR
- (iii) inability to bear weight both immediately AND in the ED
- (iv) Limitations of these rules:
 - Presence of growth plates
 - Distracting injuries or substance abuse

Q: What are fractures 'a' and 'b' called?

How are they managed?



A:

- a. Avulsion fracture of the base of the 5th MT. Occurs with inversion injury, due to traction from peroneus brevis tendon.

Management: Can be treated with supportive shoes or tensor; many people opt for a below knee walking cast for comfort. (3 weeks immobilization).

- b. Much less common Jones' fracture = transverse fracture of the proximal diaphysis
 - carries a high incidence of delayed or non-union

Management: Posterior slab; outpatient ortho follow-up

A patient presents with a swollen, tender heel after jumping off a second story balcony. X-rays show a fractured calcaneus.

Q: What other injuries must be suspected and ruled out in this patient?

A: Calcaneal fractures indicate that the patient experienced a significant axial force. Therefore, associated injuries may include:

- ankle fractures / dislocations
- knee injuries
- hip fractures / dislocations
- pelvic fractures
- lumbar spine compression fractures



Colle's Fracture:

Classically described in patient's falling on an outstretched hand (FOOSH)

These are purely extra-articular fractures involving the distal radius

Near anatomic reduction is imperative to minimize post-traumatic OA

The maintenance of volar tilt (angulation of the articular surface of radius on the lateral x-ray) and radial length (the length of the radius in relation to the ulna on the AP x-ray) delineate the need for reduction

Q: Which two parameters are of importance in deciding which Colle's fractures require reduction

A: Volar tilt (at least neutral, anatomic 20 degrees volar tilt)
Radial length (at least as long as the ulna)

SUMMARY

- Displacement or dislocation is described by the position of the distal fragment relative to the proximal fragment
- Certain pelvic fractures can cause blood losses of 3 litres or more, a fractured femoral shaft 1.5 litres, and a tib-fib fracture 3/4 litre
- Compartment syndrome may result following any fracture, especially those involving the forearm or lower leg, and usually presents with pain (out of proportion) +/- paresthesias, and may progress to pulselessness and paralysis
- Anterior shoulder dislocation can cause axillary nerve disruption ,which supplies sensation to the lateral aspect of the shoulder, and motor to the deltoid
- Joint effusions identified on lateral elbow X-rays may represent occult radial head or suprachondylar fractures

- Emergent management of orthopedic injuries in the ED involve obtaining and maintaining fracture reduction
- The Ottawa Ankle Rules are a decision tool that were developed in order to reduce the number of ankle and foot films
- A Jones' fracture (transverse fracture of the proximal diaphysis of 5th metatarsal) carries a high incidence of delayed or non-union
- Calcaneal fractures are associated with injury to the ankle, knee, hip, pelvis and lumbar spine

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48: WOUND CARE

Kevin Skarratt

OBJECTIVES:

1. To review key aspects of the history and physical when assessing a wound.
2. To learn the appropriate use for anesthetic when managing a wound.
3. To learn how to best clean a wound.
4. To examine the various methods of wound closure.
5. To review the indications for antibiotics and immunization.
6. To provide discharge instructions specific to wound care.

INTRODUCTION

Soft tissue wounds account for approximately 7% of all Emergency Room visits. The most frequently injured body parts are the scalp, face, hands, and fingers. The goals of wound repair are to restore function, minimize infection, and optimize cosmesis. These goals are best achieved via a thorough history and physical exam, meticulous wound cleansing and closure, adequate analgesia, proper dressing, and clear discharge instructions.

HISTORY

Bony fractures, tendon lacerations, joint capsule damage, and the presence of a foreign body are all possible in association with a soft tissue wound. A good history with particular attention paid to the force and mechanism of injury will help raise suspicion for the presence of these complications.

Infection risk can also be gauged based on history. Factors that increase the likelihood of infection include:

- Type of injury: crush injuries, deep injuries, and potentially contaminated wounds (foreign bodies, saliva, soil, feces)
- Location of the injury: due to variations in blood, scalp and facial wounds rarely get infected, upper extremity wounds have a lower infection rates than lower extremity 4% vs. 7%.
- Time of presentation: delay increases infection risk

- Medical history: immunocompromised from illness or medication, diabetes

Lastly, the patient allergy list and tetanus vaccination status must be obtained.

PHYSICAL EXAM

Proper lighting, hemostasis, and adequate anesthesia are all important for a proper physical exam. A complete physical exam includes careful and gentle visualization of deep structures as well as an assessment of distal neurological, vascular, and motor function.

Tendon lacerations are common over the dorsum of the hands and feet owing to the relative paucity of subcutaneous tissue in these areas. Always examine the patient in the position of the extremity when the injury is sustained, the tendon laceration may be proximal or distal to the skin laceration. This is done by putting the extremity through a range of motion while visualizing the deep structures. Hemostasis with a blood pressure cuff inflated 20 mm Hg above systolic blood pressure may be required to gain hemostasis.

Foreign bodies can often be detected with a careful physical exam. When necessary, x-ray, ultrasound, or CT scan are helpful adjuncts.

ANESTHESIA

Distal neurovascular function should be assessed prior to administering an anesthetic agent. Regional nerve blocks are often used in the Emergency Department as they enhance patient comfort and facilitate precise closure by avoiding swelling at the wound itself.

Lidocaine is the most frequently used anesthetic agent in the Emergency Room. It is available in 1% (10 mg/cc) and 2% (20 mg/cc) concentrations, with and without epinephrine. The addition of epinephrine aids in hemostasis via vasoconstriction as well as pain control. However, its use can increase infection rates and more importantly can cause tissue ischemia when used in distal areas. These areas can be remembered

with the rhyme “fingers, nose, penis, and toes”. There are small studies that suggest that low dose epinephrine can be used in healthy patients in these areas, however, this has not been studied in an ED setting, where most patients present with many comorbidities. Presently, lidocaine/epinephrine in extremities is avoided.

Lidocaine has a near-instantaneous onset of action and lasts 20-60 minutes. The maximum dose of lidocaine without epinephrine is 3-5 mg/kg. The maximum dose of lidocaine with epinephrine is 5-7 mg/kg. The signs and symptoms of lidocaine toxicity include dizziness, perioral numbness, seizures, cardiovascular collapse, and death.

Infiltration of lidocaine into skin and soft tissue is painful. The discomfort can be ameliorated by injecting slowly with a small (27 g) needle, buffering the solution with sodium bicarbonate and warming the solution prior to injection. Allergy to lidocaine is rare but possible. 1% aqueous diphenhydramine (Benadryl) may be used as an alternate agent.

Topical anesthetic agents are also used in the Emergency Room. They include LET (Lidocaine-Epinephrine-Tetracaine) and EMLA (Eutectic Mixture of Local Anesthetics). These agents are particularly useful in pediatric patients. They take upwards of 20 minutes to cause anesthesia and must be avoided around mucous membranes to avoid inadvertent systemic exposure.

CLEANSING

Thorough cleansing is an important part of wound care. Any debris should be gently removed with forceps, irrigation, or moistened gauze. Non viable tissue should be judiciously debrided by an experienced physician.

Clean, non sterile gloves are acceptable for wound cleanse and closure, as they have not been shown to increase the risk of infection when compared to sterile gloves. Hair can be slicked out of the way with a petroleum-based ointment. If removal is required, clipping with scissors at a length of 1-2 mm is suggested. Shaving should be avoided as it increases the risk of infection, and in the case of eyebrows, may cause permanent hair loss.

Surrounding skin may be cleansed with chlorhexidine or providone-iodine. It should not be used in the wound and must be not come in contact with the eyes. Normal saline or tap water is acceptable for

cleansing the wound itself. Irrigation at a pressure of at least 7 psi with normal saline or tap water has been shown to significantly reduce infection rates. A 35 cc syringe attached to an 18 g needle will generate 7-8 psi and is therefore a convenient method of irrigation in the Emergency Room.

CLOSURE

Wounds may be closed in a primary, secondary, or delayed fashion. Primary closure refers to closure immediately after the injury. Most wounds managed in the Emergency Room receive primary closure. It is preferable to close all wounds within 8-12 hours of injury. However, this may vary based on the presenting case. A clean, simple facial laceration on a young healthy patient can likely be closed at 24 hours without undue risk of infection, conversely, a contaminated crush injury to the foot of an elderly patient with diabetes may require a different clinical approach.

Secondary closure involves leaving the wound open and allowing it to close over time. This option is reserved for wounds that present late, or are otherwise at increased risk of infection. Delayed closure refers to leaving the wound open for 4-5 days, then closing if there are no signs of infection.

There are 4 main materials used for wound closure. They are: sutures, staples, tapes, and skin adhesives.

Suturing is the most common wound closure method employed in the Emergency Department. Sutures allow for precise approximation of wound edges and result in the strongest closure. Sutures are classified as nonabsorbable or absorbable. Nonabsorbable sutures are used on the skin and must be removed. Common nonabsorbable sutures used in the Emergency Room are nylon (Ethilon) or polypropylene (Prolene, Surgipro). Absorbable sutures are used on deeper structures and are left in place to dissolve. The most common absorbable sutures used in the Emergency Department are chromic gut and Vicryl. The body absorbs them in weeks to months, respectively. In terms of suture size, the choice depends on required strength and desired cosmetic result. Typically facial wounds are closed with 5-0 or 6-0; hands 4-0 and 5-0, feet, and other limb wounds with 4-0; trunk wounds or wounds under high tension (joints, gaping) with 3-0 or 4-0.

Suturing techniques include simple interrupted percutaneous, running or continuous percutaneous, deep dermal (buried), continuous subcuticular,

horizontal mattress, and vertical mattress. A detailed description of these techniques is beyond the scope of this manual. The most widespread suturing methods used in the Emergency Room are simple interrupted percutaneous and deep dermal.

The timing of suture removal is influenced by the body part involved, risk of dehiscence and the desire to optimize cosmesis. The table below summarizes typical suture size and timelines for removal.

Location	Suture Size	Removal (days)
Face	5-0 or 6-0	5
Scalp	4-0	7-10
Limbs	4-0	10-14
Trunk	4-0 or 3-0	7-14
Joints	4-0 or 3-0	14

Other methods of closure:

Staples are rapid to apply and have a low rate of infection. However, they allow for a less accurate closure and can be uncomfortable during healing. They are best suited for scalp lacerations.

