ESSENTIAL EMERGENCY PROCEDURAL SEDATION AND PAIN MANAGEMENT

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Dedication

To my parents and brothers for their support and inspiration over the years, and supporting my ideas; my teachers, mentors, and colleagues who have taught me the ropes of being a physician and an educator; my friends for their understanding and support.

In memory of my father who was a scholar in his own right. His integrity and compassion have made me who I am.

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Preface

Procedural Sedation and Analgesia (PSA) is commonly utilized in the Emergency Depart-ment (ED) for patients undergoing painful procedures. It is now an essential inter professional skill required in the ED for effective and timely patient management.

This handbook is designed to provide the reader with a guide on the subject of proce-dural sedation and analgesia. With increasing competencies required by emergency room clinicians, the need for an organized approach to PSA that is consistent with best practices is essential.

This book is divided into three sections. The first part deals with procedural sedation. A step-by-step approach to PSA is outlined along with medications commonly used. The second part deals with pain management and regional anesthesia. It is important for the clinician to understand the importance of treating pain and the best modalities. The final section deals with a systems based approach to pain management using current evidence. Also in the text are special chapters dedicated to pediatric care and chronic pain which are not well described in general emergency medicine texts.

The simple format of the book makes it easy to read and access information quickly.

Acknowledgments

Contents

Procedural Sedation

1

Introduction to Procedural Sedation

Rahim Valani

Procedural Sedation and Analgesia

- Procedural sedation is the technique of administering a sedative or dissociative agent to induce a state that allows the patient to tolerate unpleasant procedures.
- Also known as procedural sedation and analgesia (PSA) if analgesia is administered concomitantly.
- Now advocated as a core competency in emergency medicine.
- Sedation is a continuum (see Figure 1.1 and Table 1.1):

INCREASING DEPTH OF SEDATION

MILD SEDATION   MODERATE SEDATION   DEEP SEDATION   GENERAL ANESTHESIA

DISSOCIATIVE ANESTHESIA
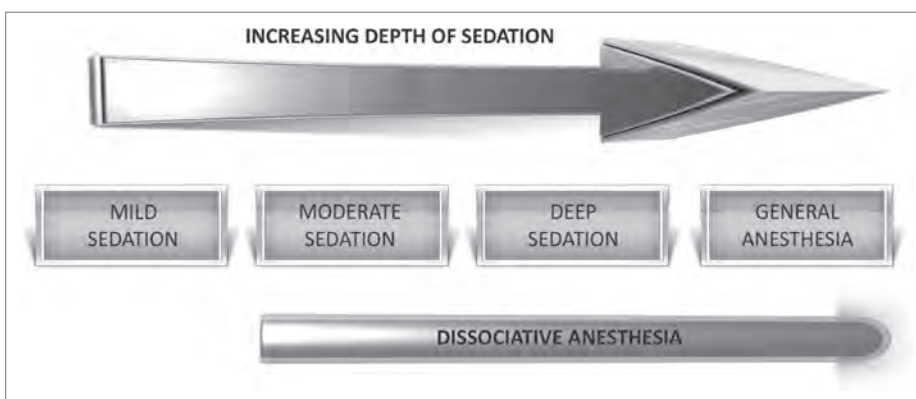
FIGURE 1.1: Sedation is a continuum, and a patient can easily move from deep sedation to general anesthesia.

- PSA is a common practice in the emergency department (ED). The goals of PSA are as follow:
  - Provide the patient with a safe environment where a painful or unpleasant procedure is required.
  - Alleviate patient anxiety.
  - Minimize physical discomfort.
  - Maximize amnesia.
  - Control motor behavior and movement if necessary so as to perform painful/unpleasant procedures such as a lumbar puncture or fracture reduction.
  - Minimize the risk of the procedure, and ensure safe discharge of the patient from the ED.

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TABLE 1.1: Continuum of sedation: le vels of sedation and analgesia

	Responsiveness	Airway	Ventilation	Cardiovascular function
Mild sedation (anxiolysis)	Responds to verbal commands	Unaffected	Unaffected	Unaffected
Moderate sedation	Purposeful response to verbal or tactile stimuli	No inter vention required	Adequate	Usually maintained
Deep sedation	Purposeful response to painful stimuli	Intervention may be required	May be inadequate	Usually maintained
General anesthesia	Unarousable	Intervention required	Inadequate	Impaired

- Appropriate policies and protocols enable a safe practice within the ED .
  - Ensure quality control while minimizing risks and adver se outcomes.
  - Preprinted order s and monitoring sheets should be a requirement for an y department perfor ming PSA.
- There is an increased need for the use of PSA in the ED due to the following:
  - Hospital o vercrowding – there ma y be a potential to a void an admission to the hospital b y performing the procedure in the ED and discharging the patient home safely.
  - Limited a vailability of anesthesia – the anesthesia team ma y be in the operating room, and not a vailable for PSA either in the ED or in the operating room.
  - Increased training of emergency ph ysicians in PSA.
- The most common procedures using PSA perfor med in the ED are as follow:
  - Orthopedic procedures (most common, and includes dislocations, fracture reductions, and immobilization/splinting).
  - Abscess incision and drainage.
  - Laceration repair.
  - Cardioversion.
  - Foreign body remo val.
  - Lumbar puncture.
  - Endoscop y.
- The role of PSA is expanding in the ED as it has been shown to:
  - Increase con venience of the patient – the y no longer need to w ait to go to the operating room.
  - Accessibility – procedures can now be done in the ED so as to facilitate easier access.
  - Cost-effectiveness b y reducing w ait times, ear lier patient discharge, and avoidance of operating room per sonnel and time.
- Exclusion criteria and contraindications for ED sedation include:
  - Sedation time >30 minutes.
  - Patient with a potentially diff cult airway (see Chapter 2).

- Patient with ASA functional class III or greater (see Table 4.2).
- Patients who are hemodynamically unstable.
- A patient who is known to be intubated shortly for another procedure/surgery.
- Lack of personnel experienced in airway management, advanced life support, and unfamiliarity with medications.
- Lack of appropriate monitoring equipment.
- Patient has a known allergy or sensitivity to the choice of drugs.

■ Complication rate of PSA estimated to be <1 percent in the hospitals and specialties routinely practicing PSA.
  - Adverse event rate is estimated at 2–3% in pediatric patients.
  - The most common complication is respiratory depression and airway obstruction.
  - Medication error is another common cause.
    ▶ Prescription error rate if 5.5 per 1,000 ED medication orders written.
    ▶ The majority are due to dosing errors. Others are due to similar packaging of drugs, illegible orders, and drug interactions.

■ Many of the complications related to PSA can be prevented by the following:
  - Appropriate monitoring and recognition of respiratory depression or arrest.
  - Adequate monitoring.
  - Ensuring the correct dose of medications (avoid drug calculation error).
  - Careful titration of medications.
  - Appropriate patient assessment.
  - Avoiding drug–drug interactions.
  - Personnel present who are trained in airway management and resuscitation.

Steps for Procedural Sedation and Analgesia (See Chapter 4)

Preparation

■ Determine the need for the procedure, and also the following:
  - Availability of space and personnel in the department to safely conduct the procedure and sedation.
  - Sedation should take place in a central, well-maintained area of the ED that has capabilities for resuscitation and airway management.

■ Provide the patient with appropriate information regarding the procedure, the need for sedation (informed consent should include benefts, risks, and limitations of therapy), anticipated changes, and expected duration of PSA.
  - Written or verbal consent should be documented.

■ Prepare necessary equipment for monitoring (see Chapter 6).

Personnel

■ Appropriate personnel trained in PSA and resuscitation must be present.
■ Require personnel for the following:
  - Monitoring of patient.

- Physician responsible for medications used during PSA and airway management (can also use respiratory therapists for airway management).
- Person who will actually perform the procedure (different from the one giving the medications).
- Additional personnel/assistants as required.

Patient Assessment (See Chapters 5 and 6)

- Want to ensure a thorough history and physical examination.
- Pay particular attention to ASA functional status (see Table 4.2), airway assessment, and other anticipated complications.
- Assess the risk of conducting the procedural sedation in the ED versus the need to take the patient to the operating room.

Equipment and Presedation Interventions (See Chapters 2, 3, and 6)

- Medications and reversal agents drawn and ready at the bedside.
- Airway equipment, including oral airways, bag-valve mask ventilation, and intubation equipment prepared.
- Patient with full cardiorespiratory monitoring (see Chapter 6).

Procedural Sedation and Analgesia (See Chapter 5)

- See Chapter 3 on pharmacology of procedural sedation.
- The patient must be monitored continuously with objective physiological monitors and qualitative clinical assessment.
- Patient needs appropriate monitoring, including:
  - Continuous cardiac monitoring.
  - Pulse oximetry.
  - Capnography.
  - Properly trained support should continuously monitor the patient and an appropriately trained emergency physician or other credentialed specialist (like a respiratory therapist) should be present.
- The choice of the agent depends on various factors:
  - Experience of practitioner.
  - Preference of practitioner.
  - Requirements imposed by the procedure.
  - The likelihood of producing a deeper level of sedation than anticipated.

Postsedation Recovery (See Chapter 5)

- Ensure that the patient is fully awake and alert for safe discharge from the ED.
- Printed instructions for family members/caregivers.

Summary

- PSA is an important skill for emergency physicians.
- Appropriate personnel with the necessary skill set are necessary for safe practice in the ED.

Suggested Reading

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2 Airway Assessment and Management for Procedural Sedation

Angela Stone and Mark Freedman

Introduction

- Ensuring adequate patient ventilation and oxygenation during procedural sedation is essential.
    - Most common adverse event during procedural sedation is respiratory depression.
    - Appropriate assessment involves recognizing challenges in airway management and preparing for respiratory depression and apnea.
- Procedural sedation requires a dedicated physician and nurse skilled in airway management and knowledgeable about potential complications.

Airway Assessment

- Assessment of the patient's airway is essential to anticipate challenges and complications to managing potential respiratory depression (see Figure 2.1).
- The goal of airway evaluation is to identify characteristics that may predispose a patient to airway obstruction, difficult bag-valve mask (BVM) ventilation, or a difficult intubation.
- Predicting these challenges will allow preparing for appropriate airway adjuncts and tailor clinical observation.

Patient History

- A brief assessment of the patient's medical history may alert you to conditions that may predispose to challenges in airway management:
    - History of difficult intubations in the past.
    - Modified oral or airway anatomy (genetic or prior surgery).
    - History of airway problems (e.g., reactive airway disease and sleep apnea).

Physical Examination

- A thorough physical examination is necessary to adequately assess a patient's airway.
- The patient should be observed and examined and the following documented in the airway assessment:
    - Look (front and side profile, in mouth).
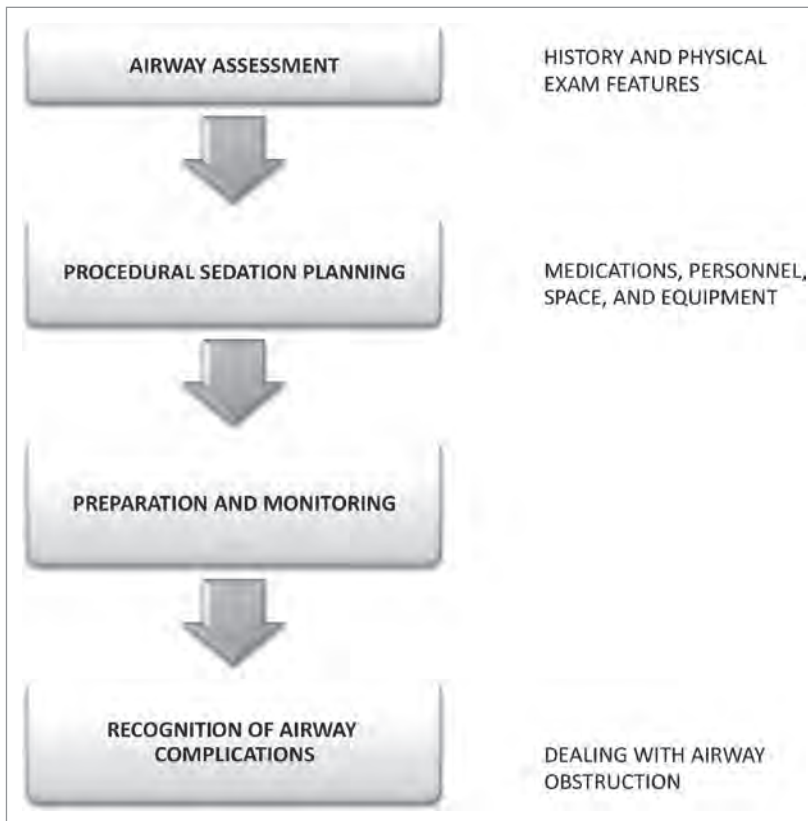
FIGURE 2.1: Overview of airway assessment and management during procedural sedation.

- Teeth (prominent incisors, crowns, dentures, loose or chipped teeth).
  - Cervical spine mobility.
- Have the patient flex and extend the head and neck.
  - Mouth Opening.
- Have the patients open their mouth as wide as possible.
- The patient should be able to insert three fingers between the incisors ideally.
  - Size of the mandible is equal to hyomental distance and thyromental distance.
    - During laryngoscopy, the tongue is pushed into the mandibular space by the blade.
      - If the mandible is too small, there will be insufficient room for the tongue to be displaced forward while the posterior tongue and epiglottis will obstruct the view of the glottis.
    - Hyomental distance – distance from hyoid bone to mentum (chin).
      - Three fingerbreadths of the patient is equal to adequate distance.
    - Thyromental distance – distance from the thyroid cartilage (Adam's apple) to the undersurface of the mandible.
      - Two fingerbreadths of the patient is equal to adequate distance.

- Oral Access
  - ▶ The size of tongue in relation to the oral cavity is assessed using the Mallampati classification.

Mallampati Classification

- The patient should be examined sitting with the head of the bed in a neutral position, the mouth opened as wide as possible, and the tongue protruded maximally.
- Visibility of the oral and pharyngeal structures (i.e., uvula, tonsillar pillars, and soft palate) are used to predict difficulty with ventilation and intubation.
  - For example, Class IV (hard palate visible only) suggests potentially difficult ventilation and intubation (Figure 2.2).
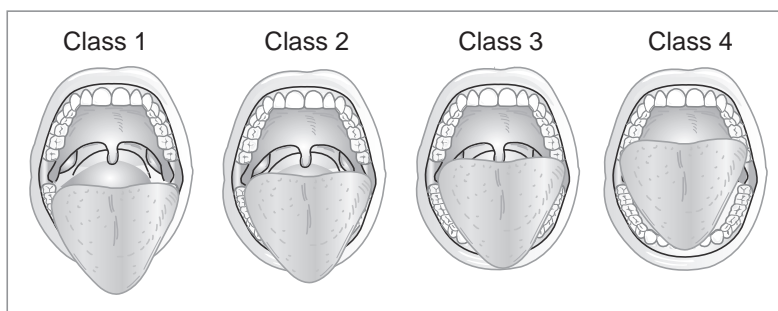
FIGURE 2.2: Airway assessment using the Mallampati classification.

Predicting Upper Airway Obstruction and Difficult Ventilation

- Predictors of difficult BVM ventilation include:
  - Beard and facial hear.
  - Obesity (BMI >26).
  - Older patients (age >55 years).
  - Edentulous.
  - History of obstructive sleep apnea.
- Two or more of the above has a 72% specificity and 73% sensitivity for difficult BVM ventilation.

Preparation and Monitoring

- Appropriate monitoring during procedural sedation is crucial, and should include the following:
  - Pulse oximetry.
  - Capnography.
  - Clinical observation.
- See Chapter 6 on monitoring during PSA.

Recognition of Airway Complications

Causes of Airway Obstruction

- Airway compromise from obstruction can occur at any level of the oropharyngeal–tracheobronchial passage.
  - Upper airway.
- Tongue (due to decreased level of consciousness causing tongue to displace posteriorly or modified anatomy).
- Soft-tissue swelling.
- Blood or vomitus.
- Direct injury.
  - Larynx.
- Foreign material.
- Soft-tissue swelling.
- Direct injury.
  - Lower airway.
- Secretions, edema, blood.
- Bronchospasm.
- Aspiration of gastric contents.
- Propfol and ketamine can be used simultaneously yet from separate syringes thus allowing their independent titration.

Recognizing Airway Obstruction

- Look for:
  - Chest/abdominal movement during sedation.
  - Condensation in the face mask.
- Listen at mouth and nose for breath sounds and abnormal sounds (gurgling, stridor, wheezing).
- Abnormal sounds due to airway obstruction:
  - Snoring – obstruction of upper airway by tongue.
  - Gurgling – obstruction of the upper airway with blood, secretions, vomit, etc.
  - Wheezing – narrowing of the lower airways/bronchospasm.
  - Silence – complete airway obstruction (i.e., laryngospasm).
- Feel at mouth and nose for expired air.

Airway Management

- BVM ventilation is a skill that is typically easy to perform. All personnel who are performing PSA need to have this skill.
- When challenges occur, the best response is that of a staged response, rather than immediately resorting to advanced airway placement.
- The vast majority of the time, noninvasive measures to treat airway obstruction (i.e., repositioning, airway adjuncts, BVM techniques) is all that is needed, and advanced airway placement can be avoided.

- In the event that advanced airway placement fails, knowing methods to trouble shoot difficulties with BVM ventilation is essential.
    - A combination of repositioning, placing an oral airway, and two-handed technique focusing on good jaw thrust is almost always successful.
    - These techniques focus on preventing the tongue from obstructing the upper airway.

Maneuvers to Open the Airway

- Head tilt, chin lift.
    - This maneuver is used to anatomically "open" the airway or place the patient in a sniffing position.
- Jaw thrust.
    - Inability to ventilate due to airway obstruction is often times corrected by using the chin-lift, jaw-thrust maneuver.
    - Ensure that the correct size face mask for a BVM is placed on the patient and held in place.
    - The mandible is elevated so as to pull forward the connecting soft tissues/tongue to relieve the obstruction.

Bag-Valve Mask Ventilation

- Knowing how to effectively ventilate a patient using a BVM (i.e., AmbuBag, Laerdal Bag) is a life-saving skill.
- Effective ventilation depends on good positioning, maneuvers that open the patient's airway, and using adjuncts to overcome airway obstruction.

Oral and Nasal Airways

- Oral and nasal airways are useful adjuncts to airway management in patients who are in moderate to deep sedation and are difficult to ventilate.
    - They act to open the airway in patients who are prone to obstruction due to body habitus or deep sedation.
    - Avoid pushing in or forcing an oral airway in a patient who is gagging.

Oral Airways

- Not tolerated in awake or mildly sedated patients.
- Appropriate size is by measuring from the front teeth of the patient to the angle of the mandible.

Nasal Airways

- Better tolerance in awake, mildly sedated patients than oral airways.
- The length of the airway is measured from the nares to the tragus of the ear.
- It should be lubricated and placed by advancing the airway straight back and close to the medial wall of the nares.
- Exercise caution in those patients who may be on anticoagulants as bleeding may occur.

Advanced Airway Techniques

Laryngeal Mask Airway

- The laryngeal mask airway (LMA) is an ovoid silicone mask with an inflatable rim that is inserted blindly into the pharynx.
- Its advantages are the following:
  - Ease of use.
  - Extremely high success rate with little training.
  - Low complication rate.
- Works as a potential alternative to endotracheal intubation when personnel are inexperienced or as a temporizing role.

Endotracheal Intubation

- If appropriate patient history, physical examination, planning, and use of noninvasive airway management skills are performed, intubation should not be necessary during PSA.
- The decision to intubate should be based on three essential criteria:
  - Failure to maintain or protect airway.
  - Failure to oxygenate or ventilate.
  - Anticipate the need for intubation.

Summary

- Having the essential skills for airway management is necessary for PSA in the emergency department.
- Appropriate planning and selection of patients can avoid potential airway complications.

Suggested Reading

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3

Pharmacology of Procedural Sedation

Vince Teo

Choosing Medications for PSA

- Procedural sedation has relied on a variety of phar macological agents for the following:
  - Give sedation (sedatives).
  - Relieve pain (analgesics).
  - Cause a dissociative state (dissociative agents).
- These agents, either alone or used in combination allow the patient to better tolerate any pain or discomfor t associated with the procedure.
- The ideal phar macological agent for procedural sedation is able to produce:
  - Optimal sedation and analgesia rapidly .
  - Has a shor t duration of action to facilitate a quick reco very without recollection of procedure.
  - Does not cause an y adverse e vents (such as respirator y depression).
- Current classes of medication emplo yed include:
  - Benzodiazepines (e.g ., midazolam).
  - Opioids (e.g ., fentanyl and mor phine).
  - Propofol.
  - Etomidate.
  - Ketamine.

Agents for Use in Procedural Sedation (Table 3.1)

Midazolam

- Benzodiazepines promote the binding of the inhibitor y neurotransmitter, gamma-aminobutyric acid (GABA) to GABA receptor s, enhancing their activity .
- Midazolam is similar to other benzodiazepines exhibiting the following proper ties:
  - Sedation.
  - Amnesia.
  - Anxiolysis.
  - Anticonvulsant.
  - Muscle relaxant.
- It has a rapid onset and shor t duration of action without active metabolites.

TABLE 3.1: Medications used for procedural sedation

Drug	Dose		Onset	Duration	Contraindications
Midazolam	Initial: 0.02–0.1 mg/kg (max 2.5 mg [1.5 mg in elderly]) Repeat 25% of dose q 3 min Cumulative max 5 mg (3.5 mg in elderly)	Inject slowly over 2 min (use 1 mg/mL)	1–2 min	30–60 min	
Propofol	Initial 0.5–1 mg/kg IV May repeat 0.5 mg/kg increments q 3–5 min	Shake well Inject slowly over 3–5 min	<1 min	3–10 min	Allergy to soybean or egg products Hypersensitivity to EDTA or sodium metabisulfte
Etomidate	0.2 mg/kg	Over 30–60 sec	<1 min	3–5 min (full recovery 5–15 min)	
Ketamine	IV 1–2 mg/kg Then 0.25–0.5 mg/kg q 5–10 min		1 min	15–10 min	HTN Increased ICP Psychosis
Fentanyl	1–1.5 mcg/kg IV titrate 1 mcg/kg q 3 min		1–2 min	30–60 min	
Atropine	0.5–1 mg q 5 min (bradycardia) 0.4–0.6 mg (salivation or secretions)	Rapid IV push	Rapid		Closed angle glaucoma Tachycardia Obstructive GI disease or ileus Myasthenia gravis
Naloxone	0.1–0.2 mg IV		1 min IV 10–15 min IM	15–30 min	
Flumazenil	0.1–0.2 mg IV may repeat in 1 min	Infuse over 15 sec Cumulative max 0.05 mg/kg (or 1 mg)	<1 min	45 min	

Indication/Properties							
Sedation	Amnesia	Anxiolysis	Analgesia	Dissociation	Pros	Cons	Other
✓	✓	✓			Longer safety record/ experience with use Anticonvulsant properties	Requires 2nd agent for analgesia Hypotension Respiratory depression	
✓	✓				Rapid onset + recovery Anticonvulsant and anti-emetic properties	CV depression Hypotension can cause rapidly deepening sedation	No analgesic properties
✓	✓				No histamine release Minimal CV, respiratory effects	Myoclonus Injection site pain Transiently lowers cerebral blood flow (slight to moderate ↓ ICP usually just several minutes)	No analgesic properties
			✓	✓	Safety data in paeds	Emergence delirium (especially adults) Myoclonus Laryngospasm; Hypersecretions Agitation Nystagmus CV stimulation	
			✓		Minimal CV depression; reversible Proven safety	Requires 2nd agent for sedation Repeat dose usually required Cough Hiccups Vomiting Itchiness	
							May be given as pretreatment to prevent excessive salivation with ketamine
						Very safe	Not routine
						Can cause seizures Benzodiazepine withdrawal	Not routine

- Dosing:
  - Initial 0.02–0.1 mg/kg (maximum 2.5 mg [1.5 mg elder ly]).
  - May repeat 25% of dose e very 3 minutes (maximum of 5 mg cumulative dose [3.5 mg elder ly]).
- Availability and administration:
  - 1 mg/mL – 10 mL vial containing 10 mg .
  - 5 mg/mL – 1 mL vial containing 5 mg .
  - Direct Injection IV slowly o ver 2 minutes (1 mg/mL concentration should be used to help facilitate this).
- Onset:
  - 1–2 minutes.
- Duration:
  - 30–60 minutes.
- Pharmacokinetics:
  - Midazolam has a rapid onset and shor t duration of action without active metabolites.
  - It is highly lipophilic, resulting in a relatively large volume of distribution (compared to other benzodiazepines).
    - ▶ Thus, its half-life increases signif cantly in obese patients.
  - It is metabolized in the liver , a substrate of the CYP3A4 isoenzyme, and is excreted primarily in the urine.
- Contraindications:
  - Hypersensitivity to midazolam or an y component of the for mulation (benzyl alcohol).
- Adverse effects:
  - Respirator y depression (dose and infusion rate dependent).
  - Apnea (dose and infusion rate dependent).
  - Hypotension.
  - Deep sedation.
  - Impaired coordination.
  - Diminished ref exes.
- Monitoring:
  - See Chapter 6 on monitoring during procedural sedation.
- Special considerations and pear ls:
  - Wait at least 2 minutes to assess response before administering subsequent doses.
  - Midazolam does NOT ha ve any analgesic proper ties.
    - ▶ For painful procedures, consider second agent for analgesia.
  - Patients premedicated with an opioid (e.g ., fentanyl) should ha ve dosages reduced by ~25%.
  - Co-administration with other CNS depressants (benzodiazepines, barbiturates, opioids, other sedatives) will increase sedation and the risk of respirator y depression.

- Concomitant use of CYP3A4 inhibitor s (e.g., azole antifungals, erythromycin, clarithromycin, verapamil, propofol) ma y increase le vels of midazolam.
- Patients with hepatic dysfunction or se vere CHF ma y have experienced prolonged effects.
- When used in combination with fentan yl, respirator y depression ma y occur in up to 25% of patients.

Propofol

- Propofol is an ultra-shor t-acting non-opioid, non-barbiturate sedative-h ypnotic agent.
- Its exact mechanism of action is unknown, but it is thought to enhance the binding of GABA to its receptor sites.
- It has sedative, amnestic, anticon vulsant, and anti-emetic proper ties.
- It does not ha ve analgesic proper ties.
- Main benef ts are that it has a quick onset and shor t duration of action resulting in a rapid reco very.
    - However, due to its potency , there is a risk to the patient of quickly progressing to deep sedation.
- Dosing:
    - Loading dose: 0.5–1 mg/kg IV .
    - May repeat 0.5 mg/kg IV e very 3–5 minutes.
- Availability and administration:
    - 10 mg/mL – 20 mL, 50 mL, 100 mL vials a vailable.
    - Propofol is a vailable as an emulsion. Shak e well prior to use.
    - Inject IV slowly o ver 3–5 minutes.
- Onset:
    - <1 minute.
- Duration of action:
    - 5–10 minutes (full reco very within 10–15 min).
- Pharmacokinetics:
    - Propofol is a vailable in a so ybean oil emulsion.
    - Although it achie ves therapeutic concentrations in the CNS rapidly , it is rapidly redistributed to muscle and fat tissue, resulting in an effective duration of action (~10 min) much shor ter than its half-life (15–45 hr).
- Contraindications:
    - Hypersensitivity to so ybean oil or egg products. (Emulsif er in the for mulation is derived from egg).
    - Hypersensitivity to EDT A or sodium metabisulf te (preparations of propofol contain either one of these agents as a preser vative).
- Adverse effects:
    - Hypotension (ma y occur in up to 30% of patients).
    - Bradycardia.
    - Respirator y depression/apnea (up to 25%).
    - Site injection pain (15–20%).

- A rapid deepening of sedation.
- Spontaneous musculosk eletal mo vements (twitching , jerking or hands, arms, feet and legs) (3–10%).
■ Monitoring:
  - See Chapter 6 on monitoring during procedural sedation.
■ Special considerations:
  - Strict aseptic technique is impor tant when preparing and administering propofol since the lipid vehicle is capable of suppor ting bacterial growth.
  - Propofol does not ha ve analgesic proper ties – alw ays ensure that analgesic agent is also given to the patient for painful procedures.
  - If site injection pain is an issue,   may pre-inject site with lidocaine.
  - Hepatic failure – ma y require low er dosage to be used,   as patients ma y recover more slowly due to decreased elimination.
  - Monitor closely , as le vel of sedation can easily progress to deep sedation.

Etomidate

■ Etomidate is an ultra-shor t-acting non-barbiturate, non-opioid, non-benzodiazepine sedative-hypnotic.
■ It has a rapid onset of action,   and shor t recovery time (comparable to propofol).
■ Furthermore, it does not promote histamine release and has minimal effects on the cardio vascular and respirator y systems, making it an appealing option for procedural sedation.
■ Dosing:
  - 0.1–0.2 mg/kg IV bolus.
  - 0.05 mg/kg IV for subsequent doses (if necessar   y).
■ Availability and administration:
  - 2 mg/mL – 10 mL ampoule containing 20 mg .
  - Use undiluted.
  - Direct injection IV slowly o ver 30–60 seconds.
■ Onset:
  - <1 minute.
■ Duration of action:
  - 3–5 minutes (full reco very 5–15 min).
■ Contraindications:
  - Hypersensitivity to etomidate.
■ Adverse effects:
  - Transient pain at site of injection (30–80% of patients).
  - Transient m yoclonus, uncontrolled e ye mo vements (20–60% of patients).
  - Transient reduction in adrenal cor tisol production for 4–8 hour s.
  - Transient decrease in cerebral blood f ow.
■ Monitoring:
  - See Chapter 6 on monitoring during procedural sedation.
  - Signs of adrenal insuff ciency (hypotension, hyperkalemia).

- ■ Special considerations:
  - ● Etomidate is highly irritating, and pre-administration with lidocaine can be considered.
  - ● Fentanyl decreases etomidate elimination.
  - ● Premedication with fentanyl or midazolam can reduce myoclonus.

Ketamine

- ■ Ketamine is a phencyclidine derivative that works as an NMDA receptor antagonist and also has been found to bind to opioid mu receptors at higher doses.
- ■ It produces a dissociative state and is a rapid-acting anesthetic with profound analgesic properties.
- ■ It has minimal cardiovascular and respiratory depression.
- ■ Ketamine is able to preserve protective airway reflexes, which may be advantageous when fasting is not assured.
- ■ Unlike other agents that follow the typical sedation continuum, ketamine's dissociative state occurs when a certain dosage threshold is met (usually 1–1.5 mg/kg).
- ■ Safety and efficacy for procedural sedation has been well documented for children.
- ■ A much higher incidence of emergence reactions in the adult population has limited its widespread use.
- ■ Dosing:
  - ● 1–2 mg/kg IV.
- ■ Availability and administration:
  - ● 10 mg/mL – 20 mL vial containing 200 mg.
  - ● 50 mg/mL – 10 mL vial containing 500 mg.
  - ● Dilute dose to 10 mL with NS.
  - ● Direct injection IV slowly over 2–3 minutes.
- ■ Onset:
  - ● 1 minute.
- ■ Duration of action:
  - ● 5–10 minutes (note analgesic effect outlasts anesthesia effect).
- ■ Contraindications:
  - ● Hypersensitivity to ketamine.
  - ● Children <3 months of age.
  - ● Active pulmonary infection.
  - ● Cardiovascular disease (angina, heart failure, aneurysm, uncontrolled hypertension).
  - ● Glaucoma and acute globe injury.
  - ● History of airway instability, tracheal surgery/stenosis.
  - ● Psychosis.
  - ● Porphyria.
  - ● Thyroid problems.
  - ● Conditions in which an elevation in BP would be detrimental.

- ■ Adverse effects:
  - ● Hypertension and tach ycardia (more common with rapid administration).
  - ● May cause h ypotension in patients in shock that is catecholamine depleted.
  - ● Emergence reactions (~12% of adults).
  - ● Excessive salivation.
  - ● Transient lar yngospasm (0.4%).
  - ● Nausea and vomiting (~6–7%).
  - ● Respirator y depression.
- ■ Monitoring:
  - ● See Chapter 6 on monitoring during procedural sedation.
  - ● Cardiac function in patients with increased BP or decompensated cardiac function.
- ■ Special consideration:
  - ● Best suited for shor t procedures that do not require sk eletal muscle relaxation.
  - ● Pretreatment with a benzodiazepine can potentially reduce emergence reactions b y 50%.
  - ● Benzodiazepines ma y also blunt sympathomimetic effects of k etamine.
  - ● Excessive salivation ma y be treated with atropine or glycop yrrolate (or pretreated to pre vent this).
  - ● Patients experiencing transient lar yngospasm ma y need to be manually bagged (See Chapter 4 on complications of procedural sedation).
  - ● Avoid in patients who are predisposed to psychotic beha vior.
  - ● Concomitant use of CYP3A4 inhibitor s (e.g ., azole antifungals, er ythromycin, clarithromycin, verapamil, propofol) ma y increase le vels of k etamine.
  - ● Concomitant use of CYP2C9 inhibitor s (e.g ., NSAIDs) ma y increase le vels of ketamine.

Fentanyl
- ■ Fentanyl is a highly potent (100 times more potent than mor phine) synthetic opioid.
- ■ Opioids are able to pro vide reliable analgesia.
- ■ Fentanyl has fa vorable characteristics for use in procedural sedation such as rapid onset, shor t duration of action, and less cardio vascular depressive effects than the other opioids.
- ■ It also has no active metabolites, and causes much less histamine release (compared to mor phine).
- ■ Dosing:
  - ● 1–1.5 mcg/kg IV initial.
  - ● Subsequent doses of 1 mcg/kg q 3 minutes.
- ■ Availability and administration:
  - ● 50 mcg/mL – 2 mL ampoule containing 100 mcg .
  - ● 50 mcg/mL – 5 mL ampoule containing 250 mcg .

- Use undiluted.
- Direct Inject IV slowly o ver 15 seconds (a void "bolus").
- Onset:
  - 1–2 minutes.
- Duration of action:
  - 30–60 minutes.
- Contraindications:
  - Hypersensitivity to fentan yl.
  - Severe respirator y depression or acute respirator y distress (e.g., acute asthma).
  - Increased intracranial pressure.
- Adverse effects:
  - Respirator y depression/apnea.
  - Hypotension.
  - Bradycardia.
  - Muscle and glottic rigidity .
  - Nausea and vomiting .
  - Euphoria.
  - Pruritus.
- Monitoring:
  - See Chapter 6 on monitoring during procedural sedation.
- Special considerations:
  - Inject slowly o ver at least 15 seconds – Rapid IV injection ma y cause muscle rigidity, respirator y depression, or cardio vascular collapse.
  - Respirator y depression or excessive sedation can be rapidly re versed with the competitive antagonist naloxone.
  - Symptomatic h ypotension ma y not be fully re versed with naloxone use, as histamine release contributes to h ypotension.
  - Pruritus can be managed with antihistamines (e.g ., diphenhydramine).
  - Should not be used alone as fentan yl is a pure analgesic.
  - Treat nausea/vomiting with an y of the following:
    - ▶ Ondansetron 1–4 mg IV.
    - ▶ Dimenhydrinate 25–50 mg IV.

Combination Therapy

- Combining agents ma y increase the risk for adver se effects associated with each drug individually.
- When administering multiple agents, the agent that poses a greater risk of respirator y depression should be administered f rst.
- The longer acting of the two agents should be administered f rst.
- A suff cient amount of time should be allow ed to pass after administration of the f rst agent to e valuate its effect prior to administering the second agent.

Fentanyl and Midazolam

- Opioids and benzodiazepines w ere the f rst combinations used together for PSA.
- They combine the sedative, amnestic, and anxiolytic proper ties of benzodiazepines with suff cient analgesic proper ties of opioids.
- Fentanyl and midazolam ha ve been used in combination effectively for procedural sedation in the emergency depar tment (ED) for man y years.
- When combining an opioid and benzodiazepine, the risk of respirator y and cardiac depression increases.
  - Respirator y depression has been obser ved in up to 25% of patients.
- Titration of dosing to clinical effect can help minimize adver se events.
- Fentanyl should be administered f rst among the two agents as it poses a greater risk of respirator y depression. The midazolam dose can then be titrated.
- Example of titration:
  - Inject fentanyl 1 mcg/kg IV slowly o ver 15 seconds.
  - Wait 1 minute.
  - Inject midazolam 0.02 mg/kg IV slowly o ver 2 minutes.
  - Monitor sedation le vel.
  - Give additional midazolam 0.02 mg/kg q 3 minutes if needed.

Ketamine and Propofol ("K etofol")

- The theoretical benef ts of this combination include:
  - A rapid onset of sedation and analgesia with a fast reco very time.
  - The sympathomimetic proper ties of K etamine should mitigate Propofol-induced hypotension.
  - Propofol might also counteract the nausea and emergence delirium associated with K etamine.
- Ketamine pro vides a profound analgesic effect and causes a dissociative state.
- Studies looking at low-dose k etamine used in conjunction with propofol with other agents used in PSA ha ve been diff cult to assess due to their small sample size and heterogeneity in regimen and procedures.
- A prospective study demonstrated safely using a low-dose 1:1 k etofol mixture with a median single dose of 0.75 mg/kg k etamine + 0.75 mg/kg propofol (mixed in 1 syringe).
- Adverse events included transient h ypoxia, emergence delirium, and insuff cient sedation requiring adjunctive doses or medications.

Premedication Agents

Atropine

- Indications:
  - Symptomatic sinus bradycardia.
  - Inhibit salivation or secretions (often caused b y ketamine).

- ■ Dose:
    - ● Bradycardia: 0.5–1 mg q 5 minutes (maximum 2 mg).
    - ● Treatment or pretreatment for salivation/secretions: 0.4–0.6 mg .
- ■ Availability and administration:
    - ● 0.4 mg/mL – 1 mL ampoule.
    - ● 0.6 mg/mL – 1 mL ampoule.
    - ● 0.1 mg/mL – 10 mL pre-f lled syringe containing 1 mg .
    - ● Use undiluted.
    - ● Direct Inject IV rapid injection.
- ■ Onset:
    - ● IV – rapid.
- ■ Contraindications:
    - ● Hypersensitivity to atropine.
    - ● Closed (nar row) angle glaucoma.
    - ● Tachycardia.
    - ● Obstr uctive GI disease or ileus.
    - ● Myasthenia gra vis.

Glycopyrrolate
- ■ Indications:
    - ● Inhibit salivation or secretions (often caused b y ketamine).
    - ● Decrease gastric acid secretion.
- ■ Dose:
    - ● Treatment or pretreatment for salivation/secretions: 0.04 mg/kg (to a maximum of 0.1 mg).
    - ● Can be repeated e very 3 minutes as needed.
- ■ Availability and administration:
    - ● 0.2 mg/mL concentration.
- ■ Onset:
    - ● IV – 1 minute.
- ■ Contraindications:
    - ● Hypersensitivity to glycop yrrolate.

Reversal Agents

- ■ Reversal agents should not be emplo yed to speed up reco very time, and their routine use should be a voided.
- ■ The duration of action of these agents are often much shor ter than the agents that caused the sedation.
    - ● This can result in an unexpected retur n of sedation in a seemingly reco vered patient.
- ■ Use of re versal agents can also pro voke abr upt return of pain or anxiety .

- Reversal agents should be reserved for prevention of serious complications of procedural sedation agents, such as respiratory depression or cardiorespiratory complications.

Naloxone

- Naloxone is generally reserved for use to reverse narcotic-induced respiratory depression, apnea, chest wall rigidity, pruritus, and hypotension.
- Dose:
  - 0.2 mg IV.
  - May repeat q 3 minutes.
- Onset:
  - IV – 1 minute.
- Duration of action:
  - 15–30 minutes.
- Contraindications:
  - Hypersensitivity to naloxone.
- Adverse effects:
  - Narcotic withdrawal.
  - Analgesic cessation.

Flumazenil

- Flumazenil should be reserved for use to reverse serious respiratory depression (in conjunction with assessment of airway and ventilation support if necessary).
- Benzodiazepine reversal via flumazenil may precipitate seizures in some patients.
- Flumazenil may also provoke panic attacks in those with underlying panic disorder.
- Dose:
  - 0.2 mg IV slowly over 15 seconds.
  - May repeat at 1-minute interval.
  - Maximum cumulative dose: 1 mg.
- Onset:
  - <1 minute.
- Duration of action:
  - 30–45 minutes.
- Contraindications:
  - Hypersensitivity to flumazenil.
  - Use of benzodiazepines to control seizures or increased ICP.
  - Use with caution in patients who may be dependent on benzodiazepines or alcohol.
- Adverse effects:
  - Seizures.
  - Nausea/vomiting.
  - Hyperventilation.
  - Emotional liability, anxiety.
  - Sweating.

Suggested Reading

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4 Procedural Sedation and Recovery

Alexandra Stefan and Rahim Valani

Introduction

- Ensure that adequate time, personnel, equipment, and location are available for procedural sedation.
- Explain procedure to the patient, and obtain informed consent.
- Consider other options if the PSA procedure is deemed to be unsafe at present:
  - Delay procedure.
  - Scale back targeted depth and use regional anesthesia.
  - Consult anesthesia to have the procedure done in the operating room.
- See Figure 4.1 for overview.
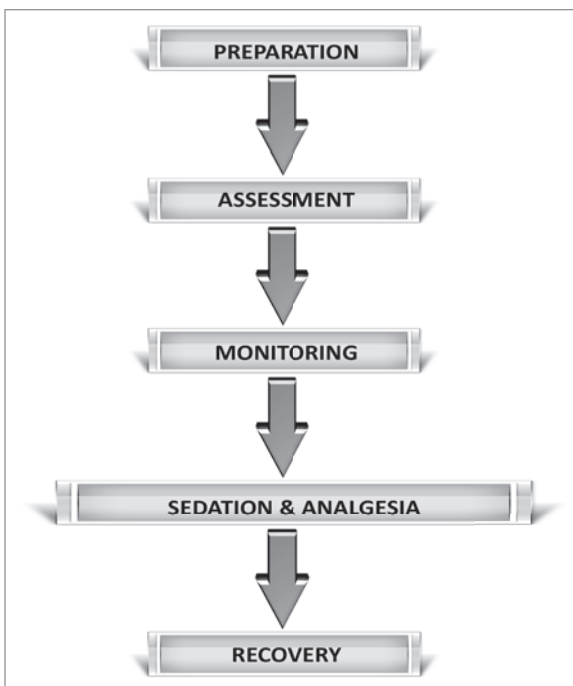
FIGURE 4.1: Overview of procedural sedation and analgesia in the emergency department.

Presedation Assessment

- Informed consent for the procedure:
  - Discuss with the patient all inter ventions that will be pro vided, including risks, benef ts, potential side effects,   and treatment alter natives.
  - Obtaining consent for PSA separate from consent for procedure.
- Document that the risks and benef ts of sedation ha ve been discussed.
- Patient assessment:
  - No evidence for routine preprocedural in vestigations.
  - Complete a thorough histor y, including anesthesia histor y and physical exam (see Table 4.1).
- Histor y should include at a minimum:
  - Prior histor y of cardiac or respirator y illness.
  - Previous adverse events/experience with sedation or general anesthesia (GA).
  - Allergies.
  - Medications.
  - Alcohol/smoking/illicit dr ug use.
  - Last oral intak e.
- Include and document: cardiopulmonar y and airway assessment; mental status and baseline vital signs.
- Complete airw ay assessment is essential to deter mine suitability for PSA in the emergency depar tment (ED) and potential complications (see Chapter 2).