Tape is fast and painless to apply. It is useful for small, linear lacerations under low tension.

Tissue adhesive (cyanoacrylate) has strength similar to 5-0 sutures and is therefore typically used in low tension wounds. Cosmetic results and infection rates are similar to sutures. It is particularly useful in pediatric patients to avoid the discomfort of suturing.

ANTIBIOTICS

Antibiotics are not routinely prescribed for wounds in the Emergency Department. However, in the setting of grossly contaminated wounds, immunocompromised hosts, severe crush injuries, open fractures, joint involvement, through-and-through intraoral lacerations, and mammalian bites antibiotics are likely to be used. Prophylaxis for mammalian bites is dependant on site of injury and likelihood of infection.

TETANUS IMMUNIZATION

Tetanus is a clinical condition caused by the anaerobic bacterium *Clostridium tetani*. *C. tetani* resides in soil and other anaerobic environments. It has an incubation period of 7-21 days and typically manifests with muscle spasm. Wounds characteristics that increase the risk of tetanus include: delayed presentation, deep wound, gross contamination, stellate pattern, ischemia, infection, and crush or puncture injuries.

Most patients who contract tetanus are over age 50 and have not been immunized. Risk factors for lack of immunization include age over 70, those born in a developing country, and lack of high school education. Tetanus vaccination can be safely given days after a wound is sustained. However, patients at risk of tetanus with no prior immunization require immediate passive immunization with tetanus immune globulin (TIG). The table below provides a simplified approach to the indications for vaccination and passive immunization.

Immun. Status	Vaccine (Td)	Passive Immun (TIG)
Complete (<10 yrs since last Td)	NO	NO
Complete <td>YES</td> <td>NO</td>	YES	NO
Incomplete <td>YES</td> <td>YES</td>	YES	YES

DRESSINGS

Dressings serve to protect the wound, minimize infection, and provide the best possible environment for healing. A key biological process in wound repair is reepithelialization of the skin defect. This process is impeded by a thick, dry scab and enhanced with proper oxygenation. In addition, too much fluid at the wound site will lead to skin maceration that also impedes healing. Therefore, a dressing should keep the wound moist, well oxygenated, and absorb excess fluid. This can be achieved with a film-like transparent dressing, or absorptive foam dressing if more secretions are expected. Alternatively, the combination of an antibiotic ointment or non-adhesive gauze covered by cotton gauze is a perfectly acceptable method to keep the wound moist, clean, and well oxygenated.

Two final notes regarding dressings: Wounds involving joints should be splinted for comfort and to promote healing. Lastly, drains are sometimes employed in the Emergency Room in wounds that are expected to have a large amount of continued purulent discharge. Although drains come in many varieties, the most frequently used is simple packing with ribbon gauze.

DISCHARGE INSTRUCTIONS

Patients should be given clear discharge instructions. Ideally a handout for later review accompanies verbal instructions.

The central issues surrounding instructions to patients are: cleansing, timing of suture removal, signs of infection, and expected cosmetic result.

Dressings should be left undisturbed for 24-48 hours after wound repair. Subsequently they can be removed and the wound can be gently cleansed with soap and water or a 50/50 blend of hydrogen peroxide and water. Antibiotic ointment and a dressing can then be applied daily.

The timing of suture removal depends on the location of the injury as outlined above.

Patients should be informed to seek medical attention if signs of infection appear, namely redness, swelling, increasing pain, or pus.

Finally, patients should be informed that unfortunately all wounds result in scars. The scar will typically fade over 6 to 12 months, during which time sun exposure should be avoided.

CLINICAL PEARL

A scalp wound that is not contaminated or bleeding excessively can be closed by bringing together the hair on both sides of the wound and making a single twist; no actual knot is required. The twist is then secured with tissue glue. This is called 'The Hair Apposition Technique'.

SUMMARY:

- The need to care for wounds is commonplace in the Emergency Department. The goal is to restore function, minimize infection, and optimize cosmesis.
- In general, all wounds should be closed within 8-12 hours of injury.
- Always examine the patient in the position of the extremity when the injury is sustained, especially when approaching tendon injuries.
- In healthy patients, clean as opposed to full aseptic technique has not been shown to increase the risk of infection, specifically when choosing gloves.
- The discomfort of injecting Lidocaine can be minimized by injecting slowly with a small (27 g) needle, buffering the solution with sodium bicarbonate and warming the solution prior to injection
- Antibiotics are required only in specific situations.
- Review all dressing care with your patient and caution them that wounds result in scars, however many will fade over 6-12 months.

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49: COMMUNICATION

Howard Ovens

OBJECTIVES

1. To understand the various barriers to effective communication in the Emergency Department
2. To know how to establish rapport with your patients
3. To organize the patient interview so as to maximize effective communication
4. To learn effective communication strategies with professionals in the Emergency Department
5. To be prepared to manage difficult patients

INTRODUCTION

It has been stated that “Effective communication between doctor and patient is a central clinical function. Good effective communication can improve patient and physician satisfaction and has been shown to improve health outcomes. Poor communication leads to complaints and is a major factor in litigation. Despite the documented importance of communicating well, serious communication problems are common in clinical practice.”

BARRIERS TO EFFECTIVE COMMUNICATION IN THE EMERGENCY DEPARTMENT

There are many reasons why the ED is a particularly difficult environment for a physician to establish effective communication with patients. The patient and their family usually arrive in the ED anxious (often with good reason), wait for what seems like a long time, are surrounded by noise, chaos, and often some unsettling scenes. They do not know who their physician will be, or when they will be seen. Privacy and confidentiality may be difficult to preserve during the interview and examination. There may be distracting noises, the physician is often interrupted, and may be obviously rushed. The patient may be in pain, or their thought processes affected by illness, drugs or alcohol. Patients and their families are unaware of other patients, who also require the attention of the nursing and medical staff, and of the necessity for establishing priorities.

You may encounter many other barriers in attempting to develop clear and comfortable communication with patients in Emergency Departments. Some patients

will have thoughts or fears about their illness which are widely discordant from your own. Reassurance to anxious patients will only be effective once rapport has been established and an adequate assessment has been performed. These are needed to convince the patient you are both competent and concerned with their welfare. In some cases, there may be language barriers or divergent cultural backgrounds and beliefs. Professional interpreters are rarely available and often objective interpreters (e.g. volunteers or other hospital staff) cannot be found leaving us dependent on family members. Use of family members may compromise patient confidentiality and can result in incomplete or inaccurate histories. It may also place relatives in awkward situations (such as ‘breaking bad news’ to a loved one).

All of us, no matter how open-minded, will form some clinical judgments that have been affected by the patient’s age, gender, race, dress, hygiene, or appearance in ways we may be reluctant to acknowledge. For example, would you approach a 22 year old scruffy man covered in tattoos with a headache with the same thoughts as a 55 year old lawyer in a business suit? Which one is more likely to be stoically enduring the pain of a subarachnoid hemorrhage and which is malingering to supply a prescription drug habit?

Sometimes patients will exhibit denial. A common example is the person who assures us they only have heartburn. Their trip to the ED at 3:00 a.m. suggests they realize it could be more serious. Other times physicians employ denial. We may be reluctant to consider difficult, time consuming, or painful possibilities in our patients. These may include child abuse, self-inflicted injury or illness, or a disease and/or problems we feel unprepared to address.

After discharge, even the most intelligent or attentive patients commonly forget details of our advice. If they have questions or further problems, they may be uncertain where to turn, or how to contact the Emergency Physician. All of these challenges must be addressed in your approach to the patient in the ED.

ESTABLISHING RAPPORT

Rapport is a harmonious relationship; a state of mutual understanding. Rapport can be established

surprisingly quickly, or made much more difficult, based on the patient's first impressions. Therefore, preparing to establish rapport must begin even before you arrive in the ED. A shift in the ED can be an emotional and physical marathon. You should arrive rested, alert and nourished. Personal business should be tended to before your shift to allow you to concentrate on the tasks at hand. You may encounter patients of many different socio-economic and ethnic backgrounds and of varying ages. In general, it is recommended that you wear comfortable but conservative professional attire; covering with a clean lab coat remains a good approach. Clothes make a better impression than greens; but, if chosen, greens should be clean and fit well. Whatever you choose, it should be possible to distinguish you as a physician from amongst the other team members.

Before walking into the patient's room, review their chart including nurse's notes and personal information. When you enter the room find a comfortable position, preferably at, or close to the patient's eye level. Make eye contact with the patient and introduce yourself, your position, and your role. As a clerk you might say:

"Hello, I'm Emma Pathy, a student doctor working here in the Emergency Department. I would like to talk with you and examine you, and then I'll discuss your problems with my supervisor."

Try to pronounce the patient's name (confirm that you are correct) and acknowledge the information already on the chart. If appropriate apologize for any delay, or thank the patient for their patience;

"Ms. Vinnyboonbotts - did I say your name correctly?
- I've read what you told the nurse about your abdominal pain. I see you've been waiting almost 3 hours, I'm sorry for the delay." Some people are more comfortable saying, "I see you've been waiting; thank you for your patience." The message is not that you are taking responsibility for the delay, but to acknowledge their experience.

Thus, before the first question you have communicated to the patient that you are a professional, that they have your full attention, and that you care about their experiences. You are off to a great start.

Sometimes in the ED we start treatment or investigations before, or during history taking. Unless the patient is comatose (do not assume they cannot hear you) communication remains important - even while you address the ABC's. For example, you go to see a patient with an exacerbation of asthma in severe distress. The nurse has an O₂ mask already on the

patient and is starting an intravenous. You arrive, briefly listen to the patient's chest, and order a bronchodilating inhalation. While doing this you can introduce yourself, tell the patient you see they are in distress, and explain your initial plans while telling them you will talk more when their breathing improves. Your early treatment efforts can be seen as a method of establishing rapport, demonstrating to the patient that you can recognize their distress and meet their immediate needs.

THE PATIENT INTERVIEW

'Hidden agendas' are a term applied to patient concerns not revealed until late in the visit. This may also result from overly directed questioning by the physician, rather than resistance or denial by the patient. Emergency Department interactions are ripe for the occurrence of unexpressed concerns. For example, time pressures may lead the physician to direct the interview excessively. Information given to the Triage Nurse or ambulance attendants may mislead the physician as to the nature of the chief complaint. Although one should acknowledge the information already on the chart, even for the most deceptively straightforward appearing problems, you should begin with an open-ended question. Allow the patient to complete their reply (the 'opening statement') before interrupting or asking for clarification. If the patient pauses, you should nod and frequently encourage them with statements such as:

"Uh-huh", "I see" or "Go on".

This indicates you are still listening. For example, you might begin by saying:

"Now Ms. Vinnyboonbotts, I've had a chance to read your chart and review what you told the nurse about your chest pain; but, would you mind telling me in your own words why you came to hospital today?"

Once the patient has completed their opening statement you should ask them if they have additional information to impart ("Is there anything else bothering you?"). Then, if appropriate, summarize what you think you heard ("So this pain is sharp, gets worse when you breathe and started suddenly yesterday - is that right?").

Next comes the directed part of the interview. The opening statement and information on the chart, together with the patient's overall appearance, will have suggested a differential diagnosis to you. You now ask questions to add information, which will

allow you to refine your hypothesis (essentially completing the history of present illness and determining relevant points from past medical history and systems review). Questions should be worded in simple English without use of medical terminology.

Patients who come to the Emergency Department will have a high incidence of psychosocial problems. These may include drug and alcohol abuse, anxiety or depression, domestic violence, and even legal problems. In most cases, information regarding these issues should be actively sought. The questions should be in simple language, direct and delivered in a non-judgmental way. If given in the same tone of voice and as part of a series of questions, most patients will accept them well and reply honestly. If the patient expresses surprise, embarrassment, or confusion over a question, usually all that is necessary is a very general, impersonal explanation of your purpose:

"Unhealthy use of alcohol is such a common problem that we ask everyone about it."

"Some women who say they fell down stairs were actually pushed, and I want to be sure you're not in any danger".

When you feel you have all the information you need, you should conclude with another opportunity for the patient to speak ("Is there anything else you wanted to add before I examine you?"). Then explain what you would like to examine and what you expect from the patient ("To sort out what's causing your pain I'd like to examine your chest, heart, and abdomen. Can you please sit on the stretcher?"). If they seem uncomfortable or hesitant in any way, then stop. Give a more complete explanation of what you plan to do and why. Ask for explicit consent to continue (e.g. "I'd like to listen to the front and back of your chest with my stethoscope, is that okay? You look uncomfortable.").

In some cases you will be running out of questions, yet you will feel you do not have a clear understanding of why the patient came to the ED or what they expect of you. In order to exclude life-threatening illness, much of the interview will follow a "disease-centred model". When you feel stuck or confused, a switch to "patient-centred" questioning may be helpful. You might ask the patient:

"What do you think is wrong?"
 "What aspect of your chest pain is troubling you?"
 "How do you think I can help you?"

After concluding the history and physical examination, you should briefly tell the patient what you've found, what your plans are, and what you expect of them. For example:

"I don't hear anything wrong listening to your chest - but you may have a broken rib. I'm going to check with my supervisor and arrange a chest x-ray. The technician will come here to get you - please wait here and I'll see you after the x-ray."

In many cases information elicited from the patient is inadequate. This may be due to patient confusion, intoxication, dishonesty, language barriers, or ignorance about their medical history. Collateral information is routinely crucial to emergency care. Paramedics, police, nurses, patient's family, the medical records, witnesses to an incident or event, staff in another institution, other hospital's previous records, and other physicians may all provide useful information in many situations. Inconsistent reports and failed attempts at procuring collateral information should be documented.

WRAPPING UP

When presenting the patient with your assessment and recommendations make sure your language is jargon-free, blame-free, and that you address the patient's prime concerns. Give patients an explicit opportunity to ask questions and react to your advice. Some patients will disagree with your plans or request a different treatment. Others will defer to you on every decision, big or small. If the patient disagrees, try to elicit what they want and why they want it. At this point you may be willing to negotiate a middle ground. Sometimes this is not possible. If so, you will have to explain your rationale for refusing. In these cases it may be helpful to explain to the patient where they might get another opinion and document carefully what happened.

All too often we are called upon to break bad news to patients or families. As a general rule ensure private, uninterrupted time. Go to a quiet room or pull the drape, ask not to be interrupted except in an emergency. Forecast what you are about to say so that the patient can prepare themselves. When done well, the person should be able to guess what you are about to say:

"Hello, I'm Dr. Doe. I was one of the people caring for your mother. She came here because of chest pain and we found she was having a heart attack. Despite our treatments her heart stopped beating. We called the special team we have for these situations, and

tried everything we could to get her heart to start again, but her heart was badly damaged", (pause) "I'm sorry", (pause) "Your mother has died".

If the meaning of the news is not yet certain (e.g. you have found a nodule in a smoker's lung), you should be vague, but honest. Follow the patient's lead in determining how much this patient wants to know at this time. If you tell a patient that you have found "something" on their chest x-ray, and you will send them to a specialist to determine what it is, some people will not ask any questions. Others will ask what the "something" is. You should gradually get more specific in response to the patient's cues:

"There are many things that could look like this shadow on your lung."

"Like what?"

"Well, some are pretty benign, others are more serious."

"Like what?"

"Well, it could be a cyst, a benign tumor, an infection, or it could be a cancer, we don't know yet."

"What do you think?"

"We don't know what you have, but in your specific circumstances, the chance that it is cancer is very real. However, it might not be. If it is cancer, the doctor we're sending you to will be able to give you the best possible advice and care."

Always leave the patient with hope and reassure them that they will not be facing their problems alone.

Regardless of how trivial the patient's complaint, or how busy you are, every patient should leave the ED with an idea of their diagnosis, what to expect, what to do if things do not go as planned, and who to follow-up with.

PROFESSIONAL COMMUNICATION

The emergency medical record is a crucial document. After the patient's visit it may provide critical information to other caregivers. Information contained in it may be used for legal purposes unrelated to health care (compensation and insurance claims, criminal proceedings) or to assist in the adjudication of complaints, inquests, or lawsuits. It may also be used to guide physician or hospital reimbursement or for research or quality assurance activities. Every one of us must develop the ability to efficiently record in a legible manner, the relevant information regarding history, examination, investigation results, treatments and responses, discharge diagnosis and instructions, along with any other information necessary to satisfy the myriad

functions of the emergency record. Also, never forget that patients are entitled to see their records. Keep all notations professional, and avoid irrelevant or pejorative comments. For example, if an intoxicated patient is difficult, you might record, "patient uncooperative, profane, and disruptive" rather than "patient obnoxious" or "unpleasant". The first phrase is based in fact, whereas the second records judgments.

In general, junior housestaff begin by being too inclusive in the recording of their history and physical, but deficient in the recording of their thoughts and discharge advice. As you become confident in a problem oriented approach, your choice of relevant positive and negative findings to record will reflect your consideration of the appropriate differential diagnosis. Similarly, when presenting a case to your supervising physician, it is beneficial if you begin with a brief statement of your provisional assessment, followed by supportive positive and negative findings, discordant findings, and relevant past history. This forces you to think about the case, commit to an opinion, and support it. It also allows us to evaluate your evidence. If the case has you stumped, begin by stating this as well. For example:

"This 35 year old male has a good story for appendicitis. His pain started 2 days ago and settled in his RLQ and is associated with chills and nausea...", then go on to relate his past history, physical exam, test results and your plan.

"I have a young woman with multiple complaints that really have me stumped. She just returned from holiday and has vague GI complaints, but also feels weak, light-headed, has some paresthesias, and this is really odd - she describes hot things feeling cold and cold things feeling hot." (This is a pathognomonic description of ciguatera toxin poisoning). Given your uncertainty you could go on to describe a detailed systems review and past history, and a fairly complete physical examination.

Just as important as clear and effective documentation is good communication with other members of the team. Courtesy, clarity, honesty (and on busy days brevity) will serve you well when dealing with clerical staff, nurses, orderlies, and other team members. Demonstrate respect for each person and their role. Give orders or requests in a clear and specific manner to avoid misunderstanding. If you are unsure of something, say so openly. Never bluff or guess. When requesting a consultation, articulate the nature of your concern and the urgency of the problem, along with a summary of tests and

treatments performed so far. Contact family physicians if you have information that may benefit them or their patients. Avoid deprecating remarks regarding another physician's care. More often than not these remarks are based on insufficient information and rarely serve a useful purpose. Finally, the Emergency Department will stress and challenge you in many ways. There are effective techniques for dealing with abusive patients, rude colleagues, or tired consultants without loss of professional demeanor.

DIFFICULT PATIENTS

Difficult patients are usually defined as patients who provoke negative emotional reactions (hatred, fear, frustration, anger) from physicians. Common patient types in this category include chronic alcoholics, chronically suicidal patients, somatizers (hypochondriacs), and malingerers. These patients are found in disproportionate numbers in EDs and cause stress far out of proportion to their numbers among ED staff. The patients are drawn to the ED because of easy access (open 24 hours, nobody is turned away) or an inappropriate sense of urgency. These patients are often handled poorly.

Young physicians may be upset at a display of negative emotions toward a patient by their teachers, or may be uncomfortable with their own negative feelings (believing a "good" doctor accepts and "loves" all patients). Success in these challenging situations requires an ability to acknowledge and accept negative emotions as important clinical

information, while formulating a professional treatment strategy that is realistic to the environment of an ED and the prognosis of the patient.

SUMMARY

- The Emergency Department presents significant barriers to effective communication.
- Most problems can be overcome with simple strategies rigorously applied.
- The challenge is to consistently utilize these strategies under difficult circumstances.

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50: END OF LIFE ISSUES

Howard Ovens, Debbie Selby

OBJECTIVES

1. To understand the common challenges in end of life issues in the emergency department
2. To effectively identify and manage pain, dyspnea, nausea/vomiting, constipation, and delirium in the palliative patient
3. To understand advanced directives and substitute decision makers
4. To understand the ethical issues of resuscitation
5. To know how best to ‘break bad news’

INTRODUCTION

End of life (EOL) situations in the emergency department (ED) are challenging and varied. They range from unexpected deaths to the final stage of a chronic illness. Challenges that arise include:

- lack of a prior relationship with the patient or their family
- limited information on the patient’s baseline health
- little information about the patient’s wishes
- time constraints within a busy department and the need to make rapid decisions despite uncertainty

In order to meet these challenges competently, a range of skills is required. Significant communication skills are needed to clarify the wishes of patients and families tactfully and efficiently. There are also specific skills required to manage end of life symptoms and provide supportive care to the dying.