TABLE 4.1: Patient assessment

History:
- Cardiac/respirator y illness?
- Previous histor y of general anesthetic?
- Medications/allergies?
- Alcohol/smoking/dr ugs?
- Last oral intak e?

Predictors of diff cult airway?
- Previous diff cult airway
- Snoring
- Sleep apnea
- Stridor
- Rheumatoid ar thritis

Physical exam:
- Vital signs
- Cardiac/respirator y exam
- Body mass index

Predictors of diff cult airway:

Head and neck	Jaw	Mouth
■ Shor t neck	■ Macro/retrognathia	■ Small opening
■ Limited extension	■ Trismus	■ Loose teeth
■ Hyoid mental distance	■ Malocclusion	■ Edentulous
■ Deformity		■ Mallampati Grade (see F igure 2.2)

TABLE 4.2: American Society of Anesthesiologists (ASA) ph ysical status classif cation

Class		Sedation risk
I	Normal, healthy	Minimal
II	Mild systemic disease without functional limitation	Low
III	Severe systemic disease with functional limitation	Intermediate
IV	Severe systemic disease, which is a constant threat to life	High
V	Moribund patient who ma y not sur vive without the procedure	Extremely high

- ■ Deter mine safety of procedural sedation with histor y and physical examination.
- ■ Patients with ASA ph ysical status class III or greater (see Table 4.2), or those with potential diff cult airway should not be done in the ED due to increased risk.

Preprocedural Fasting

- ■ Risk of aspiration with PSA is less lik ely than in GA.
- ■ Overall risk of 1:3,500 for aspiration and 1:125,000 for subsequent mor tality.
- ■ Prospective obser vational study identif ed no difference in adver se e vents between patients classif ed by preprocedural fasting status.
- ■ No study to date has deter mined a necessar y fasting period before initiation of PSA.
- ■ Guidelines and consensus statements recommend var ying fasting periods based on specif c substances, that is, solids ver sus f uids ver sus clear f uids, but no suff cient e vidence to deter mine absolute recommendations.
- ■ Table 4.3 provides a summar y of the guidelines of the American Society of Anesthesiologists, which are a safe and conser vative approach.
- ■ Clear liquids ingested up to 2 hour s do not adver sely affect gastric pH and volume, therefore pose minimal risk.
  - ● Examples of clear f uids include: w ater, fruit juice, soda, tea, and coffee.
  - ● By contrast, par ticulate matter causes pulmonar y damage on aspiration and is thus considered higher risk.

TABLE 4.3: Summar y of the American Society of Anesthesiologists preprocedure fasting guidelines

Ingested material	Minimum fasting period (hr)
Clear liquids	2
Breast milk	4
Infant for mula	6
Full f uids	6
Light meal	6

Note: These recommendations ha ve been de veloped for health y patients undergoing elective surgical procedures.

- Routine administration of antacids does not decrease risk of complications.
- Sedation depth affects likelihood of maintenance of airway reflexes and is thus linked to aspiration risk.
- There is no specific evidence that sedation length in ED affects risk of aspiration.
- Consider timing and depth of PSA in absence of a fasting period.
  - May want to consider delaying the procedure if inadequate fasting period.
- Four-step assessment in the ED:
  - Assess patient risk of aspiration: *standard versus higher risk.*
    - Higher risk includes:
      - Potential for difficult or prolonged ventilation.
      - Extremes of age (>70 years or <6 months).
      - Higher ASA classification.
      - Conditions predisposing to GERD (bowel obstruction, hernia).
  - Assess timing and nature of last oral intake.
  - Assess the urgency of procedure.
- **Emergent:** cardioversion of life-threatening dysrhythmia, reduction of markedly angulated fracture/dislocation, vascular compromise, intractable pain.
- **Urgent:** care of dirty wounds, human bites, hip dislocations, lumbar puncture.
- **Semiurgent:** care of clean wounds, shoulder reduction, foreign body removal.
- **Nonurgent/elective:** ingrown toenail.
  - Determine depth and length of PSA (see Chapter 1 on sedation continuum).

Personnel Requirements Needed for PSA in the ED

- Recognize that the feared complications of PSA include hemodynamic and airway emergencies and thus adequate support must be present.
- PSA at both moderate and deep levels shown to be safe/effective when performed by ED physicians.
- Individual performing moderate/deep sedation must be trained to perform the following:
  - Administer pharmacological agents to desired level of sedation.
  - Monitor patients to maintain desired level of sedation.
  - Manage complications observed during this process.
- No literature evidence on specific number of people that must be present.
  - Current recommendations are to have a support person dedicated to patient monitoring during moderate/deep sedation.
  - Having a separate person doing the procedure.

Equipment and Supplies Necessary for PSA

- Consider the possible complications of PSA when selecting necessary equipment.
  - Allergic reactions.
  - Respiratory arrest.
  - Cardiac arrest.

- Suppor tive equipment includes:
  - Oxygen.
  - Suction.
  - IV access needed for the procedure and maintained until reco very (this ma y not be necessar y if PSA pro vided by other routes, such as IM in children).
  - If no IV used, equipment and a qualif ed per son able to establish IV access should be a vailable throughout the procedure.
  - Medications including re versal agents (see Chapter 3).
  - Advanced life-suppor t medications and equipments (including BVM and intubation equipment). See Chapter 2, Airway Assessment and Management for Procedural Sedation, for detailed description.

Monitoring (See Chapter 7)

- Vital signs should be documented including pre- and postprocedure status.
- Monitoring should be targeted to detect ear ly signs of h ypotension, bradycardia, apnea, airway obstr uction, or hypoventilation.
- Monitor and document (see Chapter 5):
  - Level of a wareness as a guide to depth of sedation.
  - Vital signs.

Airway Equipment

- See Chapter 2.

Procedural Sedation

- A variety of phar macological agents can be used to obtain desired le vel of sedation (see Chapter 3, Phar macology of Procedural Sedation).
- The choice of agent depends on:
  - Type of procedure.
  - Desired depth of sedation.
  - Patient factor s.
  - Physician comfor t with par ticular agent.
- Slow titration of dr ugs to the desired effect is essential to minimize complications.
  - Rapid administration is more lik ely to produce h ypotension and respirator y depression.
- Route of administration:
  - Intravenous (IV) administration of sedative analgesic agents increases the likelihood of satisfactor y.
  - IV access should be maintained until patient is no longer at risk of complications.
  - If not using IV route for initiation of sedation, consider obtaining IV access after initial sedation.

- Consider and adjust for longer time required for absor ption if IM/PO medications before increasing dose b y those routes.

Recovery Postsedation: Criteria for Safe Discharge

- Need to monitor adver se e vents including h ypoxemia, apnea, airway obstr uction, cardiovascular e vents, and emesis.
  - These e vents are generally related to moderate–deep sedation (rate <5%).
- Decreased stimulation, delay ed dr ug absor ption, and slow elimination place patients at risk during the reco very period.
- The reco very area should ha ve access to resuscitation equipment.
- It is the responsibility of the ph ysician administering the sedation to ensure that these patients are safe for discharge.
- Duration and frequency of monitoring is individualized; it will depend on:
  - Level of sedation.
  - Overall patient condition.
  - The nature of the inter vention.
- Patients should be monitored until the y retur n to baseline mental status and are no longer at risk for cardiorespirator y depression; follow oxygenation, ventilation, and vital sign.
- Guidelines for discharge:
  - The patient is aler t and oriented (presedation baseline le vel).
  - Stable vital signs.
  - Tolerating f uids.
  - Patient is ambulator y.
  - Airway is patent, with protective ref exes intact.
  - Suff cient time postadministration of IV medications.
  - Discharge patients accompanied b y a responsible adult and counsel regarding procedure complications.
  - Instr uctions given to a void any activity that requires coordination or judgment.
  - Written instr uction on when to retur n to the ED for an y complications.

Summary

- Procedural sedation is safe when perfor med by skilled per sonnel.
- Strict adherence to presedation assessment, adequate monitoring , and postsedation discharge criteria can decrease risk of complications.

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Monitoring During Procedural Sedation

Sharon Ramagnano

Introduction

- All patients undergoing procedural sedation in the emergency depar tment (ED) should ha ve continuous monitoring until ready for discharge.
  - Continuous monitoring of vital signs and clinical presentation of patient constitute a minimum requirement for patient safety monitoring .
- Vital signs should include:
  - Heart rate
  - Respirator y rate
  - Blood pressure
  - Oxygen saturation
- Capnography is more sensitive for detecting inadequate ventilation/apnea and should now be a standard practice.

Pre-procedural Preparation

- Staff should ensure that the following equipment is set up in wor king condition prior to proceeding with the procedure:
  - Oxygen
  - Suction
  - Resuscitation equipment, that is, airway/intubation tra y
  - Hemodynamic monitoring—includes BP , pulse, cardiac monitor, oximetr y, and capnography
  - Sedative re versal agents on hand
  - Intravenous (IV) access established and maintained
- The equipment listed in Table 5.1 should be readily a vailable during sedation, while ensuring adequate monitoring of the patient.

Monitoring During the Procedure

- Appropriate monitoring during procedural sedation is cr ucial (see F igure 5.1).
- Monitoring should be targeted to detect ear ly signs of h ypotension, bradycardia, apnea, airway obstr uction, or hypoventilation.

TABLE 5.1: Equipment necessary during the procedural sedation

Intubation tray	Nasal airway
Various ETT tube sizes	Laryngeal masks
Laryngoscope	Lidocaine spray
Stylette	Emergency crichotomy kit
Tape	Defibrillator
Syringes	Cardiac monitoring
Masks	Continual blood pressure, pulse and oxygen saturation
Ambu bag	Capnography
Suction	Intravenous maintenance
Oxygen	Intravenous fuids
Oral airway	Blood gas syringes

- Regular review and documentation of vital signs is necessary for safe practice.
  - Consensus guidelines recommend recording every 5 minutes once sedation is established.
  - May decrease frequency once patient is awake and alert to the point of discharge.

Clinical Assessment
- No monitoring device replaces clinical assessment of the sedated patient.
- The level of awareness should be used as a guide to depth of sedation.
  - Check response to verbal or painful stimuli, eyelash response, etc. (see Chapter 1).
- Continuous visual inspection of chest-wall motion and air movement is especially important to confrm adequate ventilation.
- Signs of inadequate ventilation should be sought, including:
  - Inadequate or infrequent respirations.
  - Apnea.
  - Cyanosis.
  - Stridor.
  - Snoring.
  - Other signs of upper airway obstruction.
- Monitoring of *respiratory status* has two components: ventilation and oxygenation.
  - Ventilation status can be monitored by clinical observation and auscultation or by capnography, while oxygenation is followed by pulse oximetry.
  - See Chapter 6 on complications related to inadequate oxygenation versus ventilation.

Supplemental Oxygen

■ Supplemental oxygen is almost univer sally applied during procedural sedation in the ED, despite a lack of clinical e vidence to suppor t its use.

■ It is thought to decrease the incidence and se verity of hypoxemia due to airw ay complications.

● It may also dela y the detection of respirator y depression b y pulse oximetr y. Therefore, clinical obser vation of respirator y activity is k ey.

● Supplemental oxygen should also be considered when capnograph y is present.

Pulse Oximetr y

■ Pulse oximetr y provides rapid, noninvasive and continuous estimation of ar terial oxygen saturation (SaO $_2$).

■ Its use is univer sally recommended during procedural sedation, and studies ha ve shown an excellent cor relation betw een ar terial hemoglobin oxygen saturation (SaO$_2$) and pulse oximetr y oxygen saturation (SpO $_2$).

■ Although the mechanism of pulse oximetr y is complex, a basic under standing allows the recognition of potential limitations.

● Transmission oximetr y is based on differences in the optical transmission of oxygenated and deoxygenated hemoglobin (Hb).

■ Advantages:

● Easy to use.

● Straightforw ard inter pretation of w aveform (see F igure 5.1).
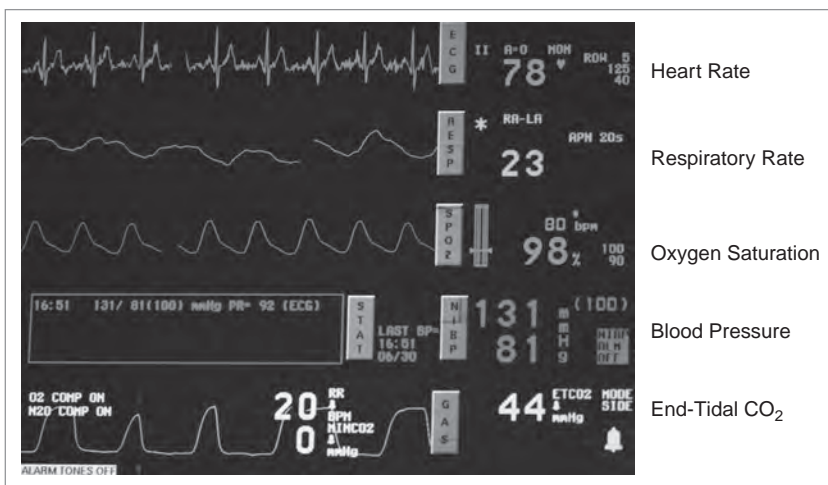
● No risk to patient.

● Inexpensive.

FIGURE 5.1: Standard protocol during procedural sedation should include complete cardiorespirator y and end-tidal CO $_2$ monitoring.

- ■ Limitations:
  - ● Falsely *low* SpO_2.
    - ▶ Hypoperfusion of the extremity .
      - ○ Hypothermia, decreased cardiac output, vasoconstriction secondar y to vasopressor use.
    - ▶ Movement ar tifact (e.g ., shivering) especially during h ypoperfusion.
    - ▶ Incorrect sensor application.
    - ▶ Highly calloused skin.
    - ▶ Artif cial nails or nail polish.
    - ▶ Presence of abnor mal hemoglobin or cer tain toxins bound to hemoglobin.
      - ○ Methemoglobinemia (reading usually around 85% despite tr ue low or high PaO_2).
  - ● Falsely *elevated* SpO_2
    - ▶ Carbon monoxide (CO) toxicity .
    - ▶ Cyanide poisoning .
      - ○ Reduces oxygen extraction from ar terial blood.

Capnography

- ■ Capnograph y is the measurement of carbon dioxide (CO_2) in each breath of the respiratory cycle.
- ■ The capnograph displa ys a w aveform of CO_2 measured in mm Hg and the value of CO_2 at the end of exhalation, known as the end-tidal CO_2 ($ETCO_2$).
- ■ There is a ver y close cor relation, in health y patients, between $ETCO_2$ and ar terial CO_2 par tial pressure (P aCO_2).
  - ● $ETCO_2$ is ~2–5 mm Hg less than P aCO_2.
- ■ Calorimetric monitor s use color scales to estimate the range of $ETCO_2$ using pH-sensitive f lter paper.

TABLE 5.2: Capnographic changes with common ventilator y patter ns during procedural sedation

Ventilatory patter n	Capnographic changes
Periodic breathing	Normal patter n punctuated b y apneic pauses May occur with deep sedation
Hypoventilation	High amplitude wide capnogram
Hyperventilation	Low amplitude nar row capnogram
Apnea	Loss of capnogram
Bronchospasm	Curved ascending phase and upsloping of the alveolar plateau

The paper changes from pur ple (<4 mm Hg CO_2) to tan (4 to 15 mm Hg CO_2) to yellow (>20 mm Hg CO_2).

- This is often used to conf rm tube placement following endotracheal intubation.

■ Quantitative monitor s aspirate samples of gas through a small catheter and are incorporated into nasal prongs or face masks to facilitate CO_2 monitoring during procedural sedation.

- Limitations include secretion plugging , air leaks, which ma y dilute the sample, and a 2–3-second dela y in response times.

■ During procedural sedation, an increase in $ETCO_2$ may be the f rst sign of inadequate ventilation and has been found to precede a fall in pulse oximetr y or clinical signs of respirator y depression.

- Identif ers of h ypoventilation using capnograph y include:
  - ▶ An $ETCO_2$ >50 mm Hg
  - ▶ An absent w aveform
  - ▶ An increase of 10 mm Hg compared to baseline

■ It is unclear whether ear lier detection of h ypercapnia without h ypoxemia alter s clinical outcome.

■ Many conditions affect ventilation perfusion ratios in the lung and can therefore widen the $PaCO_2$–$ETCO_2$ gradient.

- This results in an inaccurate representation of P aCO$_2$ by $ETCO_2$.
  - ▶ Some causes include PE, asthma, cardiac ar rest, hypovolemia, and COPD .
  - ▶ Although it ma y not adequately represent P aCO$_2$ in critically ill patients, it is still valuable to detect trends and sudden airw ay events.
  - ▶ For procedural sedation perfor med in the ED , patients are typically health y and the $ETCO_2$ should accurately represent the P aCO$_2$.

Recording of Monitored P arameters

■ Monitored parameter s should be documented at a minimum:
- Before the beginning of the procedure.
- After administration of sedative/analgesic agents.
- On completion of the procedure.
- During initial reco very.
- At the time of discharge.

■ If recording is perfor med automatically, de vice alar ms should be set to aler t the care team to critical changes in patient status based on the nor mal preset conf guration capabilities.
- All alar ms should be set to *on.*

Staff Availability for P atient Monitoring and Safety

■ A designated individual, other than the practitioner perfor ming the procedure, must be present to monitor the patient throughout the procedural sedation and for after care.

- In the ED, a minimum of two staff (nurse, physician, or respiratory therapist) must be available to monitor the patient and assist with the procedure.
- Members of the team must be trained in the recognition of complications associated with IV sedation, specifically:
  - Drug complications/interactions
  - Know the role of pharmacological antagonists
  - Skills in airway management
  - Venipuncture skills
  - Arrhythmia recognition

Post-procedure Monitoring

- Patients should be observed until they are no longer at increased risk for cardiorespiratory depression.
- Vital signs and respiratory function should be monitored at regular intervals until patients are suitable for discharge.
- Discharge according to specified criteria with verbal and written discharge instructions (see Chapter 4).
- Staff must ensure that a complete discharge instruction sheet or information is provided to the patient and family.
- The discharge instructions should include at a minimum:
  - Procedure performed
  - Medications received pre-, during, and post-procedure
  - Any prescriptions required post-discharge
  - Any adverse events that occurred during the procedure
  - After care for the presenting problem that the patient arrived with
  - When to return to the family physician for follow-up and when to return to the ED if required
  - Instructions regarding dressing changes/cast care/suture removal, etc.
  - Tetanus status (if given ensure that this information is provided for the patient to alert their family physician for their medical records)

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6

Adverse Events and Complications of Procedural Sedation

Angela Stone and Mark Freedman

Overview

- The complication rate of procedural sedation is estimated to be <1% in the hospitals and specialties routinely practicing procedural sedation and analgesia.
  - Adverse event rate is estimated at 2%–3% in pediatric patients.
  - The most common complication is respiratory depression and airway obstruction.
  - Medication errors.
- Many of the complications related to PSA can be prevented by:
  - Appropriate monitoring and recognition of respiratory depression or arrest.
  - Adequate monitoring.
  - Ensuring the correct dose of medications (avoid drug calculation error).
  - Careful titration of medications.
  - Appropriate patient assessment.
  - Avoiding drug–drug interactions.
  - Personnel present who are trained in airway management and resuscitation.
  - Management of potential complications requires early recognition. Therefore, clinical observation and appropriate monitoring during the procedure is essential in order to identify and treat possible adverse effects (see Chapter 5).
- Most complications during procedural sedation can be managed noninvasively.

Propofol

Respiratory Depression

- Propofol causes dose-related apnea and respiratory depression.
- No clinically significant events reported in studies with PSA, with most cases managed by bag-valve mask ventilation.
- Recognition of respiratory depression is essential.
  - Managed with jaw thrust, repositioning of the airway, or assisted ventilation for a brief period.
  - The need for intubation after the use of propofol in the emergency department (ED) has not been reported.

- Propofol is repor ted to ha ve the low est rate of respirator y depression when compared with methohexital, fentanyl/midazolam, and etomidate.

Hypotension

- Propofol commonly results in a drop in blood pressure,   which is often transient.
- More commonly seen with rapid bolus,   for example patients who are h ypovolemic, or ha ve poor cardio vascular reser ve.
- When compared with etomidate, propofol has been found to induce greater hypotension, although this is transient and of unknown clinical signif   cance.
- Can be treated with T rendelenburg positioning , f uid bolus, or shor t-acting vasopressor (phen ylephrine).

Injection P ain

- Injection pain has been repor ted in up to 70% of patients.
- Warning the patient often alle viates anxiety when pain is felt with the injection.
- Lidocaine, either mixed with propofol (1 mL of 1% lidocaine in 19 mL propofol) or given with a r ubber tour niquet in place 30–120 s before injection (0.5 mL/kg) has been found to pre vent injection pain.

Ketamine

Laryngospasm

- Rare but potentially life-threatening side complication.
- Unrelated to age, sex, under lying medical condition, or dose.
- Associated with procedures that stimulate h yperactive gag ref ex through direct instr umentation or secretions appear to represent a higher risk.
- Risk factor s include:
  - Upper airw ay infection.
  - Age betw een 3 and 12 months.
  - Active pulmonar y disease including asthma.
- Laryngospasm often manifests as h ypoxia and decreased chest w all mo vement not responsive to maneuver s to open the upper airw ay.
- Consider bag-valve mask and positive pressure ventilation as f   rst line approach – breaks most cases of lar yngospasm.
  - In se vere circumstances ma y require urgent paralysis and intubation.

Hypersalivation

- Ketamine stimulates salivar y and tracheobronchial secretions.
- In children, coadminister with atropine (0.01 mg/kg; min 0.1 mg   , max 0.5 mg) or glycopyrrolate (0.005 mg/kg; max 0.25 mg).
- The use of atropine appear s to be unnecessar y in adult patients in the ED .

Nausea and V omiting

- Vomiting occur s in 0%–9% of patients receiving k etamine.
- In children, the incidence appear s to be age-related, with a higher risk in patients aged 5 year s and older.

- Vomiting most often occur s during the late reco very phase when the patient is awake and aler t.
- Protective airw ay ref exes are maintained during dissociative anesthesia with ketamine, therefore, signif cant aspiration is extremely rare.
  - Ketamine is therefore prefer red over other agents for urgent or emergent procedures when fasting is not assured.
- Delayed vomiting ma y occur after discharge, and patients should be infor med about this (see Chapter 4 on discharge instr uctions).

Clonus and Hyper tonicity

- Ketamine does not produce muscle relaxation, and random pur poseless movements, hypertonicity, and clonus are not unusual.
- These mo vements are usually unrelated to painful stimuli and do not need to be treated.

Sympathomimetic Effects

- Ketamine inhibits the reuptak e of catecholamines, resulting in mild to moderate increases in blood pressure, hear t rate, and myocardial oxygen consumption.
- Potential risk to patients with coronar y ar tery disease.
  - Actual risk is unknown due to limited experiences in this population of patients.

Emergence Phenomenon

- Ketamine is associated with a hallucinator y "emergence reaction" known as emergence delirium.
- It occur s in up to 30% of adult patients and is much less common in children.
- Risk factor s:
  - Age >16 year s.
  - Female sex.
  - Rapid IV administration.
  - Use of large doses.
- Hallucinations can be pleasurable or more commonly frightening lik e nightmares.
  - Use of benzodiazepines concur rently with k etamine is belie ved to blunt, but not entirely eliminate this reaction in adults.
  - In pediatric patients, unpleasant reco very reactions are uncommon and are typically mild. There is no e vidence to suggest the use of proph ylactic administration of benzodiazepines in this population.
  - Consider their use only when treating unpleasant emergency reactions, should they occur.

Ketamine and Propofol (K etofol)

- Ketofol is a mixture of both k etamine and propofol used to achie ve procedural sedation.

- The reasoning behind using both agents is that the y are theoretically synergistic.
  - The sympathomimetic proper ties of k etamine should mitigate propofol-induced hypotension.
  - Propofol might also counteract the nausea and emergence delerium associated with k etamine.
- The most common dosing mixture is a 1:1 combination of both dr ugs at doses of 0.50–0.75 mg/kg (10 mg/mL).
  - Propofol and k etamine can be used simultaneously yet from separate syringes thus allowing their independent titration.
- The pitfall of this combination is that k etamine lacks the dose-dependent progressive effect typical of propofol.
- The dose used in most studies (0.75 mg/kg) is considered subdissociative, not achieving its characteristic trancelik e state, and acts instead lik e a simple analgesic.
  - Dissociative doses of k etamine are usually betw een 1.0 and 1.5 mg/kg IV .
- Studies ha ve shown that the combination is effective and appear s safe for procedural sedation.
- Adverse e vents included transient h ypoxia, emergence delirium, and insuff cient sedation requiring adjunctive doses or medications.

Etomidate

Respiratory Depression
- A brief period of apnea ma y occur following etomidate administration.
  - Risk factor s include:
    - ▶ Rapid administration (<80 sec).
    - ▶ Higher doses (>0.2 mg/kg).
    - ▶ Older patients (>55 year s of age).
- At typical procedural sedation doses, respirator y depression is rare and if it does occur is usually transient and mild.
- No studies ha ve reported the need to intubate a patient after its use in procedural sedation.

Myoclonus
- Myoclonus is a characteristic and common side effect of etomidate occur ring anywhere from 0%–21% of cases.
- It usually lasts <1 minute and can be minor and focal or se vere and associated with full body rigidity , tonic clonic activity , hypoventilation, and h ypoxia.
- Myoclonus-induced respirator y depression has not been repor ted with ED use.

Nausea and V omiting
- The incidence of nausea and vomiting with etomidate ranges from 0%–5%.
- The risk of nausea and vomiting appear s to be dose-related and is generally not a problem with doses used for procedural sedation.
- There ha ve been no documented cases of aspiration.

■ Randomized controlled trial comparing propofol to etomidate found that etomidate induced vomiting more frequently than that seen with propofol use during procedural sedation.

Adrenal Suppression

■ Etomidate is known to cause adrenal suppression, even following a single dose.
- The mechanism is via inhibition of 11-beta-hydroxylase activity.

■ Unlikely of any clinical significance in the setting of PSA.

Pain on Injection

■ Pain on injection is most likely caused by the propylene glycol solvent.
■ Methods to decrease discomfort include:
- Using larger veins.
- Mixing 1 cc of 1% lidocaine with each 10 mL of etomidate.
- Flushing with saline.
- Pretreating with fentanyl.

Midazolam

Respiratory Depression

■ Midazolam causes central respiratory depression through decreased sensitivity to carbon dioxide.
■ It is dose-dependent, peaking at 3 minutes after IV administration.
■ More pronounced in:
- Elderly patients.
- Patients with COPD.
- When coadministered with other respiratory depressants.

■ Consider reduced dosage in patients with COPD and in patients older than 60 years.

Prolonged Sedation

■ Most commonly occurs in elderly patients.
■ Reversal of the sedating effects of benzodiazepines is achieved using flumazenil, which is benzodiazepine antagonist with an onset of action of 1–2 minutes.
- It is given as an IV titration starting with a bolus of 0.5 mg in adults followed by 0.1 mg until a desired response is achieved.
- Can precipitate seizures in patients who are benzodiazepine dependent. Consider noninvasive support of ventilation until patient's respiratory status improves.

Paradoxical Agitation

■ Paradoxical reactions to benzodiazepines are occasionally seen in young children.
■ Reactions include agitation, combativeness, and inconsolability.
■ Treatment is supportive with airway and blood pressure management.

- Flumazenil has been shown to be quick acting and effective in abating paradoxical reactions in children.
  - Starting dose of 0.01 mg/kg (up to 0.2 mg).
  - It may be repeated to achieve the desired level of consciousness to a maximum dose of 1.0 mg or 0.05 mg/kg.
- Haloperidol may be a safe alternative to fumazenil but is less commonly used due to its risk of extrapyramidal side effects.

Fentanyl

Respiratory Depression
- Fentanyl reduces the responsiveness of the brain-stem respiratory center to carbon dioxide.
- Maximal respiratory depression occurs 5 minutes after intravenous administration.
- The magnitude is dose dependent and is intensified by the coadministration of other respiratory depressants, particularly midazolam.

Muscular and Glottic Rigidity
- Occurs only with high doses of fentanyl and has never been reported with its use in the ED for PSA.
- Management includes reversal with naloxone or paralysis with succinylcholine.

Seizures
- Also associated with high doses and has not been reported in ED patients.

Pruritus
- Mild facial pruritus is common.
- There is little or no histamine release with fentanyl use, therefore anything more serious (urticaria or anaphylactoid reactions) are seldom seen.

Midazolam and Fentanyl
- The key to avoiding complications is the titration to a desired effect.
- The combination of drugs may accentuate the potential side effects of each.
- Because opioids pose a greater risk of respiratory depression, it has been suggested that when using in combination with a benzodiazepine the opioid should be given first and the benzodiazepine titrated to effect.

Summary
- Adverse event rates for PSA in the ED are low, and most can be managed noninvasively.
- The most common complication of PSA is respiratory depression.
- Different medications used for PSA have adverse reactions and complications that can be managed.

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7 Pediatric Procedural Sedation

Savithiri Ratnapalan

Pediatric Procedural Sedation and Analgesia

- Procedural sedation is the technique of administering a single drug or a combination of drugs with sedative, analgesic, or dissociative properties to induce a state that allows children to:
  - Tolerate painful or unpleasant procedures
  - Stay still for some non-painful procedures
- The most common reasons for pediatric procedural sedation are:
  - Orthopedics (most common, and includes fracture reductions, dislocations, and immobilization/splinting)
  - Laceration repair
  - Foreign body removal
  - Lumbar puncture
  - CT scans

Challenges in Children

- Assessing the level of sedation in a child may be harder than in adults.
  - A child can easily move from moderate sedation to general anesthesia with minimal increases in sedative agents.
- Children come in various sizes and need age-appropriate equipment.
  - Ensure age- and size-appropriate equipment, including:
    - Blood pressure cuffs for different ages.
    - Doppler blood pressure monitors.
    - Pulse oximetry probes.
    - Intravenous canula.
    - Airway equipment – masks, resuscitation bags, and intubation equipment.
- Children range from neonates to teenagers, and need specific dose calculations.
  - Obtain accurate weight of patient of appropriate dosing.
  - Drug doses should be calculated and checked by at least two healthcare professionals.
  - Most common cause of drug error is dose inaccuracies.

- Children should come with a parent or guardian.
  - Consent from the parent and assent from child (as appropriate) should be obtained before the procedure.
- Children need simple explanations and clear instr uctions.
- Children need a calm monitored en vironment for procedural sedation and for recovery.

Pediatric Characteristics That Need Consideration

- Young infants ha ve relatively less oxygen reser ve (greater oxygen consumption).
  - Hypoxemia occur s more rapidly .
  - Appropriate size bag and mask ventilation should be a vailable.
- Pediatric patient sizes ma y var y from 2.5–100 kg . The "pediatric crash car t" is bigger and should ha ve age-appropriate equipment.
- Airway sizes ma y var y unpredictably among pediatric patients of same age and w eight.
- At times, three different-sized endotracheal tubes should be a vailable for the patients of the same age (the calculated size and a size smaller and larger).
  - The appropriate uncuffed endo-tracheal-tube size ma y be deter mined by the following for mula (age in year s):
    - ▶ 4 + (1/4) (age)
    - ▶ Subtract 0.5 for the appropriate size cuffed ETT
      - ○ For example, for a 4-year-old child: uncuffed ETT size = 4 + (1/4)4 = 5
      - ○ So, cuffed ETT size = 5 − 0.5 = 4.5.
  - The appropriate depth of ETT inser tion can be approximated b y:
    - ▶ Over 1 year of age:
      - ○ Oral: 13 + (1/2)age
      - ○ Nasal: 15 + (1/2)age
    - ▶ Infants (w eight in kg):
      - ○ Oral: 8 + (1/2)(w eight)
      - ○ Nasal: 9 + (1/2)(w eight)
- Small children ha ve small airw ays.
  - Since resistance to air f ow is in versely propor tional to the four th power of the radius of the airw ay, 1 mm of concentric edema in a ne wborn trachea (radius ~2 mm) increases resistance about 16 times.
  - The presence of upper respirator y tract infection should be assessed prior to sedation, adjuvant agents to reduce secretions ma y have to be used, and vigilance in airw ay monitoring obser ved.
- There are anatomic differences betw een the infant and the adult upper airw ay:
  - Infant lar ynx:
    - ▶ More superior in neck.
    - ▶ Epiglottis shor ter, angled more o ver glottis.
    - ▶ Vocal cords slanted: anterior commissure more inferior .
    - ▶ Larynx cone-shaped: nar rowest at subglottic cricoid ring .
    - ▶ Softer, more pliable: ma y be gently f exed or rotated anterior ly.

- Infant tongue is relatively larger .
- Infant head is relatively larger : naturally f exed in supine position.
- Caution in intubation: extension of head ma y result in tracheal extubation, while f exion may lead to main stem intubation.
- Young infants (less than approximately 2–3 months) are obligate nose breather s.
- Infants and young children ha ve limited hepatic glycogen storage and are more prone to hypoglycemia when fasted for prolonged periods.
  - Consider star ting an intravenous maintenance f uid such as D5% N Saline if the child has fasted or is expected to fast for a long period.
- Gastroesophageal ref ux is common in infants. W atch out for vomiting post sedation.

Policies and Protocols for a Safe P ediatric Sedation Within the ED

- Trained personnel in pediatric sedation and airw ay management.
- Accurate weight measurement, drug dose calculations, and a protocol mandating two persons sign-off on dr ugs.
- Age-appropriate equipment to monitor and manage potential adver se effects of sedation in children.
- Preprinted orders and monitoring sheets should be a requirement for an y department performing PSA.
- Documentation of consent.

Exclusion Criteria and Contraindications for ED P ediatric Sedation

Patient Criteria

- ASA classif cation >II.
- History of known airw ay problems: snoring , obstr uctive sleep apnea, large tonsils or adenoids, tracheomalacia, tracheostenosis, congenital abnor malities involving the airway (e.g., Down syndrome, Pierre Robin syndrome, Treacher Collins syndrome, and Crouzon's disease).
- Cardiovascular disease: repaired or unrepaired congenital hear t disease and congestive hear t failure.
- Severe neurologic disease, severe hypotonia, and evidence of increased intracranial pressure.
- Severe renal or liver disease.
- Severe gastroesophageal ref ux and previous esophageal surger y or injur y.
- Patients at increased risk of pulmonar y aspiration of gastric contents (e.g ., full stomach).
- Potential neck injur y, limitations in mo ving neck/opening mouth/ja w movement.
- History of known sedation failure.
- Home oxygen therap y/or home ventilation.
- Sickle cell disease.
- Baseline vital signs indicate SaO_2 <95% in room air.

Procedure Criteria
■ Sedation time greater than 30 minutes

Provider/Facilities Criteria
■ Lack of personnel experienced in pediatric sedation and airway management
■ Lack of resuscitation drugs or age-appropriate equipment
■ Lack of monitoring facilities during recovery
■ Lack of an experienced physician to do the procedure and/or supervise the procedure

Commonly Used Drugs and Combinations
■ Various single and combination drugs have been used with good safety profiles.
■ See Table 7.1.

Choice of Drugs
■ Sedative drugs used and the depth of sedation needed depend on the patient characteristics and procedures.
  ● Patient characteristics: age, fasting status, comorbid conditions, maturity, level of cooperation and level of anxiety.
  ● Procedures: painless or painful, severity of pain, urgency of the procedure, level of complexity, how much motion control is needed, and length of the procedure.

Adverse Reactions and Complication
■ See Chapter 6.
■ Type 1 IgE-mediated allergic reaction to procedural sedation is unusual.

TABLE 7.1: Sample medications that can be used for procedural sedation in pediatric patients

Level of sedation	Drug choices
Mild	Midazolam PO, Intranasal
	Nitrous oxide
Moderate	Midazolam IV
	Etomidate IV
	Midazolam IV and Fentanyl IV $\pm$ nitrous oxide
Moderate to deep sedation	Propofol
Dissociative sedation	Ketamine

- More common reactions are:
    - ▶ Histamine release (Morphine, Meperidine).
    - ▶ Nasal pruritus (Fentanyl).
    - ▶ Paradoxical reactions (Benzodiazepines, Barbiturates).
    - ▶ Emergence reactions to Ketamine.
    - ▶ Laryngospasm reported with both Ketamine and Propofol.
- Children experience higher rates of respiratory depression and hypoxia than adults.
- Adverse events in children occur more frequently in:
    - Younger children.
    - Sedation performed in non-hospital-based facilities.
    - With the use of three or more drugs or drugs with long half-lives.

Commonly Used Pharmacologic Agents for Procedural Sedation and Analgesia in Children

Midazolam

- Short-acting benzodiazepine.
- Provides sedation, anxiolysis, and amnesia (*no* analgesic effects).
- Minimal hemodynamic effects (mild hypotension with compensatory tachycardia).
- Dose- and infusion-dependent respiratory depression and apnea especially with opioids (Table 7.2).

Nitrous Oxide

- Provides anxiolysis, amnesia, and mild analgesia.
- Noninvasive, rapid onset, short duration of action.
- Generally used in concentrations of 20%–50% mixed with oxygen.
- Free flow nitrous oxide versus demand valve (Entonox ®– 50% nitrous/ 50% oxygen.
- Scavenging device essential.
- Indications: minor procedures, for example, intravenous access, laceration repair, burn debridement, and as an adjunct for more painful procedures, for example, fracture reduction.

TABLE 7.2: Midazolam dosing in pediatric patients

Route of administration	Dose
Oral	Weight <20 kg, 0.5–0.75 mg/kg
	Weight >20 kg, 0.3–0.5 mg/kg (max 20 mg)
Intranasal	–0.1–0.3 mg/kg (max 1 mL)
Intravenous	0.05–0.15 mg/kg titrate slowly to effect

- Avoid in patients with pneumothorax,   bowel obstr uction, intracranial injur y, and cardiovascular compromise.

Ketamine

- Dissociative agent that produces a trance-lik e cataleptic state.
    - Profound analgesia.
    - Amnesia.
    - Retention of protective airw ay ref exes, spontaneous respirations,   and cardiopulmonar y stability.
- *Indications:* Procedures requiring profound analgesia and immobility , for example, fracture reduction and complex laceration repair .
- Most commonly used agent for procedural sedation in children.
- Signif cant side-effect prof les including:
    - Increased intracranial pressure.
    - Hypersalivation.
    - Tachycardia.
    - Hyper tension.
    - Nystagmus and diplopia.
    - Muscle h ypertonicity.
    - Emesis.
    - Transient apnea or respirator y depression.
    - Laryngospasm.
- Relative contraindications:
    - Head injur y associated with altered mental status.
    - Loss of consciousness or emesis.
    - Cardiovascular disease.
    - Glaucoma or acute globe injur y.
    - Psychosis.
    - Thyroid disorder.
    - Age <3 months.
    - Procedures in volving stimulation of posterior phar ynx and active pulmonar y infection or disease (URI).
- Dosing for k etamine is given in   Table 7.3 .

TABLE 7.3: Dosing of k etamine for procedural sedation in pediatric patients [a]

	Intravenous	Intramuscular
Dosage	1–1.5 mg/kg	4 mg/kg
Onset of action	1–2 min	5–10 min
Duration of action	10–15 min	15–30 min

[a]May be used in conjunction with adjuvant agents including atropine or glycop   yrrolate and/or midazolam.

Drug-Specifc Considerations in Pediatrics

- ■ Any sedative but specifcally chloral hydrate:
  - ● Prolonged sedation and airway obstruction.
- ■ Midazolam:
  - ● Paradoxical reactions in 20% of children with oral midazolam.
  - ● Intravenous midazolam can be titrated and this reaction is not common.
- ■ Ketamine:
  - ● Hypersalivation. Young children may need adjuvant drugs such as Atropine or Glycopyrrolate.
- ■ Propofol:
  - ● Hypotension may not be associated with tachycardia and could be missed if not measured.

Pearls to Avoid Adverse Events in Pediatric Patients

- ■ Choose your patient:
  - ● Take a good history.
  - ● Identify those with upper respiratory infections, signifcant underlying physical illnesses, obstructive airway disease, psychosis, and drug allergy.
  - ● Avoid sedating any child with severe systemic disease (ASA ≥3).
- ■ Know the drugs you use:
  - ● Accurate weight of the child.
  - ● Dosage should not only be weight-dependent, but also age- and disease-dependent.
  - ● Know your upper limits for each drug and its potential side effects.
  - ● Draw your own drugs and label them.
- ■ Monitor patients carefully:
  - ● Continuous cardiac monitoring and pulse oximetry plus independent observer who checks vital signs.
  - ● Continue monitoring until patient returns to baseline.
  - ● Be cautious of increased sedation after painful stimulus is removed and vomiting during recovery.
- ■ Fully prepare for any complications before starting the procedure.
  - ● Age-appropriate resuscitative equipment, reversal agents, and skilled personnel in advanced pediatric life support should be immediately available.

Steps for Procedural Sedation and Analgesia

- ■ Refer to Chapter 4.

Summary

- ■ Customize the sedation technique for the patient and the procedure to be performed.

- Ensure appropriate per sonnel and interactive monitoring .
- Potential for adver se outcomes ma y increase when three or more sedating medications are used. Knowledge of each dr ug's time of onset, peak response, and duration of action is essential.
- Drugs with longer duration of action require longer periods of obser vation. This concept is impor tant for infants and toddler s transpor ted in car safety seats.

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Pain Management and Regional Anesthesia

8 Introduction to Pain Management

Jeff Gadsden

Epidemiology of Pain Presentation

- Pain is the most common presenting complaint to the emergency department (ED).
  - In France, pain-related issues account for 67% of the presenting complaints.
- Pain is initially assessed 90% of the time but reassessments occur less frequently (48% on discharge).
  - For those assessed on discharge, 27% still had pain (8% severe).
- Delay for pain management is related to ED volumes, lack of triage nurses (especially in small departments), and initial pain intensity.
- Pain intensity follow-up is important to assess to determine analgesic effectiveness.
- Patient satisfaction is related to earlier effective pain management.
- Quality indicators for pain management include
  - Time to first dose of analgesic in all painful conditions.
  - Percentage of patients with documented pain assessment.
- The mean *expectation* for time to analgesic administration for ED patients is 23 minutes, compared with actual mean time to analgesic administration of 78 minutes.

Concepts in Pain Management

- Multimodal pain management is better than any one agent alone.
  - Adding acetaminophen to ibuprofen, for example, is better than either agent alone.
  - As well, both can be given to complement narcotic therapy.
- Dosing intervals should be according to the earliest allowable time to avoid loss of pain control and optimize pharmacokinetics (see Chapter 10).
- Once nausea is controlled, oral options for analgesics should be explored to facilitate early conversion.
- For people presenting with acute on chronic pain, management should take into consideration their current daily opiate consumption.
- "Muscle relaxants," such as Robaxacet (acetaminophen/methocarbamol) and Flexeril (cyclobenzaprine), have little evidence to support their use.

- Neuropathic pain is a challenging issue.
- May need to consider the use of nontraditional analgesics, for example, pregabalin, amitriptyline, or gabapentin.
- Consider regional nerve blocks.
  - Intercostal nerve blocks have been shown to be beneficial in rib fractures.
  - See Chapter 12.
- Prior studies have suggested that protocol-driven analgesia can be more effective than provider initiated analgesia.
- Protocolized analgesic administration facilitates a greater percentage of patients being treated in a timely fashion and should be part of every ED with appropriate supporting infrastructure.

Anatomy and Physiology of Pain

Acute Pain

- Acute pain is defined as "pain of recent onset and probable limited duration. It usually has an identifiable temporal and causal relationship to injury or disease."
- Also, it can be thought of as "physiological pain" or "useful pain."
- In contrast, chronic pain typically lasts beyond time of healing and frequently has no identifiable cause.
- Acute and chronic pain in fact may represent a continuum rather than two separate entities.