INITIAL ASSESSMENT

When faced with uncertainty or incomplete information in the ED, we must almost always err on the side of intervention. Decisions can always be made later to withdraw life support, which is usually less difficult and controversial than is widely believed.

Following initial assessment and/or stabilization of the patient, there is often a brief interlude during which we can gather more information. Optimally, we speak directly to the patient or their immediate next of kin to rapidly clarify the patient’s or family’s understanding of the underlying illness, the event leading to emergency transport, and the existing expectations around emergency response and

resuscitation. Ask if anyone has ever discussed these issues and whether there is an existing directive. Remind family that the primary intent is to clarify what the patient has previously expressed or how the patient might direct us. It may also be helpful to gather third party information from old records or from primary physicians.

Finally, it is important to identify the goals of the emergency visit. Remember that initiating treatment may give the family and the patient time to adjust to the changed circumstances before a definitive discussion is held; although, raising the issue early may start them on the path of considering alternative approaches. The results of any discussion should be recorded in the patient record.

ACUTE SYMPTOM MANAGEMENT FOR PALLIATIVE CARE PATIENTS

Pain

Presentations to the ED with pain in palliative patients will usually be an acute change as opposed to chronic unrelieved pain. Most acute painful crises occur in patients with metastatic cancer (as opposed to other end-stage illnesses) and can be linked to a specific event. Possibilities to consider include; new pathologic fractures (especially vertebral body), bleeding into tumors, and pressure on nerve roots causing neuropathic pain. Management involves rapid pain relief with intravenous or subcutaneous narcotics while eliciting a diagnosis. Note that there is no justification to leave a patient in pain while doing an assessment or diagnostic tests.

The dose of narcotic used will depend on the patient’s current background narcotic use. For example, for a person on 360 mg of long-acting morphine a day, giving 2-5mg of morphine IV is unlikely to help their pain. A good general guide for breakthrough doses is to add up the total (24 hour) narcotic oral dose and divide by 10 to get the conservative oral breakthrough dose. Although the conversion of oral to IV or SC is usually 2:1, in an acute crisis more is needed, so start with the one-tenth dose you just calculated but give it IV or SC. For the person described above on 360 mg of morphine a day, the first dose should be 36 mg of morphine SC. For severe pain, especially if they have suffered with the pain for a while, an anxiolytic can help significantly. For example, the short-acting benzodiazepine, midazolam can be given 2-4 mg IV.

These patients are usually on much higher doses of narcotics than we are used to, are tolerant of high doses, and are in acute pain; therefore, they will not stop breathing with high doses and need aggressive measures to get their pain under control. After they have some pain relief, start looking for the cause to allow more definitive treatment.

For those presenting to the ED with more chronic pain, the general pathway for management starts with weak opioids (e.g. codeine), moving rapidly to stronger opioids (morphine, oxycodone, hydromorphone in order of potency). Start such patients on a regular (q4h) short acting narcotic with 50% of the regular dose as a q1h prn dose. The most critical step in these patients is referring them to a pain or palliative care clinic to ensure ongoing management.

Opioid Conversion/Equivalency Table	
Codeine	100 mg
Morphine	10 mg
Oxycodone	5 mg
Hydromorphone	2 mg

CLINICAL PEARL

In the management of pain for palliative patients, a good general guide for breakthrough doses is to add up the total (24 hour) narcotic oral dose and divide by 10 to get the conservative oral breakthrough dose.

Dyspnea

Severe dyspnea is terrifying for both the patient and their family and thus requires rapid control. For some, this episode may mark ‘the beginning of the end’; whereas for others there may be a readily reversible cause. Managing the dyspnea is not difficult. The difficult part is quickly deciding if it is reversible, how aggressive the patient wants to be treated, or if the only option is sedation and comfort measures.

Diagnostic possibilities to consider include; upper airway obstruction (presents with stridor), bronchial obstruction with lobar collapse (from tumor compression in cancers or mucous plugs in neuromuscular diseases), large pleural effusions, pulmonary emboli, pneumonia, or exacerbations of CHF/COPD. Regardless of the cause, oxygen, narcotics, and anxiolytics (e.g. midazolam) will help

the sensation of dyspnea, though clearly they will not fix the underlying problem. Again, starting doses will depend on the background narcotic use, as described above. For opioid naïve patients, morphine 2.5mg sc Q4h and Q1h prn is a reasonable starting dose. In most situations without a readily reversible cause, calling early for palliative care or primary physician (oncologist, neurologist, respirologist) help is generally necessary. In the meantime, establish goals of care and maximize comfort within those goals by using narcotics and anxiolytics titrated every 30 minutes as necessary.

Nausea/Vomiting

Severe nausea and vomiting presenting to the ED in the palliative setting will be related either to recent treatment (chemotherapy, radiation therapy) or to abdominal complications of malignancies such as bowel obstructions. Less commonly, the nausea and vomiting will be caused by constipation, massive ascites, or metabolic factors (uremia, high bilirubin, hyponatremia, or hypercalcemia). History, physical exam, and basic investigations will help sort this out. In the meantime, treatment decisions will depend on the most likely cause. Any form of gastric/bowel distension activates dopamine receptors causing nausea and vomiting; therefore, treatment must include dopamine antagonists such as prochlorperazine (10mg IV q6H prn) or haloperidol (0.5-2 mg IV/SC q4H prn). Dimenhydrinate (Gravol) is unlikely to make much impact in this setting as it works on a different mechanism. Chemotherapy directly activates the chemoreceptor trigger zone in the hypothalamus, which responds best to a 5-HT3 blocker such as ondansetron (8mg IV q8H prn). Similarly, radiation of the gut releases 5-HT from the intestinal wall to cause nausea and again will likely respond better to a 5-HT3 blocker than to the anti-histamine effects of dimenhydrinate.

Constipation

Many patients with metastatic cancer will be on opioids that cause constipation by decreasing gut motility. Hopefully, most will be on a bowel regimen including prokinetics such as senna. However, it is not unusual for some to become severely constipated and present to the ED. Presenting complaints may include nausea and vomiting, anorexia, bloating, abdominal pain and cramping, mimicking a bowel obstruction. Keys to diagnosis are the history, a rectal exam (assessing the need for disimpaction), and a flat plate abdominal x-ray. Generally, treatment measures need to start from below with enemas to clear the hard stool (or disimpaction if this is unsuccessful), then working from above with any of lactulose, magnesium citrate, or even polyethylene glycol solution (Golytely). This can be a truly serious

problem in people who require ongoing narcotics, and the presence of any intra-abdominal disease (e.g. carcinomatosis) can make it extremely hard to manage. These are not people who should just be sent home with a prescription for lactulose unless they have been diagnosed early in their course of illness and have minimal intra-abdominal disease.

Delirium

Delirium is very common in palliative patients (at least 85% experience delirium in the final days or weeks of life) and various common treatments (narcotics, anticholinergics, steroids, sedatives) can precipitate or aggravate delirium. Behaviour changes, hallucinations, and other symptoms are very disturbing for family members and most will therefore present to the ED. Reversible causes to consider include; infection (common), metabolic abnormalities (calcium, sodium), medications, and urinary retention (which in turn can happen because of medications). Management is aimed at treating any reversible causes and controlling the symptoms with the use of anti-psychotic medications.

Haloperidol (start with 1 mg IV or SC) combined with lorazepam works well; alternatively methotriptazine (Nozinan) starting at 6.25-12.5 mg SC provides both sedation and anti-psychotic features. Alternatively, the atypical anti-psychotics such as risperidol or olanzapine (e.g. zydis 5mg SL) can be used. If the underlying cause cannot be treated, most will need admission or urgent homecare/palliative management.

Actively Dying

Hopefully, very few patients will arrive in the ED in their final hours of life from an end-stage illness; clearly this is not the best environment for final hours of care. Principles of care focus on comfort for both the patient and their family. Pain, dyspnea, and delirium are managed as described above, understanding that the law clearly supports the use of narcotics and sedatives if given with the intent of relief of suffering, even if they may hasten death. As such, this is not the time to be conservative and leave a family with images of a loved one having died in discomfort. One final common symptom that may need management is upper airway secretions-'the death rattle'. This is generally more upsetting to family than to the patient who is most often obtunded by this point. Secretions can be managed with scopolamine 0.6 mg SC q4h prn or glycopyrrolate 0.6 mg sc q4h prn. Suctioning should be avoided. Other important considerations include; a quiet and preferably private room, use of a 'family room' for grieving relatives, an offer of chaplaincy, and very frequent nursing and physician attendance and reassurance.

ADVANCE DIRECTIVES AND SUBSTITUTE DECISION MAKERS

Patients, especially those in long term care facilities and/or those with a terminal illness, now commonly have an advance directive and/or a healthcare power of attorney. The legislation in each jurisdiction governing healthcare consent, substitute decision-making, and advance directives will vary somewhat. In general, the advance directive must have been composed when the patient was competent (unless signed by the legally empowered substitute decision maker) and have instructions that are reasonable and relevant to the current circumstances.

If the circumstances are unusual, reversible, or were likely unforeseen, it is safer to confirm the patient's wishes if possible. Rarely, if there are circumstances which seem inconsistent or not in the patient's best interests it is prudent to err on the side of full intervention while notifying authorities who can validate the information being presented.

ETHICAL ISSUES IN RESUSCITATION

Difficult situations may arise if we attempt to apply the concepts of 'brain death' and/or 'futility' within the context of an ED. Time constraints and lack of definitive information often make it inappropriate to rely on either of these to withhold resuscitation. Unless there is appropriate direction from the patient or family, or a clearly untreatable condition, the imperative is to fully attempt resuscitation. One helpful strategy to apply in this situation is the sense of a continuum of resuscitation, as opposed to an 'all-or-none' Do Not Resuscitate (DNR). For instance, the elderly nursing home patient without an advance directive or power of attorney who presents with pneumonia or sepsis will receive appropriate supportive interventions that may include intubation and/or vasopressors. If, despite such appropriate support the patient proceeds to a pulseless state not readily corrected through cardioversion, the resuscitation would certainly be appropriately terminated.

Alternatively, emergency intervention may lead to a temporary state of stability with an overall poor prognosis. Such an interlude may give the caregivers time to communicate further with the family and/or seek additional consultations on prognosis. This may lead to a more supportive approach or to actual withdrawal of the supports which were originally placed. Some have stated that it is unethical to withdraw supports once initiated, but there is actually no basis for this claim, and all resuscitative decisions

made can be ‘undone’ if family or power of attorney so direct.