Peripheral Receptors and Afferent Fibers

- Axons of primary afferent nerves end in skin, subcutaneous tissue, periosteum, joints, muscles, and viscera.
- There are *no* specialized pain receptors: free nerve endings (nociceptors) are sensitive to noxious physical stimuli.
  - These respond to chemical, mechanical, or thermal energy that threatens the integrity of the tissue.
- There are two main types of nociceptive afferents:
  - Aδ-fiber mechanothermal nociceptors:
    - ▶ Thinly myelinated (therefore, faster than unmyelinated C-fibers)
    - ▶ Responds to heat, cold, and pressure
    - ▶ Provides "first pain" information critical for protective withdrawal reflex
  - C-fiber polymodal nociceptors:
    - ▶ Unmyelinated (slow)
    - ▶ Respond to a broad range of physical (heat, cold, pressure) and chemical stimuli
    - ▶ Provides "second pain" that is classically burning in nature
- Tissue damage (e.g., trauma, infection, inflammation, or ischemia) disrupts cell structure and promotes the release of an "inflammatory soup" of chemical mediators that activate and/or sensitize nearby C-fibers (e.g., protons, bradykinin, histamine, prostaglandins, serotonin, and substance P).

■ Most tissues have both types of nociceptors; exceptions include liver, brain, and lung tissue which have no afferent pain fibers.

Visceral Referred Pain

■ Occasionally, axons from visceral afferent nerve converge onto the same second-order neuron as somatic afferents.

■ The brain is unable to distinguish between the two inputs and projects the sensation to the somatic structure.

● Examples: myocardial ischemia felt as aching pain in left shoulder; gallbladder distention felt as pain in right shoulder.

Pain Transmission

■ Mechanical or chemical signals are converted first to action potentials in the periphery.

■ Conducted along first-order neurons to the dorsal horn of the spinal cord.

■ Synapse with second-order neurons and ascend to thalamus via the spinothalamic and spinoreticular tracts.

■ Project to a number of areas including

● Periaqueductal gray matter, an area rich in opioid receptors

● The somatosensory cortex

● The frontal lobes

● The hypothalamus

■ This variety of destinations is congruent with the idea of pain as a complicated process that involves sensory, emotional, and social aspects.

Central Sensitization (Windup)

■ Following trauma in the periphery, the dorsal horn exhibits enhanced responsiveness to further stimuli, a phenomenon termed "windup."

■ Caused by continued barrage of nociceptive input to dorsal horn neurons and a gradual increase in spinal cord neuronal activity.

■ This results in an increased responsiveness to normally innocuous mechanical stimuli (allodynia) and a zone of hyperalgesia in uninjured tissue surrounding the site of injury.

■ Loco-regional techniques (e.g., nerve blocks) that interrupt the barrage of afferent input effectively prevent windup if provided early and for the appropriate duration (see Chapter 12).

■ Windup can also be partially modulated by the use of NMDA-receptor antagonists (i.e., ketamine).

Modulation of Pain

■ The experience of pain can be modulated by cortical events such as high levels of stress, distraction, or intense excitement (e.g., in battlefield or sporting injuries).

■ The release of endogenous opioids (endorphins) is thought to be responsible for these occurrences.

- Similarly, anxiety or depression can enhance the experience of pain for a given stimulus and may be related to reduced discharge of descending inhibitory pathways.
- The gate theory of pain modulation (Melzack and Wall) states that the transmission of impulses into the dorsal horn is controlled by a spinal gating system.
- This is controlled by the relative activity in large or small fibers.
- Activity in large fibers tends to inhibit transmission, whereas small fiber activity stimulates transmission (i.e., opens the gate).
- The coincident flooding of the dorsal horn with cutaneous touch and pressure sensation may close the gate for smaller nociceptive input (Figure 8.1).
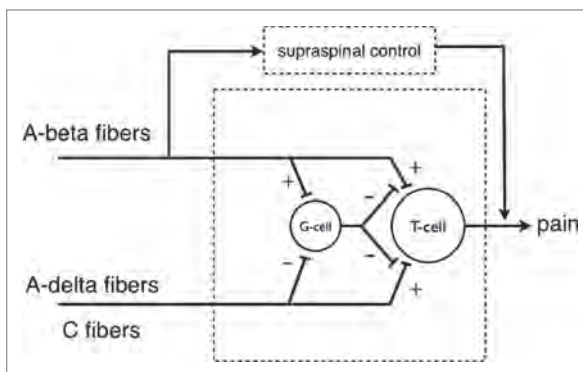
FIGURE 8.1: The gate control theory of pain. The gate cell (G-cell) is capable of modulating the activity of the afferent activity to the transmitter cell (T -cell). Input from large, nonnociceptive fibers (e.g., A-beta fibers carrying touch and pressure impulses) activates the gate, which prevents further input from nociceptive afferents. There are also cortical and subcortical inputs that influence the gate mechanism.

Progression of Acute to Chronic Pain

- The continuum of acute to chronic pain:
  - Chronic pain is increasingly being referred to as "persistent pain. "
  - Patients with chronic pain often relate onset of their pain to an acute injury.
  - Chronic pain is common after surgical procedures (see Table 8.1).
  - Increasing recognition that nervous system exhibits remarkable plasticity – consistent afferent nociceptive input can result in permanent neurophysiologic change.
- Preventive analgesia.
  - Defined as the persistence of analgesic treatment efficacy beyond its expected duration.
  - Refers to the minimizing of central sensitization and windup by the provision of quality analgesia that is continued for as long as the sensitizing stimulus persists.

TABLE 8.1: Incidence of chronic pain after surgery

Type of operation	Incidence of chronic pain (%)
Amputation	30–85
Thoracotomy	5–65
Mastectomy	11–57
Inguinal hernia	5–63
Cesarean section	6–55
Coronary artery bypass	30–50

- Most effectively achieved by the use of multimodal analgesia.
- The following have all been shown in meta-analyses to be effective in minimizing the incidence of persistent pain:
  ▶ Gabapentin
  ▶ Local anesthetics (i.e., nerve blocks, epidural analgesia)
  ▶ Nonsteroidal anti-inflammatory drugs
  ▶ NMDA antagonists (e.g., ketamine, dextromethorphan)
- The combination of multiple agents allows for complementary modes of action while reducing the dosage (and therefore side effects) of any one individual agent.

Adverse Physiological Effects of Acute Pain

Acute Pain and the Injury Response
- Acute pain activates the neurohumoral and immune response to injury.
- This is an adaptive survival response that, if prolonged, can have adverse effects on outcome.
- The hormonal/metabolic response includes increased cortisol, catecholamines and glucagon, and decrease in insulin sensitivity.
- As pain is one of the major triggers of the injury response, and the duration of the response is related to the duration of the stimulus, effective pain relief can have a significant impact on these adverse consequences.

Adverse Physiologic Effects (See Figure 8.2)
- Hyperglycemia is proportional to the extent of the injury response.
- Injury leads to upregulation of membrane glucose transport proteins glut-1, 2, and 3 which are located in brain, endothelium, liver, and some blood cells.
- Results in cellular glucose overload, glycosylation of proteins such as immunoglobulins, and production of oxygen free radicals.
- Lipolysis results in increased free fatty acids which depress myocardial contractility and increase myocardial oxygen consumption.
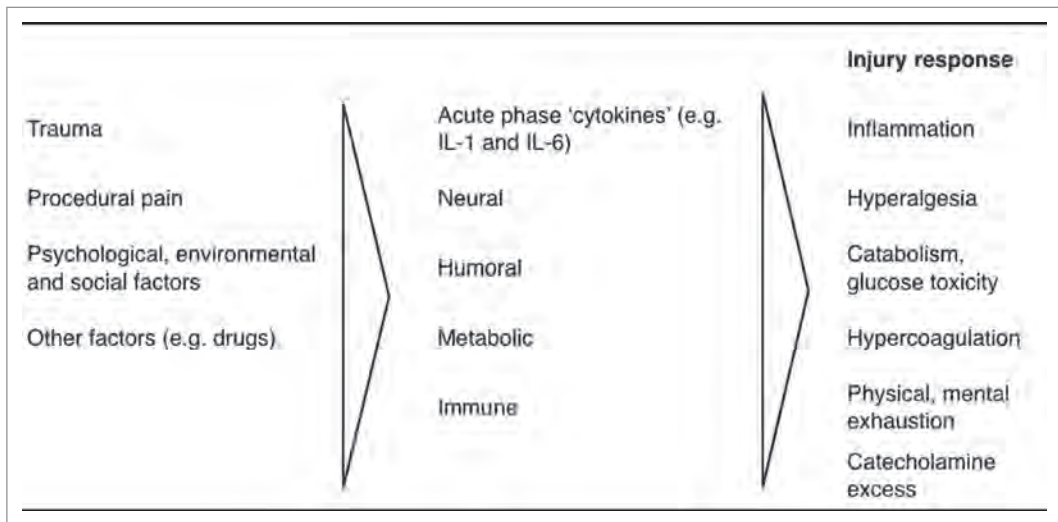
FIGURE 8.2: The injury response.

- Accelerated protein breakdown and amino acid oxidation lead to a negative nitrogen balance and poor wound healing, impaired immune function, and diminished muscle strength.
- Pain activates sympathetic efferent nerves causing increases in heart rate, inotropy, and blood pressure and increasing risk for myocardial ischemia.

Pharmacogenomics and Acute Pain
- Susceptibility to pain conditions appears to have genetic variability.
- Correlation of gene expression with a drug's efficacy of toxicity can lead to optimization of analgesic therapy.

Loss of Pain Sensation
- Hereditary syndromes exist in association with loss of pain sensation.
- Example: "channelopathy-associated insensitivity to pain," caused by a variant in the voltage-gated sodium channel. These individuals cannot propagate action potentials on peripheral nerves and are unable to feel pain.
- Other familial peripheral and autonomic neuropathies have been described such as hereditary sensory and autonomic neuropathy type IV (HSAN-4), a severe autosomal recessive disease characterized by childhood onset of insensitivity to pain and anhidrosis.

Reduced Sensitivity to Pain
- Associated with variants in genes encoding the mu-opioid receptor, catechol-*O*-methyl-transferase (COMT), and transient receptor potential (TRPV$_1$).
- These patients frequently report increased pain scores and require greater than usual doses of opioid analgesics.

Drug Metabolism

- Drug metabolizing enzymes are a major target for identifying associations between an individual's genetic profle and drug response.
- The CYP2D6 gene is highly polymorphic and infuences the metabolism of codeine, oxycodone, and tramadol.
  - For example, CYP2D6 genotypes predicting ultra-rapid metabolism resulted in about 50% higher plasma concentrations of morphine and its glucuronides following oral codeine compared with the normal population.
  - Morphine toxicity and death have been reported in a breastfed neonate whose mother was an ultra-rapid metabolizer of codeine.
  - Similarly, 1%–3% of Caucasians are poor metabolizers of NSAIDs such as ibuprofen and naproxen; these patients are at risk for increased adverse effects from elevated blood levels of the parent drug.

Summary

- Pain is a common presentation to the ED.
- Adequate assessment and management of pain is an important part of patient care.

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9 Chronic Pain Management in the ED

Rahim Valani

Introduction

- Chronic pain is def ned by the Inter national Association for the Study of P ain as pain without the biological value that has per sisted be yond the nor mal healing time.
    - Usually def ned as pain lasting for more than 6 months.
- It is estimated that 35% of the US population suffer from chronic pain, which costs $40 billion per year .
- Approximately 21% of people with chronic pain are dissatisf ed with their cur rent pain management.
- Stages of progression from acute to chronic pain:
    - Stage 1 – acute phase.
        - ▶ Initial psychological distress that is expected – fear and anxiety .
    - Stage 2 – exacerbation of psychological problems.
        - ▶ When pain lasts o ver 2–4 months.
        - ▶ Patient's response includes anger , distress, and somatization.
        - ▶ Social, f nancial, and en vironment factor s pla y an impor tant role in how the patient copes with this phase.
    - Stage 3 – acceptance of the "sick role. "
        - ▶ Physical deconditioning occur s in all three stages.
- The most common chronic pain conditions in general practice include:
    - Irritable bow el syndrome.
    - Osteoar thritis.
    - Lower back pain.
    - Chronic pelvic pain.
    - Migraine headaches/tension headaches.
    - Fibromyalgia.
- It is impor tant to complete a thorough pain histor y for e very patient who presents to the ED , including:
    - Pain characteristics – onset, location, quality, radiation, se verity, temporal prof le, and alle viating factor s.

- Effects on daily living – Can the y cope with daily activities of living such as bathing, dressing themselves, doing the laundr y, and cooking?
- Effects on their family/friends.
- Effects on their emplo yment – Are they able to continue to wor k? What are the limitations? Can the y be accommodated for limited duties?
- Changes in recreational activities.
- Psychological impact of pain – mood, sexual function, sleep, etc.
■ Physical examinations should include:
  - Inspecting for :
    ▶ Signs of inf ammation.
    ▶ Trophic changes.
    ▶ Deformities.
  - Range of motion of affected joints and muscles.
  - Palpation for tender ness, crepitus, and warmth.
■ Issues sur rounding the adequate treatment of chronic pain:
  - Poor knowledge b y health care professionals.
  - Inadequate pain assessment.
  - Not recognizing multidimensional cause of pain.
  - Poor documentation.
  - Misconceptions about the use of opioids.
  - Patient's reluctance to repor t pain.
■ Opioid used for non-cancer and chronic pain.
  - Opioids continue to be one of the most prescribed medications for chronic pain.
  - Three largest increases o ver the 10 year s (1997–2006) include the use of:
    ▶ Methadone (1,177% increase).
    ▶ Oxycodone (732%).
    ▶ Fentanyl (479%).
■ Consider the following steps in prescribing opioids for chronic pain:
  - Comprehensive initial e valuation.
  - Establish need for opioid, either as a supplemental medication or lack of response to prior medication.
  - Assess risk of star ting and using opioid medication.
  - Establish treatment goals with patient.
  - Obtain infor med consent and agreement with patient.
  - Initial dose adjustment phase in f rst 3 months, followed by frequent reevaluation to titrate dose accordingly .
  - Outcomes – dose adjustments, steady state, or discontinuing medications.
■ Consequences of chronic pain:
  - Inability to perfor m nor mal activities of daily living .
  - Feeling of hopelessness.
  - Fear of activities that can exacerbate the pain.

Myofascial Pain

- Estimated to affect up to 54% of individuals.
- Regional painful muscle/soft tissue condition related to specific trigger points and related pain.
  - Also referred to as myofascial trigger point pain.
- Hallmark is localized trigger points with focal tenderness.
  - Usually tight bands typically located in the center of the rigid muscle.
  - Pain on sustained compression over the tender point.
  - Local twitch response within the band of muscle on plucking palpation across the fibers.
- Immediate response to injection of local anesthetic is characteristic.
- Classified as primary or secondary.
  - Primary – from a specific cause with ongoing pain with continued use of that muscle.
  - Secondary – pain referred from a primary site due to mechanical stress or inflammation.
- Common posterior myofascial trigger points are located at:
  - Levator scapulae.
  - Trapezius.
  - Rhomboids.
  - Quadratus lumborum.
  - Piriformis.
- Etiology:
  - Posttraumatic.
  - Chronic repetitive injuries.
  - Muscle wasting – malignancy and stroke.
  - Muscle ischemia – arterial obstruction.
  - Environmental – heat- or cold-related injuries.
- Differential diagnosis includes:
  - Non-myofascial trigger point (fibromyalgia):
    - Myofascial trigger point patients have fewer systemic complaints compared to patients with fibromyalgia.
  - Musculoskeletal diseases – tendinitis, arthritis, and occupational myalgia.
  - Systemic diseases – arthritides (rheumatic, psoriatic), infection.
  - Psychiatric.
  - Drug reaction.
- Chronic myofascial pain can persist after the acute event due to:
  - Dorsal horn neural plasticity.
  - Ongoing self-perpetuating ischemic changes.
  - Development of circuits from the trigger point to the spinal cord.
  - Prognosis is directly related to the duration of symptoms.

- Treatment options:
  - Any pharmacotherapy must be done in conjunction with physical therapy for best results.
  - Primary treatment is to restore functional activity through performance and stretching exercises.
    - ▶ Spray with cool vaporizer followed by muscle stretches.
    - ▶ Deep pressure soft tissue massage.
    - ▶ Post-isometric relaxation. (The muscle is in a stretched position with isometric contraction against minimal resistance. This is followed by relaxation and a gentle stretch.)
    - ▶ Reciprocal inhibition.
  - Non-steroidal anti-inflammatory drugs (NSAIDs).
  - Injection of local anesthetic to trigger point site.
  - Other adjuncts: ultrasound, iontophoresis, TENS, and thermotherapy.
  - Botulinum toxin A injection.

Fibromyalgia

- Chronic pain condition that involves muscles and myofascial tissues, accompanied by trigger points.
- Prevalence is estimated at 1%–3% in the general population and is more common in females aged between 20 and 50 years.
- Pathophysiology is poorly understood.
- American College of Rheumatology definition:
  - Generalized pain involving three or more sites for 3 months or longer.
  - Exclude other conditions.
  - Reproducible pain over 11 of the 18 fibromyalgia tender points.
- Treatment should incorporate:
  - Patient education.
  - Other medications to control non-pain symptoms such as depression.
  - Aggressive use of cognitive-behavioral approaches.
  - Pharmacological treatments include:
    - ▶ Analgesics – NSAIDs and opioids.
    - ▶ Antidepressants:
      - ○ 5HT and norepinephrine are the main neurotransmitters involved.
      - ○ TCAs and SNRIs (Venlafaxine, Duloxetine, Milnacipran) seem to be most effective in treating fibromyalgia.
    - ▶ Alpha2-delta ligands:
      - ○ Gabapentin and Pregabalin limit neuronal excitation and enhance inhibition.
    - ▶ Growth hormone:
      - ○ Evidence that about one-third of patients with fibromyalgia have a functional growth hormone deficiency.

- Nonpharmacological treatments:
  - ▶ Physical therapy.
  - ▶ Movement and exercises.
  - ▶ Cognitive behavioral treatments.
  - ▶ Complementary/alternative treatment.

Complex Regional Pain Syndrome

- ■ Incidence varies from 5.5–26.2 per 100,000.
- ■ It is more common in women and with increasing age (peaks in the fifth to seventh decade).
- ■ Categorized as type I (no nerve lesion identified) and type II.
  - ● Type I is more common.
  - ● Type I was formerly known as "reflex sympathetic dystrophy."
- ■ Pain out of proportion to the inciting event is characteristic.
- ■ Fractures are the most common precipitating event, and usually in the upper limb.
  - ● Increased pressure and complaints of cast tightness during cast immobilization are predictors of complex regional pain syndrome (CRPS).
- ■ Symptoms are varied:
  - ● Inflammation.
  - ● Impaired motor function.
  - ● Trophic changes.
- ■ Exact etiology is complex, and includes:
  - ● Autonomic (sympathetic) nervous system hyperactivity:
    - ▶ Increased sweating.
    - ▶ Trophic changes.
    - ▶ Extreme vasoconstriction.
    - ▶ Coldness in the limb.
  - ● Somatic nervous system:
    - ▶ Pain and sensory disturbances.
  - ● Inflammation.
  - ● Hypoxia:
    - ▶ Decreased capillary oxygenation.
    - ▶ Elevated lactate levels.
    - ▶ Extreme vasoconstriction.
  - ● Psychological factors.
- ■ Treatment options:
  - ● Glucocorticoids.
  - ● NSAIDs – usually primary therapy.
  - ● GABA agonists – Baclofen is an option for dystonia associated with CRPS.
  - ● Bisphosphonates – benefit on pain, swelling, and mobility.

Whiplash-Associated Disorder

- Caused by an acceleration–deceleration mechanism of energy transfer of the neck.
- Incidence is estimated at 70–328 per 100,000 in Canada and the USA.
- Most patients improve within the f rst 3 months.
- It is the most common cause of neck pain associated with musculo-ligamentous conditions.
- The Quebec Task Force grades of whiplash-associated disorder (W AD):
  - Grade 0 – no neck symptoms or physical signs.
  - Grade 1 – neck pain, stiffness, or tenderness; no physical signs.
  - Grade 2 – neck symptoms and musculoskeletal signs (decreased range of motion and point tenderness).
  - Grade 3 – neck symptoms and neurological signs (decreased or absent deep tendon ref exes, muscle weakness, sensory def cits).
  - Grade 4 – neck symptoms with fracture or dislocation.
- Chronic pain develops in 15% to 20% of whiplash injuries.
- Chronic pain and disability are complex presentations due to injury-related factors (physical) and psychosocial factors (event-related factors).
- The Quebec Task Force def ned late whiplash syndrome as presence of pain, restriction of motion, or other symptoms at 6 months or more after the injury.
  - Hinders activities of daily living .
    - ▶ Subcategories include:
      - ○ Local cervical syndrome.
      - ○ Cervicogenic headaches.
      - ○ Cervicogenic vertigo.
      - ○ Cervico-brachial syndrome.
      - ○ Behavioral manifestations.
- Predictors of poor prognosis include:
  - High baseline neck pain intensity .
  - Presence of headache.
  - High WAD Grade.
  - Lack of secondary education.
- Characteristics of collision (rear-ended, side impact, frontal), gender, and age are not clear indicators of poor prognosis.
- Assessment and management based on W AD-Plus Model:
  - Grade of WAD.
  - Time since injury.
  - Pain experience.
  - Chronicity factors – depends on socioeconomic status, prior medical status, symptom severity, psychosocial factors, use of health care, and compensatory factors.
- Abnormal central pain processing has implications for management of myofascial trigger points and tight bands.

- Manual therapy and exercise are effective noninvasive interventions for neck pain.
- Interventional treatments include:
- Steroid injection in epidural space, trigger points, or facet joints:
    - Botulinum toxin injection to muscle trigger points.
    - Percutaneous radiofrequency treatment.
    - Chiropractic care can improve cervical range of motion and pain in the management of WAD.

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Pharmacology of Pain Management

- Numerous classes of medications are available for the treatment of pain, and the treatment can be tailored depending on the nature and severity of pain (i.e., acute pain, chronic pain, and neuropathic pain).
- An effective choice of medication for treating pain should include consideration of:
  - Allergies or sensitivities to medications.
  - Recent or previous history of pain medication used (what worked, what has not worked, and why).
  - Formulations of medications available (i.e., oral liquids, oral tablets, sustained-release oral tablets, injectables, and topicals).
  - Appropriate dosing, dose conversions, and relative potency.
- Ineffective management of acute pain can not only lead to patient/caregiver dissatisfaction, but also may potentially lead to progression to chronic pain.
- Classes of medications used for the management of pain include:
  - Non-opioid analgesics.
  - Opioid analgesics.
  - Co-analgesics/adjunctive agents.
  - Topical anesthetics.

Non-opioid Medications

- Non-opioid medications are used for the treatment of mild to moderate pain.
- The most common non-opioids are acetaminophen and nonsteroidal anti-inflammatory drugs (NSAIDs).

Acetaminophen

- Acetaminophen is a non-opioid analgesic that also has antipyretic properties, used for mild to moderate pain, and for fever.
- Acetaminophen inhibits synthesis of prostaglandins in the central nervous system (CNS), and inhibits peripheral pain signal neurotransmission.
- As an antipyretic, acetaminophen acts on the heat-regulating center in the hypothalamus, inducing vasodilation and sweating to disperse body heat.

- Routes: oral (PO) or rectal (PR).
- Dosing:
  - PO: 10–15 mg/kg/dose q4h PRN.
  - PR: 10–20 mg/kg/dose q4h PRN.
- Onset: PO – 15 minutes, PR – 30 minutes.
- Peak effect: PO – 30–60 minutes, PR – 2 hours.
- Duration: PO/PR – 4–6 hours.
- Pharmacokinetics:
  - Well absorbed orally, slower absorption and effect rectally.
  - Metabolized in liver via glucuronidation, and by P450 enzyme CYP2E1 to metabolite NAPQI (hepatotoxic in acute overdose).
  - Excreted by the kidneys.
- Contraindications:
  - Liver dysfunction, hypersensitivity to acetaminophen.
- Significant drug interactions:
  - Phenytoin, barbiturates (i.e., phenobarbital), and carbamazepine may decrease the levels and effect of acetaminophen.
  - Minor increase in bleeding risk with warfarin.
- Adverse effects:
  - Nausea/vomiting (may take with food).
  - Rash.
  - Hepatotoxicity with chronic use or acute overdose.
  - Renal injury with chronic use.
- Monitoring:
  - Pain and/or fever reduction, liver enzymes and liver function tests (LFTs) with chronic use.
- Acetaminophen special considerations and pearls:
  - Lack of action on prostaglandins in the peripheral nervous system (PNS) likely related to lack of anti-inflammatory action.
  - No gastric irritation/ulceration compared to NSAIDs.
  - Extended-release 650-mg tablets also available, but usually not used for acute pain in children.
  - Caution different concentrations of oral drops (80 mg/mL) versus oral liquid suspension (32 mg/mL).
  - Be aware that acetaminophen is a component of multiple over-the-counter and prescription products, limit acetaminophen content from all sources to ≤75 mg/kg/day.
  - Ensure that the foil is removed from suppositories prior to insertion; half doses should be achieved by cutting suppositories lengthwise symmetrically.
  - PR absorption is slower and less complete than oral absorption (see dosing above).
  - Oral liquid may be instilled rectally (limit volume to 80 mg/1 mL).

Nonsteroidal Anti-inflammatory Drugs

- NSAIDs are used for treatment of mild to moderate pain.
- NSAIDs inhibit the peripheral synthesis of inflammatory prostaglandins, and have analgesic, anti-inflammatory, and anti-pyretic properties.
- Typical NSAIDs used in the pediatric population are ibuprofen, naproxen, and ketorolac.
- Routes: PO, PR; IM/IV (ketorolac).
- Dosing:
  - Ibuprofen: PO – 5–10 mg/kg/dose PO q6–8h.
  - Naproxen: PO – 5–10 mg/kg/dose PR BID PRN.
    - ▶ Rectal dosing: less than 50 kg–250 mg/dose PR, greater than 50 kg–500 mg/dose PR (maximum 1,000 mg/day).
  - Ketorolac: IV/IM/PO 0.5 mg/kg/dose q6h PRN (see special considerations below; maximum 15–30 mg/dose).
- Administer PO with food to decrease stomach irritation.
- Onset: PO – 30–60 minutes, PR – 2 hours, IM/IV – 30 minutes.
- Peak effect: PO – 2–4 hours, PR – 4 hours, IM/IV – 2–3 hours.
- Duration – PO/PR/IM/IV – 6–8 hours.
- Pharmacokinetics:
  - Well absorbed orally, slower absorption rectally.
  - Metabolized by liver, eliminated by kidneys.
- Contraindications:
  - Hypersensitivity to NSAIDs.
  - Severe asthma triggered by NSAID use.
  - Acute active bleeding.
  - Elevated bleeding risk.
- Significant drug interactions:
  - Anti-platelet agents, anticoagulants, steroids (ASA, clopidogrel, warfarin, prednisone, etc. – additive bleeding risk).
  - Anti-hypertensive medications (ACE inhibitors, B-blockers – reduced anti-hypertensive effect).
  - ACE inhibitors, diuretics (increased risk of hyperkalemia).
  - NSAIDs may increase the levels of aminoglycosides (i.e., gentamicin), cyclosporine, digoxin, haloperidol, lithium, methotrexate, quinolones (i.e., ciprofloxacin), and vancomycin.
  - Levels and effects of NSAIDs may be increased by tricyclic antidepressants (i.e., amitriptyline), selective serotonin reuptake inhibitors (SSRIs) (i.e., fluoxetine), serotonin/norepinephrine reuptake inhibitors (SNRIs) (i.e., venlafaxine), and probenecid.
- Adverse effects:
  - GI irritation/nausea/vomiting/heartburn (take with food).
  - Rash and pruritus.
  - Dizziness.
  - Fluid retention.

- ◼ Monitoring:
  - ● Reduction of pain/fe ver/inf ammation.
  - ● Blood pressure/edema for cardiac patients.
  - ● Liver enzymes/LFTs, CBC, and renal function for chronic use.
- ◼ NSAID special considerations and pear ls:
  - ● Relative increasing potency of NSAIDs generally ibuprofen → naproxen → ketorolac.
  - ● Naproxen helpful in rheumatic or inf ammatory disorder s.
  - ● Ketorolac to be used parenterally IV or IM if patient not able to tolerate other PO NSAIDs, and to be used shor t term only (i.e., <5 days) due to GI ir ritation/bleeding risk. The IM route is also painful.
  - ● May transition to PO k etorolac if needed, but prefer red to use ibuprofen as PO agent.

Opioid Medications

Codeine

- ◼ Codeine is an opioid analgesic inhibiting neurotransmission in the ascending pain pathway, and an antitussive (in low er doses), acting centrally on the medulla.
- ◼ Codeine is a *pro-drug*, which does not ha ve signif cant phar macological activity until metabolized b y cytochrome P450 CYP2D6 enzymes in the liver to the active metabolite, mor phine (~10% metabolized to active mor phine).
- ◼ Codeine should be used with caution for pediatric pain management due to variable phar macokinetics and unpredictable eff cacy and toxicity .
- ◼ Routes of administration: PO , IM, subcutaneous (Subcut).
- ◼ Dosing:
  - ● PO analgesia: 0.5–1 mg/kg/dose q4–6h PRN.
  - ● PO antitussive: 0.15–0.3 mg/kg/dose q4–6h PRN.
  - ● IM/Subcut: 0.5–1 mg/kg/dose q4–6h PRN.
  - ● Maximum analgesic dose: 1.5 mg/kg/dose, or 60 mg/dose.
  - ● Maximum antitussive dose: 20 mg/dose.
  - ● Adjust dose for renal impair ment.
  - ● Sustained-release products used for chronic pain and dosed based on average daily requirements of immediate-release preparations, usually dosed q12h.
- ◼ Product availability and administration:
  - ● PO liquid: codeine phosphate – 5 mg/mL.
  - ● PO *immediate-release* tablets: codeine phosphate – 15 and 30 mg .
  - ● PO *sustained-release* tablets (for chronic pain): codeine contin (codeine sulfate trih ydrate) – 50, 100, 150, and 200 mg .
  - ● IM/Subcut: 30 mg/mL ampoule q4–6h PRN.
  - ● IM or Subcut no dilution required, do not administer IV .

- Onset: PO – 30–60 minutes; IM/Subcut – 10–30 minutes.
- Peak effect: PO – 60–90 minutes (4 hour s for sustained release);
  IM/Subcut – 30–60 minutes.
- Duration: PO and IM/Subcut – 4–6 hour s (12 hr for sustained-release PO tablets).
- Pharmacokinetics:
  - Readily absorbed orally , ~60% bioavailability (see Section IV: PO Con version, Table 10.3).
  - Low protein binding .
  - Metabolized b y CYP2D6 in liver into mor phine-active metabolite.
  - Eliminated primarily b y the kidne ys.
- Contraindications:
  - Hypersensitivity/allerg y to codeine or mor phine.
- Signif cant dr ug interactions:
  - Other CNS depressant/respirator y depressant medications (i.e., other opioids and benzodiazepines).
  - Quinidine (inhibits CYP2D6, decreased effect of codeine).
- Adverse effects:
  - Excessive sedation.
  - Respirator y depression.
  - Bradycardia.
  - Hypotension.
  - Dizziness.
  - Drowsiness.
  - Constipation.
  - Urinar y retention.
  - GI upset (ma y take with food).
- Monitoring:
  - HR, RR, BP, reduction in pain score, level of sedation and constipation.
- Codeine special considerations and pear ls:
  - Respirator y depression can occur e ven at therapeutic doses (not necessarily only at toxic doses).
  - Codeine does not necessarily ha ve less adver se effects than stronger opioids.
  - Genetic polymor phism – variable phar macokinetics and unpredictability .
    - ▶ P450 CYP2D6 enzyme that metabolizes codeine to mor phine for therapeutic effect is highly polymor phic.
    - ▶ Up to 28% of Nor th African, Ethiopian, or Saudi Arabian patients; 10% of Caucasian patients; 3% of African American patients, and 1% of Chinese, Japanese, or Hispanic patients are "ultra-metabolizer s" – meaning more than expected codeine is metabolized to mor phine (excessive sedation, respirator y depression, or e ven death ma y result).
    - ▶ "Slow or poor metabolizer s," on the other hand, may experience ineffective pain relief with codeine.

- Genetic testing for CYP2D6 polymor phism is not readily commercially available, thus it is preferential to prescribe oral or parenteral mor phine directly (rather than producing mor phine indirectly through codeine metabolism).
- IV administration of codeine is not recommended due to signif cant histamine release and potential for se vere vasodilation, hypotension, and cardiac/ respiratory arrest.
- IM/Subcut administration of codeine is also not recommended as painful and poorly tolerated.
- Multiple combination products containing codeine are a vailable; be a ware of potential for additive sources of codeine.
- Prescribed in mg , not mL for oral liquids.
- Do not cr ush sustained-release products used for chronic pain.
- Consider stool softener or mild laxative to mitigate constipation.

Morphine
- Morphine binds to the μ-opioid receptor s in the CNS, inhibiting neurotransmission in the ascending pain pathw ay.
- Routes of administration: PO , IV, IM/Subcut, and PR.
- Dosing:
  - Intermittent parenteral doses: 0.05–0.1 mg/kg/dose IV/IM/Subcut q2h PRN.
  - Intermittent oral/rectal doses: 0.2–0.5 mg/kg/dose PO/PR q4h PRN.
  - Continuous infusion: 0.1 mg/kg IV bolus loading dose, then 10–40 mcg/kg/ hr IV/Subcut infusion, with 0.1 mg/kg/dose q1h PRN for breakthrough pain.
  - Sustained-release products used for chronic pain and dosed based on average daily requirements of immediate-release preparations.
  - Adjust dose in renal impair ment.
- Product availability and administration:
  - PO liquid: mor phine – 1 mg/mL.
  - PO immediate-release tablets: 5, 10, 20, 25, 30, 40, 50, and 60 mg .
  - PO q12h sustained-release tablets (MS-Contin): 15, 30, 60, and 100 mg
  - PO q24h sustained-release capsules (Kadian): 10, 20, 50, and 100 mg .
  - PO extended-release capsules (M-Eslon): 15, 30, 60, and 100 mg .
  - Suppositor y: 5, 10, 20, and 30 mg .
  - Injectable: 2, 10, and 50 mg/mL.
  - Inject slowly o ver 2 minutes, dilute with NS or D5W to a maximum concentration of 5 mg/mL (rapid IV administration ma y produce excessive histamine release, hypotension, respirator y/cardiac depression). Ensure respiratory suppor t is readily a vailable.
  - Wean continuous infusions slowly to pre vent withdrawal syndrome.
  - For chronic pain, sustained-release capsules ma y be opened and sprinkled on food (pellets to be sw allowed whole).
- Onset: PO/PR – 30 minutes (60 minutes for controlled-release PO preparations); IV/IM/Subcut: 5 minutes.

- Peak effect: PO/PR – 60 minutes (~4 hr for controlled-release PO preparations); IV/IM/Subcut – 10–20 minutes.
- Duration: PO/PR – 4 hour s (12 hr for controlled-release PO preparations); IV/IM/Subcut – 3–4 hour s.
- Pharmacokinetics:
  - Good oral absor ption, bioavailability ~40% (see Section IV: PO con version section, Table 10.3).
  - Moderate protein binding .
  - Morphine is the active metabolite of codeine (via CYP2D6),   and also produces active metabolite (mor phine-6-glucuronide) when mor phine is metabolized in the liver by glucuronidation (does not in volve P450 CYP enzymes).
  - Eliminated primarily by the kidne ys.
- Contraindications:
  - Hypersensitivity/allerg y to mor phine or codeine.
- Signif cant dr ug interactions:
  - Other CNS depressant/respirator y depressant medications (i.e.,   other opioids, benzodiazepines).
  - Morphine may increase le vels of SSRIs, thiazide diuretics.
  - Succinylcholine, amphetamines, and antipsychotics ma y increase the le vels/ effects of mor phine.
  - MAO inhibitor s may increase the effect of mor phine.
- Adverse effects:
  - Excessive sedation.
  - Respirator y depression.
  - Bradycardia.
  - Hypotension.
  - Dizziness.
  - Drowsiness.
  - Pruritus.
  - Constipation.
  - Urinar y retention.
  - GI upset (ma y take PO with food).
- Monitoring:
  - HR, RR, BP, oxygen saturation, reduction in pain score, level of sedation, and constipation.
- Morphine special considerations and pear ls:
  - Preser vative-free mor phine is a vailable for epidural or intrathecal use.
  - High-alert medication for medication er rors associated with abbre viations – not to be prescribed as MSO $_4$ for mor phine sulfate (interchange er ror with magnesium sulfate – MgSO $_4$).
  - Prescribe in mg , not mL for oral liquid.
  - Do not cr ush sustained-release products for chronic pain.
  - Consider stool softener or mild laxative to mitigate constipation.

Fentanyl

- Synthetic opioid analgesic (increased potency compared to morphine), which is short acting.
- Binds to μ-opioid receptors in CNS to inhibit ascending pain pathways.
- It has sedative and analgesic properties.
- Routes of administration: IV/IM/Subcut, intranasal, and topical patch.
- Dosing:
  - IV/IM/Subcut intermittent: 0.5–1 mcg/kg/dose q1h PRN.
  - IV continuous infusion: 1–2 mcg/kg bolus, then 0.5–3 mcg/kg/hr, with 1–2 mcg/kg/dose q1h PRN for breakthrough pain.
  - Transdermal patch: change q72h (moderate to severe chronic pain, with stable opioid requirements; not for opioid naïve patients): conversion of stable opioid dose to equivalent dose of fentanyl in consultation with pharmacist – individualize dosing.
  - Intranasal: 1.5 mcg/kg/dose, q5 minutes PRN.
- Product availability and administration:
  - Injectable: 50 mg/mL.
  - Transdermal patches: 12, 25, 50, 75, and 100 mcg/hr.
  - IV pushes slowly over 2–3 minutes, undiluted or diluted in 10 mL NS or D5W; IM/Subcut undiluted. May also be given by Subcut as a continuous infusion.
  - Transdermal patches used for chronic pain with stable opioid requirements, changed q72h.
  - Wean continuous infusions to prevent withdrawal syndrome.
- Onset, peak effect, and duration: See Table 10.1.
- Pharmacokinetics:
  - Quick onset, short duration with intermittent dosing.
  - Highly lipid soluble (prolonged effects with continuous infusion).
- Contraindications: hypersensitivity to fentanyl, opioid naïvety for transdermal patches.
- Significant drug interactions:
  - Other CNS depressant/respiratory depressant medications (i.e., other opioids, benzodiazepines).
  - MAO inhibitors (increased effect of fentanyl).
  - Fentanyl may increase the effects of β-blockers, diltiazem, verapamil, SSRIs, thiazide diuretics.

TABLE 10.1: Pharmacokinetics of fentanyl via different routes

	IV/Intranasal route	IM/Subcut route	Transdermal
Onset	2–5 min	5 min	6 hrs
Peak effect	5 min	30 min	24 hrs
Duration of action	30–60 min	1–2 hrs	72 hrs

- ■ Adverse effects:
  - ● Excessive sedation.
  - ● Respiratory depression.
  - ● Chest wall rigidity (rapid IV administration).
  - ● Dizziness.
  - ● Drowsiness.
  - ● Pruritus.
  - ● Rash (topical patch).
  - ● Hypokalemia.
  - ● Constipation.
  - ● Urinary retention.
- ■ Monitoring: HR, RR, BP, oxygen saturation, reduction in pain score, and level of sedation.
- ■ Fentanyl special considerations and pearls:
  - ● Intermittent IV pushes should be administered *slowly* (over 2–3 minutes, if possible) to prevent chest wall rigidity and respiratory depression.
  - ● Chest wall rigidity/apnea from rapid IV administration may be reversed with neuromuscular blocker. Be prepared to intubate the patient.
  - ● Respiratory depressant effect may outlast analgesic effect.
  - ● More lipid soluble than morphine, thus more potent.
  - ● No histamine release, thus less bradycardia and hypotension than morphine.
  - ● Increased body heat increases topical absorption of fentanyl from patch.
  - ● Only certain manufacturer's patches may be cut (matrix vs. reservoir delivery).
  - ● Atomizing intranasal delivery device preferred for increased tolerability and absorbable surface area (vs. drops to nares).

Hydromorphone
- ■ Synthetic opioid analgesic (increased potency compared to morphine), and antitussive.
- ■ Binds to μ-opioid receptors in the CNS for analgesia, and acts centrally on the medulla for cough suppression.
- ■ Route of administration: PO, IV/IM/Subcut, and PR.
- ■ Dosing:
  - ● IV/IM/Subcut intermittent doses: 10–20 mcg/kg/dose IV q2h PRN.
  - ● Continuous IV/Subcut infusion: 20 mcg/kg bolus, then 2–10 mcg/kg/hr infusion, titrated to effect.
  - ● PO immediate-release tablets, PO liquid, PR: 0.04–0.08 mg/kg/dose q3–4h PRN.
  - ● Sustained-release products used for chronic pain and dosed based on average daily requirements of immediate-release preparations, usually dosed q12h.
- ■ Product availability and administration:
  - ● Injectable – 2, 10, 20, 50 mg/mL.

- PO immediate-release tablets – 1, 2, 4, and 8 mg.
- PO liquid – hydromorphone 1 mg/mL.
- PR immediate-release suppository: 3 mg.
- PO sustained-release capsules (q12h, hydromorph contin) – 3, 6, 12, 18, 24, and 30 mg.
- For chronic pain, sustained-release capsules may be opened and sprinkled on food (pellets to be swallowed whole).
- IV pushes undiluted or diluted to 10 mL with NS or D5W over 2 minutes.
■ See Table 10.2 for onset, peak effect, and duration.
■ Pharmacokinetics:
- Well absorbed orally, some variability with IM absorption (may have slower onset IM).
- Metabolized in the liver via glucuronidation to inactive metabolites.
- Primarily eliminated by the kidneys.
■ Contraindications: hypersensitivity to hydromorphone.
■ Significant drug interactions:
- Other CNS depressant/respiratory depressant medications (i.e., other opioids, benzodiazepines).
- Hydromorphone may increase levels of SSRIs and thiazide diuretics.
- Succinylcholine, amphetamines, and antipsychotics may increase the levels/effects of hydromorphone.
- MAO inhibitors (increased effect of hydromorphone).
■ Adverse effects:
- Excessive sedation.
- Respiratory depression.
- Bradycardia.
- Hypotension.
- Dizziness.
- Drowsiness.
- Elevated intracranial pressure.
- Pruritus.
- Constipation.
- Urinary retention.
- GI upset (may take PO with food).

TABLE 10.2: Pharmacokinetics of hydromorphone for different routes of administration

	IV/Subcut route	IM	PO/PR	Sustained-release PO
Onset	5 min	15 min	15 min	60 min
Peak effect	10 min	10 min	30–60 min	4 hr
Duration	4 hr	4 hr	4 hr	12 hr

- Monitoring: HR, RR, BP, oxygen saturation, reduction in pain score, level of sedation, and constipation.
- Hydromorphone special considerations and pearls:
  - Better absorbed orally than morphine.
  - Prescribe oral liquids in mg, not mL.