BREAKING BAD NEWS

Notification of death to a family is one particular instance of ‘breaking bad news’. Interviews with families suggest that this can be a very critical time, and managing these issues well is a vital skill for emergency providers.

Key Steps to Follow

- Try to ensure 5-10 minutes of uninterrupted time in a quiet, private place.
- Sit down.
- Introduce yourself, your role, and anyone else in the room (it is often helpful to have a chaplain or social worker present who can remain with the family when you leave).
- Find out what the family knows about the patient’s situation and what they have been told so far.
- Explain in clear, simple language what has transpired and that their loved one has died. Avoid jargon and medical talk, but do use the word ‘dead’ or death’.
- Leave plenty of silences both to allow family to absorb what you are saying and to allow questions.
- Acknowledge their emotions and respond empathically to their questions; reassure them as appropriate that unnecessary suffering was avoided.

Once the family has had opportunity to ask questions they may need direction as to what to do next.

Issues to Address

- Viewing the deceased - prepare family who wish to view the deceased as to what they will see (tubes, lines, post-mortem changes), place the body in a quiet room covered by a sheet, and have a staff person (nurse, chaplain or social worker) stay unobtrusively in the room with the family.
- Gather the patient’s personal belongings and offer them to the family.
- Ask them who else should be contacted among their family and friends. Offer assistance including access to a private telephone.
- Ask them to notify the funeral home of their choice.
- Organ donation should be addressed. A full discussion on the issues is outside the scope of this chapter but information can be found at <http://www.giftolife.on.ca/>.

<http://www.giftolife.on.ca/>. It is important that the family has an opportunity to donate any suitable organs or tissues and that any information given to them is accurate. If uncertain, obtain assistance or further information.

- If the coroner is to be notified, explain that to the family. Each jurisdiction will have slightly different indications and processes for reporting deaths. In general, any death due to assault, accident, or involving possible medical malpractice should be reported. Any sudden and/or unexplained death is best reported. If in doubt, contact the coroner’s office for guidance.
- Depending on the policies in your hospital, offer or discuss a medical autopsy to determine cause of death (if it is a coroner’s case the coroner will decide on whether they want a forensic autopsy and will arrange it.)

Finally, remember that death in the ED can and will affect you and other members of the ED team. Some deaths will have more impact than others. Death of children will often cause more emotional trauma for care-givers, while any case that reminds someone of themselves, their family, or of some tragedy in their own life will have more impact. This will be magnified if they have a sense of responsibility in the death due to less than optimal care. It is critical to identify these feelings and talk about them to a department chief, colleague, or other support person.

SUMMARY

- Following initial assessment and/or stabilization of the patient there is often a brief interlude during which information should be gathered from the patient’s family and chart in order to clarify the family’s understanding of the illness and process, advanced directives, and expectations.
- Palliative patients are usually on much higher doses of narcotics than we are used to, are tolerant of high doses, and are in acute pain; therefore aggressive measures are needed to get their pain under control
- Dyspnea in the palliative patient can be managed with oxygen, narcotics, and anxiolytics (e.g. midazolam) while an underlying cause is being established
- Anti-nausea medications should be chosen based on their mechanism of action matching the mechanism of illness
- Delirium is very common in palliative patients and can be treated with anti-psychotic medications
- In order for an advanced directive to be valid it must have been composed when the patient was

- competent and must have instructions that are reasonable and relevant to the current circumstances
- Always err on the side of intervention when faced with a patient without clear advanced directives
 - Unless there is appropriate direction from the patient or family, or a clearly untreatable condition, the imperative is to fully attempt resuscitation
 - When ‘breaking bad news’ to families, sit down and introduce yourself and anyone else in the room, find out what the family knows about the situation, explain things in clear simple direct language, acknowledge their emotions and respond empathically to their questions
 - Be prepared to counsel the family regarding viewing the deceased, funeral home arrangements, organ donation, autopsy and coroner involvement

- It is critical to identify your own emotions after the death of your patient and address them appropriately

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51: DOMESTIC/INTIMATE PARTNER VIOLENCE

Sheila Dunn, Jessica Potvin

OBJECTIVES

1. To review the role of the Emergency Department (ED) in partner violence
2. To identify victims of partner violence in the ED
3. To learn how to be supportive and assess danger when partner violence is disclosed
4. To review the documentation, safety planning and follow-up of victims of partner violence

INTRODUCTION

While there are many forms of domestic or intimate partner violence, this chapter will focus on the problems of partner assault. This extremely common problem can be very subtle in its presentation. All genders and sexualities are affected by partner violence however women are affected most and present with more severe injuries.

Emergency Departments are a frequent contact point for assaulted women within the health care system. Women who are experiencing Intimate Partner Violence are seen in Emergency Departments more frequently than patients with appendicitis, diverticulitis or joint dislocations; but, only 10% of these women are identified as assaulted. Of women presenting to an Emergency Department, 16-30% were injured by their partner.

FACTS ABOUT INTIMATE PARTNER ASSAULT

- annual prevalence amongst women living with a partner who assaults them is about 8%.
- pregnant and adolescent women are at particular risk
- 60% of female homicide victims are killed by their (ex)partner/spouse
- all cultural groups, all ages, and all economic classes are affected as are gay/lesbian relationships
- abuse is likely to be repeated once it has occurred

- women are in increased danger after a separation, divorce or change in the relationship
- separation increases the chances a woman will be murdered by her partner by a factor of four
- abuse is not caused because the abuser uses alcohol, although alcohol is often a co-factor.
- Intimate partner violence includes physical assault, sexual assault, assault against pets or property, verbal assault, stalking and threatening.

ROLE OF THE EMERGENCY DEPARTMENT

The role of the Emergency Department Staff is to:

- identify the problem
- assess, support and treat
- document
- plan safety
- ensure there is follow up

IDENTIFYING THE PROBLEM

Abuse may be identified by the patient, but more often is not disclosed. Learn how to ask any woman who presents with symptoms which may be related to abuse, about the possibility of abuse. Some Emergency Departments have universal screening of all clients registered to inquire if they are in a “safe situation”. Interview the patient alone, away from the partner, family, or friends. If the partner insists on being present, state that departmental protocol requires him out of the examining room for at least some of the time.

Suggestive Injuries

- bilateral multiple bruises or lacerations, may be in different stages of healing
- patterned injuries (show imprint of striking object)
- periorbital hematoma
- nasal fracture or fractured mandible

- injuries to the arms
- perforated tympanic membrane
- cigarette burns
- injuries to the abdomen during pregnancy, threatened abortion
- injuries to the breasts or genitals
- injuries out of keeping with the stated cause

If you find injuries of this nature ask “Has anyone hurt you?” or “In my experience, these types of injuries are often the result of abuse or assault. Has this happened to you?”

Somatic Symptoms

- often chronic and not well defined
- chest pain, palpitations, shortness of breath
- abdominal or pelvic pain
- dizziness, numbness
- fatigue

Psychosocial Symptoms/Presentations

- depression, anxiety
- substance abuse
- suicide attempts
- sleep disturbance

If somatic or psychological symptoms ask, “The kinds of symptoms you are having are sometimes associated with intimate partner violence. Do you ever experience abuse or violence in your home?” or “Can you tell me if anyone is hurting you or making you feel bad about yourself?”

Even if the patient does not disclose that violence is happening, they know for future that the Emergency Department is a safe place she can go to for help. Often those in intimate partner violence situations do not realize they are in a violent situation and often have to be asked several times before they disclose.

IF ABUSE IS DISCLOSED

In Ontario, there are several Sexual Assault and Domestic Violence Treatment Centres. These centres provide 24/7 coverage of an RN specially trained to assess intimate partner violence. In Toronto, these centres are located at Women’s College Hospital and Scarborough Grace Hospital. You can refer clients to the hospital or the RN can attend the hospital where the client is located. Services provided include:

- Risk assessment

- Safety planning
- Injury documentation and photography
- Facilitate police involvement
- Children’s Aid involvement if necessary
- Follow-up services,

Be Supportive

- tell the patient that you are glad she could tell you about it
- affirm that it is a crime
- be supportive and non-judgmental
- identify that abuse is a major health problem
- affirm that the perpetrator is responsible for the abuse - many survivors feel responsible or blame themselves

Assess Danger

- What happened and when?
- How often does it occur and when was the last episode?
- What is the pattern? Is it getting worse or more frequent?
- Are weapons involved?
- Are there threats, particularly death threats?
- Does the partner use alcohol or drugs? Alcohol and drug use have been proven to increase lethality in intimate partner violence and is used as a factor when conducting risk assessments.
- Are there children involved? Where are they? Are they in danger or have they been abused?
- Have the police or CAS been involved? If there are children under the age of 16 in the home that are witnesses to the abuse, it is mandatory in Ontario to report to the CAS.
- Do they have suicidal thoughts?

If there is immediate danger to the patient in the Department, notify Security and contact the police.

ASSESSMENT AND TREATMENT

- ABCs as always
- Identify and treat injuries as found
- Provide non judgmental support
- Ask about sexual assault
- Assess the risk to the patient as above.
- Help the victim to clarify their needs
- Review options available to the patient so they can make informed choices

- Develop a safety and follow-up plan and inform her of community resources
- Refer to a specialized Treatment Centre if the client wishes.

DOCUMENTATION

- Document your findings as if you are going to court. Detail the mechanism of injury if the woman can recall it, e.g. "states hit on left cheek by closed fist". Physical findings can then be related back to the mechanism of injury.
- Be detailed, and use body diagrams with clear descriptions of size, colour, shape, location of the injuries
- Ideally, if the patient is agreeable, a police photographer should record the injuries

PLANNING SAFETY

- Discuss with the patient their risk of being subjected to violence in the future. Violence often increases with intensity and frequency over time.
- Help develop safety strategies:
- Offer to contact the police. If the patient was brought in by police, there is Mandatory Reporting for Intimate Partner Violence and charges will be laid.
- Where will the patient stay after discharge? Offer to contact a shelter,. If going home,

• SUMMARY

DO'S	DON'TS
<p>ASK about abuse IDENTIFY patients who have experienced abuse ACKNOWLEDGE the seriousness of the problem EXPRESS belief in the patient THINK about immediate safety STRESS that no one deserves abuse; assault is a crime GIVE the patient a list of resources in your community</p>	<p>BLAME OR SHAME the patient MORALIZE IGNORE the disclosure of abuse MINIMIZE the effect of abuse ALIGN yourself with the abuser ASK "Why don't you leave?"</p>

Adapted from Hotch D, et al. Domestic violence intervention by emergency department staff. CAEP, 1995.