Methadone

- Long-acting opioid, binds to μ-opioid receptor in CNS.
- Similar analgesic potency to morphine, but much longer duration of action and produces less sedation than morphine.
- Antitussive activity, minimal sedation.
- Route of administration: PO.
- Dosing:
  - 0.1–0.2 mg/kg PO q6h PRN.
  - Adjust dose in renal impairment.
- Product availability and administration:
  - PO immediate-release tablets: 1, 5, and 10 mg.
  - PO oral liquid concentrates: 1 and 10 mg/mL.
  - Oral liquids must be dispensed as diluted oral concentrate in juice to prevent use for injection.
- Onset: 30–60 minutes.
- Peak effect: 2–4 hours.
- Duration: 6–8 hours.
- Pharmacokinetics:
  - Well absorbed orally.
  - Highly protein bound.
  - Metabolized in liver by CYP P450 enzymes, and by *N*-demethylation to inactive metabolites.
  - Eliminated by the kidneys.
- Contraindications: prolonged QT interval, hypersensitivity to methadone.
- Significant drug interactions:
  - Caution with medications that also prolong the QT interval (additive effect as methadone can also prolong QT).
  - Azole antifungals, alfuzosin, amphetamines, ciprofloxacin, MAO inhibitors, quinine, SSRIs, and succinylcholine may increase the levels of methadone.
  - Carbamazepine, phenytoin, barbiturates, and protease inhibitors/non-nucleoside reverse transcriptase inhibitors (NNRTIs) may decrease the levels of methadone.
- Adverse effects:
  - Respiratory depression.
  - Bradycardia.
  - Hypotension.
  - Prolongation of QT interval/Torsade de pointes.
  - Dizziness.

- Drowsiness.
- Diaphoresis.
- Constipation.
- Urinary retention.
- Methadone special considerations and pearls:
  - Pain management dosing different than narcotic addiction dosing (q6h PRN vs. q24h, respectively).
  - Less sedation than other opioids.
  - Can accumulate with chronic use due to high degree of protein binding, thus a decrease in dose or frequency may be required after 5–7 days.
  - Potential for medication error with different concentrations of oral liquids, prescribed in mg, not mL.
  - There are regulatory requirements for methadone prescription in many jurisdictions.

PCA Opioids

- Patient-controlled analgesia (PCA) is a method for delivering continuous infusions of opioid analgesics such as morphine, hydromorphone, or fentanyl.
- PCA allows a set dose of opioid to be administered on-demand to the patient, at a set interval via an infusion pump.
- Continuous infusion (i.e., mcg/hr) can also be run via a PCA pump, and the rate of the continuous infusion adjusted based on the record of on-demand doses used.
- PCA is usually via IV or Subcut route.
- The advantage of PCA is a more stable and patient-specific delivery of pain control.
- Patients must be assessed for ability to understand and use PCA correctly.
- PCA syringes are usually prepared by the Pharmacy Department, under laminar flow in a sterile hood and are affixed with high alert labels to indicate for PCA pump use only.

Example of PCA Morphine

- Morphine: 125 mg/25 mL syringe (5 mg/mL diluted with NS).
- PCA dose: usually 10–30 mcg/kg/dose.
- Lockout interval: 6–10 minutes.
- Maximum limit per 4 hours: ~1,200 mcg/kg/4 hr or as directed by Acute Pain Service.
- Continuous infusion: 0–30 mcg/kg/hr.
- Higher doses may be required for chronic use/opioid tolerance (Table 10.3).

Co-analgesics/Adjunctive Agents

- Co-analgesics or adjunctive agents are medications used along with opioids and non-opioids to control pain.
- Use of an adjunctive agent may decrease the required dose for an opioid analgesic (opioid-sparing).

TABLE 10.3: Relative potency of opioids and IV:PO con versions

	Medication	Ratio comparison based on standard of mor phine 10 mg IM [a]	
		Equipotent Oral Strength[b] (mg)	Equipotent parenteral strength (mg)
Most potent	Fentanyl	N/A	0.1–0.2
	Hydromorphone	4–6	2
	Morphine	20–30	10
	Methadone	Single dose: 20 Chronic use: 2–4	N/A
Least potent	Codeine	200	120

[a]Based on adult data, unknown direct applicability to pediatric population. Refer to usual initial doses for opioid naïve pediat ric patients, and use relative ratio of potency to co vert betw een medications or routes of administration.
[b]For immediate-release oral preparations.

- Adjunctive agents are medications that w ere originally de veloped for the treatment of another medical condition, but ha ve been found to ha ve analgesic proper ties as well.
- The major use of adjunctive agents is for the treatment of neuropathic pain.
- Adjunctive agents can be selected based on the nature of pain, or the adverse effects that ma y actually be benef cial depending on the patient' s presentation.
- Adjunctive agents should alw ays be titrated to an effective dose slowly , and then tapered gradually to discontinue.

Amitriptyline

- Amitriptyline is a tricyclic antidepressant medication, which can also be used to control chronic and neuropathic pain.
- TCAs blunt neuropathic pain b y inhibiting reuptak e of serotonin and norepinephrine in the CNS, blocking conduction in sodium channels, blocking adenosine receptor s, and NMD A receptor s.
- Routes of administration: PO .
- Dosing:
  - Amitriptyline: 0.1 mg/kg PO QHS, increase as tolerated o ver 2–3 weeks to 0.5–2 mg/kg PO QHS.
- Product a vailability and administration:
  - Amitriptyline: 10, 25, 50, and 75 mg tablets.
- Onset: se veral weeks of consistent dosing and compliance to achie ve adequate relief of neuropathic pain, some relief within 7 da ys.
- Pharmacokinetics:
  - Well absorbed orally .
  - Highly protein bound (non-dialyzable in toxicity).
  - Metabolized in liver to active metabolite, nortriptyline.
  - Excreted primarily b y kidneys.

- ■ Contraindications:
  - ● Hypersensitivity to TCAs.
- ■ Signif cant drug interactions:
  - ● Use of MAO inhibitor within 14 days (risk of serotonin syndrome, hypertensive crisis) – contraindicated.
  - ● Alfuzosin, bupropion, cimetidine, ciprofloxacin, divalproex, duloxetine, lithium, MAO inhibitors, amphetamines, metoclopramide, protease inhibitors, quinine, SSRIs, terbinafine, and valproic acid may *increase* the levels and effects of amitriptyline.
  - ● Barbiturates, carbamazepine, St. John's wart may *decrease* the levels of amitriptyline.
- ■ Adverse effects:
  - ● Sedation – tolerance to sedation usually develops within a few weeks.
  - ● Anticholinergic effects – tolerance to anticholinergic effects usually develops within a few weeks.
  - ● Anxiety, worsening depression, suicidal ideation.
  - ● Postural hypotension.
  - ● Tachycardia.
  - ● Weight gain.
  - ● Photosensitivity, rash.
  - ● Discoloration of the urine (blue-green).
- ■ Monitoring:
  - ● Decrease in severity of neuropathic pain over several weeks.
  - ● Mood, suicidal behaviors, or ideation.
- ■ TCA special considerations and pearls:
  - ● Caution use in pediatric patients with mood disorder, due to potential initial worsening of depression or suicidal ideation.
  - ● Narrow therapeutic range, thus toxic in overdose.
  - ● Routine therapeutic drug monitoring not necessary, but levels may be helpful in suspected toxicity.
  - ● Amitriptyline may be useful if the patient complains of constant burning pain and trouble sleeping (adverse effect of sedation helpful).

Gabapentin and Pregabalin
- ■ Gabapentin and pregabalin are anti-convulsant medications that can be used for chronic and neuropathic pain in pediatrics that is described as "shooting" pain.
- ■ Mechanism of action is not fully understood.
  - ● Likely binds to undefined neuroreceptors or a carrier protein.
  - ● Pregabalin may modulate calcium channels involved in neuropathic pain.
- ■ Gabapentin and pregabalin are structurally similar to the inhibitory neurotransmitter, gamma-amminobutyric acid (GABA), although they do not bind to the GABA receptors.
- ■ Route of administration: PO.

- Dosing:
  - Gabapentin:
    - Day 1 : 5 mg/kg/dose PO QHS.
    - Day 2 : 5 mg/kg/dose PO BID .
    - Day 3 : 5 mg/kg/dose PO TID .
    - Titrate to effect.
    - Usual dose 8–35 mg/kg/da y divided TID .
  - Pregabalin: optimal dosing is not w ell def ned.
    - May empirically star t at 25 mg PO BID , increased b y 25 mg/da y to a usual maximum of 150 mg PO BID , or 6 mg/kg/da y (titrate to low est effective dose).
    - Adjust dose in renal impair ment.
- Product availability and administration:
  - Gabapentin – 100-, 300-, and 400-mg capsules, 600- and 800-mg tablets, 100-mg/mL oral liquid.
  - Pregabalin – 25-, 50-, 75-, 150-, 225- and 300-mg PO capsules.
- Onset: Se veral weeks of consistent dosing and compliance to achie ve adequate relief of neuropathic pain, some relief within 7 da ys.
- Pharmacokinetics:
  - Rapid oral absor ption via facilitated transpor t proteins.
  - Low protein binding .
  - Not metabolized (no inactive or active metabolites).
  - Excreted in urine and feces.
- Contraindications
  - Hypersensitivity to gabapentin or pregabalin.
- Signif cant dr ug interactions:
  - Ketorolac ma y decrease the le vels of gabapentin.
  - Gabapentin ma y increase the effects of CNS depressants.
- Adverse effects:
  - Peripheral edema.
  - Restlessness, emotional lability , poor concentration.
  - Increased risk of suicidal beha vior and ideation.
  - Dizziness.
  - Fatigue.
  - Dry mouth.
  - Weight gain.
  - Pruritus.
  - Constipation.
  - Diplopia.
- Monitoring
  - Decrease in se verity of neuropathic pain o ver se veral weeks.
  - Suicidal beha viors or ideation.

- Gabapentin and pregabalin special considerations and pear ls:
  - Bioavailability decreases with increasing doses (i.e.,   giving a higher dose does not always result in greater clinical effect – transpor t proteins for absor ption are saturable).
  - Pregabalin may be titrated more rapidly than gabapentin (better tolerability of pregabalin).
  - Gabapentin oral liquid should be refrigerated.
  - Administering with food can increase absor ption.
  - Titrate doses slowly , and taper gradually to discontinue.

Baclofen

- Baclofen is a non-paralytic sk eletal muscle relaxant used as an adjuvant medication for patients with per sistent muscle spasm and related musculosk eletal neuropathic pain.
- Baclofen is an agonist to the GABA inhibitor y neurotransmitter receptor , inhibiting afferent neurotransmission and muscle contraction.
- Routes: PO (intrathecal in se vere cases).
- Dosing:
  - Titrate slowly to effect.
  - Less than 2 year s old: 10–20 mg/da y divided q8h, increase dose q3 da ys in increments of 5–15 mg/da y to reach maximum of 40 mg/da y.
  - Two to 7 year s old: 20–30 mg/da y divided q8h, increase dose q3 da ys in increments of 5–15 mg/da y to reach maximum of 60 mg/da y.
  - Greater than 8 year s old: 30–40 mg/da y divided q8h, increase dose q3 da ys in increments of 5–15 mg/da y to reach maximum of 120 mg/da y.
- Product availability and administration:
  - PO tablets: 10 and 20 mg .
  - PO compounded suspension 5 or 10 mg/mL (not commercially a vailable), refrigerate.
  - 0.05 mg/mL and 0.5 mg/mL injection for intrathecal use – requires initial test dose, may be used as continuous intrathecal infusion.
  - Administer PO with food.
- Onset – 3–4 days.
- Peak effect – 5–10 days.
- Duration – 8 hour s.
- Pharmacokinetics:
  - Rapid oral absor ption.
  - Minimally metabolized in the liver (little active or inactive metabolites).
  - Largely excreted in urine and feces.
- Contraindications:
  - Hypersensitivity to baclofen.
- Signif cant dr ug interactions
  - Baclofen increases effect of CNS depressants.

- ■ Adverse effects:
    - ● Hypotension.
    - ● Chest pain/palpitations.
    - ● Dizziness.
    - ● Insomnia.
    - ● Headache.
    - ● Slurred speech.
    - ● Rash, pruritus.
    - ● Dry mouth.
    - ● Anorexia.
- ■ Monitoring: decreased muscle rigidity and spasm, tolerance of dose titration.
- ■ Baclofen special considerations and pear ls:
    - ● Abrupt withdrawal may potentiate seizure activity or hallucinations.
    - ● Be aware of potential for er ror with multiple compounded oral liquid concentrations (5 and 10 mg/mL).
    - ● Prescribe oral liquid in mg , not mL.

Topical Anesthetics

LET Gel
- ■ LET gel contains Lidocaine 4%, Epinephrine (1:2,000) 0.1%, Tetracaine 0.5% in a cellulose gel (without cellulose is LET solution, liquid for mulation).
- ■ LET is used for anesthesia of lacerations, most commonly lacerations of the face and scalp in pediatric patients.
- ■ Administration: apply 1–3 mL of solution with cotton-tipped applicator around edges and directly into wound.
- ■ Duration to apply/onset of anesthesia: 20–30 minutes.
- ■ Duration of anesthesia: 30 minutes after application complete.
- ■ LET gel special considerations and pear ls:
    - ● Preferred agent for laceration repair o ver TAC, and less painful than local inf ltration of lidocaine: see Chapter 11.
    - ● Should not be used on mucous membranes or end-ar teriolar peripheral extremities (i.e., f nger tips, nose tip, penis, ears) due to vasoconstriction with epinephrine.
    - ● Avoid in patients with h ypersensitivity to amide anesthetic agents.
    - ● Most studies of LET gel for laceration repair are in children greater than 2 years of age.
    - ● Should be labeled " *not* for injection," refrigerated and protected from light.
    - ● Not eff cacious for intact skin.

TAC
- ■ TAC is a topical anesthetic, containing Tetracaine 0.5%, Epinephrine 0.05%–0.1%, and Cocaine 11.8%.

- TAC is not a preferred agent for topical anesthesia due to expense, concern of toxicity, and regulatory restriction surrounding use and storage of cocaine.
- LET is equally efficacious, with less adverse effects and restrictions.
- Application: apply 2–5 mL directly to wound with cotton or gauze.
- Duration to apply/ onset of anesthesia: 10–30 minutes.
- Duration of anesthesia: 30 minutes after application complete.
- TAC special considerations and pearls:
  - Rare severe adverse effects: seizure, CNS stimulation, peripheral vasoconstriction, tachycardia, and MI.
  - Should not be used on mucous membranes or end-arteriolar peripheral extremities (i.e., finger tips, nose tip, penis, ears) due to vasoconstriction with epinephrine.
  - Tetracaine and cocaine are an ester anesthetics (avoid in patients with hypersensitivity to ester anesthetics).
  - Not efficacious for intact skin.

Eutectic Mixture of Local Anesthetics

- Eutectic mixture of local anesthetics (EMLA ®) contains 2.5% lidocaine and 2.5% prilocaine.
- As a eutectic mixture, when combined together, lidocaine and prilocaine dissolve into a liquid preparation, and are available commercially formulated as a cream or as a topical patch.
- Used primarily for reducing pain associated with minor procedures such as venipuncture, vaccinations, and superficial skin procedures (i.e., laser treatments, electrolysis, surgical debridement), as well as relief of pruritus.
- Application:
  - Apply a thick layer (1–2 g/10 cm^2) of cream to intact skin, cover with nonadhesive occlusive dressing (i.e., Tegaderm).
  - Apply patch directly on area to be anesthetized.
- Duration to apply/onset of anesthesia: 1–2 hours.
- Duration of anesthesia: 0.5–2 hours after application complete.
- EMLA special considerations and pearls:
  - Not a preferred agent for topical anesthesia in the emergency department due to long onset of action; efficacy depends on duration of application.
  - Should be used only on intact skin.
  - Significant adverse effects such as contact dermatitis and methemoglobinemia (especially if applied to large areas, repeated doses, nonintact skin, or children <3 months of age).
  - Caution with medications that may also induce methemoglobinemia (i.e., sulfonamides).
  - Other adverse effects: blanching, erythema, and edema at site of application.
  - Avoid on mucous membranes.
  - Avoid in patients with hypersensitivity to amide anesthetics.

Tetracaine (Ametop ®)

- Topical anesthetic commercially available as cream containing tetracaine 4%.

- Used primarily as topical anesthetic for venipuncture or IV cannulation.
- Application: apply la yer of cream (1 g/30 cm ^{2} – 6 × 5 cm) to intact skin, cover with nonadhesive occlusive dressing (i.e., Tegaderm).
- Duration to apply/onset of anesthesia: 30–45 minutes.
- Duration of anesthesia: 4–6 hour s (application ma y be repeated after 5 hr , not to exceed 2 g/24 hr s for children).
- Tetracaine special considerations and pear ls:
  - Avoid in patients with h ypersensitivity to ester anesthetics.
  - Not recommended for children less than 1 month of age.
  - Adverse effects: er ythema at application site (vasodilation), edema, and pruritus at application site. Rare blistering ma y occur.

Lidocaine (Maxilene ®)

- Topical anesthetic commercially a vailable as cream for topical application containing lidocaine 4% or 5% in a liposomal for mulation.
- Cream used primarily as topical anesthetic for venipuncture, relief of pain, and pruritus associated with minor skin ir ritations (i.e., superf cial bur ns, insect bites, and scrapes).
- Application of cream: r ub pea-sized amount into area to be anesthetized for 30 seconds, and then co ver with remaining amount of dose in a thin la yer. Occlusive nonadhesive dressing ma y be used, but is not necessar y.
  - Age less than 1 year , or weight less than 10 kg = 1 g per site.
  - Age 1–6 year s, or weight 10–20 kg = 2 g per site.
  - Age greater than 7 year s, or weight greater than 20 kg = 2.5 g per site (1/2 of 5 g tube).
    - ▶ Maximum dosing: ma y repeat for each procedure as necessar y, up to a maximum of three applications per da y, spaced b y at least 2 hour s.
- Duration to apply/onset of anesthesia: 20 minutes.
- Duration of anesthesia: 1 hour after completion of application.
- Lidocaine special considerations and pear ls:
  - Do not use in patients with h ypersensitivity to amide anesthetics.
  - Produces less vasodilation than Ametop (potentially less er ythema), and less vasoconstriction than other topical anesthetics containing epinephrine (i.e., LET and T AC), thus ma y lead to greater visibility and success for IV star ts.
  - Not recommended for children <1 month of age.
  - Only for use on intact skin.

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11 Local Anesthetics

Brian Levy and Jonathan Sherbino

Introduction

- Local anesthetics are commonly used in the emergency department (ED) for laceration repair and regional analgesia.
- Inca Indians first used cocaine, derived from *Erythroxylon coca* bushes found in the Andes during cranial trephination.
- Small nerve fibers are more sensitive to local anesthetics, while myelinated fibers are blocked before nonmyelinated fibers.

Biochemistry

- Generic structure of local anesthetic agents.
  - Aromatic ring (lipophilic) – intermediate chain – hydrophilic tail.
- Anesthetic properties determined by:
  - pKa – amount of local anesthetic that penetrates through the tissues.
  - Partition coefficient – intrinsic lipid solubility.
  - Degree of protein binding.
  - Type of intermediate chain determines two basic types: the "esters" and the "amides."
    - ▶ Amino-esters:
      - ○ "Esters" include cocaine, procaine, tetracaine, and chloroprocaine.
      - ○ Esters are hydrolyzed by plasma pseudocholinesterase.
    - ▶ Amino-amides:
      - ○ "Amides" include lidocaine, mepivacaine, prilocaine, bupivacaine, and etidocaine.
      - ○ Easy memory trick: amides have the letter "i" occurring twice in the generic name.

Anatomy of Nerves

- Structure of peripheral nerves.
  - Bundles of individual nerve fibers or fasciculi are encased in a longitudinal array of collagen fibers known as the endoneurium.
  - Buried within the endoneurium are the actual nerve fibers comprised of an axon or multiple axons, which may or may not be myelinated.

- Neuron resides within the fasciculi encased in the endoneurium
  - In general, neurons contain dendrite(s), which act as signal collector s that monitor the en vironment, receive signals from other neurons and feed information to the neuron body .

Physiology

- Neural impulses are conducted b y the axon, which conducts signals from the cell body to a synapse.
- In unmyelinated ner ve f bers, conduction mo ves as a "ripple" along the entire surface of the axon.
- Myelin sheath insulates the axon,   speeding impulse conduction.
  - Impulses skip from node to node along these m   yelinated axons, depolarizing the entire inter vening axon segments all at once (salutator   y conduction).
- Neural transmission is made possible b   y specialized voltage-gated sodium channels, which contain a pore allowing selective ion mo   vement.

Mechanism of Action of Local Anesthetics

Effect of pKa

- Upon tissue inf ltration, the lipid-soluble nonionized por tion of anesthetic diffuses through the tissue, ultimately across the lipid bila yer axonal membrane.
- Nerve tissue is lipophilic (up to and including the axons' m   yelinated sheathes, which are simply fat).
  - The higher the propor tion of nonionic molecules, the greater the degree to which the anesthetic can penetrate the tissue.
  - Only the nonionized por tion of the anesthetic solution can penetrate ner   ve tissue through the axon.
- Clinical effect of given dosage also impacted b   y pH of the tissue into which the anesthetic is infused:
  - When the pH of the solution or tissue containing the anesthetic is   *greater than* the dr ug's pK_a, then a greater propor tion of the anesthetic molecules in solution will be in nonionized for m. *Hence, the lower the pK_a, the faster the onset of anesthesia.*
  - Nonionized for m is more lipophilic, *therefore enhancing neural tissue penetration and speeding onset of action.*
  - Inf amed tissue and abscesses tend to ha   ve low pH, which unfa vorably impacts local anesthetic penetration.
- Once within the axoplasm, a por tion of the dr ug re-ionizes, and this ionic por tion is thought to enter the sodium channels where it slows the mo   vement of sodium ions, thereby pre venting the for mation/f ow of action potentials.

Effect of Intrinsic Lipid Solubility

- Lipid solubility is typically expressed as "par   tition coeff cient."
  - Partition coeff cient compares solubility of agent in a nonpolar solvent with solubility in a polar solvent such as w   ater.

- The greater the par tition coeff cient, the greater the potency , and more rapid the onset.

Effect of Protein Binding

- Duration of blockade deter mined by intrinsic protein binding of agent.
  - Higher protein binding causes tighter bonding to sodium channel receptor s and greater duration of blockade.

Effect of Vasoconstrictor s (e.g ., Epinephrine)

- Benef ts of adding vasoconstrictor s include the following:
  - Slows systemic absor ption, allowing increased maximum dosages without increased risk of systemic toxicity .
  - By slowing systemic absor ption via local decrease of blood f ow, duration of action lengthens.
  - Vasoconstrictor s reduce local blood f ow, promoting hemostasis and impro ve visualization of f eld.
  - Epinephrine typically added in concentrations of 1:100,000 or 1:200,000.
    - ▶ Epinephrine does not prolong action of bupivacaine.
    - ▶ Traditional texts continue to recommend against use of epinephrine in areas of body perfused only b y end ar terioles:
      - ○ More recent literature re view (regarding digital inf ltration) refutes this long-standing "prohibition" as "medical m ythology."

Effect of Ner ve Anatomy

- When local anesthetics inf ltrate a peripheral ner ve, they diffuse from the outer surface "mantle" of the ner ve toward the inner f bers "core."
  - In general, the mantle f bers inner vate more proximal str uctures anatomically , and core f bers inner vate more distal str uctures.
  - Expect faster onset of ner ve block more proximally than distal blocking action.

Local Anesthetic Agents

- See Table 11.1 .

Short-Duration Agents

- Procaine:
  - Largely replaced b y lidocaine due to high incidence of h ypersensitivity reactions.
- Chloroprocaine:
  - Most frequent use has been in shor t-duration epidural anesthesia.
  - Believed to be the least toxic local anesthetic to the central ner vous system (CNS) and cardio vascular system.
    - ▶ Prior contro versy suggesting neurological def cits after large inadver tent subarachnoid injection.

TABLE 11.1: Local anesthetics

Name generic (trade)	Class	Concentration (%)	Maximum dose (with epinephrine)	Onset	Duration (min)
Short acting					
Procaine (Novocaine)	Ester	1–2	7 mg/kg (9 mg/kg)	10–15	20–30 (30–45)
Chloroprocaine (Nesacaine)	Ester	1–2		6–12	15–30 (30)
Moderate acting					
Lidocaine (Xylocaine)	Amide	1–2	4–5 mg/kg (7 mg/kg)	5–15	30–60 (120)
Mepivacaine	Amide	0.5–1	4–5 mg/kg (7 mg/kg)	5–15	45–90 (120)
Prilocaine (Citanest)	Amide	0.5–1	8 mg/kg	15–25	30–90 (120)
Long acting					
Bupivacaine (Marcaine)	Amide	0.25–0.5	2 mg/kg (3 mg/kg)	15–30	120–240 (180–240)
Ropivacaine (Naropin)	Amide	0.2–0.5		1–15	120–240 (180–240)
Topical tetracaine [a] (Pontocaine)	Ester			3–10	30–60

[a]Topical tetracaine has a fast onset of action and duration. Used primarily for rapid ophthalmic and pharyngeal anesthesia.

- ○ Traced to bisulfte preservative no longer contained in current formulations.
- ○ Lumbar spasms reported with preparations of chloroprocaine that contained EDTA, which is no longer part of current formulations.

Moderate-Duration Agents
- Lidocaine:
  - Most multipurpose and versatile of local anesthetics.
    - ▶ Fast onset and relatively short duration make it ubiquitous agent for lacerations, foreign body removal, abscess drainage, lumbar punctures, catheter insertions, and so forth.
  - Variety of concentrations are available from 0.5%–4%, however, 2% is particularly useful when minimal volume is desirable (e.g., fngers).
  - Now restricted for intrathecal use due to concern that even small doses can induce "transient neurologic symptoms" (TNS), which involves onset of pain in the lower extremities from a few hours to 24 hours after apparently uncomplicated administration of spinal anesthesia.
- Mepivacaine:
  - Lower relative incidence of TNS than lidocaine.
  - Alkalinization with bicarbonate, as with lidocaine, may speed its onset of action.
  - Relative contraindication in pregnancy due to slow fetal hepatic metabolism.

- Prilocaine
  - Rapid metabolism and low er acute CNS toxicity relative to lidocaine suggested potential as an inf ltrative agent.
  - At doses >600 mg, ortho-toluidine, a metabolite of prilocaine, con verts hemoglobin to methemoglobin.
    - ▶ Of par ticular concer n in topical for mulations, where dosing for ENT procedures is not strictly adhered to.

Long-Duration Agents

- Bupivacaine:
  - Slow onset, long duration.
  - Str ucturally similar to mepivacaine with longer duration of action.
  - Typically used at concentrations of 0.5%–0.75% for major ner ve conduction blocks.
  - At 0.25% concentration, stronger sensor y than motor blockade mak es it ver y useful for local anesthesia.
    - ▶ Well be suited as spinal/epidural agent for obstetrics and postoperative anesthesia.
    - ▶ Has been mixed with faster onset, shor t-duration agents such as chloroprocaine to increase speed of onset.
      - ○ Doing so appear s to considerably shor ten the duration of block.
  - Highly cardiotoxic, likely due to high protein binding and lipid solubility .
    - ▶ May cause conduction blocks, activation of reentrant pathw ays and refractory ventricular ar rhythmias including ventricular tach ycardia and ventricular f brillation.
      - ○ Potential to induce refractor y cardiac ar rest at concentration of 0.75% if inadver tently injected intra venously.
  - Use with epinephrine does not extend duration of block but does reduce plasma uptak e.
    - ▶ Recommended for inf ltration with bupivacaine in order to pro vide forewarning of inadver tent intravascular administration.
    - ▶ Include epinephrine as a mar ker, subject to ph ysician judgment, when inf ltrating potentially toxic dosages.
      - ○ Intravascular injection of epinephrine 10–15 μg/mL in adults causes ≥10 beat/min increase in hear t rate and/or ≥15 mm Hg increase in systolic blood pressure.
- Ropivacaine:
  - Relatively ne w agent designed to retain the long-acting proper ties of bupivacaine with less cardiotoxicity .
    - ▶ Str ucturally almost identical to bupivacaine with one fe wer carbon in its hydrophilic tail.
  - At low concentrations, ropivacaine ma y provide an e ven greater ratio of sensor y to motor block than bupivacaine.

- Currently, there is ongoing debate regarding comparable safety and relative toxicity of ropivacaine ver sus bupivacaine in obstetrical labor and intraoperative anesthesia.

Local Anesthetic Inf ltration

- Pain during injection is a common complaint.
- Several means to reduce pain during inf ltration include:
  - Using a f ne needle (27–30 gauge).
  - Distracting patient as you inf ltrate.
  - Inf ltrate slowly (30 sec/mL) from proximal tow ard distal direction.
  - Inf ltrate inside the wound edge to reduce pain relative to injecting into intact skin.
  - Warm anesthetic solutions (to ~42°C).
  - Alkalinize the solution.
    - ▶ Raises anesthetic solution pH.
      - ○ Increases propor tion of molecules in nonionic state, therefore:
        - ➢ Faster onset
        - ➢ Small amount required for conduction blockade
    - ▶ Add sodium bicarbonate (44 mEq/50 mL) to lidocaine in a 1:10 ratio (1 mL bicarbonate added to 10 mL lidocaine).
      - ○ Increases rate of degradation at room temperature, decreasing shelf life by 7 days (therefore, make up as you go to a void this).
      - ○ More lipid-soluble agents such as bupivacaine precipitate easily; hence, use ratio of 1:50 (0.1 mL bicarbonate to 5 mL bupivacaine).

Side Effects/Complications of Local Anesthetics

Local Anesthetic Systemic T oxicity

- Term coined b y the American Society of Regional Anesthesia and P ain Medication (ASRA).
- Tends to affect:
  - Females greater than males.
  - Extremes of age: 16% of cases w ere below 16 year s of age and 30% w ere older than 60.
- Median time from f rst injection to symptoms: 53 seconds to 60 minutes.
  - Shorter onset thought to be from intra vascular injection.

Pathophysiology

- Local anesthetic systemic toxicity (LAST) is thought to be a function of:
  - Patient factor s – baseline health, concur rent medications, for example, comorbid cardiac, pulmonar y, renal, hepatic, metabolic, and neurologic disease.

- Choice of anesthetic:
  - ▶ Esters are metabolized through plasma pseudocholinesterase and water-soluble metabolite is excreted.
  - ▶ Patients with sensitivity to succinylcholine, taking cholinesterase inhibitors, or those with myasthenia gravis at increased risk from esters.
  - ▶ Cocaine is exception, which is partially hepatically cleared.
  - ▶ Amides are metabolized through the liver, so caution should be used in administering to patients with hepatic or renal compromise.
- Agent:
  - ▶ Greater danger with more lipid-soluble agents (high potency agents, e.g., bupivacaine).
  - ▶ Ninety percent of cases of LAST involved bupivacaine, ropivacaine, and levobupivacaine.
    - ○ Bupivacaine and etidocaine are more cardiotoxic due to high lipid solubility and ability to blockade specific myocardial sodium channels.
    - ○ Bupivacaine carries even greater cardiotoxicity in pregnancy.
    - ○ Epinephrine exacerbates bupivacaine cardiac toxicity.
    - ○ Neonates are at greater risk for bupivacaine toxicity due to lower levels of albumin leaving more free drug in system, and reduced hepatic blood flow allowing amides not to be fully metabolized.
  - ▶ Amount:
    - ○ Rapid infiltration carries greater risk of toxicity than slow infiltration.
    - ○ Application of all topical at once leads to greater toxicity than application in stages or layers.
  - ▶ Location of block:
    - ○ Toxic levels most often induced by inadvertent intravascular administration.
    - ○ Highly vascularized sites more vulnerable (such as for intercostals blocks).
    - ○ Decreasing order of systemic absorption as follows:
      - ➢ Intercostals.
      - ➢ Intratracheal.
      - ➢ Epidural/caudal.
      - ➢ Brachial plexus.
      - ➢ Mucosal.
      - ➢ Distal peripheral nerves.
      - ➢ Subcutaneous.
    - ○ Physician's response to signs and symptoms:
      - ➢ Timeliness of detection by the healthcare provider.
      - ➢ Beware of tachycardia and dysrhythmias from systemic absorption of epinephrine.

Prevention

- Use the lowest *dose* required for the procedure.
- Aspirate with each injection to detect inadvertent intravascular injection.
- Administer incrementally in 3–5-mL aliquots, allowing approximately one circulation cycle (30–45 sec; longer for distal lower extremities) between injections.
  - Patients with a compromised ejection fraction warrant even slower administration to avoid toxic stacking of doses.
- Use ultrasound guidance where large doses will be administered in order to avoid intravascular injection.
- Monitor patients who have required larger doses post-procedure, since toxic reactions may evolve, in particular, when injecting into areas of swollen tissue.

Signs and Symptoms

- Presentation is highly variable – be on guard!

CNS Toxicity

- Patient presentation varies widely, and these occur in only ~20% of cases.
  - Early symptoms include:
    - ▶ Lightheadedness.
    - ▶ Circumoral numbness.
    - ▶ Metallic taste.
    - ▶ Agitation.
    - ▶ Slurred speech.
  - Late symptoms include:
    - ▶ Seizures.
    - ▶ CNS depression.
  - Seizures are the most common sign!
  - LAST induced by direct intravascular injection can bypass milder symptoms and proceed direction to seizures with rapid/simultaneous ignition of cardiac sequelae.

Cardiac Toxicity

- Incidence is less common than CNS toxicity.
- Consequence of direct myocardial sodium channel blockade.
  - May manifest as slower PR or QRS intervals.
  - Sodium blockade can trigger reentrant pathways causing VT or ventricular fbrillation.
- Toxicity is potentiated by concomitant hypoxia, hypercarbia, and/or acidosis.
- The effect is dose dependent.
- Early symptoms include:
  - Hypertension.
  - Tachycardia.
  - Ventricular arrhythmias.

- Late symptoms include:
  - Profound decreased contractility .
  - Bradycardia.
  - Hypotension.
  - Asystole.
- Cardiac toxicity may or may not be preceded by seizures or CNS symptoms.
- Bupivacaine and etidocaine are more cardiotoxic due to high lipid solubility and ability to blockade specif c myocardial sodium channels.
  - Bupivacaine car ries even greater cardiotoxicity:
    - ▶ During pregnancy.
    - ▶ When used with epinephrine.
  - In neonates due to low er levels of albumin lea ving more free dr ug in system, and reduced hepatic blood f ow allowing amides not to be fully metabolized.
- Beware of tach ycardia and dysrh ythmias from systemic absor ption of epinephrine.

Local and Allergic Effects
- True allergies to amides/ester s are rare.
  - Reactions more common with ester metabolite para-aminobenzoic acid (P ABA).
  - Much less common are reactions to amides, which are lik ely due to methylparaben that is used as a preser vative in amides.
- Systemic toxicity , vaso vagal reaction, and systemic absor ption of epinephrine are more common.
- True allergic reactions generally in volve cutaneous or upper airw ay signs.
- Prevention:
  - Switch to a preser vative-free solution of the opposite class.
    - ▶ For example, if the reaction occur s due to ester s, consider cardiac lidocaine, which is preser vative-free.
    - ▶ Other alter natives:
      - ○ Benzyl alcohol with saline or epinephrine.
        - ➢ A solution of 0.9% benzyl alcohol with 1:100,000 epinephrine can be made by adding 0.2 mL epinephrine 1:1,000 to a 20-mL vial of nor mal saline containing benzyl alcohol 0.9%.
        - ➢ Small double-blind randomized controlled trial (RCT) found that the above preparation results in signif cantly less pain than buffered lidocaine, although the duration of action w as substantially less.
      - ○ Diphenhydramine.
        - ➢ Similar chemical str ucture similar to local anesthetics, but differ s adequately to a void cross-reactivity .
        - ➢ A 0.5% solution can be made b y making a 10:1 dilution using nonpreser vative containing nor mal saline with 5% diphenhydramine (i.e., 50 mg/mL).
        - ➢ Small double-blind RCT compared 1% lidocaine to 0.5% diphenhydramine for minor laceration repair found that except on

the face 0.5% diphenhydramine provides equivalent analgesia to lidocaine with no increased pain of injection.

➢ At 1% concentration, diphenhydramine has been found to cause increased pain and/or burning on injection.

➢ Disadvantages to diphenhydramine:
  ○ Tissue sloughing of cutaneous layer has been reported in concentrations of 1% or higher.
  ○ Vesicle formation, prolonged anesthesia, paresthesias at concentrations of 2–5%.

Treatment of Severe Systemic Symptoms

■ Be prepared for advanced airway management as necessary.

■ Seizures should be promptly aborted with benzodiazepines.

■ Hyperventilation may be effective by transiently reducing $PaCO_2$ causing vasoconstriction with consequent slowing of drug delivery to the brain.

■ Cardiac arrest should be treated according to advanced cardiac life support (ACLS) guidelines with the following modifications:
  ● Use small initial doses of epinephrine.
  ● Amiodarone is the preferred agent to treat ventricular arrhythmias.
  ● Lidocaine or procainamide is *not* recommended.
  ● Blockers and calcium channel blockers are *not* recommended.

■ Intralipid therapy:
  ● Early infusion of 20% lipid emulsion may create a "lipid sink" or serve as an energy store for cardiac myocytes.
    ▶ Dose:
      ○ Twenty percent lipid emulsion.
      ○ Initial bolus of 1.5 mL/kg of lean body mass. A second bolus can be considered after 3–5 minutes if necessary.
      ○ Follow with infusion of 0.25 mL/kg/min continued for 10 minutes following the reestablishment of hemodynamic stability.
        ➢ In the event of refractory cardiotoxicity, increase infusion rate to 0.5 mL/kg/min.
        ➢ The current recommended ceiling of lipid emulsion treatment is 8–10 mL/kg, administered over 30 minutes.
      ○ NB: Propofol is not considered an advisable alternative because it has its own intrinsic cardiorespiratory toxicity in high doses.
      ○ In cases of refractory cardiovascular toxicity, consider use of cardiopulmonary bypass until tissue levels of local anesthetics have cleared.
      ○ Patients with signs of cardiotoxicity should be monitored for at least 12 hours, since toxic local anesthetic effects can persist or recur even posttreatment.

Topical Anesthetics

- Several topical agents are a vailable.
- See Chapter 11 for more details.
- Specif c agents

Cocaine

- Available for prescription as cocaine h ydrochloride in topical preparations ranging from 2%–10%.
- Rapidly absorbed through mucous membranes; peak plasma le vel achie ved in 15 –60 minutes.
- Applications:
  - Vasoconstrictive – primar y cur rent use in ENT applications (e.g ., epistaxis).
  - Potent decongestant for swollen mucous membranes.
  - May be used to numb mucosa in preparation for subsequent submucosal inf ltration of lidocaine.

Tetracaine, Adrenaline, Cocaine (T AC)

- Consists of 0.5% tetracaine; 0.05% epinephrine, and 11.8% cocaine.
- Onset 10–30 minutes after applying .
- Applications:
  - Nonmucosal skin lacerations, especially to the face and scalp.

Lidocaine, Epinephrine, Tetracaine (LET) Gel or Solution

- Mixture consisting of 4% lidocaine, 0.5% epinephrine, and 0.1% tetracaine.
- Onset 20–30 minutes from application.
- Applications:
  - Nonmucosal skin lacerations to face and scalp.
  - Slightly less effective on extremities.
  - Does not wor k on intact skin.

Eutectic Mixture of Local Anesthetics

- Made up of 25 mg/mL of lidocaine and 25 mg/mL of prilocaine, plus a thick ener, emulsif er and distilled w ater to adjust pH to 9.4.
- Anesthetizes to 3 mm within 60 minutes and 5 mm within 120 minutes; as a dental preparation, onset is ~2 minutes.
- A study found that 85% of children treated with eutectic mixture of local anesthetics (EMLA) required no fur ther anesthesia for suturing; how ever, a 90-minute w aiting period is required for adequate penetration, which can limit its use.
- Applications:
  - FDA indication is for use on intact, nonmucosal skin.
  - May be used on extremities but not to the palms and soles.

Liposomal 4% or 5% Lidocaine Cream (Maxilene)
- Onset of action in 30 minutes.
- In general requires about 1/2 the time to achie ve anesthesia than EMLA cream.
- Applications:
  - Used commonly for pediatric procedures:
    - Venipuncture.
    - Port-a-cath inser tion.
    - Peripheral intra venous inser tion.
    - Other painful procedures.
  - Temporary relief from minor cuts and abrasions.
  - Penile meatotom y.

Agents for Specif c Conditions
- Minor hemor rhoid pain, pruritus ani.
  - Prescription medications:
    - Proctosedyl – h ydrocortisone, framycetin sulfate, cinchocaine hydrochloride, aesculin.
      - Available in ointment and suppositories.
        - Anusol-HC 2.5% h ydrocortisone with anti-inf ammatory, antipruritic, vasoconstrictive proper ties.
        - RectaGel HC – Lidocaine 2.8% and h ydrocortisone acetate 0.55%.
  - Over the counter :
    - Anusol Plus Ointment, suppositories (contain pramoxine).
    - Sandoz Anuzinc Plus.
- Pharyngitis:
  - Benzydamine 0.15% (requires prescription).
    - An indazole nonsteroidal anti-inf ammatory drug (NSAID) with analgesic, antipyretic, and anti-edema proper ties.
      - Unlike other NSAIDs, benzydamine h ydrochloride does not inhibit cyclooxygenases (CO X) but:
        - Stabilizes membranes, resulting in local anesthesia.
        - Inhibits the production of pro-inf ammatory cytokines.
        - Inhibits the generation of reactive oxygen species b y neutrophils.
        - Inhibits leuk ocyte aggregation and adhesion.
        - Exhibits antimicrobial proper ties.
        - Also used 1 da y prior to radiation therap y and continued during treatment to a void radiation-induced mucositis.
        - Not commercially a vailable in the United States.
        - Not established for use in patients <6 years of age.
        - Gargle or rinse 15–30 seconds, then expel e very 1.5–2 hours.
        - Trade names: Apo/Ratio/Dom/No vo/PMS-Benzydamine; Sun-Benz, Tantum.

- Phenol 1.4–1.5% (no prescription required):
  - ▶ Antiseptic/analgesic.
  - ▶ Commercially a vailable as Chloraseptic spra ys.

Suggested Reading

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12 Regional Nerve Blocks

Brian Levy and Jonathan Sherbino

Introduction

- Regional nerve block is a common procedure in the emergency department (ED).
- Can be used for reduction of pain and/or facilitation of painful procedures (e.g., suturing, fracture, or dislocation reduction), especially in anatomical areas, where a large anesthetic field can be achieved distal to the block.
- Advantages of using a nerve block include the following:
  - Allows smaller amounts of local anesthetic to be utilized (in comparison to local infltration), where a wound involves a broad area in a given nerve distribution (e.g., multiple facial lacerations on ipsilateral side).
  - May avoid tissue distortion caused by local infltration, particularly in cosmetically signifcant areas (e.g., the vermillion border of the lip).
  - Avoids wound margin distortion caused by infltration of large volumes of anesthetic.
  - Avoids the pain and anxiety of multiple injections.
  - May eliminate the need (and attendant risks) for procedural sedation.
- Disadvantages include the following:
  - Requires patient cooperation, especially where effcacy of block depends on patient's ability to detect slight paresthesias.
  - Typically, it is a blind procedure in most EDs (performed without aid of nerve stimulator or ultrasound guidance).
  - Increases risk of anatomical injury (e.g., pneumothorax).
  - Damage to peripheral nerve (1.9 per 10,000).
  - Increases risk of ineffective block (erroneous placement of anesthetic).
  - Risk of systemic toxicity (7.5 per 10,000) from inadvertent intravascular injection.
  - Hematoma.
  - Local infection.
  - Pain at site of injection.

Contraindications

Absolute Contraindications

- Patients suffering from psychosis or dementia,   thereby making procedure diff cult or patient unable to give infor med consent.
- Infection superf cial to area of inf ltration.
  - Do not inject through infected tissue.
    - ▶ Less effective anesthesia in infected tissue,   especially in an abscess due to acidic en vironment.
    - ▶ Injection into or through infected tissue can induce local or systemic spread of infection.
- Hypersensitivity to local anesthetics – occur s in ~1% of the population.
- Known allerg y.
- On anticoagulation (see below).

Relative Contraindications

- Patients unable to communicate during procedure.
  - Severe pain ma y indicate intraneural injection, which can produce ischemic nerve injur y.
- Patient objection to remaining a wake.
- Preexisting neuropath y.
- Histor y of malignant h yperthermia.
- Uncontrolled seizure disorder s.
- Coagulation disorder s or anticoagulation therap y.
  - Absolute contraindication to peripheral ner ve blockade when it in volves:
    - ▶ Passage of needle deep within muscle mass.
    - ▶ Paravertebral blocks or approaches.
    - ▶ Risk of noncompressible ar terial/venous puncture.
    - ▶ Risk of hematoma with subsequent local mass effect on airw ay.
    - ▶ Postsurgical thromboproph ylaxis is not a contraindication to preoperative blockade.

Preparation

- Appropriate histor y and physical examination, including (but not limited to):
  - Anticoagulant therap y.
  - Sensor y or motor def cits, especially in area to be anesthetized.
  - Uncontrolled seizure disorder s.
  - Evaluation of distal neuro vascular status prior to regional.
    - ▶ Skin color, temperature, capillar y ref ll time, and pulses.
    - ▶ Sensation.
    - ▶ Motor function.
- Explanation of the procedure to the patient.
  - Informed consent should be recorded in the char t.

- Prepare appropriate monitoring and resuscitation equipment to detect and response to possible complications of the procedure.
- Position patient proper ly.
- Identify and mar k landmar ks (using ultrasound where possible).
- Antiseptic (e.g ., chlorhexidine) skin preparation.
- Surgical draping of the f led f eld.
- Universal precautions (mask, glo ves, eye shields, and contact precautions as indicated).
- Consider placement of intra venous catheter in the e vent the patient requires supplemental and/or emergency treatment.
- Supplemental:
  - Consider topical premedication of area to be injected to help reduce pain of injection.

General Techniques

- In every procedure, aspirate at each location prior to injection to a void intravascular injection of the local anesthetic.
- Local inf ltration of puncture site with 1% or 2% lidocaine facilitates procedure.
- Inject perineural region not intraneural.
  - Perineural injection ma y invoke very brief sensor y paresthesia indicating anesthetic reaching ner ve distribution, however, despite old adage "no paresthesias, no anesthesia" paresthesias ma y also mar k intraneural injection, especially per sistent paresthesia during injection.
  - Paresthesias during injection ma y por tend residual neuropath y even if not injected in the intraneural region.
  - Upon encountering paresthesias (prior to inf ltration), relocate needle to prevent intraneural injection.
    - ▶ Intraneural injection ma y cause ner ve ischemia and damage via ele vated nerve sheath pressure.
    - ▶ May cause prolonged and intense pain.
    - ▶ Immediately ter minate injection and reposition needle a fe w millimeter s away, wait for pain to subside, and reinject.
- Three-ring syringe simplif es aspiration, improves control, and eases ref lling.
- Choice of agent – see Chapter 11.
- Identifying point of inf ltration:
  - ▶ Using anatomical landmar ks.
  - ▶ Ultrasonograph y.
    - ○ Advantages:
      - ➢ Shor tens procedural time requirements.
      - ➢ Reduces blind needle passes.
      - ➢ Reduces dosages of anesthetic required to achie ve block.
      - ➢ Allows visualization of neighboring str uctures, for example, pleura, avoiding pneumothorax.

> ➢ Avoid intravascular injection.
> ➢ Accuracy.
> ○ Nerve stimulators:
> ➢ Not routine practice in most EDs.

- Maintain high index of suspicion for local anesthetic systemic toxicity (LAST) – see Chapter 11.
  - Addition of epinephrine 1:200,000 (5 μg/ml) producing tachycardia may signal clinician of impending systemic toxicity.
  - See Chapter 11 for additional details on avoiding, recognizing, and managing LAST.
- Educate patient on:
  - Management of postprocedure pain.
  - Identifcation of wound infection.
  - Other complications as appropriate.

Head and Neck Regional Blocks

Supraorbital and Supratrochlear Nerve Block

- Anatomy and region of sensory coverage (see Figure 12.1).
- Branch of trigeminal nerve (CN V).
  - Originates in midbrain comprised of three branches:
    - ▶ Opthalmic V1.
    - ▶ Maxillary V2.
    - ▶ Mandibular V3.
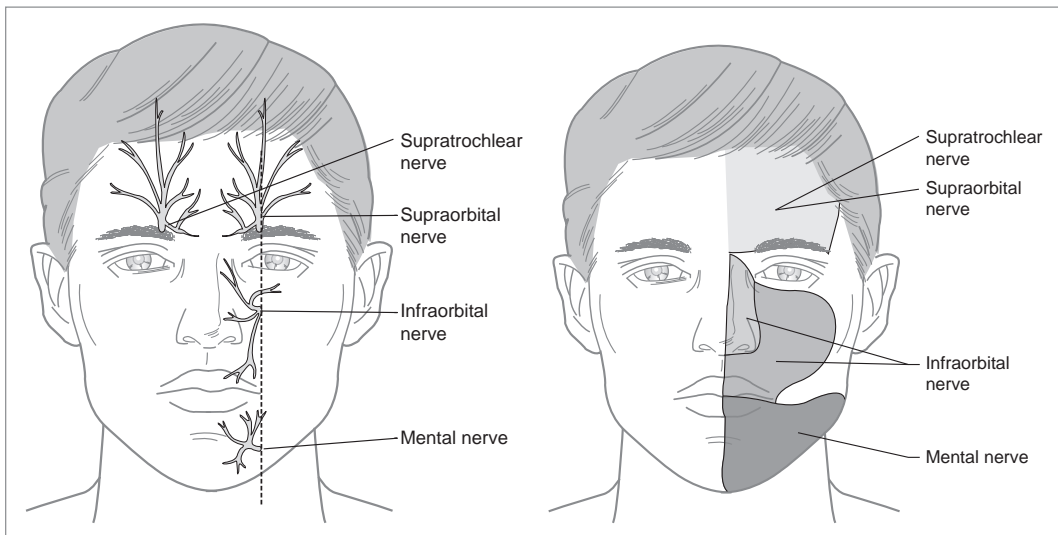
FIGURE 12.1: Anatomy and sensory distribution of the supraorbital, supratrochlear, infraorbital, and mental nerves.

- Supraorbital ner ve protr udes through supraorbital foramen directly superior to the pupil and abo ve the superior orbital rim.
- Supratrochlear ner ve exits skull just inferior to the orbital rim and 5–10 mm medial to supraorbital foramen.
- Supraorbital ner ve supplies most of forehead and supratrochlear supplies ipsilateral surface area of the nose and nasal bridge.
- Forehead supplied b y supraorbital and supratrochlear ner ves:
    - Both branch from ophthalmic ner ve division (V1) of cranial ner ve V (CN V), the trigeminal ner ve.
    - By blocking the supraorbital and infraorbital ner ves, complete anesthesia of periorbital area is achie ved.
    - By blocking supraorbital and supratrochlear ner ves bilaterally , entire forehead is anesthetized from ver tex of scalp to bridge of nose.
- Applications:
    - Wound repair, soft tissue exploration.
- Approach (see Figure 12.2) .
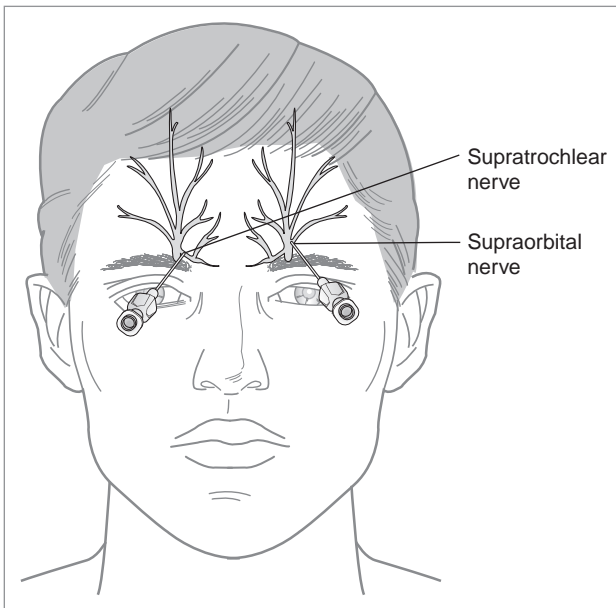
FIGURE 12.2: Approach to supraorbital and supratrochlear ner ve block.

Auriculotemporal Block
- External ear and sur rounding tissue is inner vated by:
    - Auriculotemporal ner ve anterosuperior ly and anteromedially extending to more lateral regions of the cheek and temple.
    - Greater auricular ner ve, inner vating inferoposterior aspects.
    - Minor occipital ner ve inner vating posterior auricular surface and sur rounding tissue.

- Auricular branch of vagus (Alderman's nerve and Arnold's nerve) innervates concha, external auditory canal, and most areas immediately surrounding auditory meatus.
- Applications:
  - Suture of large laceration of ear or surrounding tissue.
  - Excision and drainage of hematoma in area.
- Approach (see Figure 12.3):
  - Prepare ear and surrounding tissue with antiseptic.
  - Infiltrate a track surround the entire ear.
    - Spirating, infiltrating 3–5 mL of anesthetic as the needle is withdrawing.
    - Before needle is entirely withdrawn, reorient the needle, fully inserting it just anterior to the ear. After aspirating, infiltrate an additional 3–5 mL along an anterior track as the needle is withdrawn.
    - Next, introduce the needle just below the ear lobe in the sulcus, and repeat the above procedure anteriorly and posteriorly, thus creating another "V"-shaped tract of anesthetic.
- Maximum anesthesia occurs within about 10–15 minutes.
- The superficial temporal artery is medial to the ear and crosses the zygomatic arch. Aspirate to ensure that it is not punctured prior to infiltration.
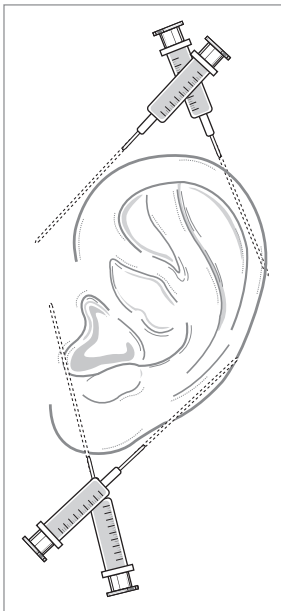
FIGURE 12.3: Auricular nerve block.

Infraorbital Block

- Anatomy (see Figure 12.1):
  - Division of maxillary nerve, V2 of the trigeminal nerve.
  - Exits infraorbital foramen 5–10 mm inferior to the orbital rim and superior but sagittally aligned to the maxillary canine teeth (tooth 6 on the patient's right and tooth 11 on the patient's left).

■ Applications:
  ● Anesthetizes medial cheek from the low er eyelid r unning caudally to include the ipsilateral upper lip, and including the medial cheek r unning laterally to a line dra wn ver tically at the lateral canthus of the ipsilateral e ye.
  ● Nasal bridge and nasal folds are generally not par t of the geograph y anesthetized in this approach.
  ● Wound closure.
  ● Pain relief.
  ● Debridement.
■ Approach:
  ● Intraoral:
    ▶ Position patient supine or sitting .
    ▶ If possible, provide topical anesthesia with a cotton-tipped applicator applied to the oral mucosa superior to the maxillar y canines, then dr y and retract the upper lip.
    ▶ Stabilize and position the upper lip.
    ▶ Inject at the gingival ref ection with the needle at the maxillar y canine (tooth 6 or 11) and track superior ly to a point approximately halfw ay between the entr y site and the orbital rim (i.e., this should be just inferior to the infraorbital foramen), and inject 3–5 mL of anesthetic.
      ○ The anesthetic should be injected adjacent to but not directly into the infraorbital foramen.
      ○ Direct injection into the foramen ma y result in sw elling of the low er eyelid or possible intraneural injection.
  ● Alter native intraoral approach (see Figure 12.4):
    ▶ If uncer tain of landmar ks or the block is not successful, inf ltrate ~5 mL of anesthetic solution intraorally in a fanlik e patter n within the upper buccal margin.
      ○ While lacking precision of a single-targeted injection, a 10–15-second massage of the tissues immediately subsequent to the injection is likely to yield similar anesthesia.
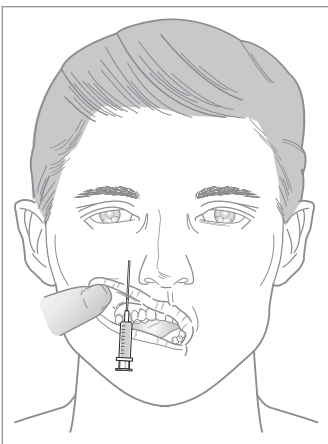
FIGURE 12.4: Approach to infraorbital ner ve block (intraoral approach).

- Extraoral:
  - ▶ Needle passes closely to facial ar tery and vein.
    - ○ Do not use vasoconstrictor s.
    - ○ Crucial to aspirate before injecting anesthetic.
  - ▶ Landmark the infraorbital foramen.
  - ▶ Prepare skin in sterile fashion.
  - ▶ Advance the needle through the skin, subcutaneous tissue, and quadratus labii superioris muscle.
  - ▶ Inject the anesthetic (2–3 mL); inf ltrated tissue will sw ell.
  - ▶ Massage the area for 10–15 seconds.

Inferior Alveolar Block and Intraoral Mandibular Block

- ■ Anatomy:
  - Mandibular division of trigeminal ner ve (V3) gives rise to:
    - ▶ Inferior alveolar ner ve
      - ○ Gives rise to mental ner ve.
      - ○ Innervates pulp of mandibular teeth from third molar to central incisor .
    - ▶ Buccal ner ve.
    - ▶ Auriculotemporal ner ve.
- ■ Applications:
  - Anesthetizes all teeth on ipsilateral side of mandible.
  - Anesthetizes the body of the mandible and the low er por tion of the mandibular ramus.
  - Floor of the mouth and anterior two-thirds of the tongue.
  - Anesthetizes anterior mandibular periodontium and low er lip and chin b y blocking mental ner ve.
- ■ Useful in:
  - Provision of anesthesia for multiple mandibular teeth in anesthetized quadrant and anterior two-thirds of tongue and lingual soft tissues.
  - Patients with dentoalveolar trauma.
  - Postextraction pain.
  - Dry sock et.
  - Pulpitis.
  - Abscess.
- ■ Approach (see Figure 12.5):
  - Patient should be seated in a chair with back (such as dental chair or ophthalmic room chair) with occiput f rmly against neck suppor t.
  - Apply topical anesthetic if a vailable.
  - Patients are anxious of dental blocks; patient ma y unexpectedly jer k on contact of needle.
    - ▶ Procedure is easiest to achie ve with a long needle (minimum 10-mL syringe permits adequate length for direct inf ltration), preferably 1 ⅜ in., 25 gauge, although 1 ⅛ in., 27 gauge ma y be e ven more comfor table for patient.
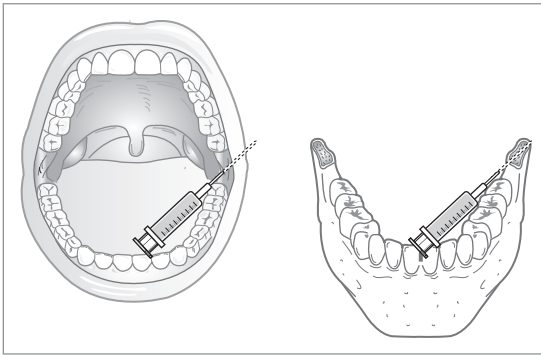
FIGURE 12.5: Approach to inferior alveolar ner ve block.

- Palpate the retromolar mandibular for nix with the nondominant glo ved thumb to identify the anterior border of the ramus of the mandible; the nondominant index f nger should be positioned just anterior to the ear .
- *The mucosa should be stretched* to maximize visibility and reduce pain of injection.
- Specif cally, note the *coronoid notch,* which is the greatest conca vity on the anterior border of the ramus of the mandible.
  - ▶ Inject at the *height* of this deepest conca vity of the ridge.
  - ▶ Move thumb medially from the coronoid notch to palpate the next prominence medially , which is known as the *internal oblique ridge.* The needle will be inser ted just medial to the inter nal oblique ridge at the deepest height of the notch.
  - ▶ Inject 1.5–2 mL of anesthetic. If analgesia is not achie ved, two more similar injections ma y be made.
    - ○ Delayed trismus and sensor y def cit ha ve been repor ted.

Mental Ner ve Block

- ■ Anatomy (see Figure 12.1):
  - Mental ner ve is ter minal sensor y branch of inferior alveolar ner ve exiting mandible through mental foramen.
  - The mental foramen is in-line with the pupil and generally lies mid way between the alveoli (tooth sock ets) and the inferior border of the mandible.
  - Innervates low er lip and chin.
  - Three branches:
    - ▶ One branch supplies skin of chin.
    - ▶ The other two branches inner vate skin and mucous membrane of low er lip.
- ■ Applications:
  - Provides anesthesia for repair of lacerations of the low er lip or chin.
  - Does not pro vide anesthesia for teeth, tongue, vestibule, or other soft tissue of the oral mucosa.
  - Easier to perfor m and less painful than other intraoral blocks.

- ■ Approach (see Figure 12.6):
  - Patient should be seated in chair with back (such as dental chair or ophthalmic room chair) with occiput firmly against neck support so that when mouth is open mandible is parallel to the floor.
  - Locate mental foramen.
    - ▶ Mental foramen should be located:
      - ○ Between teeth 21 and 22 or between 27 and 28 (between canines and first premolars in adults).
      - ○ Between first and second primary molars in children.
  - Needle should be inserted into the gingival reflection between the subject teeth, injecting approximately 2–4 mL of anesthetic solution *near, but not into the mental foramen.*
    - ▶ Injecting into the foramen can cause permanent damage to the neurovascular bundle.
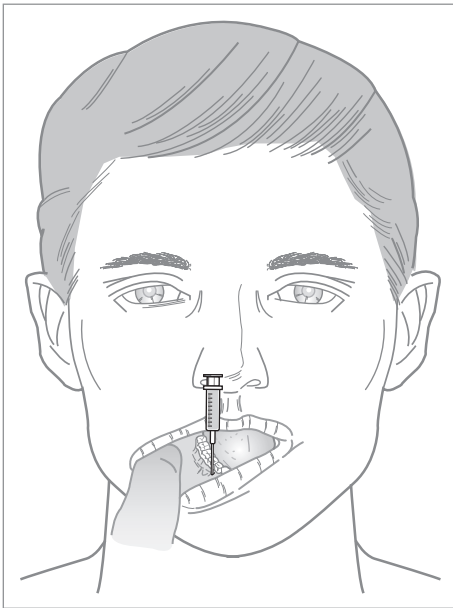
FIGURE 12.6: Approach to mental nerve block.

Superior Alveolar Block

- ■ Anatomy:
  - Division of V2, maxillary division of trigeminal nerve (CN V) further divided into:
    - ▶ Anterior superior alveolar (ASA):
      - ○ Branches from maxillary nerve immediately proximal to its exit from infraorbital foramen.
      - ○ Supplies upper incisor and canine teeth.
    - ▶ Middle superior alveolar (MSA):
      - ○ Branches from infraorbital portion of maxillary nerve.
      - ○ Innervates maxillary premolars (bicuspids) and part of first molar roots.

- ▶ Posterior superior alveolar (PSA):
  - ○ Branches from maxillary nerve just proximal to infraorbital groove.
  - ○ Innervates second and third maxillary (upper) molars and two of the three roots of the first maxillary molars.
- Lingual gingiva is *not* innervated by superior alveolar and this block will *not* anesthetize palatine structures.
- ■ Applications:
  - Anesthesia of branches associated with trauma or dental pain to teeth innervated by respective branches of ASA, MSA, or PSA.
  - Dentoalveolar abscesses.
  - Postextraction and dry socket pain.
- ■ Approach (see Figure 12.7):
  - Patient should be seated in chair with back (such as dental chair or ophthalmic room chair) with occiput firmly against neck support so that when mouth is open mandible parallel to floor.
  - ASA nerve block:
    - ▶ Retract the lip exposing the mucobuccal fold where it joins the apex of the canine tooth.
    - ▶ With the lip retracted, insert needle into the intersection of the mucobuccal fold and the canine at an angle of 45 degree, and advance the needle about 1–1.5 cm.
    - ▶ Slowly inject 2 mL of local agent; massage for 10–20 seconds.
  - MSA nerve block:
    - ▶ Retract the lip exposing the mucobuccal fold where it intersects the joint of the maxillary premolar 2 and molar 1 (teeth 3 and 4, or teeth 13 and 14 in an adult).
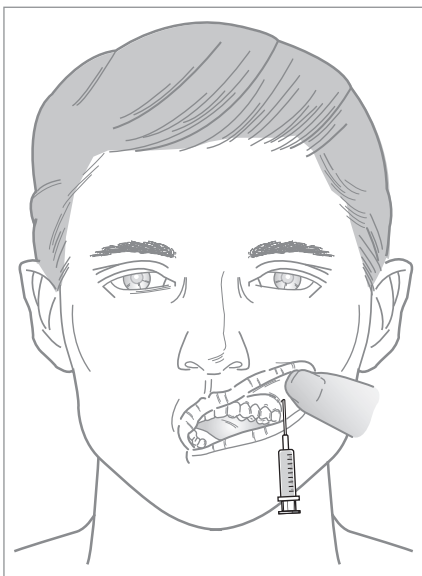
FIGURE 12.7: Approach to superior alveolar block.

> ▶ With the lip retracted, insert needle into the inter section of the mucobuccal fold and these two teeth at an angle of 45 degree, and advance the needle about 1–1.5 cm.
>
> ▶ Slowly inject 2–3 mL of local agent; massage for 10–20 seconds.

- PSA nerve block:
  - ▶ Retract the lip exposing the mucobuccal fold where it inter sects the joint of the maxillar y molars 1 and 2 (teeth 2 and 3, or teeth 14 and 15 in an adult).
  - ▶ With the lip retracted, insert needle into the inter section of the mucobuccal fold and these two teeth at an angle of 45 degree, and then advance it tow ard the posterolateral maxillar y tuberosity (up, back, and inward) along the natural cur ve of the maxilla to a depth of 2–2.5 cm.
  - ▶ If the needle hits the bone, withdraw slightly and redirect more laterally .
  - ▶ Slowly inject 2–3 mL of local agent; massage for 10–20 seconds.

Blocks of the T runk

Intercostal Ner ve Block (ICNB)

- Anatomy (see Figure 12.8):
  - Each intercostal block pro vides anesthesia to the ner ve running above and below a given rib.
  - Vein, artery, nerve (VAN) ar ranged run from superior to inferior within the costal groo ve at the inferior end of each rib.
  - Ribs 1–6 obscured b y rhomboids and scapular position making blocks diff cult.
  - Whether fracture anterior or posterior , it is best to block at "rib angle" just lateral to paraspinal muscles, or ~6 cm lateral to midline.
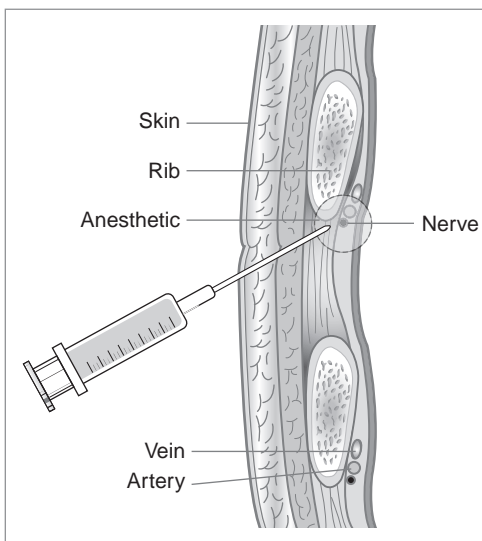
FIGURE 12.8: Approach to intercostals ner ve blocks.

- By blocking posterior to the midaxillar y line both divisions of the intercostal nerve, lateral pectoral cutaneous branch and anterior pectoral cutaneous branch are block ed.
- Indications:
  - Relief of posttraumatic pain, postoperative, or postinfectious pain emanating directly from thoracic or abdominal w alls.
  - Partial or complete substitute for opiates in cases of se vere pain in volving:
    - ▶ Rib fractures:
      - ○ Immediately relie ves pain and impro ves pulmonar y mechanics.
      - ○ Improves mobility.
    - ▶ Dislocation of costochondral joints.
    - ▶ Herpes zoster.
    - ▶ Nerve entrapment within the rectus abdominis.
    - ▶ Blockade at the posterior axillar y line pro vides relief from somatic pain but not visceral pain from thoracic or abdominal organs.
- Approach (see Figure 12.8):
  - Intercostal ner ves must be block ed proximal (posterior) to fracture site.
  - Overlapping inner vation from segments abo ve and below dictates that *the nerves above and below fractured rib(s) must be blocked.*
  - Nondominant index f nger hand palpates the inferior intercostal space and shift skin and subcutaneous tissue cephalad until the inferior edge of the rib is appreciated.
  - This technique allows nondominant index f nger to ser ve as guide and help protect against pneumothorax while helping to ensure optimal block.
  - Inser t needle angled cephalad approximately 10–15 degrees, using nondominant index f nger as a guide.
  - Penetrate skin and raise subcutaneous wheal and w ait 5–10 seconds for anesthesia, then advance until needle contacts bone.
  - Retracted skin is then released b y nondominant hand, and needle is w alked caudally *very gently* until it falls off inferior edge of rib.
  - Needle then advanced ~3 mm, which is at the costal groo ve.
  - *Aspirate to ensure neither blood nor air* (e.g., pneumothorax) is retur ned, then deposit 2–5 mL of anesthetic.
  - Patient should be obser ved for signs of systemic toxicity or pneumothorax (e.g., hypoxia, shor tness of breath or cough) for 30 minutes.
    - ▶ Consider chest x-ra y in debilitated patient or if in doubt.
    - ▶ Three-year retrospective char t study of 160 trauma patients indicates incidence of an individual block causing pneumothorax about 1.4% per individual intercostal ner ve block ed.
- Complications:
  - Bilateral intercostal blocks could impair respiration.
  - Pneumothorax.
  - Instances of multiple fractures and multiple blocks introduce potential for local anesthetic toxicity .

Upper Extremity Blocks

Wrist Block

■ Anatomy (see Figure 12.9):

● Ulnar ner ve:

▶ Originates from the medial cord of brachial plexus.

▶ Becomes superf cial in distal forear m bound by fascia to the *anterior surface* of f exor retinaculum and car pal tunnel.

▶ Travels across wrist joint passing abo ve ulnar styloid with ulnar ar tery.

▶ Innervation of f exor pollicis bre vis, abductor pollicis, palmaris bre vis, hypothenar muscles (abductor digiti minimi, f exor digiti minimi, and opponens digiti minimi), medial two lumbricals, and all interosseous muscles.

○ Sensation generally to h ypothenar surface, dorsal medial surface of palm, medial wrist, f fth digit, and medial half of four th digit.

● Median ner ve:

▶ Arises from par ts of the medial and lateral cords of the brachial plexus.

▶ Median ner ve trunk passes deep to f exor retinaculum into the car pal tunnel.

▶ NB: palmar branch of median ner ve crosses superf cial to f exor retinaculum and remains unaffected b y carpal tunnel syndrome.

▶ Motor control of thenar muscles (abductor pollicis bre vis, f exor pollicis brevis, and opponens pollicis) and f rst, second lumbricals.

▶ Sandwiched in the middle of the wrist betw een large tendons of palmaris longus and f exor carpi radialis (the prominent tendons on wrist f exion) at proximal wrist crease.

● Radial ner ve:

▶ Largest ter minal branch of posterior cord of brachial plexus.

▶ Divides into superf cial and deep branches at antecubital fossa.

▶ No motor control of hand muscles.

▶ Sensor y f bers inner vate lateral aspect of wrist and dor solateral aspects of hand.
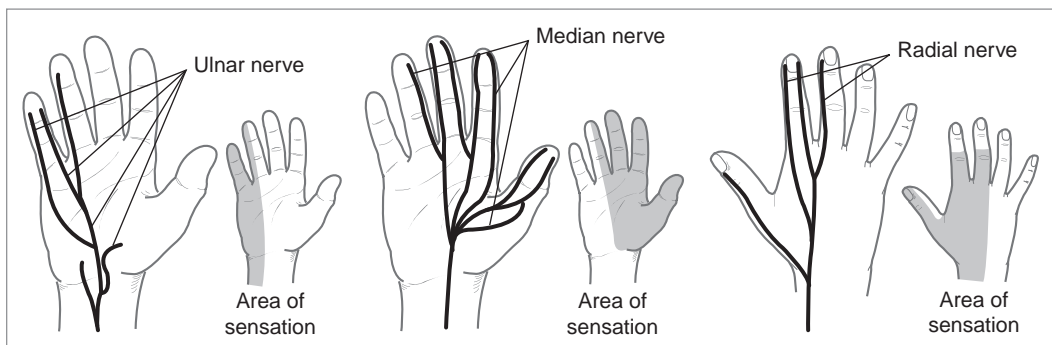
FIGURE 12.9: Anatomy and sensor y distribution of the ulnar , median and radial ner ves.

- Indications:
  - Anesthesia, depending on ner ves block ed for aspects of hands, wrists, and digits for facilitation of wound repair , casting , splinting , or hand surgeries.
  - Entrapment neuropathies.
- Blocking all of the ner ves below will anesthetize the entire hand, with the exception of a 2–3-cm patch on the volar aspect of the thenar eminence at the base of the thumb inner vated by lateral antebrachial cutaneous ner ve.
  - Anesthesia to this area ma y be pro vided by inf ltrating a wheal just proximal to this area – proximal to the f exor crease of the wrist.
- Approach:
  - Document neuro vascular status before beginning the procedure.
  - Prepare sterile site 1–2 cm proximal to the medial distal wrist crease.
  - Hand and wrist in supine position.

Radial Sensory Fibers

- On the dor sal surface, raise a wheal abo ve the radial styloid (3–5 mL of local anesthetic) and inf ltrate a subcutaneous tract as shown in Figure 12.10 . Fan out in order to create a f eld block anesthetizing the sensor y branches of the radial nerve with another 3–5 mL of local anesthesia.
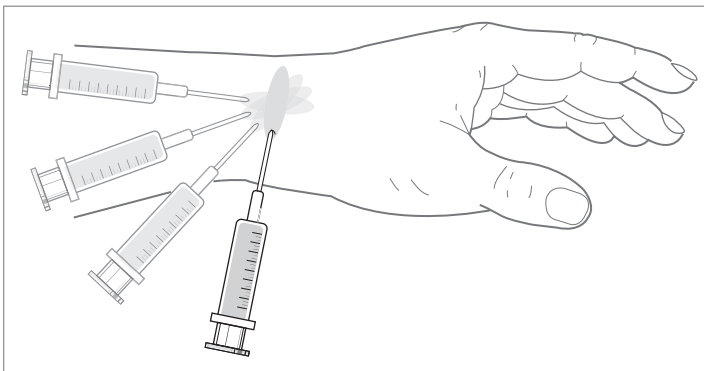
FIGURE 12.10: Inf ltration for radial ner ve block at the wrist.

Median Nerve

- Through the volar surface, inser t the needle about 1 cm ulnar to the palmaris longus (see Figure 12.11).
- Continue to advance the needle until a "pop" is felt as f exor retinaculum is penetrated, then inject 3–5 mL of anesthetic agent.
- If no pop is perceived and bone is contacted, withdraw the needle 2–3 mm, and inject the anesthetic.
- To increase block eff cacy, withdraw needle to le vel of skin, redirect needle laterally 30 degrees and again medially 30 degrees and reinser t, inf ltrating 1–2 mL of additional anesthetic in each of these two directions.
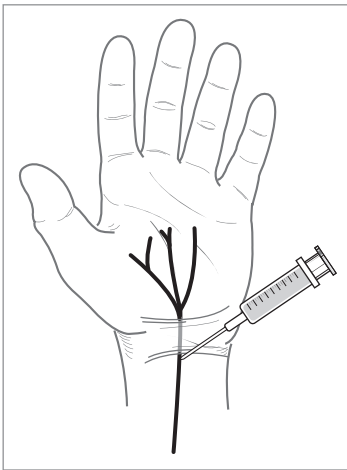
FIGURE 12.11: Median ner ve block at the wrist.

Ulnar Nerve

- Along the ulnar aspect of the wrist, inser t the needle just under the f exor car pi ulnaris tendon (see Figure 12.12).
- Advance needle to a depth of 1.5 cm, after aspiration, inject 5–7 mL of anesthetic solution.
  - Ulnar ar tery is immediately lateral to ulnar ner ve; achie ve negative aspiration before injecting .
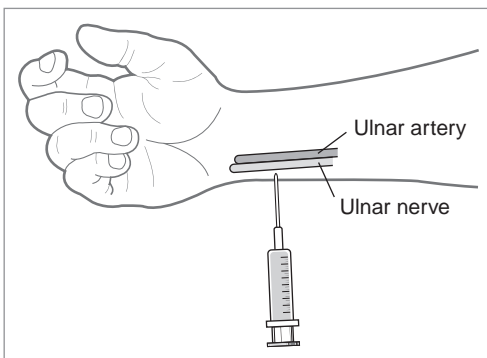
FIGURE 12.12: Ulnar ner ve block at the wrist.

Digital Blocks

- Anatomy (see Figure 12.13):
  - Volar digital ner ves emanate from median and ulnar ner ves, while the dor sal digital ner ves emanate from radial and ulnar ner ves.
  - Nerves cour se along both dor sal and ventral sides along each of the phalanges and on both medial and lateral sides.
- Indications:
  - Laceration repair.
  - Drainage of paron ychia.
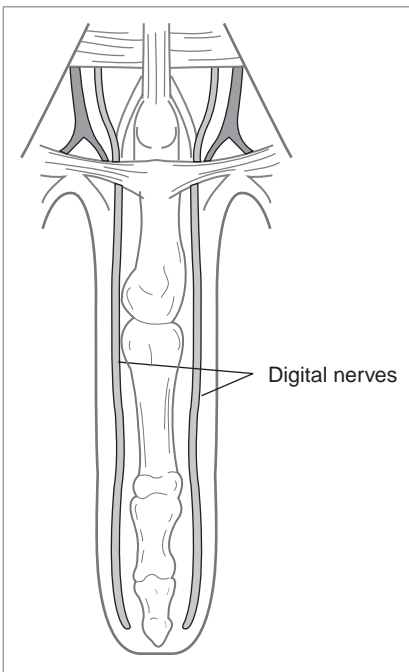
FIGURE 12.13: Anatom y of the digital ner ve.

- ● Fracture or dislocation reduction.
- ● Nail remo val and nail bed repair .
- ■ Documentation – note neuro vascular status prior to block.
  - ● Capillar y ref ll.
  - ● Two point discrimination (2–4 mm on volar pads; compare contralateral side).
  - ● Motor function.
  - ● Integrity of tendons.
- ■ Approaches (see Figure 12.14):
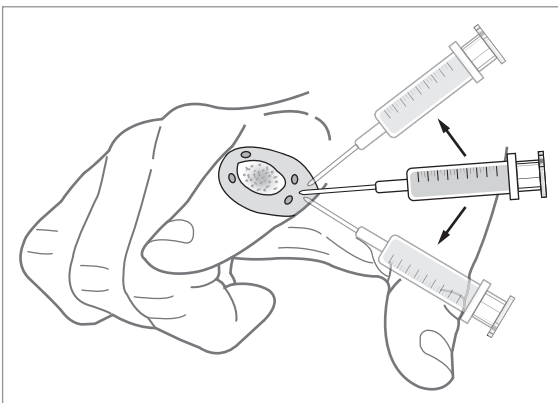
FIGURE 12.14: Approaches to digital ner ve block.

- Ring block:
  - ▶ Hand and f nger in prone position.
  - ▶ Insert needle into dor sal aspect of w ebspace at 45 degrees just distal to metacarpophalangeal (MCP) joint at the le vel where skin texture changes and advance needle tow ard volar end of the bone.
  - ▶ As bone is gently contacted, withdraw the needle 3–4 mm, aspirate, and then deposit 2 mL of anesthetic in a volar direction.
  - ▶ Withdraw and readvance needle to the dor sal aspect of the phalanx just distal to the MCP joint, aspirate, and then deposit an additional 1 mL.
  - ▶ Withdraw the needle, and repeat the procedure on the opposite side of the f nger.
  - ▶ In order to achie ve suff cient anesthesia w ait for 10–15 minutes before commencing procedure.
- ■ Transthecal/digital sheath (volar) block:
  - Requires only one injection but pro vides anesthesia to both volar and dor sal surfaces.
    - ▶ Does not anesthetize proximal dor sal surface to le vel of DIP joint.
    - ▶ Typically requires only 1.5–3 mL of anesthetic to block entire f nger versus approximately double that in a classic ring block.
    - ▶ Relatively less risk of trauma to neuro vascular bundles than classic ring block.
  - Deposit anesthetic into the f exor tendon sheath in order to anesthetize the digital ner ves.
  - Approach:
    - ▶ Inject just distal to the MCP joint (slightly proximal to the f rst digital crease).
    - ▶ Insert needle at an angle of 90 degrees to proximal digital crease *in the midline of the digit,* avoiding neuro vascular bundles.
      - ○ Needle should penetrate skin, subcutaneous tissue, tendon sheath, and the f exor tendon.
      - ○ Advance the needle until bone is gently contacted.
      - ○ Withdraw needle 2–3 mm, lea ving the tip of the needle within the f exor tendon sheath.
      - ○ Expect little or no resistance initially; inf ltrate 1.5–3 mL of anesthetic solution; expect increasing resistance and possible slight f exion as the sheath f lls.
      - ○ Patient should experience digital anesthesia within 2–3 minutes.
- ■ Complications:
  - Ischemia.
  - Neuropathy, intraneural injection.

Lower Extremity Blocks

Femoral Nerve Block
- ■ Anatomy (see Figure 12.15):
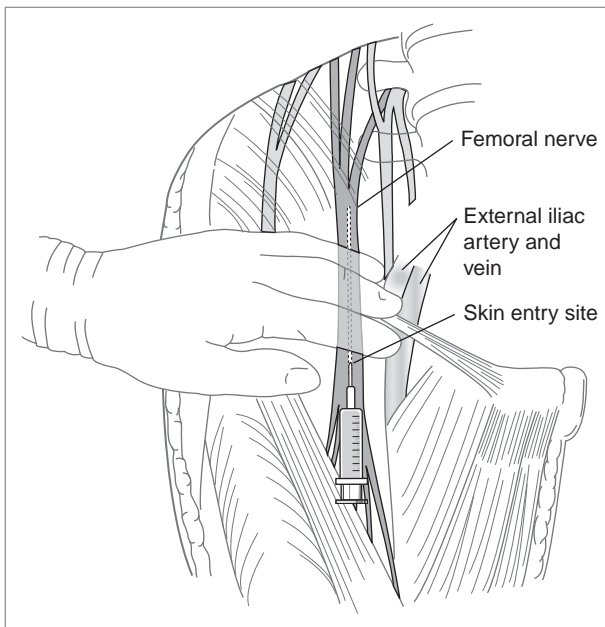
FIGURE 12.15: Approach to femoral nerve block (FNB).

- Femoral nerve is the largest terminal branch of lumbar plexus, comprised of dorsal divisions of anterior rami of L2–L4.
- Courses through psoas muscle, leaving psoas at its lateral border, and then descending into the thigh in the groove between psoas and iliacus muscles, coursing beneath the inguinal ligament.
- A few centimeters below the inguinal ligament, femoral nerve divides into anterior and posterior branches.
  - ▶ At this level, the femoral nerve is just lateral and slightly posterior to the femoral artery.
  - ▶ After dividing into anterior and posterior branches:
    - ○ Anterior branch of femoral nerve:
      - ➢ Supplies motor innervation to the sartorius and pectineus muscles.
      - ➢ Provides sensation to skin of anterior and medial thigh.
    - ○ Posterior branch of the femoral nerve.
      - ➢ Supplies motor innervation to the quadriceps.
      - ➢ Sensory innervation to the medial aspect of the lower leg (saphenous nerve).
- ■ Indications:
  - Anesthesia to anterior thigh and medial leg.
    - ▶ Reliable block to femoral and lateral femoral cutaneous nerves; inconsistently blocks obturator nerve.

- Proximal femur and hip fractures, especially in elder ly people.
  - ▶ May allow for reduced use of parenteral analgesia.
- Analgesia for procedures on knee and thigh.
- Approach (see Figure 12.15):
  - Use of ultrasound or ner ve stimulator is recommended.
  - Identify the femoral ar tery.
  - Mark the site and prepare a broad sterile f eld to include the inguinal crease.
  - Raise a wheal 1 cm lateral to the femoral ar tery and at the le vel of the inguinal crease.
  - Advance the needle and direct it slightly cephalad, stopping approximately 2–3 cm below the skin, the typical depth of the femoral ner ve.
    - ▶ If the patient experiences paresthesias o ver the anterior thigh caused by the activation of the femoral ner ve, withdraw the needle slightly just enough for the paresthesias to stop.
  - Inject 5 mL aliquots of local anesthesia, aspirating betw een each injection, to a total of 20–30 mL of local anesthetic.
  - Typical time to onset of block is 10–30 minutes depending on type and dosage of local anesthetic administered.

"Three-in-One" Block

- Used to impro ve perfor mance of femoral ner ve block (FNB) especially in relie ving pain sensation from the obturator ner ve.
- Indication:
  - To anesthetize the femoral, obturator, and lateral femoral cutaneous ner ves in patients with hip fractures.
  - Anesthesia of obturator ner ve, in par ticular, may be w eak in the typical FNB described abo ve.
  - The obturator ner ve receives sensor y impulses from the medial aspect of the thigh and pro vides motor inner vation of the adductor muscles.
- Approach:
  - Blockade perfor med in the same manner as FNB described abo ve (Figure 12.15).
  - Prior to inf ltration, the hand compresses the area just caudad to the needle to promote cephalad spread of the local anesthetic.
  - Pressure distal to the injection site continues for a total of 5 minutes during and/or after the injection is made.
  - This achie ves blockade of all three ner ves: the femoral, the lateral femoral cutaneous, and the obturator .
  - The use of ultrasound pro vides better technique.