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52: SEXUAL ASSAULT

Sheila Dunn, Jessica Potvin

OBJECTIVES

1. To identify sexual assault victims in the Emergency Department (ED)
2. To review a general approach to the care of the sexual assault victim in the ED
3. To know which investigations and prophylactic interventions are recommended
4. To provide appropriate follow-up for the sexual assault victim

INTRODUCTION

Sexual assault (SA) is defined as ‘any form of sexual activity with another person without her/his consent’. It encompasses all unwanted sexual acts from fondling to forcible penetration. Sexual assault is a crime of violence in which the assailant uses the sexual act to overpower, control, degrade or humiliate the victim.

A Few Facts

- It is estimated 1 in 4 women and 1 in 10 men will be sexually assaulted in their lifetime.
- All age groups can be victim to sexual assault, but the most vulnerable group is 16-25 year old women.
- The assault is usually by someone the victim knows.
- Only ~10% report the attack to police and only 65% of victims presenting to Sexual Assault Treatment Centres (SATC) in Toronto elect to have forensic evidence collection.
- Physical injuries are usually minor; but, about 5% of victims will have major injuries - usually non-genital.
- It is the long term psychosocial problems of depression, post-traumatic stress disorder, substance abuse, suicide, and relationship problems that are frequently the most debilitating result of sexual assault.
- Supportive and compassionate care in the early phase can significantly assist the victim in recovery.

EMERGENCY DEPARTMENT MANAGEMENT

Identifying the Problem

Sexual assault engenders many emotional responses in the victim including fear, shame and embarrassment. It is important to approach the patient in a non-judgmental and non-biased way, as you may be the first person they disclose the assault to. Your response affects their choices in the important medical and forensic options that are available to them. Drug facilitated sexual assault (DFSA) is a common occurrence, with alcohol being the substance most frequently consumed. Rarely, other substances such as Gamma-Hydroxybutyrate (GHB) and other over-the counter drugs such as Diphenhydramine are administered by perpetrators to coerce and disable victims. DFSA also results in a memory loss of the sexual assault from complete memory loss to remembering only parts of the assault.

Not all victims will present with sexual assault as the stated problem. Be aware and ask the patient about sexual assault.

Other presentations include:

- Requesting the “Morning After Pill”
- Requesting a sexually transmitted Infection (STI) check
- Presentation after an altered level of awareness state with suspicion SA may have occurred
- Suffering multiple injuries from an obvious physical assault

General Approach to Care

- Prioritize emergent medical conditions. Remember the ABCs of trauma care and initiate therapy as appropriate. If there is minimal or no trauma, try to disturb clothing and physical findings as little as possible so that evidence is preserved.
- Reassure the victim that they are safe and provide a private quiet place to wait. If possible, have a nurse or other support person stay with the victim.
- Believe the victim. Your job is not to prove that sexual assault has occurred, but to provide care and offer medico-legal documentation.

- Don't ask questions beginning with 'why'. They sound judgmental. Most victims already blame themselves in some way.
- Deliver care in a manner that helps the victim regain control over what is happening to her/him. Continued explanation and reassurance about both legal and medical procedures, and obtaining informed consent from them is essential.
- Refer the patient to appropriate experts. Most areas in Ontario have designated **Sexual Assault Treatment Centres** who have special expertise. In Toronto, the centre is located at Women's College Hospital and Scarborough Grace Hospital, however the team is mobile to other hospitals if the patient cannot be discharged due to injury or other medical concerns.
- The nurse from the treatment centre is specially trained to deliver care options that include:
Forensic Evidence Collection (Sexual Assault Evidence Kit)
Prophylactic medications for Sexually Transmitted Infections and pregnancy.
Post exposure prophylactic medications for HIV Testing
Counseling referral
Follow-up options

Involving the Police

It is the victim's choice to have evidence collected and involve the police. Remember that in Canada, unlike the US, you do not report a sexual assault to the police unless the victim requests it. For those patients under 16 you are legally required to report to the local Children's Aid Society.

COLLECTING EVIDENCE: THE SEXUAL ASSAULT EVIDENCE (SAE) KIT

The SAE Kit includes forms for consent, medical history, history of assault, physical exam treatment guidelines, plus containers for forensic evidence. It was developed to ensure standardized collection of evidence. It takes 1-2 hours of uninterrupted time to complete and, once taken, the evidence cannot be left unattended until it is given to police so that the "chain of evidence is unbroken". It is generally only done up to 72 hours post assault.

In Toronto only the Sexual Assault Treatment Centres (SATC) at Women's College Hospital and Scarborough Grace generally uses the SAE Kit. The responsibility of the referring physician is to medically stabilize the patient, if possible without disturbing evidence that could be used to prosecute an assailant. If a victim cannot be transported because

of ongoing medical treatment, the SAE Kit is taken to the victim, and the nurse from the Sexual Assault Treatment Center will complete the SAEK at that site.

As a trainee you will not be expected to use a SAE Kit. **The following is for your information only.** The SAE Kit contains forms, diagrams and containers for the:

History

- General medical history including LMP, gynecologic history and contraception used
- History of the assault, type of sexual contact and mechanism of any injuries

Collection of Evidence

- Clothes worn at time of assault.
- Blood and urine for alcohol and drug screen
- Body evidence
- Genital/Anal Evidence

Documentation of Injuries

- Documentation of emotional state
- About 60% of victims have some physical injury, most often non-genital especially head and neck and upper extremities
- Very important to accurately note type of injury (abrasion, bruise, laceration, edema, erythema), size, shape, location, colour
- Examine oral cavity for palatal petechiae, frenulum tears if fellatio occurred
- Look for petechiae of face, conjunctiva, neck, if history of strangulation
- Genital findings in 10-30% without use of colposcope
- Most common: erythema, edema, small tears around 6 o'clock at the posterior fourchette, superficial abrasions, bruises
- Vaginal vault lacerations uncommon, but serious, as can bleed profusely and can enter the peritoneal cavity
- In anal assault- edema, stellate tears, bruising
- Usually digital exam is sufficient, but do proctoscopy if laceration is a concern
- Be wary of patient with vaginal or rectal bleeding and significant abdominal pain - rule out penetration into peritoneal cavity

MEDICAL TESTING AND PROPHYLAXIS

- If not immediately referring to a Sexual Assault Treatment Centre, baseline testing can be ordered by the physician. Otherwise, all of the following tests are offered and completed by the Sexual

Assault Treatment Centre nurse.

- HCG, if any chance of pregnancy from previous sexual activity.
- Oral culture for GC, if fellatio
- Cervical culture for GC, chlamydia and vaginal culture for trichomonas, if vaginal assault. (for forensic purposes Chlamydia culture techniques are preferred over PCR techniques)
- Rectal culture for GC, chlamydia, if anal assault
- Syphilis testing
- Hepatitis B and C
- Tube for HIV testing can be held, if not sent immediately

If patient declines referral to the Sexual Assault Treatment Centre, the following can be initiated in the ED:

Pregnancy prophylaxis

Offer emergency contraception if the patient is not using reliable contraception (BCP, IUD, tubal ligation, etc.). If preexisting pregnancy is possible obtain a STAT hCG or urine pregnancy test first. Preferred treatment is Plan B: levonorgestrel 1.5 mg PO as a single dose or 0.75mg tabs x 2 (12 hours apart) if single dosing will not be tolerated. Levonorgestrel is superior to the Yuzpe method and is available OTC in many pharmacies.

STI Prophylaxis

Should be offered to all patients:

Cefixime 800 mg po (covers GC)
plus

Doxycycline 100mg bid x 7 days
or

Azythromycin 1 gm po once (Chlamydia, preferred drug)

Hepatitis B Prophylaxis

This should be offered for vaginal/anal assault or traumatic oral assault with ejaculation if unimmunized.

Hepatitis B Immune Globulin 0.06 mg/kg IM
plus

Engerix B 1 ml IM deltoid - repeat Engerix at 1 and 6 months

HIV

The physician should discuss HIV, as almost all victims are concerned and most do not ask about it. The risk of HIV transmission in most sexual assaults is low. HIV testing can be done at the time of assault

repeated in, 6 weeks, 12 weeks and 6 months to rule out seroconversion. Patients should be advised to use condoms until negative test results are obtained. A baseline sample for HIV can be held for future testing if the patient desires (e.g. if future tests are positive).

Post exposure prophylaxis for HIV is offered up to 72 hours post assault at SATCs. If started the baseline HIV sample should be sent for immediate testing. The risk of transmission varies with the nature of the assault (e.g. traumatic vaginal or anal assault by assailant). Post exposure prophylaxis (PEP) should be instituted as soon as possible to be maximally effective, within 72 hours for a duration of 28 days.

SPECIAL CONSIDERATION

Suspected assault in the pediatric age group: Involved clinicians need to have appropriate training in order to examine, interpret findings, diagnose and treat. Some cases do not need to be assessed urgently and are better seen in outpatient clinics in a multidisciplinary setting.

Urgent cases include:

- Children with acute injury
- Patients that will likely not return for outpatient assessment
- Crisis support required
- Required urgent kit collection
- Urgent medical prophylaxis/treatment required

FOLLOW-UP

- Sexual Assault Treatment Centres have follow-up programs available. These include:
Follow up STI testing
HIV testing
Monitoring or dispensing HIV PEP
Counseling referrals
- Refer to a family physician
- Follow up at 1 week for blood test/symptom monitoring if on HIV PEP, this may be done in an ID clinic as available
- Repeat STI tests 2-3 weeks after treatment, completion of hepatitis B immunization at 1 and 6 months, hepatitis C and syphilis testing 12 and 24 weeks, HIV testing at 6 weeks, 12 weeks and 6 months
- Ensure follow up counseling and assessment of mental state

SUMMARY

- Sexual Assault is defined as ‘any form of sexual activity with another person without her/his consent’. It encompasses all unwanted sexual acts from fondling to forcible penetration.
- Supportive and compassionate care in the early phase can significantly assist the victim in recovery.
- It is the victim’s choice to have evidence collected and involve the police.
- For those patients under 16 you are legally required to report to the local Children’s Aid Society.
- Medical testing may include HCG, syphilis testing, oral/vaginal/anal swabs for GC & Chlamydia, Hep B & C screening, HIV screening.
- Consider pregnancy, STD, Hep B and HIV prophylaxis.