Ankle and Foot Blocks

- Anatomy (see Figure 12.16):
  - Remember the "s" implies SENSOR Y.
    - ▶ Superf cial peroneal, Saphenous, and Sural are 100% Sensor y
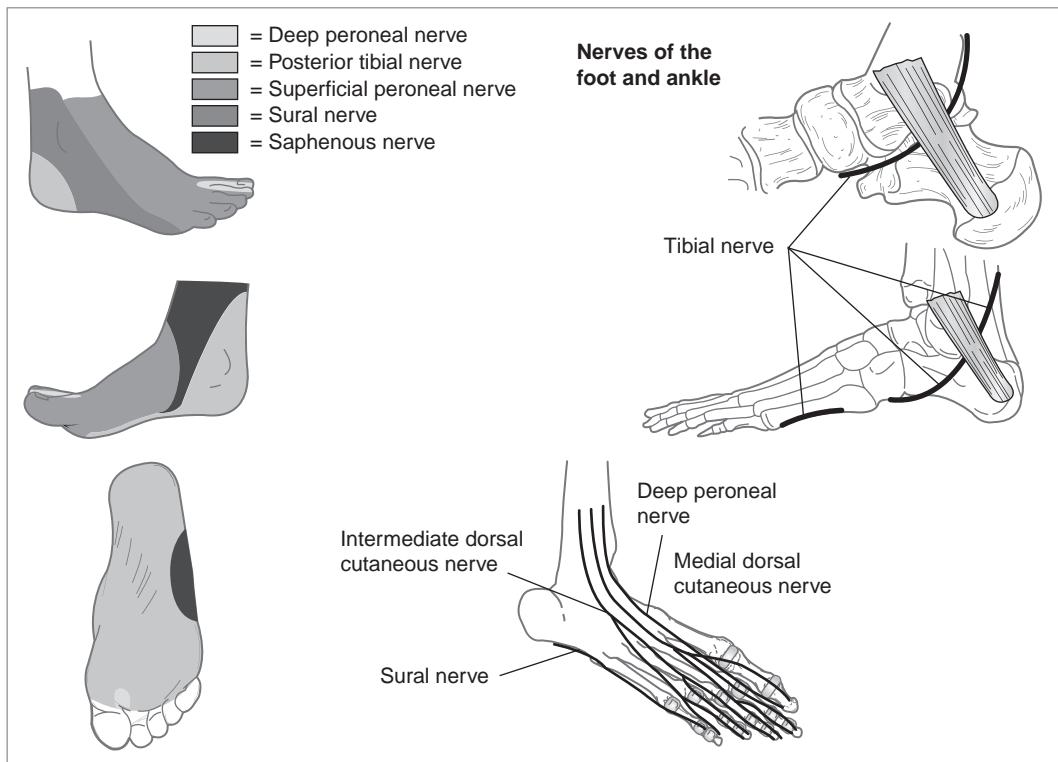
FIGURE 12.16: Inner vation of the foot.

- Complete block of sensation and motor inner vation of the foot can be achieved by blocking f ve ner ves, all of which originate with the sciatic nerve:
  - ▶ Posterior tibial ner ve:
    - ○ Divides into lateral and medial plantar ner ves, calcaneal sensor y branches, and ner ve to the abductor digiti quinti.
    - ○ Provides motor inner vation to muscles on plantar side of foot, and sensor y inner vation over plantar surfaces.
  - ▶ Superf cial peroneal ner ve:
    - ○ Branch of common peroneal ner ve.
    - ○ Purely sensor y.
    - ○ Sensor y inner vation only to dor sal surface of the foot, lateral great toe, and toes two to four .
  - ▶ Deep peroneal ner ve:
    - ○ Lateral branch inner vates extensor digitor um brevis and extensor hallucis bre vis.
    - ○ Sensor y inner vation to the f rst web space.
    - ○ Damage including from lateral injur y to knee ma y result in foot drop.

> ▶ Sural nerve:
>> ○ Branch of combined peroneal nerve (anastomotic branch) and tibial nerve (median sural nerve).
>> ○ Purely sensory.
>> ○ Sensory innervation to dorsolateral surface of foot, including toes four and five.
> ▶ Saphenous nerve:
>> ○ A terminal branch of the femoral nerve.
>> ○ Purely sensory.
>> ○ Located approximately 1–2 cm anterior to medial malleolus at the ankle.
>> ○ Sensory innervation of medial aspect of ankle, foot, and great toe.

■ Applications/indications:
- Laceration or wound exploration or repair.
- Manipulation of dislocated ankles.
- Incision and drainage of abscesses.
- Toenail repair.
- Fracture reduction.

■ Approach:
- Document neurovascular status before and immediately after block.
- When administering multiple blocks, block posterior tibial nerve first, since it is the largest.
- Ideally, the patient should be supine, with ankle supported by pillow or rolled sheet.

Posterial Tibial Block

- Nerve lies in the flexor retinaculum (tarsal tunnel), approximately 1–2 cm posterior to medial malleolus (see Figure 12.17).
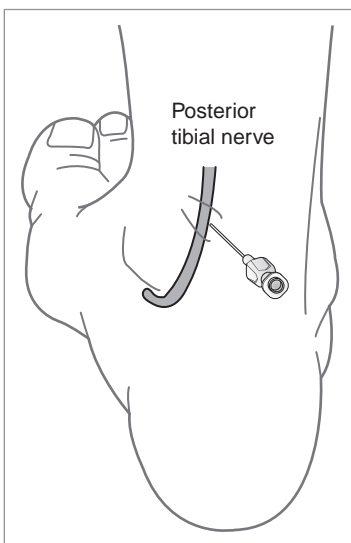
FIGURE 12.17: Posterior tibial nerve block.

- Palpate posterior tibial pulse and inject to its posterior   , aspirating to ensure against intra vascular injection.
- Inject 5–7 mL, simultaneously palpating to ensure that tendon sheath is f   lling with anesthetic.
- Wait at least 1–2 minutes before commencing other blocks to discer   n signs of systemic toxicity or h ypersensitivity.
- By waiting 10 minutes, at least par tial anesthesia of the foot ma y be achieved, potentially reducing the pain of other blocks due to o   verlap of sensor y f elds.

Deep Peroneal (F ibular) Ner ve Block
- Palpate the dor sal pedis pulse (see   Figure 12.18).
  - Dorsal pedis pulse is located betw   een the extensor hallucis longus and the extensor digitor um longus.
- Deep peroneal ner ve lies lateral to the dor sal pedis pulse.
- Needle should penetrate the skin per   pendicularly and advance to the le vel of the tarsal bones.
- Slowly inject 5–7 mL of local anesthetic.
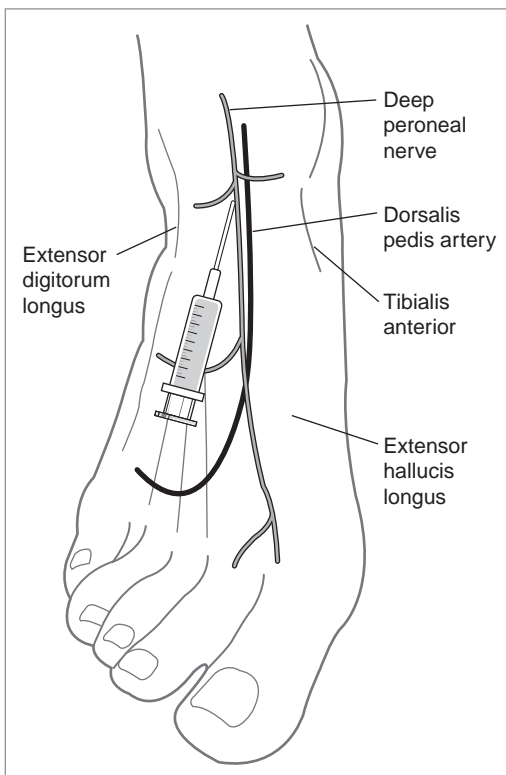
FIGURE 12.18: Deep peroneal ner ve block.

Superf cial Peroneal Nerve

■ Note the broad distribution of sensor y f bers across the anterior ankle, in addition to the lateral branch, which is appreciated b y plantar f exing the foot and the fourth toe (see Figure 12.19).

■ Anesthesia is accomplished *by subcutaneous infiltrations forming a ring between the malleoli*, with a total of 5–10 mL of local anesthetic.
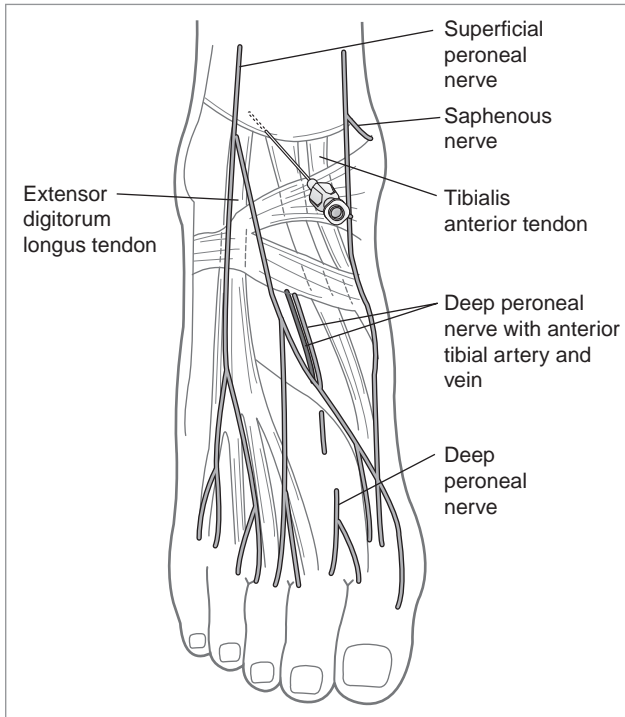
FIGURE 12.19: Superf cial peroneal ner ve block.

Saphenous Ner ve

■ Saphenous ner ve courses subcutaneously along the anteromedial ankle betw een the tibialis anterior tendon and medial malleolus (see Figure 12.20).

■ Positioned ~2 cm superior and 1 cm anterior to the medial malleolus.

■ Inject along this line subcutaneously , approximately 5–7 mL.

Sural Ner ve Block

■ Identify the area betw een the Achilles tendon and the superior border of the lateral malleolus (see Figure 12.21).

■ After aspirating , inject 5–6 mL of anesthetic into a band from the superior aspect of the malleolus to the Achilles tendon.

Superficial peroneal nerve

Saphenous nerve

Extensor digitorum longus tendon

Tibialis anterior tendon

Deep peroneal nerve with anterior tibial artery and vein

Deep peroneal nerve
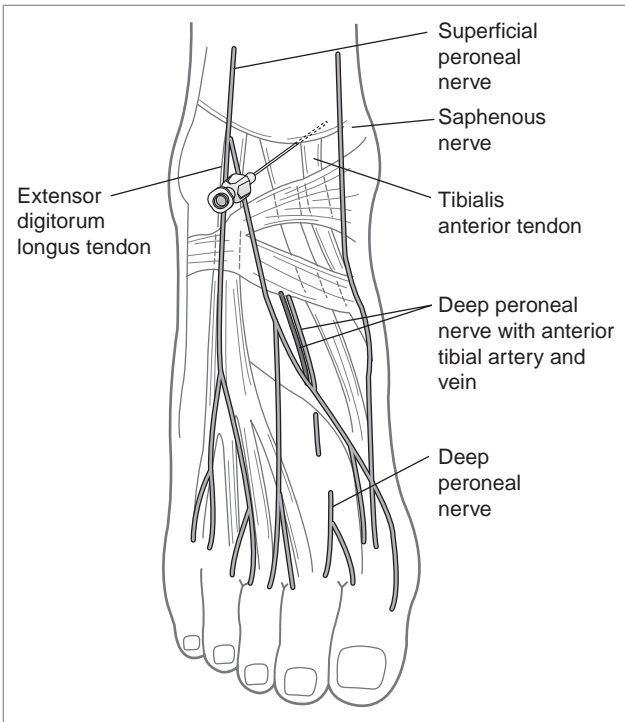
FIGURE 12.20: Saphenous ner ve block.
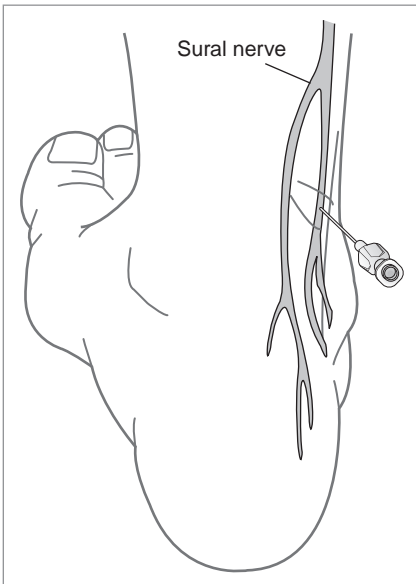
Sural nerve

FIGURE 12.21: Sural ner ve block.

Other Blocks

Bier Block (Intra venous Regional Anesthesia)

- Indications:
  - Alter native to general anesthesia for shor t procedures in volving upper and lower limbs.
    - ▶ Maximum safe duration is 60 minutes.
  - Consider for large lacerations, fracture, or dislocation reductions of forear m, wrist, hand, and other procedures on upper or low er extremities lasting <60 minutes.
    - ▶ Used by Farrell et al., safely in children as young as 2 year s and elder ly patients as old as 86 year s by emergency ph ysicians administering 1.5 mg/kg (administered as 0.5% = 5 mg/mL) "mini-dose" of lidocaine for upper extremity fractures and dislocations.
- In all cases, full neurological function retur ned within 10 minutes of the end of the procedure.
- Advantages:
  - Avoids general anesthesia.
  - Fasting not required.
  - Bloodless operating f eld.
  - Muscle relaxation.
- Contraindications:
  - Allergy or hypersensitivity to lidocaine.
  - Homozygous sickle cell disease.
  - Severe Raynaud's or Buerger' s disease.
  - Crushed or otherwise h ypoxic limb.
  - Severe obesity and vascular disease, which a tour niquet could aggra vate.
- Risks:
  - Transient paresthesias, mottling, and temperature changes of the limb.
  - Risk of systemic toxicity from premature release of tour niquet.
  - Vasovagal reactions.
- Procedure:
  - Lidocaine *without* epinephrine is the anesthetic of choice. Longer-acting agents such as bupivacaine should *not* be emplo yed.[55]
    - ▶ Dilute 1% lidocaine to half strength (0.5% = 5 mg/mL with equal par ts of normal saline).
    - ▶ Dosage in children and elder ly should be 1.5 mg/kg .
    - ▶ Dosage in health y adults can be titrated to 3 mg/kg .
    - ▶ In the e vent that insuff cient analgesia is achie ved, supplement the intravenous infusion with additional plain nor mal saline in order to stimulate circulation of the lidocaine within the extremity .
- Equipment:
  - Double-cuff pneumatic tour niquet. *Do not use standard blood pressure cuffs.*
    - ▶ The standard blood pressure cuff ma y suddenly release resulting in a systemic intra vascular anesthetic infusion.

- A 50-ml syringe with plastic tube and 22-gauge butterfly needle.
- Cotton or felt cast wrap to pad the limb beneath the tourniquet.
- Cardiac monitoring should be routine.
■ Procedure (upper limb):
  - Place the deflated double-cuff tourniquet on the patient's upper arm.
    ▶ Use cast padding under the cuff to prevent bruising.
  - Insert butterfly cannula (22 gauge or smaller) into a vein on the injured extremity *at least 10 cm distal* to the pneumatic tourniquet and close to the site of injury. Tape in place.
  - Elevate the injured extremity for 3–4 minutes and wrap it from distal to proximal with compression bandage to exsanguinate the limb's blood supply.
  - Inflate the distal tourniquet to about 250–300 mm Hg (patient's systolic blood pressure + 100 mm Hg as guide).
    ▶ In children, use systolic blood pressure +50 mm Hg.
    ▶ Avoid this procedure in elderly obese individuals suspected to have significant arteriosclerosis.
  - Infuse the calculated dose of anesthetic via the butterfly catheter.
  - When the infusion is complete, remove the IV cannula and maintain pressure and bandage the IV site.
  - Carry out the procedure.
  - If during the course of the procedure the patient experiences severe pain at the site of the tourniquet, then inflate the proximal tourniquet to the equivalent pressure of the distal tourniquet, and *only* then deflate the distal tourniquet.
  - Once the anesthetic has been infused, the tourniquet *must remain inflated* (either the distal or proximal cuff) *continuously for at least 30 minutes.*
  - Once 30 minutes has elapsed or the procedure has been completed, whichever is longer, *but not more than 60 minutes,* then the tourniquet should be released through a *cycling process:*
    ▶ Release the pressure on the tourniquet for 5–10 *seconds* and then reinflate the cuff to its occlusion pressure for 1–2 minutes.
      ○ Repeat this release/reinflate cycle three times in order to prevent a rapid bolus of lidocaine into the systemic circulation.
  - The patient will probably need additional analgesics after the procedure since the lidocaine typically dissipates quickly after tourniquet release.
  - The patient should be observed for at least 30 minutes postprocedure for systemic reactions or toxicity.

Hematoma Block
■ Concept: local anesthetic delivered by infiltration directly into the hematoma, which has formed at the site of the fracture.
■ Indications:
  - Closed fractures, especially those of the upper extremity.
    ▶ Do not perform a hematoma block through contaminated or infected soft tissues.

- ■ Advantages:
  - ● Can be executed ver y quickly.
  - ● Avoid procedural sedation.
- ■ Procedure:
  - ● Prepare the skin site o verlying the fracture with antiseptic solution.
  - ● Inject 5–15 mL of plain 1% lidocaine, or 5–10 mL of plain 2% lidocaine into the hematoma for ming at the fracture site and through the disr upted periosteum.
  - ● In this specif c case, aspirate to *ensure* that blood is retur ned in the syringe, indicating appropriate positioning within the hematoma.
  - ● Wait 5–10 minutes for onset of block.

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Pediatric Pain Assessment and Nonpharmacological Therapy

Tracy Akitt, Sadie Bartram, Sheryl Christie, and Karen Paling

Introduction

- Many factors can contribute to how a child perceives pain and copes with anxiety-provoking situations.
- The pain response for a particular patient is individual and learned through social learning and experience.
- Unmanaged pain can lead to:
  - Short-term consequences such as children displaying heightened sensitivity to subsequent medical procedures.
  - Long-term consequences such as the development of hyperalgesia.
- Pain endured as a child correlates with adult behaviors such as pain responses, fear, coping effectiveness, and willingness to seek medical attention.

Understanding Coping Mechanisms

- Adequate interventions to assess and manage childhood pain are essential.
- Use a multimodal and multisystem approach when treating a child in pain.
- Involve skilled and specially trained professionals such as child life specialists to assist with pain assessments and interventions, and also to assist pediatric patients and their caregivers to cope.
- Through appropriate assessment, coping behaviors can be identifed as to how a child responds to anxiety and distress.
  - According to Kuttner (1996), there are four types of coping behaviors:
    - *Catastrophizers* – view painful events in a very negative way. Although they ask many questions to gain information, the information is not comforting.
    - *Sensitizers* – fnd the information is comforting to them. They are able to gather lots of information to develop more coping strategies to handle painful events.
    - *Minimizers* – only process information given in small amounts. They are able to use the information to help themselves cope with situations and may require more time to develop coping strategies.
    - *Deniers* – prefer not to have any information. This can be diffcult when it is time for the stressful event to occur (i.e., surgery).

- It is impor tant to note that these coping beha viors are not static and can change o ver time depending on the situation and the suppor ts provided.

Use of Child Life Specialists in the Emergency Depar tment

■ Implementing child life programs in the emergency depar tment (ED) ha ve been shown to relie ve the stress and anxiety associated with an ED visit.

■ Procedure teaching and suppor t, guided imager y, distraction, and other techniques used during procedures ha ve been shown to:
- Improve coping and decrease upset beha vior (of patients and parents).
- Decrease medication required for sedation or analgesia.
- Improve staff eff ciency.
- Improve parent satisfaction.

Pediatric Pain Assessment

■ Pain assessment in children should be an ongoing process.

■ Assessment tools should be used to under stand the le vel of pain a patient is experiencing (see examples listed in Tables 13.1 –13.5).
- Many hospitals ha ve a variety of pain assessment tools readily a vailable for their staff. Check with your local resources.

■ Pain in children is measured b y:
- What the child sa ys.
- What an obser ver sees.
- The child's ph ysiological responses to pain.

■ A child's cognitive and language de velopment pla y a large role in their ability to "self-repor t" pain, thereby making it necessar y to rely more hea vily on obser vational and bioph ysiological measures for infants, toddler s, and patients with neurocognitive impair ments.

■ Self-repor ting considerations:
- A child's pain is measured simply b y asking the child how much he or she hurts, where it hur ts, and for how long it has hur t.
- The child is the best source of infor mation, as he or she is tr uly the only person who knows the intensity of the pain.
- A child's self-repor t, when possible, should be the primar y measure of pain assessment.

■ Factors that will inf uence a child's pain response include:
- Cognitive, emotional, and language de velopment, as this will inf uence how children perceive and under stand pain (see Tables 13.1 .–13.5).
- Developmental age and temperament.
- Previous pain experiences and a child' s memor y in relation to these experiences.
- Caregiver in volvement.

TABLE 13.1: Infant pain assessment and nonphar macological
pain management strategies

Infant (bir th–18 mo)

Cognitive perceptions of pain

0–3 mo
- No apparent under standing of pain; memor y for pain lik ely but not conclusively demonstrated; responses appear mostly ref exive.

3–6 mo
- Response to pain supplemented with sadness and anger .

6–18 mo
- Developing fear of painful situations; ear ly stages of being able to localize pain (through touching/pointing).
- Understanding of common words for pain e volving such as "owie, ouchie, booboo."
- It is impor tant to remember that words for pain var y with cultures and languages.
- Ask parents the words the y frequently use at home.

Pain assessment tools

Self-rating – Not possible.
Biophysiological – Heart rate, oxygen saturation, palm sw eat.
Observations (beha vioral):
- Cry (length and type), facial expressions, body mo vement, parent ratings/perceptions.
- Neonatal Facial Action Coding System (N-F ACS).
- Children's Hospital of Easter n Ontario P ain Scale (CHEOPS) – (1–7 yr).
- Faces, legs, activity, cr y, consolability (FLA CC) – (0 –7 yr).

Considerations for procedural preparation

Information to include:
- Important to prepare the parents of infants. Their comfor t to sta y during the procedure should be assessed, and their presence should be encouraged.
- Give parents infor mation on the steps of the procedure, the responses their child could exhibit, and where to be positioned during the procedure.

Developmental considerations:
- Infants are at a stage of building a secure attachment to a parent.
- Including parents in all aspects of care and to be their source of comfor t is highly impor tant.

Without these inter ventions:
- Infant's stress le vels can increase in absence of nur turing and comfor ting responses.
- Failing to relie ve the pain can cause mistr ust and fear tow ard caregiver s.
- Immediate effects such as ir ritability, fear, and sleep disturbance ma y occur.
- Effects include dela yed healing , impaired emotional bonding , and altered response to subsequent painful experiences.

Environmental considerations

Caregiver in volvement:
- Encourage parental par ticipation during the procedure to comfor t their child.
- Separation from parents can heighten the distress of infants.
- If the parents are not comfor table or unable to sta y, it is impor tant for them to retur n as quickly as possible.

Health care wor ker behaviors:
- Changes or missing their usual routine of sleeping , eating , and bathing can be distressing for the child.
- This routine should be respected b y the health care wor kers by planning procedures to be implemented to allow the child' s routine to continue as much as possible, and for the child' s routine care to be pro vided by their parents.

Health care setting:
- Keep the room quiet and calm (voices low).
- Minimize the number of people in the room.

(continued)

TABLE 13.1: Infant pain assessment and nonphar macological
pain management strategies (Continued)

Cognitive and behavioral distraction

Comfort suggestions:
■ Pacif er, blanket, favorite item
■ Swaddling position, massage, touch
Distraction play materials:
■ Rattles, pop-up to ys, light-up to ys, music, bubble blowing
Distraction con versation:
■ Singing , action rh ymes, positive comfor ting words
Relaxation methods:
■ Massage, touching , blowing bubbles, blowing bubbles a way

Developmental Age
■ Younger children (younger than 7 year s):
  ● Rate their pain as higher than older children and displa y more distress during a painful e vent.
  ● Level of distress and pain ratings ma y be due to:
    ▶ Their de velopmental le vel of not under standing the pur pose of a painful procedure.
    ▶ The fact that the y do not grasp that often the procedural pain will be o ver quickly.
    ▶ Their limited ability to use cognitive coping strategies.
■ Older children (older than 7 year s):
  ● Are better able to distinguish the difference betw een "pain," "unpleasantness, " and "fear ."
  ● May feel a need or ma y want to appear stoic, thus pain ratings (both self- and obser ver-reported) ma y be low er than younger children' s pain ratings.

Temperament
■ Temperament is a concept def ned by Thomas and Chess that describes a person's beha vioral style.
■ Temperament theor y looks at how children will respond to an exter nal stimulus.
  ● Three temperament categories are as follows:
    ▶ Diff cult
    ▶ Easy
    ▶ Slow to w arm up
  ● Children considered b y parents to ha ve "diff cult" temperament w ere rated as having greater pain responses than children who w ere found to ha ve "easy" temperaments when requiring immunizations.
■ Health care pro viders need to be f exible and adapt their inter ventions and modify the en vironment to better match the child' s temperament.

TABLE 13.2: Toddler pain assessment and nonpharmacological pain management strategies

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Toddler (18 mo–3 yr)

Cognitive perceptions of pain

18–24 mo
- Use the word "hurt" (language/culture specific) to describe pain and noncognitive coping strategies.

24–36 mo
- Beginnings of pain description and attribute an external cause to pain.
- Threat of immediate pain is overwhelming, particularly in situations where the child has a recent previous experience with that same or similar pain stimulus (i.e., IV start or blood work).
- Future benefit of procedure not understood.

Pain assessment tools

Self-rating – Difficult to assess as there is a large developmental range in abilities.
Biophysiological – Heart rate, oxygen saturation.
Observations (behavioral):
- Children's Hospital of Eastern Ontario Pain Scale (CHEOPS) – (1–7 yr).
- Faces, legs, activity, cry, consolability (FLACC) – (0–7 yr).

Considerations for procedural preparation

Information to include:
- Sensations and the steps of the procedure need to be explained to the parents and child.
- Use simple, developmentally appropriate language.
- Allow the child to explore the equipment used for upcoming procedures.

Developmental considerations:
- Toddlers are at a developmental stage where they want to develop a sense of autonomy.
- Allowing toddlers explore the hospital room helps them to express this need for independence.

Without these interventions:
- Can respond with resistance and uncooperativeness.
- Since they can remember painful procedure, they could react the same way in future procedures.

Environmental considerations

Caregiver involvement:
- Allow parents to comfort versus restrain their child during procedures.
- Having comfort items available (e.g., a blanket or stuffed animal) is important when parents cannot be present.
- Allow parents to provide routine (daily) care to their child.

Health care worker behaviors:
- Since toddlers need to feel they are doing things independently, it is important to give the child a role in their health care. It can be something simple like helping put on a blood pressure cuff.
- Minimize the use of restraint. If it is necessary, it should be done seconds before the procedure starts.

Health care setting:
- Exploring the room can be normal behavior for toddlers.
- This curiosity can be used as a means of distraction for the child.
- Keep the room quiet and calm (voices low), and minimize the number of people in the room.

Cognitive and behavioral distraction techniques

Comfort suggestions:
- Pacifier, blanket, favorite item.
- Position of comfort sitting "front to front" or "back to front" in parent's arms, in a chair, or on the bed.
- Massage, touch

Distraction play materials:
- Pop-up books, light-up toys, music, musical/sound toys, motion windup toys

Distraction conversation:
- Singing, action rhymes, storytelling, reading books, counting

Relaxation methods:
- Massage, touching, blowing bubbles, blowing bubbles away

TABLE 13.3: Preschool pain assessment and nonpharmacological pain management strategies

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Preschool (3–6 yr)

Cognitive perceptions of pain

36–60 mo
■ Can give gross indications of the intensity of pain and beginning to use more descriptive adjectives and attach emotional terms such as "sad" or "mad" to the pain.

Pain assessment tools

Self-rating – 36–60 mo – Gross indications of pain (no pain, a little pain, a lot of pain); can mark site of pain on a body outline.
"Poker Chips" – Children (ideally, over 60 mo) are asked to show how much pain they are having by using one to four red poker chips to represent their pain.
"OUCHER" Scale – Used in children over 36 mo in which the children point to one of six faces represented on the scale to indicate which face represents the level of pain they are having.
Biophysiological – Heart rate, oxygen saturation.
Observational (behavioral):
■ Children's Hospital of Eastern Ontario Pain Scale (CHEOPS) – (1–7 yr).
■ Faces, legs, activity, cry, consolability (FLACC) – (0–7 yr).
Noncommunicating Children's Pain Checklist (NCCPC) – To be used with developmentally delayed children 3–18 yr old.

Considerations for procedural preparation

Providing the information:
■ Disseminate the information with the child and parents present.
■ Include sensory information and the steps of the procedure.
■ Preschool aged children can have the tendency to fantasize information into scary or overwhelming thoughts. Information about procedures should be given immediately before the procedure.
■ Be honest with the child about the pain the procedure may cause, but be mindful of the words used.
Special considerations:
■ Child may perceive pain as a punishment for something they did wrong. This should be addressed in preparing the child.
■ Using real medical equipment in the preparation helps the preschooler to understand and is helpful in detecting the child's fears.
Without these interventions:
■ The preschoolers can continue to believe that they have done something wrong, and continue to view the hospital and associated procedures as punishment.

Environmental considerations

Caregiver involvement:
■ Allow parents to be the primary source of comfort.
■ Parents can also be taught how to encourage the child's coping strategies and utilize distraction techniques (see below) throughout the procedure.
Health care worker behaviors:
■ Can engage in simple communications with children of this age. This helps to build rapport and trust with both the child and the parents.
■ If restraint is necessary, it is important to tell the child that you are helping them hold still and only do so seconds before the procedure is to start.
Health care setting: Allow the child to explore the room. Keep the room quiet and calm (voices low), and minimize the number of people in the room.

(continued)

TABLE 13.3: (Continued)

Cognitive and behavioral distraction techniques

Comfort suggestions:
■ Blanket, favorite item
■ Position of comfor t sitting "front to front" or "back to front" in parent' s ar ms, in a chair, on the bed, or in sitting position with parent next to child

Distraction pla y materials:
■ Pop-up books, light-up to ys, music, musical/sound to ys, bubble blowing , motion windup to ys, search and f nd books, videos interactive technolog y devices

Distraction con versation:
■ Singing, action rh ymes, stor ytelling, reading books, counting, talking about fa vorite things, jokes/humor

Relaxation methods
■ Massage, touch, bubble blowing , blowing bubbles a way, imagining blowing out candles, imagining blowing up balloon, pinwheel blowing , par ty blowers
■ Guided imager y (imagining a special fa vorite place)

■ It is impor tant to assess and collaborate with parents with regards to how they feel their children' s temperament ma y affect their ability to cope with their pain.

Previous Experience

■ The child's pre vious experience with painful e vents must be tak en into account b y health professionals during pain assessment.

■ Bijttebier and V ertommen (1998) suggest that a child' s pain response is predominantly shaped b y the quality of the pre vious experience than the presence of previous experience with a painful e vent.
  ● Histor y of negative pain experiences:
    ▶ Show higher le vels of anxiety before the procedure.
    ▶ Display more distress.
    ▶ Less cooperative during the procedure.
  ● Histor y of positive pain experiences:
    ▶ More cooperative.
    ▶ Develop effective coping strategies, which help to reduce pain and allow the child to gain a sense of master y over the situation.

Memory

■ Infants subjected to man y painful procedures become conditioned to anticipate pain.

■ The memor y of a painful e vent may be distor ted, often o verestimating the pain felt during the procedure.

■ Memories of a painful experience can be reframed, which can help to reduce distress for subsequent procedures.

■ Consult a health care professional such as a child life specialist to help the child reframe a painful e vent, discuss the effectiveness of coping strategies used during the procedure, and set the stage for subsequent procedures.

TABLE 13.4: School age pain assessment and nonphar   macological
pain management strategies

School age (6–12 yr)

Cognitive perceptions of pain

■ Can explain where a pain hur ts by grossly explaining and pointing .
■ Can explain what happened (histor y) to better under stand what is wrong .

Pain assessment tools

Self-rating – Coping can impact their ability to decipher ph ysical pain and anxiety .
Wong-Baker FACES scale – This scale uses a series of six faces that progressively appear more uncomfor   t-
   able. The faces also ha ve a numerical value associated with them (0,   2, 4, 6, 8, 10).
"Poker Chips" – See T able 13.3.
"OUCHER" scale – See T able 13.3.
Biophysiological – Heart rate, oxygen saturation.
Observational:
■ Children's Hospital of Easter n Ontario P ain Scale (CHEOPS) – (1–7 yr).
■ Faces, legs, activity, cr y, consolability (FLA CC) – (0–7 yr).
■ Procedure Beha vior Checklist – (6–18 yr).
Noncommunicating Children's Pain Checklist (NCCPC)   – To be used with de velopmentally dela yed children
   3–18 yr old.

Considerations for procedural preparation

Providing the infor mation:
■ Parents should continue to be included in preparation,   and in volved during the procedure.
■ Be open and honest about pain and discomfor t that ma y occur.
■ If pain does occur when the y have been told it will not, the child ma y lose tr ust in the health care wor ker.
Special considerations:
■ At this age, children begin to under stand more about pain, but ha ve a fear of death and disability associ-
   ated with pain.
■ Children should therefore be encouraged to ask questions and express feelings.
■ Thinking patter ns at this age are still ver y concrete, therefore words should be chosen carefully .
Without these inter ventions:
■ Distress can be indicated b y crying, anger, fear, and withdra wal.
■ If the child has not been prepared, they may not utilize their coping strategies w ell and could become more
   anxious and lose tr ust in the care pro viders.
■ Explain sensations the y will experience during procedures before the y occur, allowing time for the child to
   ask questions.

Environmental considerations

Caregiver in volvement:
■ It has been thought that children of this age can be without their parents for a longer period of time,   but
   parental presence remains impor tant throughout hospitalization. Because of the psychosocial challenges a
   hospital visit presents, regression can be seen in the child,   and their need for parental presence continues.
■ Parents can also pla y an active role to help their children cope at this age.
Health care wor ker behaviors:
■ This age group is at a de velopmental stage where the y want to de velop a sense of accomplishment.
■ Should offer choices during the child' s hospital sta y.
■ These choices could include things lik e choosing to sit up or la y down during a procedure.
Health care setting:
■ School-aged children are able to under stand the relationship betw een e vents and experiences.
■ Keep in mind that presenting an IV tra y, for example, before being ready to star t the procedure could poten-
   tially bring about anticipator y anxiety.
■ Keep the room quiet and calm (voices low),   and minimize the number of people in the room.

(continued)

TABLE 13.4: (Continued)

Cognitive and behavioral distraction techniques

Comfort suggestions:
- Blanket, favorite item
- Position of sitting with parent next to child

Distraction play materials:
- Music, musical/sound toys, bubble blowing, motion windup toys, search and find books, videos, video games, squishy balls, interactive technology devices

Distraction conversation:
- Reality conversation, reading books, counting, talking about favorite things, jokes/humor

Relaxation methods:
- Massage, touch
- Breathing strategies – blowing bubbles, deep breathing, blowing up balloon in stomach
- Guided imagery (imagining a special favorite place)

Caregiver Involvement in Assessment

- When self-report is not possible (child is too young or cognitively impaired), caregivers should be regarded as "the expert" on their child.
- Parents often know their child best and are often able to predict how he or she will respond to pain.
- Parents will also be able to identify some of the words their children use to describe their pain to make a more accurate pain assessment.

Nonpharmacological Pain Management

- It is important to use a combination of nonpharmacological interventions.
- For procedure-related pain in children and adolescents, it is important to use a variety of cognitive behavioral interventions.
- Cognitive behavior strategies:
  - Must be used together, and not in isolation.
  - Can be used for a variety of medical procedures, adapting information appropriately for the different procedures.
- Pain interventions focus on minimizing pain and distress during the procedure by:
  - Creating a positive health care environment.
  - Encouraging parent coaching and involvement.
  - Preparing the child and family.
  - Utilizing cognitive behavioral distraction techniques.
  - Modeling relaxation strategies to aid coping.

Interventions

Health Care/Procedure Environment

- Children may encounter medical procedures for very different circumstances.

TABLE 13.5: Adolescent pain assessment and nonpharmacological pain management strategies

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Adolescent (12–18 yr)

Cognitive perceptions of pain

■ As children get older, they potentially have more words/experiences to draw upon to better describe the value of their pain (i.e., stabbing/sharp/dull).

Pain assessment tools

Self-rating – Many more self-report tools are available for this age group due to their maturing cognitive abilities. Coping can impact their ability to decipher physical pain and anxiety.
Wong-Baker FACES scale – See Table 13.3.
Biophysiological:
■ Heart rate, oxygen saturation.
Observational:
■ Some adolescents are less likely to verbalize pain, and so lack of crying or moaning in itself should not be used as the only indicator of pain.
■ Procedure Behavior Checklist – (6–18 yr).
Noncommunicating Children's Pain Checklist (NCCPC) – To be used with developmentally delayed children 3–18 yr old.

Considerations for procedural preparation

Providing the information:
■ Parents should be prepared for the responses their child may have to procedures.
■ Increased understanding at this stage, so they may also ask for, and need more details about procedures.
■ Include the steps of the procedure, the sensations to be felt, all while encouraging their questions to be asked.
Special considerations:
■ Adolescents understand more about the emotional and physical aspects of pain and its cause.
■ Have a more sophisticated understanding of the consequences of an injury or procedure and therefore could experience heightened anxiety.
■ Allow time for questions, encourage their participation, and allow some control.
Without these interventions:
■ Because of the loss of independence and control that can result from being in the hospital, adolescents could respond with anger and frustration.

Environmental considerations

Caregiver involvement:
■ Increasing need for a sense of identity and independence can result in ambivalence to their parent's involvement.
■ Adolescents can go from wanting their parents involved to feeling embarrassed about their continued involvement. Wishes for parental or no parental involvement, as well as their privacy, should be respected.
Health care worker behaviors:
■ Health care workers' roles are especially important with adolescents as the establishment of a good relationship facilitates coping and greater cooperation.
Health care setting:
■ Respect privacy, autonomy, and self-respect.

Cognitive and behavioral distraction techniques

Comfort suggestions:
■ Favorite item
■ Provide patient with the choice of parental support
Distraction play materials:
■ Music, search and find books, videos, video games, squishy balls, interactive technology devices
Distraction conversation:
■ Reality conversation, reading books, counting, talking about favorite things, positive self-statements, jokes/humor
Relaxation methods:
■ Massage, touch, progressive muscle relaxation
■ Breathing strategies – deep rhythmic breathing, watching stomach rise and fall
■ Guided imagery (imagining a special favorite place)

- For children experiencing the procedure for the first time, they:
  - ► Are often unfamiliar with the setting and will require preparation as to what to expect.
  - ► Will benefit from more explanations for various procedures.
  - ► Require support for development of new coping strategies.
- Previous experiences or chronic conditions:
  - ► Interventions depend on whether the experience was negative or positive.
  - ► Often having a familiar health care worker perform the procedure tends to decrease distress.
  - ► Require support to accommodate any coping strategies they have used in the past.
  - ► Exposure to procedural cues like seeing medical equipment can heighten a child's anxiety; therefore, make accommodations as needed.
  - ► Delaying a procedure can also heighten a child's anxiety if they are already prepared and familiar with what is happening.
- Health care workers should take the time to introduce themselves and get to know the child prior to the procedures.
- Keep a calm and quiet environment to alleviate distress.
- Children tend to be less distressed when they have some control over the situation.
  - By giving choices (to watch or look away) and being flexible about utilizing comfort positions such as sitting in a parent's lap or sitting up in a chair can aid in the child being more relaxed and less distressed.
- Children should be allowed to express their pain or discomfort by crying/shouting out as this can also result in less distress during a painful procedure.
- If a procedure is attempted unsuccessfully, then children should be offered the option of taking a break.

Caregiver Coaching/Involvement

- Children perceive parents being present during blood draw to "help the most."
- A parent's comfort level should not be assumed.
  - The parents should always be asked to determine their comfort level to be present during procedures.
  - Parents often feel helpless when observing their child in distress.
  - Parents need to feel empowered and in control of the situation in order to adequately support their child through procedures.
  - It is important to make parents aware that openly displaying emotions reinforces the child's lack of control and emotional reactivity.
- When parents and health care workers are focusing on coping-promoting behaviors, this assures that parents engage in fewer undesirable distress-promoting behaviors and promote more positive outcomes for their child.
  - The distress a child experiences can be affected by any adult responses and comments during a procedure.
  - Parents and health care providers should not focus on the distressing parts of the procedure.
  - Avoid expressing any negative thoughts or feelings about the procedure.

- Parents need preparation and coaching on how to effectively encourage their child's coping strategies.
  - Parents should be given an active role, and be prepared on how the y can best help their child.
  - Parent positioning is impor tant to consider .
  - Parents can be educated on the different distraction techniques benef cial for their child's age.
  - Parents can be encouraged to identify that the child is not alone and e veryone is wor king together to minimize the child' s pain and anxiety , can aid in calming a child, and reassure them that the y are safe and secure (i.e., "we are going to help you get through this").

Positive Reinforcement

- It is impor tant to continue to give praise for positive beha viors before, during, and after the painful procedure.
- It is impor tant to empow er the child through positive words and give the child choices so that they can feel the y are actively par ticipating in creating a positive outcome.
- Reassurance should be done in combination with other strategies.

Providing Information/Preparation

- Preparation can act as an exposure-based treatment to:
  - Reduce anticipator y anxiety and distress.
  - Reduce fear of the unknown.
- Provide the child and parent with skills to use to cope during the procedure. Children who are w ell infor med about a procedure repor t less pain than children who do not know the pur pose of the procedure.
- Preparations can differ , depending on:
  - Developmental age group.
  - Previous experience.
  - Temperament.
  - Attitude.
  - Coping skills.
- Other factor s that are considered when trained professionals, such as child life specialists, are preparing a patient for a procedure include:
  - *Timing of when the information should be provided,* recognizing and allowing time for the children to rehear se and plan coping strategies, and to ask questions.
  - *Language and terminology used,* avoiding medical jargon.
  - *Delivery of information,* presented in a nonthreatening w ay, the use of sensor y information to describe what the child will feel, and the timing and sequence of events.
  - *Tools used,* to include hands-on exploration of equipment kits, and picture preparation books de veloped for various procedures.
  - *Development of coping strategies,* to include openly talking about feelings, misconceptions, and fear s; selection of comfor t measures; rehear sal of breathing/relaxation strategies; and selection of distraction techniques.

Behavioral and Cognitive Distraction

- Cognitive and behavioral distraction involves the use of children's imagination and sense of play.
- It involves both the use of various play materials (i.e., bubbles, videos, toys) and nonprocedural related conversation (i.e., counting, singing, and discussing favorite things).
- Used to redirect the child's attention from the painful event.
- In order for distraction techniques to be effective, these activities must be relevant, developmentally appropriate, and contain observable behaviors.
  - Giving the child something concrete to focus and specific instructions on what you require them to do is much more effective and will increase understanding and compliance.

Relaxation

Breathing Exercises

- Promote relaxation.
- Encourage deep breathing, where the child is moving their diaphragm and exhaling through their mouth, and can elicit more rhythm and pacing for relaxation.
  - Very young children can also be encouraged to sing, blow bubbles, or pretend to blow out birthday candles as these activities encourage controlled breathing that promotes relaxation and distraction.

Guided Imagery

- Involves guiding children to use their imagination so that they can focus on feelings that are opposite to the feeling of pain and distress.
- Guided imagery has been shown to distract and reduce the perception of pain.

Suggested Reading

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Systems Based Approach to Pain Management

14 Headache

David Ng

Epidemiology of Headache

- The prevalence of migraines is 10%–15%, and females are affected three times more than men.
- Patients presenting with headache account for up to 4% of all emergency department (ED) visits.
  - Four percent of headaches have serious or secondary pathology.
  - 0.5% of headaches have life-threatening pathology.
- Twenty-five percent of women and 9% of men experience disabling migraines.
  - Disabling migraines cost 4–6 lost workdays a year, amounting to direct/indirect costs of 17 billion a year in the USA.

Pathophysiology of Headache

- The brain parenchyma is insensate to pain.
- Pain receptors originate in large cranial vessels, venous sinuses, proximal intracranial vessels, pia mater, and dura mater.
- Serotonin (5-HT) receptors are the main focus of pain management as they are known to modulate peptide release and regulate cerebral vessels.
- The anterior vessels are innervated by V1, while the posterior vessels are innervated by C2. Pain can be more generalized or referred to the associated dermatome.
- Pathophysiology of primary headaches remains poorly understood.
  - Current theories of primary headache pathology include:
    - ▶ Hypersensitivity of nociception of myofascial tissue.
    - ▶ Cortical neuronal depression phenomena.
    - ▶ Abnormal vascular dilatation/inflammation.

Classification of Headache

- Classified as either primary or secondary headache as per the International Headache Society (see Table 14.1).
- Primary headache originates from the pain receptors.
  - Although potentially disabling, primary headaches are not life threatening.

TABLE 14.1: Primary and secondar y headaches [a]

Secondary headache		Primary headache
Acute danger	Non-acute danger	**Primary headache**
Subarachnoid hemor rhage	Cervical stenosis	Tension
Meningitis	of C2	Migraine
Cerebral venous sinus thrombosis	Trigeminal neuralgia	Cluster
Carotid/ver tebral ar tery dissection	HTN	
Pseudotumor cerebri	Sinusitis	
Glaucoma	TMJ disorder	
Temporal ar teritis	Post-lumbar puncture	
Eclampsia	Concussion	
CO poisoning	Medication o veruse	
Brain tumor[a]	Brain tumor[a]	
Trauma[a]	Trauma[a]	

[a]Danger is dependent on degree of increased intracranial pressure, shift/effect on intracranial str uctures due to hemorrhage/mass effect.

- Secondar y headache is due to a specif c pathology that stimulates the pain receptor s.
- It is impor tant to identify which ones are life threatening to the patient.

Goals in the Emergency Depar tment

- Want to r ule out all life-threatening secondar y causes of headache.
- Key questions to consider on histor y:
  - Periodicity and how this par ticular episode e volved.
  - Associated features.
  - Behavior during headache.
  - Family histor y of migraines and aneur ysms.
  - Current medications.
  - Social situation and stressor s.
  - What the patient thinks.
- Fundoscopy should be car ried out on all patients with headache, along with a neurological exam.
- Patients presenting with the following red f ags should ha ve emergency neuroimaging in the ED:
  - New onset headaches.
  - Thunderclap headache.
  - Headache with an atypical aura (lasting o ver an hour or with motor weakness).
  - Aura without headache in a patient who is migraine naïve.
  - New headache in a patient o ver the age of 50.
  - Progressively wor sening headache.
  - Headaches that change with posture or other maneuver s that increase ICP.

- New headache in a patient with HIV , cancer, or immunodef ciency.
- Headache with fe ver.
- Headache associated with focal neurological symptoms.
■ Response to therap y should not be an indicator of benign etiolog y.

Specif c Management of Headache in the Emergency Depar tment

Primary Headaches
■ See Chapter 10 on phar macology of pain management for specif c medications.

Tension Type
■ Recurrent episodes that last from hour s to da ys.
■ Typically bilateral, non-pulsating headache with no associated features.
■ Specif c treatment:
  - Ibuprofen 200–400 mg , acetaminophen 1 gm q4hr (grade A).
    ▶ NSAIDs (Naproxen 375, diclofenac 25, ibuprofen 400 mg) ha ve similar effect to each other .
  - Caffeine 65 mg PO ma ybe a useful adjunct, but will increase GI side effects/ dizziness (grade B).
  - Ketorolac 60 mg IM for acute relief of moderate to se vere headache (grade B).
  - There is no e vidence/conf icting e vidence for the use of triptans and muscle relaxants.
  - Avoid narcotic, hypnotic combinations due to increased use of o veruse, rebound, tolerance/dependency (grade C).

Migraine Type
■ Recurrent attacks that last from 4 hour s to 3 da ys, usually ha ving one to two episodes per month.
  - Patients are asymptomatic betw een episodes.
■ Typically unilateral, throbbing associated with nausea, vomiting , photophobia (ma y or may not ha ve aura).
■ Specif c treatment:
  - Intravenous f uids and dar k/quiet en vironment.
  - Avoidance of ph ysical activity .
  - If mild, consider NSAIDs, acetaminophen (grade B).
  - If moderate or se vere pain, consider triptans or dopamine antagonists, both have about 65–70% response rate (grade A).
  - IV dexamethasone 10–25 mg shows modest effect in decreasing relapse rate at 24–72 hour s, NNT = 9 (grade A).
  - If recur rent or disabling , consider proph ylactic treatment – beta-block ers, TCAs, SSRIs, anticon vulsants (grade A).
■ Avoid opioids because the y have increased risk of rebound headache with retur n to ED compared to placebo.

Cluster Type

- Short-lasting attacks (an ywhere from 15 min to 3 hr), recurring frequently in bouts of 6–12 weeks in a year .
- Typically, se vere unilateral orbital, temporal pain lasting 15–180 minutes, associated with ipsilateral lacrimation, rhinorrhea, facial sw elling, miosis, and ptosis.
- Specif c treatment:
  - High f ow O_2 (non-rebreather mask 7–12 L for 20 min) effective in 70% of patients (grade A).
  - Sumatriptan 6 mg SC (grade A).

Specif c Anti-migraine Medications

Triptans

- Triptans are serotonin 5-HT $_{1B/1D}$ receptor agonists.
- The choice of T riptan is inf uenced by onset, route of administration, eff cacy, and rate of side effects.
- Triptans are contraindicated in patients with signif cant coronar y ar tery disease due to vasoconstrictive effects.
- Serious adver se effects include:
  - Coronar y ar tery spasm.
  - Serotonin syndrome.
  - Drug–drug interaction with MA O inhibitor s, oral contraceptives, other SSRIs, estrogen-containing contraceptive pills, and CYP3A4 inhibitor s.
- Common side effects of triptans include (triptan sensations):
  - Paresthesias.
  - Flushing.
  - Mild neck tightness or chest pressure.
- See Table 14.2 .

TABLE 14.2: Pharmacokinetic and eff cacy rates of different T riptans

Triptan	Onset	Adverse event rate (%)	Absolute sustained pain-free rate (%)	Special note
Almotriptan 12.5 mg P o	<1 hr	14.2	25.9	Least amount of recurrent headache
Sumatriptan 100 mg po 6 mg sc	1.5 hr s po 15 min sc	33.6	20	Only triptan to ha ve SC and IN routes
Rizatriptan 10 mg	1 hr	40.8	25.3	Comes in oral disintegra-tion tablet
Eletriptan 40 mg	<1.5 hr	42.3	20.9	
Zolmitriptan 2.5 mg	<1.5 hr	39.2	19	IN for mulation a vailable

ªDoses illustrated are maximum single dose, however, a second rescue dose can be administered 2 hour s later if headache has not resolved.
SC, subcutaneous; IN, intranasal.

Dopamine Antagonists
- Choice of medications include (grade A):
  - Chlorpromazine 10 mg IV.
  - Metoclopramide 10 mg IV.
  - Prochlorperazine 10 mg IV.
- Conficting studies show chlorpromazine and prochlorperazine to be superior or equivalent to metoclopramide.
- IM formulation associated with high relapse rate.

Dihydroergotamine
- Mechanism through alpha receptor blockade and serotonin agonist.
- Dihydroergotamine (DHE) 1 mg IV q1hr PRN (maximum 3 mg) or 1 spray IN q20min (grade A).
- Although clinically effective, trials show it to have increased GI side FX and to be inferior compared to dopamine agonists or triptans in effcacy.

Secondary Headaches
- Need to identify the cause and address with specifc treatment.

Subarachnoid Hemorrhage
- Headache is the predominant symptoms in these patients.
- Usually a sudden onset headache that reaches peak intensity within a few minutes.
- Majority of aneurysmal SAH occur between 40 and 60 years with mean age of 50.
- High mortality up to 50%.
- Etiology
  - Eighty-fve percent are due to bleeding from a cerebral aneurysm.
  - Ten percent from non-aneurysmal perimesencephalic hemorrhage.
- Diagnosis:
  - Urgent noncontrast CT scan.
    - ▶ Sensitivity is up to 98% if performed within the frst 12 hours.
  - If CT scan is negative, then lumbar puncture (LP) is needed to look for red cells and xanthochromia (yellow color caused by breakdown of bilirubin and oxyhemoglobin).
  - A negative CT and CT-angiogram can exclude subarachnoid hemorrhage with 99% post-test probability.
- Management:
  - Neurosurgical intervention of aneurysm.
  - Management of blood pressure to minimize re-bleeding risk is controversial (given risk of decreased perfusion/increased infarction).
  - Stop antiplatelet agents and reverse anticoagulation agents.
    - ▶ FFP.
    - ▶ 10 mg IV vitamin K, prothrombin complex concentrate (octaplex) 40 mL IV.

Thunderclap Headache

■ Sudden onset explosive headache.

■ Always consider as subarachnoid hemor rhage until pro ven otherwise.

■ Differential diagnosis includes:
  ● Cerebrovascular accident.
  ● Venous sinus thrombosis.
  ● Hypertensive emergency .
  ● Cervical ar tery dissection.
  ● Intracranial hypertension.
  ● Third ventricle colloid cyst.
  ● Intracranial infection.

■ Diagnosis:
  ● Diagnosis of exclusion.

■ Etiology:
  ● Probable role of cerebral vasoconstriction and excess sympathetic activity or sensitivity .

■ Management:
  ● Need to r ule out subarachnoid hemor rhage with CT/LP . Consider MRI for venous sinus thrombosis/cer vical ar tery dissection.
  ● If workup is negative, and primar y thunderclap headache is diagnosed, consider role of sympathomimetics (pheochromocytoma, drug abuse).

Giant Cell Temporal Arteritis

■ Usually in patients o ver the age of 55, mean age of 72 year s.

■ Incidence of 1/500 for people o ver 50 year s.

■ No adverse effect on mor tality.

■ Fifteen to 20% ha ve permanent vision loss (often at time of presentation)

■ Clinical features:
  ● New headache o ver jaw, face, eyes, tongue with features of claudication.
  ● Patients ma y present with systemic symptoms (malaise, polymyalgia, fever).
  ● Possible ele vated ESR/CRP .
  ● Visual changes.
  ● Tenderness/decreased pulse o ver temporal ar tery.

■ Etiology:
  ● Chronic vasculitis of medium, large vessels, most commonly cranial ar teries originating from aor tic arch.

■ Diagnosis:
  ● Def nitive diagnosis is with a positive temporal ar tery biopsy.

■ Treatment:
  ● Treat immediately (prior to biopsy) if visual loss or high suspicion.
  ● Prednisone 40–60 mg po OD (grade 2c) if no visual loss.
  ● Methylprednisolone 1 gm IV (grade 2c) if visual loss.

- ASA 80/100 mg /da y (grade 1b).
- Optho/rheum/neuro consultation.

Cerebral Venous Thrombosis
- More common in women (3:1).
- Frequency uncer tain, but presumed to be uncommon <1/100,000.
- Estimated 10% mor tality if treated.
- Eighty percent ha ve full reco very.
- Pathophysiolog y:
  - Thrombosis of cerebral veins, dural sinus results in increased venous pressure → cerebral edema/venous hemor rhage → cerebral ischemia
- Risk factor s:
  - Prothrombotic conditions (acquired/genetic).
  - Oral contraceptive medications, pregnancy.
  - Malignancy.
  - Infection.
  - Head Injur y.
- Clinical presentation:
  - Highly variable.
  - Eighty-f ve percent of cases present with at least one risk factor .
  - Acute, subacute, or chronic headache.
  - Headache wor se with recumbency or V alsalva maneuver.
  - Signs and symptoms of intracranial h ypertension, focal neurological def cits, encephalopath y, seizure.
- Diagnosis:
  - MRI is most sensitive.
  - Can use CT with contrast looking at the venous phase.
  - Noncontrast CT – only 70% sensitive.
- Treatment:
  - Anticoagulation with low-molecular-w eight heparin/Coumadin.

Post-LP Headache (PLPHA)
- Ten to 30% of patient post-LP will ha ve a headache.
- Pathophysiolog y:
  - CSF leakage from the dura with traction on pain-sensitive str uctures.
  - Consider cerebral venous sinus thrombosis (as LP is rare cause of this).
- Clinical features:
  - Typically self-limited.
  - Onset 12–24-hour post-LP , resolves within 14 da ys.
  - Worse with upright position.
- Prevention/treatment:
  - Use of smaller needle, bevel parallel to longitudinal dura f bers.

- No role for prolonged recumbency in proph ylaxis.
- Bed rest and oral analgesics for mild PLPHA.
- Consider caffeine sodium benzoate 500 mg in 1 L NS o ver 1 hour (grade B).
- Epidural blood patch for se vere, refractor y PLPHA (grade B).

Trigeminal Neuralgia
- Paroxysms of unilateral facial pain described as an electrical discharge, followed by a brief spasm.
- Follows sensor y distribution of trigeminal neuralgia.
- Pathophysiolog y:
  - Either primar y or secondar y due to compression from tumor or aneur ysm, or chronic inf ammation.
- Clinical features:
  - Pain occur s in brief episodes.
  - Usually unilateral.
  - Can ha ve a trigger point along the ner ve.
- Prevention/treatment:
  - Carbamazepine 100–200 mg BID (grade A)
  - Other options: Baclofen, Gabapentin, Clonazepam, Amitriptyline.
  - Surger y if ner ve decompression is an option.

Medication Overuse Headache
- Occurs when patient tak es analgesics frequently for headaches.
- Estimated to happen in 1% of adults.
- Usually occur with o veruse of triptans, ergotamines, analgesics, opioids.
- Pathophysiolog y incompletely under stood.
- Diagnosis:
  - Headache presents on ≥15 days/month fulf lling criteria C and D .
  - Regular o veruse for ≥3 months of one or more dr ugs that can be tak en for acute and/or symptomatic treatment of headache.
  - Headache has de veloped or mar kedly wor sened during medication o veruse.
  - Headache resolves or re verts to its pre vious patter n within 2 months after discontinuation of o verused medication.
- Treatment:
  - Abrupt withdra wal of analgesia with long-acting NSAID for non-opioid/h ypnotic overuse.
  - Consider taper or clonidine and medical obser vation for high-dose opioid/ hypnotic o veruse.

Pseudotumor Cerebri (Idiopathic Intracranial Hypertension)
- Annual incidence is 9/1,000,000 in the general population.
  - This increases 20 times in obese women aged 20–44.

- ■ Risk factor s:
  - ● Medications – tetracycline.
  - ● Female gender.
  - ● Obesity.
- ■ Clinical features:
  - ● Papilledema is the hallmar k of idiopathic intracranial h ypertension (IHI).
  - ● Chronic headache.
  - ● Retrobulbar pain.
  - ● Transient vision loss (75% of patients).
  - ● Pulsatile tinnitus (60% of patients).
  - ● Sixth ner ve palsy.
- ■ Papilledema can lead to per manent blindness if left untreated.
- ■ Diagnosis:
  - ● Diagnosis of exclusion: must exclude secondar y causes of increased ICP .
  - ● CT +/− MRI to r ule out abo ve.
  - ● MRI f ndings include 'empty sella, ' dilation of the subarachnoid space around optic ner ve, and posterior sclera f attening at the lamina cribrosa.
  - ● LP with opening pressure >250 mm H$_2$O.
- ■ Management:
  - ● Low sodium w eight loss program.
  - ● Acetazolamide 500 mg PO BID .
  - ● Consideration of: Furosemide 20 mg BID , glucocor ticoids, topiramate, TCAs, valproate.
  - ● Serial LPs for refractor y cases.
  - ● Ophthalmolog y and neurolog y refer ral.

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Chest Pain

Jessica Hernandez and Jarone Lee

Introduction

- Chest pain accounts for 10% of visits to the emergency depar tment (ED).
- Etiologies of chest pain range from life threatening to benign.
- Forty-f ve percent of patients with chest pain are e ventually diagnosed with acute coronar y syndrome.
  - Of these, 7% are younger than the age of 35,   and 50% are older than the age of 40.
- Noncardiac causes of chest pain include:
  - GI disease – GERD/ref ux, esophageal spasm, peptic ulcer disease, biliar y colic, pancreatitis, bowel obstr uction.
  - Respirator y – pulmonar y embolus, pneumonia, pneumothorax, pleurisy, empyema.
  - Chest w all syndromes – shingles,   soft tissue injuries,   rib fracture.
  - Nerve root compression.
  - Psychiatric – anxiety , globus, panic disorder s, somatization.
- Musculosk eletal pain accounts for 36% of chest pain complaints,   of which 13% are due to costochondritis.

Pathophysiology

- Chest pain is frequently described as the following:
  - Tightening , burning, pressure, aching , shar p, tearing , or gaseous.
- Chest pain sensation from visceral organs (esophagus,   hear t, lung, great vessels, etc.) arise from the same autonomic ganglia.
- Painful stimuli felt in the chest can refer throughout the tor   so, neck, and upper extremities.
- No one description of chest pain can be def   nitively correlated with a specif c cause.

Cardiovascular Chest P ain
Cardiac Ischemic Pain (Acute Coronary Syndrome)

- Cardiac ischemia is due to an inability to meet oxygen and nutrient demands.
  - Atherosclerosis is the leading cause of coronar   y vessel nar rowing, which leads to ischemia.

- Management:
  - ▶ Percutaneous coronar y inter vention (PCI) is the management of choice for patients with ST ele vation myocardial infarction (MI). In center s without these capabilities, thrombolytics are an option as long as there are no contraindications.
  - ▶ Other management options include:
    - ○ Aspirin – shown to impro ve mor tality with a number needed to treat of 42.
    - ○ Pain control can be achie ved with narcotic analgesia and nitrates.
    - ○ Other options ha ve shown no benef t in mor tality but are utilized: clopidogrel, glycoprotein IIb/IIIa inhibitor s.
- Cocaine-induced chest pain is caused b y vasoconstriction of the coronar y ar teries due to its direct alpha agonist effects. How ever, it is also known to accelerate atherosclerotic disease.
  - Management:
    - ▶ PCI is the management of choice for patients with signs of ischemia b y Electrocardiogram (EKG) and cardiac mar kers.
    - ▶ Aspirin.
    - ▶ For pain control, consider benzodiazepines, opiates, or nitroglycerin.

Pericardial Pain

- Pericardial pain is caused b y inf ammation of the pericardial sac (pericarditis).
- Sudden shar p pain that wor sens when supine and with inspiration.
- Look for EKG f ndings or presence of a pericardial r ub.
- EKG stages:
  - Stage 1 – diffuse ST ele vations with PR segment depression.
  - Stage 2 – nor malization of ST and PR changes; f attening of the T w aves.
  - Stage 3 – diffuse T w ave inversions.
  - Stage 4 – nor malization of EKG, or may continue to ha ve persistent T w ave inversion.
- Due to its intimate anatomical relationship,   myocarditis ma y also present similar ly.
- Etiolog y includes:
  - Neoplastic.
  - Autoimmune.
  - Infectious – TB, other bacteria, viral.
  - Uremia.
  - Post-MI.
  - Idiopathic.
- Management:
  - NSAIDs are the treatment of choice for these patients.
  - Def nitive treatment is varied and should focus on the under   lying cause.

Aortic Dissection

- Pain from an aor tic dissection occur s when there is a tear in the intimal la yer.

- Sudden onset, tearing like sensation.
- Risk factors: elderly patients, hypertension.
- CT angiography is the imaging of choice with high sensitivity and specif city, as well as helping rule out other pathology.
- Classif cation:
  - Stanford:
    - Type A – involves ascending aorta.
    - Type B – does not involve ascending (arch and descending aorta).
  - DeBakey:
    - Type I – ascending aorta, the arch, and the descending aorta.
    - Type II – ascending aorta only.
    - Type III – distal to left subclavian. Subtype **A** is above the diaphragm and subtype **B** extends below the diaphragm.
  - Management:
    - Management requires tight blood pressure control (Esmolol, Labetalol, or Nitroprusside are the preferred agents).
    - Surgery may be necessary if the tear involves the ascending aorta (Stanford Type A).
    - Manage pain as needed with opiates.

Pulmonary Chest Pain
Pulmonary Embolism

- Pain from pulmonary embolism is caused by the release of inf ammatory mediators in the lungs.
- Approximately 10% of pulmonary emboli cause pulmonary infarction, leading to ischemic pain.
- Management:
  - Def nitive management is with anticoagulation.
  - Control pain with NSAIDs, acetaminophen, and opiates.

Pneumothorax and Pneumomediastinum

- Pain from spontaneous pneumomediastinum and pneumothorax is due to air leakage into a potential space, which can cause inf ammation and compression of surrounding organs.
  - Management:
    - Def nitive management may include tube thoracostomy, surgical repair, or observation depending on the size of the pneumothorax and recurrence.
    - Control pain with NSAIDs, acetaminophen, and opiates.

Infectious, Pleurisy, and Pleural Effusion

- Pain due to infections, pleurisy, and effusions is caused by inf ammation, and is transmitted via somatic sensation from the parietal pleura.
  - Management:
    - Def nitive management may include antibiotics, immunosuppressants, or chemotherapy depending on the underlying cause.
    - Control pain with NSAIDs, acetaminophen, and opiates.

Gastrointestinal Chest P ain
- The most common cause of noncardiac chest pain.

Esophagitis
- The esophagus contains chemoreceptor s, mechanoreceptor s, and thermoreceptors.
- Esophagitis is caused b y inf ammation usually due to acid ref ux, infection, or ingestion of ir ritating substances.
  - Management:
    - For infectious esophagitis, pain control is with NSAIDs, acetaminophen, and opiates.
    - Treat under lying etiolog y with antibiotics, antifungals, or antivirals as appropriate.
    - For gastroesophageal ref ux, use acid suppressive therap y such as antacids, H_2 receptor antagonists, and proton pump inhibitor s. Also consider prokinetic agents.

Esophageal Rupture
- Esophageal perforation is caused b y medical procedures such as endoscop y, over 50% of the time.
- Spontaneous esophageal perforation is commonly due to straining or vomiting , resulting in sudden changes in intraesophageal and intrathoracic pressures causing r upture.
- Leakage of esophageal contents will ultimately cause mediastinal inf ammation and pain.
  - Management:
    - Def nitive therap y includes broad-spectr um antibiotics and surgical repair .
    - Pain control is with intra venous opiates.

Esophageal Dysmotility (Nutcracker Esophagus, Diffuse Esophageal Spasm, Hypertensive Lower Esophageal Sphincter)
- Esophageal dysmotility disorder s cause pain via distention, spasm, or increased intra-esophageal pressures.
  - Management:
    - Calcium Channel Block ers.
    - Other options include:
      - Nitric oxide-based dr ugs such as nitrates and phosphodiesterase inhibitors.
      - Tricyclic antidepressants.
      - Theophylline.
      - Botox injections.

Musculosk eletal/Neurologic
Trauma, Musculoskeletal, and Costochondritis
- The somatic intercostal ner ves perceive muscle exer tion, injury to muscle or ribs, and inf ammation of the chest w all.

- Management:
  - ▶ Control pain with NSAIDs, acetaminophen, and opiates.
  - ▶ Consider intercostal ner ve block (see Chapter 12).

Acute Herpes Neuritis

- Herpes neuritis is an acute reactivation of the varicella vir us.
- After reactivation, the vir us spreads through the peripheral ner ves causing inf ammation of skin, soft tissues, and ner ves.
- Usually affects one or two adjacent der matomes on the same side; consider immune-suppressed state if bilateral or multiple der matomes.
- Most common complication is post-her petic neuralgia.
  - Management:
    - ▶ If symptoms began within 24–72 hour s, consider the use of antivirals.
      - ○ Acyclo vir, valacyclo vir (pro-drug of acyclo vir), or famciclo vir can be prescribed for the treatment of her pes neuritis.
      - ○ Brivudine is another antiviral medication a vailable in some countries. Its use is limited due to a potentially fatal interaction with 5-f uorouracil (5-FU).
    - ▶ Control pain with NSAIDs, acetaminophen, and opiates.
    - ▶ For moderate to se vere symptoms, consider steroids.

Postherpetic Neuralgia

- Post-her petic neuralgia occur s when the pre viously damaged neurons cause spontaneous pain without ne w injury.
  - Management:
    - ▶ Control pain with NSAIDs, acetaminophen, and opiates.
    - ▶ Typically requires long-ter m management, with a combination of the following: antidepressants, anticon vulsants, topical capsaicin, MDA receptor antagonists, intrathecal glucocor ticoids, cryotherapy, and surger y.

Summary

- Chest pain is a common presentation to the ED with a variety of causes.
- Several life-threatening causes need emergent in vestigations and specif c treatment.

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16 Back Pain

Elaine Rabin and Nelson Wong

Introduction

- Acute lower back pain is most commonly defined as lower back pain of less than 6 weeks duration.
  - In the emergency department (ED), patients often present within hours to days of onset of pain.
- Lower back pain has a broad differential diagnosis.
- Eighty-five to 97% of acute low back pain is ultimately determined to be mechanical/musculoskeletal or nonspecific in nature.
  - Treatment goals in these cases include pain relief and restoration of function.

Epidemiology

- Approximately one-fourth of US adults have had an episode of low back pain in the past 3 months.
- It accounts for 3% ED visits/year.
- Burden to society *:*
  - $84–625 billion annually in direct and indirect costs in the USA.
  - Second most common cause of lost time in the work place affecting 2% of US workforce.
  - Leading cause of work disability in adults less than 45 years.
  - Up to one-third of cases become chronic and last over 1 year, limiting activity in 20%.
  - Five percent of cases account for 75% costs.

Goals in the Emergency Department

- Want to rule out life- and limb-threatening causes of back pain before presuming etiolgy to be musculoskeletal and treating for this.
- Key questions to consider on history:
  - Periodicity and how this particular episode evolved.
  - History of trauma.
  - Associated features – anesthesia, paresthesia, paralysis, fecal incontinence, urinary retention.
  - Associated symptoms – fever, syncope, diaphoresis, nausea/vomiting.

- A complete vascular and neurological examination should be carried out on all patients with back pain.
- Red flags for patients presenting to the ED with back pain:
  - Fever with back pain.
  - Associated neurological symptoms.
  - History of intravenous drug use:
    - ▶ History of cancer.
    - ▶ Immunocompromised or recent steroid use.
    - ▶ Age older than 50 or younger than 17 years.
    - ▶ Pain lasting longer than 6 weeks.
- Response to analgesia should not be an indicator of benign etiology.

Pearls

- Routine imaging of uncomplicated lower back pain (i.e., no red flags) is not indicated.
- Many asymptomatic patients will have disc bulges demonstrated on MRI, so disc bulges do not necessarily imply causality, especially without radiculopathy/sciatica.

Etiology

- Musculoskeletal
  - Muscle spasm/strain
  - Disc herniation with or without sciatic symptoms
  - Spinal stenosis
  - Degenerative joint disease
- Urologic
  - Renal colic (see Chapter 21)
  - Pyelonephritis
- Vascular
  - Aortic dissection
  - Epidural hematoma
  - Abdominal aortic aneurysm
- Infectious
  - Spinal epidural abscess
  - Osteomyelitis

Workup for suspected life- and limb-threatening etiologies of Back Pain in the Emergency Department

Cauda Equina Syndrome

- Symptoms due to compression of lower spinal nerve roots.
- Can result from compression for any reason (tumor, hematoma, etc.), but is often due to large intervertebral disc bulge into the spinal canal.

- ■ Signs/symptoms:
  - ● New, progressive or se vere lower extremity motor or sensor y def cits.
  - ● Saddle anesthesia.
  - ● Urinary retention or incontinence.
  - ● Decreased rectal tone, bowel incontinence.
- ■ Investigations:
  - ● If suspected, MRI without contrast is the prefer red imaging technique.
  - ● CT without contrast ma y be useful if MRI is una vailable.

Abdominal Aor tic Aneur ysm Rupture

- ■ Risk factor s:
  - ● Older age.
  - ● Male.
  - ● Hyper tension.
  - ● Smoking .
  - ● Atherosclerotic disease.
- ■ Signs/symptoms:
  - ● Aneurysm without r upture is often asymptomatic.
  - ● Sudden-onset, colicky pain not related to mo vement.
  - ● Hematuria.
  - ● Classic triad of h ypotension, abdominal or back pain, and pulsatile abdominal mass is found in less than half the cases.
  - ● Common clinical scenario is an older patient presenting with symptoms of renal colic without a pre vious histor y of nephrolithiasis.
  - ● Investigations:
    - ▶ Ultrasound is useful for detecting aneur ysm and large amounts of f uid due to r upture.
    - ▶ CT scan with intra venous contrast can re veal both aneur ysms and rupture.

Malignancy, Primar y Tumor or Metastases

- ■ Primary tumor s of the spine are most often lymphoma, leukemia, myeloma, ependymomas and other gliomas.
- ■ Metastases are often due to prostate, breast, and lung cancer s.
- ■ Risk factor s: Older age, cancer histor y.
- ■ Signs/symptoms:
  - ● New back pain in patients younger than 18 or older than 50.
  - ● Worse lying or sitting , and straining .
  - ● Gradual onset, unrelie ved with medications.
  - ● Pain at night.
  - ● Radicular or cauda equina symptoms.
  - ● Systemic signs of malignancy (e.g ., unexplained w eight loss, fever).
- ■ Investigations:
  - ● If suspected, MRI with and without contrast is prefer red.
  - ● CT can be useful if MRI una vailable.

Fractures

■ Due to osteoporosis, trauma, or tumor (pathological).
■ Risk factor s:
  ● Cancer.
  ● Osteoporosis.
  ● Age greater than 50.
  ● Recent trauma.
  ● Prolonged steroid use.
■ Signs/Symptoms:
  ● Midline tender ness.
  ● Radicular symptoms (par ticularly in compression fractures due to osteoporosis).
  ● Neurologic def cits cor responding to a lesion at a par ticular spinal le vel.
■ Investigations:
  ● X-ray is usually suff cient for diagnosis.
  ● Refer for fur ther imaging if pain per sists.
  ● MRI without contrast is prefer red to CT if a vailable.

Abscess (Epidural or P araspinal)

■ Risk factor s:
  ● Intravenous dr ug use.
  ● Diabetes.
  ● Immunocompromised state.
  ● Recent epidural anesthesia/injection.
  ● Recent proximal skin abscesses
■ Signs/symptoms:
  ● Fever.
  ● Leukocytosis.
  ● Radicular or cauda equina symptoms.
  ● Paralysis (late-stage f nding).
■ Investigations:
  ● If suspected, obtain MRI with and without contrast if a vailable.
  ● Otherwise CT with and without contrast for immediate diagnosis.

Management of Acute Musculosk eletal Lower Back P ain

■ Important facts regarding the a vailable e vidence:
  ● Few rigorous studies on treatment are a vailable.
  ● Available studies are mostly from primar y care and other off ce-based literature.
  ● Even those focusing on acute low er back pain often study time frames for pain relief not useful for ED visits (da ys to w eeks).
■ In practice:
  ● 80% of patients are prescribed medications, and more than one-third of patients are prescribed more than one medication.
  ● Education is a fundamental par t of treatment.

Treatments That Have Been Demonstrated to be Effective

- Acetaminophen
  - Few strong studies available.
  - Compared with NSAIDs:
    - ▶ Mixed evidence regarding relative effect (weaker analgesic effect according to data extrapolated from osteoarthritis studies
    - ▶ Safer side-effect profile in most patients.
  - Given favorable side-effect profile and low cost may be considered first-line treatment.
- NSAIDs
  - Best-studied class of medications.
  - Strong evidence of small improvements in the short term.
  - Onset within 1–2 hours.
  - No differences found among NSAIDS in effectiveness.
  - Compared to opioids:
    - ▶ Overall evidence is that NSAIDs may be equally effective and generally have a more favorable side-effect profile.
    - ▶ In one of the few ED-based studies, ketorolac and acetaminophen-codeine had similar effects, but ketorolac had fewer adverse effects.
  - Side effects:
    - ▶ The lowest effective dose should be used to minimize side effects.
    - ▶ Gastrointestinal (GI), renal, and cardiovascular.
      - ○ GI side effects are minimal at nonprescription doses.
      - ○ Other side effects include abdominal pain, diarrhea, edema, dry mouth, rash, dizziness, headache, and tiredness.
  - Evidence supports effectiveness in chronic pain as well.
  - Not helpful if sciatic symptoms are present.
- COX-2 Inhibitors
  - COX-2 inhibitors may have equivalent efficacy and fewer GI side effects than other NSAIDs, but have not been well studied to date.
  - May have increase cardiovascular disease risk.
- Muscle relaxants
  - This category is based on the FDA-approved indication; mechanism of action varies and includes centrally acting antispasmodics and peripherally acting antispasticity agents.
  - Antispasmodics
    - ▶ Strong evidence of clinical improvement, especially in the short term for patients without sciatica.
    - ▶ Cyclobenzaprine and tizanidine are the most well-studied.
    - ▶ No difference has been found among antispasmodic agents.
  - Less evidence is available for antispasticity agents.
  - Compared with NSAIDs: may be more effective but the evidence is still unclear.
  - Combined with NSAIDs:

- ▶ Unclear whether more effective than NSAIDs alone.
- ▶ Combination is associated with an increased frequency of side effects.
- Not a f rst-line agent for acute low back pain, most often considered an adjunct to analgesics.
- Side effects *:*
  - ▶ CNS: sedation, dizziness, headache, blur red vision.
  - ▶ GI: nausea and vomiting .
  - ▶ Use of some ma y lead to dependence.
  - ▶ Various dr ug-specif c serious side effects.
- ■ Benzodiazepines
  - No FDA appro val for low er back pain.
  - Compared with muscle relaxants: small number of studies repor t similar or less effectiveness to sk eletal muscle relaxants, however, not all studies demonstrated benef t.
  - Given risks of dependence and inadequate e vidence for chronic low er back pain it is not recommended for long-ter m use.
  - Side effects *:*
    - ▶ Sedation.
    - ▶ Respirator y suppression.
- ■ Opioids
  - Few trials and no systematic re views.
  - No differences found among specif c opioids.
  - Generally considered effective.
  - Due to side effects and potential for abuse, most guidelines and systematic reviews recommend as second line and for limited cour se of treatment for acute low er back pain refractor y to pre vious therap y.
  - Compared with NSAIDs: mixed e vidence regarding whether more effective (see abo ve).
  - Side effects:
    - ▶ GI: nausea, vomiting .
    - ▶ Sedation and respirator y suppression.
    - ▶ Potential for abuse in patients predisposed to addiction.
    - ▶ Potential for dependence.
- ■ Tramadol
  - Centrally acting with some effect at mu receptor s.
  - Similar to opioids: effective for pain relief but with less GI effect and dependence.
  - Side effects *:* headache, nausea.
- ■ Superf cial heat (heating pad, heat wrap)
  - Heat wrap signif cantly reduced pain ver sus placebo.
  - Some e vidence that 8-hour heat wrap ma y be more benef cial than NSAIDS, acetaminophen.
  - No risk of systemic side effects.

- Rapid return to normal activity within the limits of pain.
  - Universally recommended in published guidelines.
  - Evidence supports improvement in pain relief and functional status.

Treatments That Have Been Demonstrated to be Ineffective
- Exercise
- Bed rest
- Systemic corticosteroids

Treatments with Unclear Effectiveness
- Systemic and local steroid injection.
  - Use reserved for the treatment of epidural compression syndromes with consultation.
- Transcutaneous electrical nerve stimulation (TENS).
  - Local electrodes placed overlying skin that deliver electrical stimulation.
  - Widespread use as a treatment modality for over 30 years.
- Topical NSAIDs.
- Topical anesthetics.
  - Lidocaine patches caused dizziness in some patients.
- Cold treatment.

Disc Herniation and Spinal Stenosis
- For disc herniation: look for radicular symptoms, positive straight leg raise.
- Not as much evidence available regarding therapy, but most symptoms resolve with similar treatment to above in 4 weeks.
- NSAIDs not better than placebo in patients with sciatica.
- Acute pain of spinal stenosis may be relieved with walking or exercise.
- If patient presents with at least 1 month duration of pain, and if the patient is a candidate for epidural injections, discectomy (disc herniation), or spinal surgery (spinal stenosis), consider referral for nonemergent MRI.

Chronic Lower Back Pain
- Acute on chronic exacerbations of low back pain often present to the emergency room.
- Studies of low back pain agree that chronic low back pain often tends to be multifactorial in nature.
- Antidepressant drug therapy has been shown to be useful in some patients, specifcally those with underlying depression. The evidence is mixed with regards to patients without underlying depression.
- Short-term therapy with opioids has been recommended in various clinical practice guidelines.

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17 Sickle Cell Pain

Jeffrey Glassberg and Patricia Shi

Epidemiology

- Sickle cell disease (SCD) affects nearly 100,000 individuals in the United States.
- The most common reason for emergency department (ED) visits in this population is vaso-occlusive crisis (VOC).
  - Second most common reason for ED visits is fever.
- In both the ED and inpatient settings, pain is often undertreated.

Pathophysiology

- A point mutation at codon 6 of the β-globin gene causes production of abnormal hemoglobin called hemoglobin-S.
- When both inherited β-globin genes carry this mutation, or the hemoglobin S mutation is paired with another mutation (hemoglobin C and β-thalassemia are the most common), the patient will have SCD.
- In response to tissue hypoxia and stress response, HbS forms rigid polymers, which give erythrocytes their sickle shape.
- The pathophysiology of SCD and its complications are due to many factors including:
  - Enhanced leukocyte activation
  - Increased platelet activation
  - Blood cell adhesion
  - Endothelial dysfunction
  - Chronic hemolysis
- The clinical manifestations are myriad and include:
  - VOC
  - Acute chest syndrome (ACS)
  - Splenic or hepatic sequestration
  - Stroke
  - Aplastic crises
  - Dactylitis
  - Priapism
  - Leg ulcers
  - Increased infection risk – rule out sepsis with fever

- Avascular necrosis
- Bony infarcts and osteomyelitis
- Retinopathy
- Pulmonary hypertension
■ The most common manifestation is pain (VOC).
■ Most patients experience daily pain, with the most common sites being the lower back and legs.
  - Pain can also progress to VOC where the severity of pain requires high-dose opiates, usually necessitating hospital admission.

Potentially Life-Threatening Causes

■ In a patient presenting with pain, all the non-SCD-related, life-threatening causes of pain in that region must be considered (e.g., right lower quadrant pain in a patient with SCD is still appendicitis until proven otherwise).

Acute Chest Syndrome

■ It is a serious pulmonary complication, and should be considered in any sickle cell patient presenting with chest pain.
■ Overall incidence of 10.5 per 100 patient years.
■ Most common in the 2–4 years age group.
■ ACS is defned as new infltrate on chest x-ray with one of the following:
  - Chest pain
  - Fever
  - Respiratory symptoms – dyspnea, tachypnea
  - Hypoxia
■ While ACS criteria are not distinguishable from the defnition of pneumonia, it is described as a specifc entity because:
  - The cause of ACS is not always infection.
  - Etiology is multifactorial:
    ▶ Microbial infection
    ▶ Vaso-occlusion
    ▶ Fat embolism from ischemic/necrotic bone marrow
    ▶ Thromboembolism
  - The treatment for ACS is transfusion or exchange transfusion. Without this, clinical status often deteriorates rapidly with very high mortality.
■ Pearl: ACS is usually not the presenting complaint. More commonly, it develops during the course of in-patient admission. Assess frequently for signs and symptoms of ACS.
■ Management:
  - Supportive measures – oxygen.
  - Appropriate hydration – avoid bolus of fuids. Consider maintenance fuids without risking overhydration.
  - Appropriate pain control.

- Incentive spirometr y.
- Antibiotics: third-generation cephalosporin, macrolides.
- Simple or exchange transfusion.

Vaso-occlusive Crisis

■ Hallmark clinical manifestation of SCD .
■ Caused b y local ischemia from:
  - Decreased blood f ow
    ▶ Polymerization of HbS
    ▶ Cellular deh ydration
    ▶ Increased vascular adhesion
  - Inf ammation
■ Precipitants include:
  - Infection
  - Fever
  - Acidosis
  - Hypoxia
  - Dehydration
  - Sleep apnea
  - Extremes of heat and cold
  - Asthma exacerbation
■ Clinical presentation:
  - Tenderness
  - Swelling
  - Warmth
■ Need to distinguish it from osteom yelitis and septic ar thritis. This can be diff cult with routine blood wor k or x-rays. Best option is ar throcentesis for differentiating from septic ar thritis and MRI for osteom yelitis.
■ It is the most common reason for hospital admission in sickle cell patients.
■ Effective management must include:
  - Assessment of pain
  - Treatment of the pain appropriately
  - Frequent reassessment
  - Adjustment of pain medications as necessar y
■ Barriers to pain management:
  - Limited knowledge of SCD
  - Inadequate assessment of pain
  - Biases against opioid use, with unsubstantiated fear of:
    ▶ Tolerance
    ▶ Dependence
    ▶ Addiction
    ▶ Drug seeking

- ■ Assessment
  - ● Identify pain location(s)
  - ● Determine severity
    - ▶ Use a validated pain assessment tool. The most common is the 11-point numerical rating scale (0–10), although many others exist.
  - ● Analgesics used prior to arrival
    - ▶ It is crucial to ask the type and dose of analgesics used prior to arrival in order to calculate an appropriate analgesic starting dose in the ED.
  - ● Potential triggers:
    - ▶ See list above.
  - ● Laboratory evaluation:
    - ▶ VOC may be associated with significant changes in hemoglobin levels, hemolysis, and reticulocytosis.
    - ▶ Table 17.1 provides a summary of appropriate lab testing during VOC.
  - ● Pearl: Obstructive lung disease patterns and airway hyperresponsiveness are very common in children with SCD.
    - ▶ This is a frequent trigger for VOC.
    - ▶ Assess vigilantly for wheezing even if the patient does not carry a diagnosis of asthma and have a low threshold for treating with steroids and bronchodilators.
    - ▶ Pain may be more difficult to control if asthma exacerbation is present and not treated.
- ■ Treatment (see Table 17.1)
  - ● IV fluids:
    - ▶ Always use hypotonic IV fluids (e.g., D5½ NS), unless the patient is overtly hypovolemic (e.g., diarrhea).
    - ▶ Avoid overhydration as this can potentiate atelectasis, which is associated with the development of ACS.