- Follow-up for psychological counseling, review of blood tests/cultures and immunizations must be ensured.

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*Contributor to Special Consideration section:
Heather Farina, RN BScN

53: MEDICO-LEGAL ISSUES

**Andrew McDonald, Maria McDonald,
updated by Mary Ann Badali**

OBJECTIVES

1. To understand 3 important areas dealing with legal consent in the Emergency Department:
2. Consent by a patient to a treatment plan, including substitute consent and emergency situations
3. Consent in situations of mental illness and the role of a Form 1 under the Mental Health Act
4. Consent by a patient for the release of information including some occasions when the physician must report an event without the patient's consent

INTRODUCTION

Since the health of citizens is mostly under provincial jurisdiction in Canada, most health-related legislation is under provincial jurisdiction and may vary from province to province. This chapter deals only with the laws in Ontario.

CONSENT TO TREATMENT BY A PATIENT

A person has the right to self-determination and to participate fully in decisions relating to his or her health. This is both an ethical and a legal principle throughout Canada. In Ontario, the Health Care Consent Act, 1996 (referred to as HCCA) consolidated the principles of consent to treatment to ensure that they are consistently applied to all settings. With this act, the term 'capable' (referring to mentally capable) has replaced the term 'competent'.

Generally, consent for treatment must be obtained from a patient prior to treatment, OR if the patient does not have capacity to decide about the treatment, consent must be obtained from the patient's Substitute Decision Maker (referred to as SDM). In Ontario, consent may not be required in certain emergency situations (see later in chapter). It should also be noted that consent cannot authorize an illegal procedure (for example, euthanasia).

What is capacity?

A patient is capable to give consent to treatment if the patient is:

- able to understand the information that is relevant to making a decision about treatment, and
- able to appreciate the reasonably foreseeable consequences of making or not making a decision

Some people may be able to make simple treatment decisions (e.g. whether or not to have a cast applied to a broken leg) yet incapable of making more complex ones (e.g. whether to undergo heart surgery). Also, the nature of the patient's illness may fluctuate over time as may the patient's mental capacity (e.g. if the patient has a bipolar disorder). The physician should periodically reassess the patient's capacity. If an incapable patient becomes capable, then the patient's own decision to give or refuse consent governs over decisions of the SDM.

Is there a minimum age of consent?

In Ontario, there is no longer a minimum 'age of consent'. Thus patients including those less than 18 years of age should be assessed as to their capacity to make the decision in the same way as an adult. For example, a 13 year old may be capable to give consent regarding sutures for a laceration, but might not be capable to give consent to undergo major surgery.

How is capacity determined?

A physician may presume that a patient is capable of making decisions with respect to treatment or admission to a hospital unless there are reasonable grounds to believe the patient is incapable. The following are some examples of situations where one might question the patient's capacity to give consent, which would then necessitate a capacity assessment:

- the person shows signs of confused or delusional thinking
- the person is experiencing severe anxiety
- the person's judgment is impaired by drugs or alcohol

What is treatment (and what is not)?

It is important to understand that consent from the patient (or his or her SDM) is required before treatment begins. The legislation (HCCA) defines treatment specifically, as well as setting out what is not considered to be treatment. Treatment is defined as "anything that is done for a therapeutic, preventive,

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palliative, diagnostic, cosmetic or other health-related purpose and includes a course of treatment or a plan of treatment.” The definition goes on to state that treatment does not include (and therefore consent is not required to undertake the following activities):

- assessing a person’s capacity
- assessing or examining a patient to determine the general nature of a person’s condition
- taking a patient’s health history
- communicating an assessment or diagnosis
- providing personal assistance service (daily activities like washing, eating, grooming, elimination, positioning, etc.)
- providing a treatment that “in the circumstances poses little or no risk of harm to the person” (for example, applying a bandage)

Treatment has been defined by recent Ontario cases to include “withdrawal of services”. Thus, “consent” is required to withdraw or discontinue life support measures.

The legislation to assess and restrain an agitated patient is described in both under the Mental Health Act as well as the Health Care Consent Act. Under the Health Care Consent Act, an assessment and examination of the person may be undertaken to determine the general nature of a person’s condition and this may be done without consent. There is a duty established in common law that allows a caregiver to restrain or confine a person when immediate action is necessary to prevent serious bodily harm to the person or to others. There may be occasions when restraint of the person may necessary to undertake the assessment and examination with the goal of preventing serious bodily harm to the person or to others. This is separate from assessment in the Form 1 setting.

Once a treatment plan is proposed, then consent must be obtained from the patient (or from a SDM) to proceed (unless there is an emergency as defined below).

What is required in the consent process?

The consent process requires good communication between the physician and the patient. When a court considers whether consent was obtained from a patient, it considers the whole consent process, and looks at the written consent form *only* as an indication that the verbal discussion took place. During the consent discussion, the physician is striving to provide information about the choices of treatment in a clear and understandable manner. The information should be presented in simple terms (use terms

appropriate for the patient), and must be communicated in a language that the patient understands. Also, any other barriers to communication (e.g. impaired hearing or vision, aphasia) must be addressed to facilitate the conversation.

The following elements are necessary in order to ensure that consent is valid. The consent must:

- relate to the treatment being discussed
- be informed (see below)
- be given voluntarily
- not be obtained through misrepresentation or fraud

What is ‘informed’ consent?

If a complaint were to proceed to a lawsuit, only a judge can decide during a court proceeding, whether the consent given by a patient was ‘informed’. The judge will look for evidence that the consent was valid (see list above) and that the relevant information required to make a decision was given to the patient (see list below). The information which must be communicated to the patient is the information that a reasonable person in the same circumstances would require in order to make a decision about treatment.

The information provided to the patient must include a discussion of:

- the nature of the proposed treatment
- the expected benefits of the treatment
- the material risks and side effects of the treatment (common complications with a high frequency of occurrence as well as potentially major complications for the particular individual)
- the alternative courses of action
- the likely consequences of not having treatment

The patient must also have the opportunity to ask for additional information.

How is consent documented?

There is no legal requirement that the patient’s consent be in writing. However, the policies, regulations or procedures of a hospital may require that consent forms be signed by the patient if the treatment carries significant risk. The practice of having a consent form signed by the patient or documenting the consent discussion on the hospital chart has developed to ensure that the consent process has not been overlooked. If the giving of consent is ever challenged, the onus is on the physician to prove that consent had been given.

Thus, it is to the physician's advantage to obtain a written consent from the patient or document the consent discussion on the hospital chart when the procedure is associated with any material side effects. However, having a patient simply sign a pre-printed form without a proper discussion of the harms, risks, options, etc. is not likely to be considered sufficient by a court to properly inform the patient about the treatment decision and options.

Can consent be withdrawn?

A capable patient may withdraw consent to treatment at any time. Similarly, the incapable patient's SDM may withdraw consent at any time. If consent for the treatment is withdrawn, treatment should be stopped immediately. Ask questions to explore why the consent was withdrawn, and propose alternatives to the patient or the SDM. If, for example, a patient yells "stop" during suturing, the treatment must stop. Further questioning may indicate, for example, that improved pain control will help the patient to give consent to restart treatment.

If a patient refuses treatment or withdraws consent to treatment, even if it is lifesaving, do not take it personally or get angry. Dignity and autonomy are important rights of all persons. Patients are free to choose to follow or not follow your advice. Patients are not obliged to make decisions consistent with his or her apparent 'best self interests'. If the patient refuses your advice, try to understand the reasons given by the patient, outline to the patient all other available options and seek the assistance of your staff physician.

Is consent needed in an emergency?

Consent to treatment is required from capable patients, even in an emergency. In certain cases where there is an emergency, consent may not be required for treatment, if the patient is incapable of providing the consent, and no SDM is available.

According to the Health Care Consent Act, an emergency exists if a person is apparently experiencing severe suffering or is at risk of sustaining serious bodily harm if treatment is not administered promptly. An examination to determine the general nature of a person's condition, including diagnostic procedures, is permitted without consent in order to determine whether an emergency exists (since such an examination does not fall within the definition of 'treatment'). The act goes on to state that no consent for treatment is needed in an emergency if three conditions are met:

- the patient is incapable AND
- there is an emergency AND

- delay required to get consent from a SDM will prolong the suffering or put the person at risk of sustaining serious bodily harm.

The emergency treatment of an incapable patient may only continue as long as is reasonably necessary to find a SDM. If a patient is apparently capable but the communication needed to get an informed consent cannot take place because of a language barrier or disability, emergency treatment may be provided.

Steps must be taken that are reasonable in the circumstances to find a way for the communication to take place and there must be no reason to believe that the person does not want the treatment. In this circumstance, the emergency treatment of a capable patient may only continue as long as is reasonably necessary to find a means to enable communication to take place, or to find a SDM.

The physician should record in the chart the conditions that led to the determination that the patient was incapable of consenting to the treatment, that an emergency existed, and that a delay would prolong the suffering or put the patient at risk of sustaining serious bodily harm.

If the physician knows that a mentally incapable patient would have refused treatment if capable, then emergency treatment cannot be given. For example, if the patient is carrying a wallet card stating that they do not wish blood transfusions, then that treatment should not be given, even if the patient is incapable. In the absence of prior expressed wishes or a SDM, the physician should make treatment decisions he/she believes are in the 'best interests' of the incapable patient.

"Consent" issues are particularly complicated in cases dealing with children where for religious reasons there may not be agreement on the recommended therapeutic interventions. If the parent will not provide consent for recommended treatment, Court approval may need to be considered after reporting to the Children's Aid Society.

How to find a substitute decision maker?

Ask the patient whether someone has accompanied him or her to the emergency department or whether a relative can be contacted by telephone.

The SDM should be:

- capable to give consent to the treatment (i.e. be able to understand the information and able to appreciate the consequences)
- at least 16 years of age (unless he/she is the parent of the incapable patient)

- available within a reasonable time, whether in person or by telephone
- willing to assume the responsibility of giving or refusing consent.

The SDM must follow the wishes of the incapable patient that were expressed while the person was capable. If the incapable patient's prior wishes are not known, or it is impossible to comply with the wish, then the SDM must decide according to the incapable patient's 'best interests'.

If a SDM gives consent to treatment, the physician should note the following in the chart:

- the Substitute Decision Maker's name, address and telephone number,
- the relationship of the Substitute Decision Maker to the patient.

What is the hierarchy of substitute decision making?

Ontario's Health Care Consent Act sets out a list from the highest ranked SDM to the lowest ranked. If there is a dispute between various people claiming to be the ones to make the substitute decision, then the higher ranked person is permitted to make the decision. In general, the highest ranking are SDMs legally appointed by a court or by the patient, followed by close relatives and finally by more distant relatives. The rank order of SDMs is listed in the Appendix I.

THE ROLE OF THE MENTAL HEALTH ACT IN PSYCHIATRIC CASES

The Mental Health Act of Ontario (MHA) sets out separate rules for the assessment of a person with a mental health disorder. A mental disorder is defined in the Act as 'any disease or disability of the mind'.

Patients Presenting with Mental Health Disorders

The police may exercise their authority under the Mental Health Act to bring a person to hospital if they are concerned for the person's safety or the safety of others. This does not require the person's consent. The person needs to be examined to determine if a Form 1 should be completed in the emergency department.

Alternatively, a person may be brought to the hospital with a Form 1 already completed by a physician (e.g. the Family Physician). This form allows the facility's staff to detain the person for a full assessment of mental state and potential dangerousness/risk. Again, this does not require the person's consent. A

'Psychiatric Facility' (such as a hospital with a psychiatric unit) is permitted to hold a person for up to 72 hours for psychiatric assessment if it is in possession of a properly completed Form 1 that has been signed within the prior seven days. Once a person is detained in hospital on a Form 1, the physician completing the Form 1 must advise the person of the detention and give the person a completed Form 42. This form is a written explanation of the circumstances and reasons for the detention.

Once the period of detention (up to 72 hours) for full assessment and examination begins, a psychiatrist will examine the person. If appropriate criteria are met, the person may be admitted as an involuntary patient. Other options may include admission as a voluntary or informal patient, or discharge.

Examination and Assessment under Form 1

The required information during an assessment may come from a variety of sources, such as direct examination, or from other persons. At least one of the criteria from each of the following two tests must be met: the past / present test, and the future test.

The past / present test looks at past or current events to help predict the future dangerousness. The physician must have reasonable cause to believe the person:

- has threatened or is threatening to cause bodily harm to self; OR
- has attempted or is attempting to cause bodily harm to self; OR
- has behaved or is behaving violently toward another person; OR
- has caused or is causing another person to fear bodily harm; OR
- has shown or is showing a lack of competence to care for self

For the future test, the physician must decide (using all the available information, including the past / present test) if the person is apparently suffering from mental disorder that will likely cause:

- serious bodily harm to self; OR
- serious bodily harm to others (need not be imminent); OR
- imminent and serious physical impairment of the person (imminent means in the next hours, days or even weeks, and includes unintentional harms such as wandering in the cold, or gross neglect at the hands of others).

Remember that a Form 1 is an important legal document. It must be accurate, legible and complete. Only the physician who examined the person may sign a Form 1. A Form 1 does not permit treatment to be given.

Consent for Release of Information

The relationship between a patient and a physician requires a high degree of confidentiality. Under the Medicine Act, it is professional misconduct to give information concerning a patient's condition or any professional services performed for a patient to anyone without the consent of the patient, unless "required by law" (as set out in a statute or in a court order). The Code of Ethics of the Canadian Medical Association states that it is a fundamental principle of ethical behaviour to protect a patient's right to confidentiality.

In Ontario, health specific privacy legislation clarifies these principles. The Personal Health Information Protection Act, 2004 (PHIPA) confirms the patient's existing right to control and have access to their personal health information. This Act establishes a set of uniform rules about the manner in which information may be collected, used and disclosed.

A physician should insist on being provided an original written consent or authorization (signed by the patient) for the release of specific medical information unless disclosure is "required by law". The expression "required by law" relates to the various acts and regulations which mandate obligatory reporting or a court order. It does *not* refer to a letter from a lawyer, a subpoena or summons to attend at trial, or questioning by a police officer. Be vigilant when getting requests for information about a patient's condition, whether verbal or written. Always ask for the patient's written consent (signed by the patient) unless you receive verbal confirmation from the patient personally (in which case you should make a note of the conversation in the chart).

MANDATORY REPORTING

The following list some of the more common events that require mandatory reporting, that is, without the consent of the patient.

Aeronautics Act

- reporting of a flight crew member, air traffic controller or other holder of a Canadian aviation document if the patient has a medical or optometric condition that is likely a hazard to aviation safety

Highway Traffic Act

- reporting of person suffering from a condition that may make it dangerous for the person to operate a motor vehicle

Child and Family Services Act

- reporting if a child is or may be in need of protection, or that a child is or may be suffering or may have suffered abuse

Health Protection and Promotion Act

- reporting of 'reportable' and 'communicable' diseases

The Coroner's Act

- reporting the death of persons under certain circumstances

Regulated Health Professions Act,

- reporting of sexual abuse of a patient, professional misconduct, incompetence, and incapacitation by a member of any health professions college.

Generally, statutes requiring mandatory reporting also provide protection from lawsuits for those making reports acting in good faith.

SUMMARY

- patients have the right to give consent or refuse a proposed treatment
- when a patient is incapable to give consent, a Substitute Decision Maker must be sought
- document consent discussions on the hospital record or on a consent form to demonstrate a consent discussion has taken place and the patient grants his/her consent
- know the circumstances when consent to treatment may not be necessary
- the Mental Health Act and its forms serve to protect patients' rights, and document decision-making regarding involuntary detention in hospital
- get written consent from patients before releasing their health information, unless required by law

APPENDIX I

Order of Substitute Decisions Makers according to the Health Care Consent Act of Ontario (1996)

Legally Appointed Representatives

1. Legal Guardian of the patient (appointed by the court) who has authority to give or refuse consent to treatment
2. Power of Attorney for Personal Care (may be a relative or anyone chose by the patient)
3. Representative appointed by the Consent and Capacity Board

Relatives

4. Spouse (includes those who have lived together for one year; or are parents of a child; or entered into a cohabitation agreement) or Partner (if close relationship of primary importance and lived together for one year), but not if living separate and apart

5. Child or Parent
6. Parent with only a right of access
7. Brother or Sister

Others

8. Other relative (includes people related by blood, marriage or adoption)
9. Public Guardian and Trustee (a public agency).

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54: PREHOSPITAL CARE

Brian Schwartz
Updated by **Jordan Chenkin**

OBJECTIVES

1. To understand the levels of prehospital care providers
2. To review the responses phases of 'a call'

INTRODUCTION

Prehospital Care and transport in the city of Toronto are provided by Toronto Emergency Medical Services. The Emergency Medical Services (EMS) system may also involve other agencies in providing a 'tiered response' for potentially serious calls, such as cardiac arrest. This involves selective dispatching of firefighters or police in addition to paramedics to certain calls.

The Province of Ontario also supports an advanced air ambulance system ('Ornge'), involving both fixed wing and helicopter aircraft, utilizing the levels of providers described below. While most air medical transports are inter-hospital, helicopters also respond to trauma and medical patients at the scene in rural areas.

Various jurisdictions (rural, urban, suburban) have different systems, based on geography, population, and skill levels of providers. The system outlined below is the one that serves the population and hospitals in Toronto.

LEVELS OF PROVIDERS (Province of Ontario)

Emergency Medical Responders (EMR)
Usually non-medical public safety personnel, i.e. firefighters, police. Can quickly respond to the scene of an emergency to provide initial care before more advanced resources are available. Scope of practice/skills:

- CPR
- First aid
- Oxygen
- Assist ventilation
- Automated Defibrillation

First response is often performed by lay responders with automatic external defibrillators (e.g. lifeguards, flight attendants, security personnel).

Primary Care Paramedic (PCP)

Scope of practice/skills:

- Patient assessments
- Basic airway management (including oropharyngeal airway and suction)
- O₂ - mask, cannula or bag-valve mask (BVM)
- CPR
- Semi-automatic external defibrillation
- Basic trauma care including immobilization for spinal / extremity injuries, hemorrhage control, triage skills and appropriate transport (i.e. trauma triage guidelines), extrication principles
- Basic procedures such as blood glucose determination, 12-lead ECG
- Administration of some drugs (epinephrine for anaphylaxis, salbutamol, ASA, NTG, glucagon for hypoglycemia)
- May include starting and maintaining intravenous lines

Advanced Care Paramedic (ACP)

PCP plus additional skills:

- Advanced airway management (i.e. endotracheal tube placement, cricothyrotomy)
- Cardioversion, manual defibrillation, transcutaneous pacing
- Administration of emergency drugs (i.e. IV epinephrine, naloxone, dopamine)
- Treat cardiac emergencies according to ACLS standards
- Other advanced procedures: needle thoracostomy, intraosseous insertion

Critical Care Paramedic (CCP)

ACP plus additional skills:

- Expanded medication list (inotropic infusions)
- Rapid sequence intubation
- Foley catheter insertion, NG tube, invasive monitoring (CVP, arterial lines)

RESPONSE PHASES 'A CALL'

Pre-arrival

- Public Education - EMS access and when to call 911, CPR, public access defibrillation
- System Access - 911 call receiving centre - transfer to appropriate agency (police, ambulance or fire)
- Emergency Medical Dispatcher - uses algorithm to determine type of response required. May also provide caller with pre-arrival instructions.
- Emergency vehicle deployment - primary or advanced paramedics with tiered response if necessary

On Scene

- EMR and paramedic interventions
- Medical intervention - basic life support, advanced life support, medical control (medical directives and/or direct medical oversight by a base hospital physician on radio or phone)

Transport

- In general, want to minimize on-scene time and transport as quickly as possible (ie. 'load and go')
- Ongoing care and essential interventions provided during transport
- Notification of receiving facility prior to arrival

Transfer of Care

Verbal and written report to Emergency Department staff by ambulance crew.

Data Analysis and Quality Management

Continuous quality improvement

- Measuring patient care process and clinical outcomes
- Improving care through clinical education and systems management

SUMMARY

- There are 4 levels of prehospital care providers: emergency medical responders, primary care paramedic, advanced care paramedic and critical care paramedic
- There are 5 response phases to 'a call': pre-arrival, on scene, transport, transfer of care and data analysis/quality management

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