TABLE 17.1: Management of acute pain crisis

- ■ Labs:
  - ● CBC, reticulocyte count
  - ● ALT, LDH, fractionated bilirubin if worsened icterus
  - ● Type & screen if Hb ≥1 g/dL is below baseline
- ■ D5½ NS with 20 mEq KCl/L at 1–1½ times maintenance (100–150 mL/hr)
  - ● Reassess every 24 hr
- ■ Quickly achieve and maintain pain control
- ■ Adjuvant analgesics: acetaminophen and diphenhydramine/ hydroxyzine
- ■ Laxatives: docusate and senna
- ■ Deep venous thrombosis prophylaxis with low-molecular-weight or regular heparin
- ■ Pulse oximetry only to keep O_2 saturation ≥92%
- ■ Incentive spirometry with chest or back pain
- ■ Continue outpatient folate and hydroxyurea

- IV opiates
  - ▶ Intravenous opiates (morphine, hydromorphone, and fentanyl) are the cornerstone of pain management for VOC.
  - ▶ At some centers, hydromorphone is preferred due to its low side-effect profle and lack of active metabolites. Selection of opioid is institution and physician dependent.
  - ▶ Meperidine is not recommended and, in patients with renal insuffciency, it is contraindicated due to its active metabolite normeperidine, which causes seizures.
  - ▶ Initial IV opiate dosing should be adjusted based on outpatient usage.
  - ▶ In the initial phase, reassess pain every 15–30 minutes and redose opiates until pain is controlled.
  - ▶ Pain usually improves after two to three doses of IV opiates if administered in rapid succession.
  - ▶ After relief is obtained, it must be maintained with round-the-clock opiate dosing, not just on a prn basis. Two options are listed in Table 17.2, along with tapering recommendations.
  - ▶ Pearl: Do not start PCA until the patient's pain has been controlled by an initial bolus (or boluses) of IV opiates.
    - ○ Without this, the PCA will be inadequate.
    - ○ Weaning should occur after the frst 24 hours and be started in the mornings rather than evenings.
  - ▶ Adjuvant medications
    - ○ Acetaminophen and diphenhydramine are indicated because they have opiate sparing effects.
    - ○ Long-term NSAID use should be avoided.
      - ➢ Single bolus dose ketorolac has shown some potential. Further studies are needed in its use.
      - ➢ NSAID use may hasten decline in renal function, especially if renal dysfunction is already present.
      - ➢ Creatinine is not a reliable indicator of subtle renal dysfunction in patients with SCD. SCD patients have supranormal proximal tubule function, which can result in normal creatinine values even when signifcant renal dysfunction is present.

TABLE 17.2: Around the clock options for adults (>50 kg)

Patient controlled analgesia (PCA) with hydromorphone (1 mg/mL)	Basal 0.1 mg/hr Demand 0.1–0.3 mg q8 min
Long-acting opioid agonist (controlled-release morphine or oxycodone or transdermal fentanyl)	Base initial dosing on short-acting opioid requirements. Rescue doses of 10–15% of the total 24-hr dose or 50% of the 4-hr dose should be the same opioid as the ATC medication, available q1–2 hr prn
Tapering opioids	Wean dose by 10–20% every 8 hr as tolerated to keep pain score <5. Once opioid dose 25–30% of initial level, can switch to equianalgesic oral opioids and consider discharge

▶ Incentive spirometr y is indicated for all patients with V OC.

▶ DVT prophylaxis: heparin or low-molecular-w eight heparin.

    ○ Low-molecular-weight heparin has been shown to safely reduce the severity and duration of painful crises.

    ○ Unless contraindicated, it should be administered to all admitted patients.

▶ Supplemental O_2 only to k eep oxygen saturation abo ve 92%.

▶ Transition to oral opiates.

    ○ When PCA dosing is at 25–30% of its initial le vel, the patient can be transitioned to oral opiates.

    ○ If the patient prefer s a longer acting opiate, star t it se veral days before discharge because it ma y take three to four doses to reach steady state le vels.

Priapism

■ Mean age of onset is 12 year s.

■ Occurs as a result of V OC of the penis, which causes obstr uction of the venous drainage.

■ Classif cation:

    ● Prolonged if it lasts more than 3 hour s

    ● Stuttering if it lasts for a fe w minutes, lasts less than 3 hour s, and resolves spontaneously

■ Complications of recur rent episodes include f brosis and impotence.

■ Identify the following at time of presentation:

    ● Time of onset

    ● Last episode and treatment

    ● Trauma

    ● Infection

    ● Drug use – sympathomimetics, alcohol, phosphodiesterase inhibitor s

■ Treatment:

    ● Fluids – if prolonged, then consider IV f uids

    ● Analgesia – consider parenteral analgesia (see Section on V aso-occlusive crisis and Chapter 10)

    ● Alpha agonists (pseudoephedrine 30–60 mg PO)

    ● Beta agonists (T erbutaline 5–10 mg PO)

    ● Aspiration of cor pus ca vernosum

Suggested Reading

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18 Dental and Ocular Pain

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Dental Pain

Epidemiology

- Dental complaints represent 0.4–10.5% of emergency department (ED) visits.
- Dental complaints can be categorized as (1) orofacial pain, (2) orofacial trauma, and (3) infections.
- Traumatic causes are often secondary to falls, accidents, assaults, or motor vehicle collisions.

Orofacial Pain

- Dental caries
- Periodontal disease (gingivitis)
- Postextraction alveolar osteitis (periosteitis/dry socket)
- Postoperative pain
- Acute necrotizing ulcerative gingivitis (Vincent disease)
- TMJ dysfunction

Orofacial Trauma

- Dentoalveolar trauma
- Dental fractures
- Concussions/luxations/avulsions
- Facial fractures
- Soft-tissue lacerations
- TMJ dislocation

Infections

- Dental abscesses
- Ludwig angina
- Deep neck abscesses
- Cellulitis

Clinical Assessment of Dental P ain

- Before instituting analgesia in an y form ensure that cause of pain has not compromised airw ay:
  - Sublingual hematoma
  - Expanding hematoma
  - Brawny neck (Lud wig angina)
  - Trismus
  - Drooling
  - Neck immobility

Dental Caries

- Represents the loss of integrity of the tooth enamel.
- Pain management consists of oral NSAIDs.
- Regional block ma y be appropriate in select situations.
- Management consists of r uling out other causes (i.e., abscess) and refer ral to a dentist.

Postextraction Alveolar Osteitis

- Otherwise known as dr y sock et, caused b y disr uption of the clot from the sock et, exposing alveolar bone.
- Presents in 2–5% of extractions, usually 3–4 da ys afterwards.
- Pain management consists of NSAIDs, regional ner ve block, and oil of clo ves.
- Regional ner ve blocks are often required to pro vide nor mal saline ir rigation and application of packing .
- Antibiotics ma y be required and refer ral to a dentist within the next 24 hour s.

Dental Abscess

- Secondar y to bacterial infection (*Streptococcus* species and oral anaerobes) from untreated dental caries.
- Left untreated can spread to deep neck spaces.
- Regional ner ve blocks for the affected region are appropriate within the ED .
- NSAIDs with the possibility of an opioid are also appropriate in pain control management.
- Def nitive management includes incision and drainage, tooth extraction, and antibiotics.

Ludwig's Angina

- Infection of submental, sublingual, and submandibular spaces, with ele vation and displacement of the tongue, which can lead to airw ay compromise.
- Poor dental h ygiene, dysphagia, odynophagia, trismus, and edema are common signs and symptoms.
- Pain management consists of opioids with close monitoring of airw ay compromise.
- Regional blocks are not indicated in this condition.
- Def nitive management includes IV antibiotics, and emergent refer ral to ENT for surgical inter vention as indicated.

Acute Necrotizing Ulcerative Gingivitis
- Also known as T rench mouth.
- Triad of pain, ulcerated interdental papillae, and gingival bleeding .
- Etiolog y is poor ly under stood, but associated in immunocompromised hosts, with *Treponema, Fusobacterium, Selenomonas,* and *Prevotella* commonly found.
- Pain management consists of systemic opioids, oral rinses with viscous lidocaine.
- Def nitive management includes w arm saline rinses, chlorhexidine rinses, and appropriate antibiotics.

TMJ Dislocation
- Secondar y to direct trauma, laxity of ligaments of joint, extreme opening of the mouth, dystonic reactions.
- Anterior dislocation of the condyles that become trapped in the anterosuperior eminence.
- Previous dislocations predispose to fur ther episodes.
- Def nitive management of joint reduction will pro vide analgesia.
- Proper reduction will often require procedural sedation to alle viate pain, muscle spasm, and patient resistance.

Mandibular/Maxillary Fractures
- Pain management consists of systemic opioids and the use of regional ner ve blocks where indicated.
- Management consists of ensuring there is no airw ay compromise and prompt referral to a maxillofacial surgeon.

Tooth Avulsions, Concussions, and Luxations
- Secondar y to falls, direct trauma, spor ting injuries.
- Def nitions:
  - Concussion – no displacement or loosening of teeth. There is cr ush injur y to adjoining tissue.
  - Luxation – dislocation of teeth.
    - Can be intr usive, extr usive, lateral, lingual, or buccal.
  - Avulsion – loss off tooth from the sock et.
- Regional ner ve block in the ED ma y provide the most comfor t for the patient (see Chapter 12).
- NSAIDs, soft diets are appropriate as outpatient pain management.
- Stabilization of the tooth and refer ral to a dentist is required.

Dental Fractures
- Secondar y to falls, direct trauma, and spor ting injuries.
- Seventy percent is to the central incisor s.
- Ellis classif cation:
  - Class I – in volves only the enamel
  - Class II – exposure of the dentin
  - Class III – fracture that includes exposure of the pulp
  - Class IV – root fracture

- Regional ner ve block in the ED ma y provide the most comfor t for the patient.
- NSAIDs, soft diets are appropriated as outpatient pain management.
- Referral to a dentist is required for both cosmetic and str uctural repairs.
- Higher Ellis class requires more urgent refer ral.

Considerations for Anesthesia for Dental P ain

- Does the patient ha ve any allergies?
- Are they on anticoagulation?
- Is there a need for immediate homeostasis?
- How long is analgesia required?
- What are the requirements for postprocedural analgesia?

Analgesia Options for Dental P ain

- Procedural sedation.
  - Consider use in TMJ dislocation.
- Topical anesthesia.
  - Available in man y preparations (e.g ., liquids, sprays, and viscous gels).
- Gels are shown to be more effective in pro viding pain control.
  - Benzocaine (6–20%).
- Rapid onset: ~30 seconds.
- Duration: 5–15 minutes.
- Poor systemic absor ption.
  - Lidocaine (2–5%)
- Onset: ~2–5 minutes.
- Duration: 15–45 minutes.
  - Injectable anesthetics
- Determine if large area is required (i.e., ner ve block vs. local inf ltration) – see Chapter 12 for ner ve blocks.
- Recommend topical anesthetic to be applied to area of injection to decrease pain of injection.

Specif c Dental Blocks

- See Chapter 12.
- Supraperiosteal Injection.
  - Indication:
    - ▶ Provides anesthesia to one tooth (pulp, root, buccal mucosa) for fractures, subluxations, and dr y sock ets.
- Greater palatine ner ve block.
  - Indication:
    - ▶ Palatal laceration, maxillar y teeth anesthesia.
    - ▶ Affects posterior aspect of unilateral hard palate and o verlying soft tissues.

- ■ Nasopalatine block.
  - ● Indication:
    - ▶ Augmentation of supraperiosteal injection of the anterior maxillar y teeth, and anesthetize anterior palatal mucosa for palatal laceration repair.
    - ▶ This block will affect the anterior por tion of the hard palate, from the left to right premolar s.
- ■ Infraorbital ner ve block.
  - ● Indication:
    - ▶ Useful for repair s of area from low er eyelid, lateral nose, and upper lip, as well as pro viding anesthesia to incisor s and canines.
    - ▶ Provides anesthesia to the anterior superior , middle superior alveolar nerve, and the infraorbital ner ve.
- ■ Inferior alveolar ner ve block.
  - ● Indication:
    - ▶ Useful for dr y sock et pain, postextraction pain, or pulpitis.
    - ▶ Anesthesia to unilateral aspect of mandible, mandibular teeth, anterior two-thirds of the tongue and f oor of the mouth.
- ■ Supraorbital ner ve block.
  - ● Indication:
    - ▶ Forehead and upper e yelid repair.

Ocular Pain

Epidemiology
- ■ Two percent of ED patient visits are for various ocular complaints including primary ophthalmologic, infectious, and traumatic causes.
- ■ Primary complaints include:
  - ● Glaucoma.
  - ● Infectious causes include conjunctivitis, her petic infection, stye, chalazion, periorbital/orbital cellulitis, and cor neal ulcer.
  - ● Traumatic causes include subconjunctival hemor rhage, corneal abrasion, and foreign body.

Considerations in the Management of Ocular P ain
- ■ Histor y or contact with her pes
- ■ Chemical exposure
- ■ Contact lens use
- ■ Ruling out foreign body
- ■ Treating under lying condition (glaucoma)
- ■ Providing systemic/topical antibiotics
- ■ Providing tetanus immunization for abrasions

Clinical Assessment of Ocular P ain
- Before instituting analgesia in an y form ensure that vision-threatening diagnosis is ruled out:
  - Retinal ar tery or vein occlusion
  - Open/closed angle glaucoma
  - Temporal ar teritis
- Ensure that complete ophthalmic examination is prefor med. This ma y include:
  - Visual acuity
  - Intraocular pressure
  - Funduscopy
  - Visual f elds
  - Pupils
  - Extraocular mo vements
  - pH
  - Slit lamp with f uorescence

Glaucoma
- Increased intraocular pressure as a result of either o verproduction or decreased resor ption of aqueous humor .
- As pressure increases, irreparable optic ner ve damage can occur .
- Use of NSAIDs and/or opioids systemically in conjunction with treatment to decrease intra-ocular pressure (IOP).
- Management of increased IOP:
  - Carbonic anh ydrase inhibitor s (Acetazolamide) – decrease aqueous production.
  - Topical beta-block ers (Timolol) – decrease aqueous production.
  - Hyperosmotic agents (Glycerin, Mannitol) – causes f uid shift from e ye space into the vascular space, resulting in an osmotic dieresis.
  - Miotic agents (pilocar pine) – causes ciliar y muscles to contract thus opening the space in the trabecular meshwor k to allow increased absor ption of aqueous humor .

Conjunctivitis
- Caused b y either bacterial (*Streptococcus pneumoniae, Haemophilus influenzae, Neisseria gonorrhoeae*) or viral infection.
- For pain management, use topical NSAIDs, such as 0.5% k etorolac.
- Anti-histamines can be used for pr uritus, which is common in viral conjunctivitis.
- Treat respective infections with appropriate antibiotics.

Herpes Zoster Ophthalmicus
- Rash that follows der matomal lines.
  - Look for Hutchinson' s sign with lesions at the tip of the nose.
- Dendritic lik e lesions are seen using f uorescein staining .
- Regional blocks can pro vide temporar y relief.

- If pain is ongoing , opioids, tricyclic antidepressants,   or gabapentin can be used to decrease duration or se verity of post-her petic neuralgia.
- Requires urgent ophthalmologic consultation requiring intra   venous antivirals.

Stye/Chalazion/Blepharitis

- Common presentation to urgent care clinics.
- Generally, secondar y to staph ylococcal infection.
- Warm compress to affected e ye helps alle viate symptoms.
- Treatment with antibiotic ointments and "no tear   s" shampoo.

Corneal Abrasion

- Defect of the nor mal cor neal epithelium caused b y trauma (f ngernail, twig, etc. or after foreign body remo val).
- Account for up to 10% of e ye complaints.
- Topical anesthetics are helpful for pain control in the ED   .
- The use of antibiotic ointments and anti-inf   ammatory eye drops (k etorolac 0.5%, diclofenac 0.3%) are helpful for pain management as w   ell as for the treatment of the under lying condition.
- Eye patches are not indicated. A meta-analysis demonstrated no difference in healing and no reduction in pain.

Corneal Ulcer

- Cor neal infection often secondar y to contact lens use.
- Pseudomonas ver y common in this type of infection.
- Topical anesthetics ma y be used initially in the ED but should not be continued. They are lik ely to mak e the ulcer wor se.
- Fluoroquinolone e ye drops are required q1h. Cycloplegic drops ma   y also be used for pain management.
- Eye patching should not be used!
  - Patching does not reduce pain in adults or pediatric population,   and ma y exacerbate the under lying condition (cor neal abrasion, ulcers).
  - There is little role for e ye patching in the ED .
- Referral to an ophthalmologist on an emergent basis.

Foreign Body

- Need to r ule out possibility of globe injur y (e.g ., high-velocity metal piece).
- Topical anesthetic ver y helpful in the ED .
- Must ensure remo val of foreign body b y examining entire e ye including under the lids.
- Irrigation initially ma y be helpful. Often using a small gauge needle or a bur   r is required.
- As for cor neal abrasions, antibiotic and anti-inf ammatory ointments ma y be helpful.

Topical Agents for Ocular P ain

Artificial Tears

- Two drops 4–6×/day as needed.
- Duration of use should be limited.

Topical NSAIDs

- Useful in inf ammatory changes such as conjunctivitis.
- Avoid in cor neal ulcerations and her petic infections.
  - Diclofenac at 0.1% (1–2 drops TID-QID)
  - Ketorolac 0.5% (1–2 drops TID-QID)

Topical Glucocor ticoids

- Useful in acute anterior uveitis, conjunctivitis, episcleritis, and scleritis.
- Should only be emplo yed after other treatments ha ve been exhausted.
- Prolonged use associated with increased risk of cataracts,   glaucoma, and infection.
  - Fluorometholone 0.1% (1 drop BID-TID)
  - Prednisolone 1.0% (2 drops QID)

Topical Cycloplegics

- Useful in conditions where blepharospasm is present.
- One percent cyclopentolate.

Topical Anesthetics

- Useful in acute ED setting .
- Should not be prescribed as outpatient as it car ries a high risk of secondar y keratitis.

Summary

- Review anesthetic considerations for each patient,   to help guide what type of anesthetic/analgesic would be most benef cial for your patient.
- Consider ner ve blocks, as the y require less anesthetic, less distor tion to tissues, and provide good anesthesia if done cor rectly.
- Do not forget to treat the under lying cause of pain.
- IV opiates should be used in cer tain cases where other means of analgesia will not be suff cient enough.
- Topical anesthetics for ocular pain are useful in the acute setting   , but patients should not be discharged with them as the y carry a high risk of complications with prolonged use.

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19 Prehospital Pain Management

Michelle Welsford and Greg Soto

Introduction

- When it comes to managing acute pain emergency prehospital practitioners, similar to emergency department (ED) personnel, are often falling short.
- Inadequate analgesia, referred to as oligoanalgesia, remains a major problem in both prehospital and Emergency Department (ED) care.
- Inadequate pain control takes several forms:
  - Delayed or nonreceipt of analgesia
  - Use of ineffective analgesia (e.g., non-steroidal anti-inflammatory drugs (NSAIDs) where opioids are indicated)
  - Underdosing analgesics
  - Failure to combine analgesics
  - Failure to use adjuncts
- Two important reasons not to delay analgesia in the field are as follow:
  - Analgesia administration is associated with decreased need for subsequent opioids.
  - If analgesia is delayed until ED arrival, time to administration can be significantly delayed (up to 2 hours in one study).
- Pain is associated with increased morbidity:
  - Increased patient suffering from the unpleasant experience.
  - Can lead to delayed wound healing.
  - Increased metabolic rate.
  - Altered immune response.
  - Lowered pain threshold for subsequent painful experiences.
  - Increased association with PTSD.
- The goal of prehospital pain management is to start the pain relief (not necessarily eliminate) – without significant complications.
- Several important emergency medical service (EMS) advocacy groups have called for improvements in prehospital pain management.
  - The National Association of EMS Physicians, the American College of Emergency Physicians, and the EMS Outcomes Project have all advocated for this.

Reasons for Oligoanalgesia

- Reasons cited for inadequate pain management in the out-of-hospital setting include:
  - Poor under standing of pain
  - Underestimation of pain
  - Poor assessment (poor under standing of a vailable pain assessment tools)
  - Provider biases – bar riers such as sex, age, race, ethnicity, language, and socioeconomic status
  - Fears related to opioid dependence and dr ug-seeking beha vior
  - Poor choice of analgesic
  - Inadequate protocols
  - Online medical control ph ysician attitudes and practices
    - ▶ Concer n of on scene dela y

Pain Assessment T ools

- Patients ma y not receive analgesia if the y are not questioned regarding their pain.
- Emergency pro viders often underestimate a patient' s analgesic requirements.
  - Have also been found to discredit patients' pain based on their own beliefs.
- Pain should be assessed and reassessed to ensure appropriate treatment.
- There are multitudes of pain scales tested and used in hospitals but pain scales used prehospital need to be simple, quick, and reliable.
- The verbal rating scale (VRS) and numerical rating scale (NRS) ha ve both been validated in the ED for adults and ha ve been shown to be easy , quick, discriminating , and reliable.
  - The NRS is lik ely the most commonly used tool for pain assessment and reporting in prehospital care because it does not require an y equipment or charts.
  - Provider verbally asks the patient to rate their pain on a scale of 0–10.
- For children, there are se veral scales including the pictorial F aces P ain Scale, and for younger children obser vational/beha vioral scales can be used (see Chapter s 13 and 22).

Patient Monitoring for Analgesia Administration

- In addition to assessing the patient' s perception of pain, it is impor tant for prehospital pro viders to under take a full assessment.
  - This includes:
    - ▶ Vital signs prior to analgesia administration (blood pressure, hear t rate, respirator y rate, SpO_2, and ECG rh ythm).
    - ▶ Continually reassess the patient' s pain scores.
    - ▶ Repeat vital signs periodically following analgesia administration.
- It has been established that prehospital pro viders do not consistently document pain encountered in the f eld.

- It is impor tant to document:
  - Initial pain score – identif es need for analgesia.
  - Response to initial treatment.
  - Ongoing need for fur ther treatment.
- Knowledge of the side effects of the specif c agents will also guide the reassessments so that these can be recognized ear ly and managed (e.g ., respirator y depression, hypotension, or nausea and vomiting with opioids).

Contraindications and Cautions for Analgesia Administration

- There are se veral relative contraindications to administration of analgesics in the f eld:
  - Hypotension
  - Hemodynamic instability
  - Allergies
  - Comorbidities that can lead to complications with specif c agents (e.g ., renal failure and NSAIDs)
- Extra caution with abo ve contraindications is necessar y in the prehospital setting since there is:
  - Less a vailability of backup
  - Fewer re versal agents a vailable
  - Fewer agents to counteract complications
- There are also some medical conditions that are best treated with other therapies rather than only analgesics.
  - For example, ongoing cardiac ischemia where treating with analgesics alone may prevent administration of other more suitable medications.

Quality Impro vement and Medical Over sight in P ain Management

- Quality impro vement programs ha ve been shown to impro ve assessment of pain and better medical directive compliance.
- Quality impro vement programs should include initial and ongoing education and retrospective re view to assess if management is appropriate and effective.
- Education should in volve:
  - The role of prehospital pain management
  - The assessment of pain se verity
  - Nonpharmacologic approaches to pain management
  - Pharmacolog y of analgesics
  - Patient monitoring and assessment
  - Management of side effects or complications

Nonpharmacologic P ain Management

- There are se veral nonphar macologic adjuncts that EMS pro viders can utilize to reduce pain.

- Therapeutic communication techniques can be used ver   y successfully to calm, provide reassurance, and/or distract the patient.
  - ▶ This may involve reassurance, guided imager y, or breathing techniques.
  - ▶ These techniques are rarely for   mally taught at present, but rather are par t of the "ar t" of paramedicine and commonly used b   y experienced EMS providers.
- Distraction such as music can also be used successfully for procedures that are mildly/moderately painful such as IV initiation or wound cleansing/dressing   .
- Physical modalities such as splinting/immobilization and/or positioning/ elevation of an extremity ma y reduce the pain associated with an injur   y.
- Heat or cold application can modulate pain perception in man   y injuries.
  - ▶ Heat application has also been shown to dramatically reduce pain in prehospital patients with presumed biliar   y colic.
- ■ Rubbing, massaging , or providing stimulation to an area proximal to the injur   y may sometimes reduce the perception of pain in the cerebral cor   tex (updated gate theor y).

Pharmacologic P ain Management

- ■ See Chapter 10.

Opioids

- ■ Commonly used for moderate to se   vere pain.
- ■ Usually administered intra venously in the prehospital care system with relatively quick onset and ability to titrate to effect.
- ■ Morphine and fentan yl are the most common prehospital opioids.
- ■ Since there are no signif   cant differences in outcomes with mor   phine and fentan yl, most prehospital systems should choose only one agent,   however, the fe w differences between them ma y be used to deter   mine preference in specif c situations.
- ■ Naloxone is an opioid re   versal agent that has signif   cant prehospital experience showing that it is safe and can re   verse the respirator y depression and apnea side effects.
- ■ Widespread abuse of opioid medications means that the security of these medications and the safety of the pro   viders is an additional concer   n.
- ■ Federal legislation go verns the storage and procurement of these medications making them more administratively complex.

Morphine

- ■ Most commonly used opioid in prehospital care.
- ■ May also be administered intramuscular   ly and subcutaneously , but this results in a delayed onset, less agility with titration,   and a prolonged action.
- ■ In addition to its use for painful injuries or other tr   uncal pain, it is also commonly used for cardiac ischemic pain when nitroglycerin is not effective.

Fentanyl

- ■ Although fentan yl is less commonly used b y ground ambulances, it is as effective as mor phine and ma y have a fe w advantages.

- Fentanyl is not commonly used for cardiac ischemic pain that is nonresponsive to nitroglycerin not because of concern of poor outcomes, but rather lack of evidence of benefit.
- Shorter duration of action compared to morphine makes it more preferential for procedural or short-term pain and/or for patients whose hemodynamic status may change (severe trauma patients).
- Additional route of intranasal administration makes this agent potentially attractive for pediatric use (no need for intravenous initiation).

NSAIDs and ASA

- These agents are infrequently used by EMSs.
  - Oral preparation results in delayed onset.
  - Usually for mild to moderate pain only.
- Single dose or acute use for most patients is very safe with GI upset and nausea being the most common side effects and renal failure/dysfunction being rare with single acute use.
- NSAIDs are contraindicated in patients with allergies (caution due to ASA and NSAID cross-reactivity allergies including those patients with asthma and atopy).
- There are no reversal agents for NSAIDs.
- There are many concerns with chronic use or in some patients with comorbidities such as risk of GI bleed, renal dysfunction, and potential cardiovascular risks.
- ASA is commonly used by prehospital providers for its anti-platelet actions for patients with possible acute coronary syndromes but not as an analgesic.
- There are no specific monitoring requirements for NSAIDs other that usual reassessment of the patient and their pain.

Acetaminophen

- Similar to NSAIDs, acetaminophen is available in oral forms only in North America.
- Is commonly used for mild to moderate pain although it can be combined with opioids such as codeine, oxycodone, or tramadol to be used for moderate to severe pain.
- Its oral formulation and onset of action limits its use and practicality in prehospital care.

Nitrous Oxide

- Nitrous oxide is an inhaled agent administered as a 50/50 nitrous oxide/oxygen mixture that has analgesic, sedative, and dissociative actions.
- Time of onset is 3–5 minutes with duration of action of 3–5 minutes.
- It requires a tank and special mask with demand valve that allows the patient to self-administer. Patients should receive supplemental oxygen as well.
- The mask must not be secured to the face, but rather self-administered by the patient.
  - As the patient experiences adequate analgesia and/or sedation, the mask will fall from the patient's face preventing oversedation.

- Nitrous oxide is commonly used b y EMS ser vices, but there are some concer ns that have led to its remo val by some EMS pro viders and/or entire states/ provinces.
  - Concer n of exposure to ambient gas in the closed space of an ambulance (chronic exposure has been link ed to health effects), and the abuse potential by providers.

Pharmacologic Adjuncts to P ain Management

- Sedative agents such as benzodiazepines (lorazepam, midazolam, diazepam) ma y be used in conjunction with other analgesics b y prehospital pro viders for some emergency procedures (cardio version, pacing, etc.).
  - It is impor tant to know that these agents are *not* analgesics and should be used with analgesics for painful procedures.
- Other adjunctive medications include some antinauseants used with opioids, where the analgesic effect is additive.
- The effects on sedation, respirator y depression, and blood pressure are additive with opioids and so must be used cautiously .

Prehospital Sedation

- Indications for sedation:
  - Procedural sedation: cardio version, transcutaneous pacing , intubation (pre and/or post).
  - Pain management adjunct: splinting , extrication, etc.
  - Management of phar macologic toxicity and/ or withdrawal: sympathomimetic toxicity, depressant /alcohol withdra wal.
  - Anxiolysis.
  - Pharmacologic restraint in combative and psychotic patients.
- Contraindication and cautions for sedation:
  - Prehospital sedation uses similar medications as in the ED , but the environment is vastly different.
  - As opposed to the ED , prehospital sedation is usually accomplished b y only one (or occasionally two) pro vider that is responsible for the medication, monitoring, and procedure.
  - The prehospital en vironment has the additional restrictions of the following:
    - ▶ Limited space
    - ▶ Limited backup medications
    - ▶ Limited backup equipment
    - ▶ Lack of backup per sonnel
  - Therefore, prehospital sedation should be done ver y cautiously and only when necessar y for life-threatening conditions (cardio version of an unstable rh ythm) or for the pro vider's safety (combative patient).
  - Only providers with education and experience in advanced airw ay management and ventilation should administer sedation.

- Prehospital drug-assisted intubation (DAI) requires special caution.
  - ▶ The National Association of EMS Physicians Position Statement on the topic recommends that DAI can be harmful and should not be for all providers, but rather only for specially trained and supervised providers that have additional education and CQI.
- Patient care monitoring should include everything discussed under the analgesia section including continuous ECG rhythm, oxygen saturation, HR, RR, and BP, and may also include end-tidal CO_2.

Nonpharmacologic Approach to Anxiolysis

- There are therapeutic techniques, which will assist and enhance sedation although these focus more on reassurance, calming, and guided imagery.
- In the prehospital setting these may not be effective alone for procedural sedation, but they may be invaluable in the anxious patient.

Pharmacologic Agents for Sedation

- See Chapter 3.

Benzodiazepines

- Central neuronal inhibition causing sedation, anxiolysis, and anticonvulsant activity.
- Dosage is very individual and must be titrated carefully to effect.
- Larger doses can cause respiratory depression or excessive sedation requiring intubation although the dosages where this occurs have a wide variation.
- Midazolam may be the ideal agent for prehospital use because of its short onset, short duration, and multiple routes for administration.
- Routes include IV, IO, IM, intranasal, and buccal (between the teeth and the cheek).
- Other agents include lorazepam and diazepam.

Opioids

- As discussed earlier, these agents are analgesics but can also provide some sedation.

Nitrous Oxide

- As discussed above, this agent is useful as an analgesic and also has sedative and dissociative properties but with very short duration of action, so only useful for very short procedures (splinting).

Ketamine

- Little EMS use/experience except in physician-based EMS systems.
- Has analgesic properties at lower doses and dissociative properties at higher doses; does not inhibit respiratory drive and protective reflexes.
- Also has a mild bronchodilatory action, which has lead to its use for intubation induction in asthmatic patients in hospital.
- Can be administered IM and IV.

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20 Trauma and Musculoskeletal Pain

Tomislav Jelic, Hareishun Shanmuganathan, Christian La Rivière, and Shelly Zubert

General Principles in the Trauma Patient

- Trauma management should include appropriate pain management for the patient.
- See Chapter 8 on the neurobiology of pain and its effects.
- Effects of uncontrolled pain include:
  - Stress response – sympathetic and catabolic drive.
    - ▶ Tachycardia.
    - ▶ Increased tissue oxygen consumption.
    - ▶ Hypercoagulability.
    - ▶ Immunosuppression.
    - ▶ Activation of inflammatory mediators.
    - ▶ Hyperglycemia.
  - Increased thromboembolic events.
  - Agitation.
  - Pulmonary complications (acute lung injury, acute respiratory distress syndrome).
  - Infections.
  - Increased length of stay.
  - Increased mortality.
  - Increased risk of chronic pain syndromes and posttraumatic stress disorder.
  - Difficulty in managing physiological parameters of the patient, including:
    - ▶ Ventilator intolerance.
    - ▶ Hemodynamic instability.
    - ▶ Gastrointestinal dysfunction.
    - ▶ Renal dysfunction.

Stable Trauma Patient

- The first priority is to ensure that the airway, breathing, and circulation are adequate.
- By definition, a stable patient has vitals that are stable and are close to/at the normal limits, GCS 13–15.
- No acute organ dysfunction.

Nonpharmacological Measures

- Patient reassurance.
- Patient positioning .
  - Keep weight off of the injured area.
  - Protect the injured area.
    - For example, dressings or clean drapes over wounds.
- Supporting the injured area.
  - Splints/supportive bandages or tapes.
  - Backslabs or casts.
  - Elevating injured extremities (when possible) to decrease edema.
- RICE (rest, ice, compression, elevation).

Pharmacological Pain Management

- Assess the patient for allergies or potential drug interactions.
- Use the WHO Analgesic Ladder .
  - For mild to moderate pain, you can start with PO medications first, beginning with acetaminophen or NSAIDs.
  - If pain is severe, use IV medications, titrated to effect to achieve analgesia with minimal side effects (remember that there is no hard-and-fast dose "ceiling" with opioids).
- To maintain analgesia, ensure that the patient is on regular pain medication, and avoid playing "catch up. "
- Frequent reassessment of pain and overall clinical status is the key.
- Specific recommendations:
  - IV boluses, repeated every 5–15 minutes, of morphine, fentanyl, and hydromorphone to achieve rapid pain control (see Chapter 10 on pharmacology of pain management).
  - Maintain ongoing analgesia with regularly scheduled doses of IV or PO morphine or hydromorphone.
    - Fentanyl, while having less cardiorespiratory effects than morphine, is typically too short acting to achieve ongoing pain control, unless an IV infusion is started.
    - Consider the use of patient-controlled anesthesia (PCA) (see Chapter 10 for PCA choices).
- See Chapter 10 on the side effects of narcotics.
- Trauma pearls:
  - Do not use transdermal opioids (i.e., the fentanyl patch) as absorption of the drug in the acute trauma patient will vary considerably.
  - Do not use extended-/sustained-release opioid drugs in the acute setting as analgesia requirements will vary considerably in the acute setting and the risk of opioid overdose is significant.
  - The application of regional blocks for specific injuries will often reduce the amount of systemic analgesia that is required (see Chapter 12 for specific regional nerve blocks).

Unstable Trauma Patient

- As earlier, securing and maintaining the ABCs take priority over analgesia.
- By definition, an unstable trauma patient has disrupted vitals, severe ongoing hemorrhage, and/or evidence of acute organ dysfunction.
- Nonpharmacological measures as described above are the first step.
- Consider regional blocks as they avoid the side effects of systemic opioids (pruritus, hypotension, respiratory depression), however, they introduce the risks of local anesthetic toxicity and peripheral nerve injury.
- Need to balance pain control with ongoing management and side effects of the medications.
- Parenteral therapy is preferred for moderate to severe pain.

Ketorolac

- Reduces the production of prostaglandins and thromboxane, thereby decreasing pain.
- Platelet dysfunction, gastritis, and renal impairment are side effects that are dose dependent.
- There is controversy as to whether NSAIDs impair bone, tendon, and ligament healing.
  - This effect has been demonstrated in animal studies (based on histologic examination of the fracture healing site).
  - There are no human randomized controlled trials that have shown this effect.
  - Cohort studies have shown divergent effects and often factors such as the doses used and complicating factors such as smoking have not been described.
  - It is our recommendation that demonstrated analgesia benefit of NSAIDs outweighs the theoretical, but not demonstrated, risk of impaired healing.

Opioids

- Opioids are the mainstay of analgesia in the trauma setting.
- Intramuscular or subcutaneous administration of opioids is not recommended in a trauma setting as trauma patients will have highly variable tissue perfusion and drug delivery (and thus onset and duration) is unpredictable.
- For procedural pain (e.g., placement of a chest tube), short-acting pain medications such as fentanyl is recommended as the duration of action of the drug must parallel the relatively short duration of the procedure.
- Both visceral pain and opioids themselves may induce nausea. Concomitant treatment with anti-emetics should be considered.

Ketamine

- Provides analgesia and due to its sympathetic effects, it contributes to hemodynamic stability.
- It has been shown in sub-analgesic doses to synergistically improve fentanyl's efficacy.

- Research perfor med on ventilated patients in which CO_2 was monitored show ed no increase in intracranial pressures when k etamine is used. Hence, the old doctor's myth about k etamine use and raised ICP should be discarded.
- Ketamine is a cardiac stimulant (via sympathetically mediated mechanisms), so its use in patients with cardio vascular disease is a possible problem as it can cause increased m yocardial oxygen demand.
  - Pretreatment with benzodiazepines has been shown to reduce the cardiovascular effects of k etamine.

Special Populations

The Pregnant T rauma Patient
- NSAIDs
  - Salicylates in par ticular are trapped within the fetal circulation, due to the fetus' relatively higher pH.
  - The use of NSAIDS in the pregnant patient is not recommended due to adverse mater no-fetal outcomes.
- Acetaminophen
  - First-line analgesic (and antip yretic) in the pregnant patient.
  - Assuming nor mal liver function, there is no dose adjustment required and the 4 g/24 hr ceiling is appropriate.
- Opioids
  - The primar y concer n with opioids in the pregnant patient relates to their use in the perinatal period.
  - Administration of opioids during labor ma y lead to neonatal respirator y and CNS depression.
  - Chronic high-dose use of opioids can induce an addiction/withdra wal syndrome in the neonate.

The Elder ly Trauma Patient
- Elderly patients require careful monitoring , titration, and selection of medications due to the following:
  - Decreased dr ug metabolism and excretion due to age-related decline in renal and hepatic function.
  - Altered volume of distribution – the y have relatively less lean muscle tissue and more fat.
  - Less cardio vascular and respirator y reser ve.
    - Consequently, they are not as tolerant of cardio vascular and respirator y side-effects produced b y analgesics, particularly opioid medications.
  - Due to the sympathetic drive induced b y pain, they are more susceptible to cardiac demand ischemia in the setting of uncontrolled pain.
- Approach to pain control.
  - Start with low er doses of analgesics and then titrate to effect using additional, but relatively smaller doses of analgesics.

- For ongoing analgesia, use smaller doses and longer dosing intervals than you would in a younger patient.
- The elderly requires more frequent reassessment of pain and analgesic side effects compared to young patients.

The Pediatric Trauma Patient

- The basic neurological and hormone response to pain in the pediatric patient is effectively the same as in the adult patient.
  - Physiologic differences (see Chapters 13 and 22):
    - Neurological.
    - Relatively fewer inhibitory neurotransmitters in the spinal cord, which normally help to attenuate the perception of pain.
    - Less developed inhibitory synapses within the brain, which also help to attenuate the perception of pain.
- The clinical response to pain in the pediatric patient shows marked differences to the adult.
  - Neonates and infants exhibit a lower threshold to pain with repeated exposures as opposed to the habituation shown by adults.
  - Older children report increased perception of pain with repeated painful stimuli.
  - This is likely due to cognitive immaturity. Young children do not have the maturity to understand and differentiate the source and purpose of the painful stimulus. For example, the pain of the insertion of an IV to facilitate treatment compared to the pain of a fracture.
  - Oligo-analgesia has been associated with an increased development of PTSD in pediatric trauma patients.
- Drug metabolism considerations in children.
  - Hepatic metabolism
    - Liver becomes fully functional at 1 month of age.
    - At ages 2–6, the liver is relatively larger compared to the overall body mass, so drug metabolism is actually increased during this time period.
  - Renal metabolism
    - Renal blood flow, glomerular filtration, and tubular secretion are all reduced during the first year of life. Thus, renal excretion and metabolism of drugs is relatively reduced in the first year of life.
    - Renal function normalizes after the first year.
  - Drug distribution
    - Children have less body fat – lipid-soluble drugs are available in higher concentrations in the plasma.
    - Children have decreased protein binding – thus opioids and local anesthetics have relatively higher bioavailability in children.
- Recommendations in the pediatric trauma patient (see Chapters 13 and 22 for specific recommendations).
  - Assessing pain
    - Parental assessment of a child's pain should also be considered as they typically know their child the best.

- ▶ Clinical signs and symptoms of pain.
  - ○ Crying, grunting, and moaning . How ever, the pediatric patient ma y simply remain silent due to a fear of fur ther injections.
  - ○ Tachycardia, hypertension, and tach ypnea.
  - ○ Young children ma y exhibit a paradoxical vagal response to pain – bradycardia and attenuation of the nor mal sympathetic response. This is especially tr ue in periods of prolonged pain.
  - ○ Increased muscle tone, agitation.
- ● Approach to treating pain
  - ▶ Evidence shows that pediatric trauma patients are consistently undertreated for pain due to fear s of o verdose.
  - ▶ If you are uncer tain about the le vel of a child' s pain, a trial of a small dose of analgesia is appropriate. Examine the child afterw ards for objective e vidence of an y benef cial effect.
    - ○ Decrease in le vel of tach ycardia, hypertension, and tach ypnea.
    - ○ Decreased agitation and/or more interactive with you during the exam.
    - ○ Decreased muscle tone or voluntar y guarding during the exam.
- ■ Pharmacologic approaches – see Chapter 22.

The Renal or Hepatic F ailure Patient
- ■ Opioids are primarily metabolized b y the liver and are renally excreted.
- ■ Hepatic failure
  - ● Begin with low er doses and longer dose inter vals and titrate up, as opioids and benzodiazepines will ter minate their action primarily b y redistribution as opposed to metabolism.
  - ● Avoid mor phine and codeine as these opioids ha ve active metabolites. The ideal opioids in patients with hepatic failure are fentan yl and hydromorphone.
- ■ Renal failure
  - ● Morphine and codeine ha ve active metabolites that are renally excreted, and thus will accumulate in the renal failure patient.
  - ● Fentanyl and hydromorphone are the ideal choices as the y have inactive metabolites.

Specif c Injuries and Specif c Recommendations for P ain Control

Blunt Chest T rauma
- ■ Rib fractures
  - ● Systemic analgesia to pre vent splinting and respirator y failure.
    - ▶ Achie ve acute pain control with opioids.
    - ▶ Then give regular oral and/or IV analgesia.
    - ▶ Intercostal blocks or epidural analgesia is an option in the stable patient.

Extremity Fractures

■ Splinting and casting
  ● The definitive treatment for reducing the majority of the pain associated with a fracture.
■ Elevation
■ Hematoma block (see Chapter 12)
  ● Most appropriate for Colles', Smith's, and metacarpal fractures.
■ Peripheral nerve block (see Chapter 12)
  ● Wrist block – phalangeal and metacarpal fractures, finger crush/amputation injuries.

Strains/Sprains

■ Splinting/taping.
■ Tensor bandage.
■ NSAIDs and/or acetaminophen. Consider codeine.
■ Walking aids – crutches may be necessary for brief period.

Burns

■ Nonpharmacological methods:
  ● If the burn is <30 minutes old, cooling the affected area with water (10–25 degrees celsius) helps to reduce the pain dramatically.
■ Do not use ice as this will make the burn injury worse.
  ● A moist dressing over the burn site is also effective.
  ● Avoid air currents over the burn.
  ● Avoid reinjury – heat sources or refreezing in the case of cold burns.
■ Pharmacological methods:
  ● For small area and relatively minor burns, simple oral analgesics such as acetaminophen or ibuprofen should be adequate. Consider codeine.
  ● For large/severe burns in the acute setting, parenteral opioids are the most appropriate method of pain relief.
  ● Intravenous lidocaine:
    ▶ Mechanism of analgesia: inhibits neuronal activity of afferent neurons within the dorsal horn of the spinal cord.
    ▶ A bolus dose of 1 mg/kg, then an infusion rate of 1–4 mg/min.
  ● Benzodiazepines are useful in reducing the anxiety associated with the burn injury.
    ▶ Remember that benzodiazepines and opioids have additive effects in causing respiratory depression and hypotension.

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21 Abdominal Pain

Sean Moore

Introduction

- Abdominal pain is the most common complaint in the emergency depar tment (ED), accounting for one in nine patients who present.
- Most causes are benign and self-limiting , but approximately one in six ma y be serious or life threatening .
- Diagnosis is often diff cult and may rely on elements from the patient demographics, histor y, past medical and surgical histor y, physical examination, laborator y tests, and imaging .
- The use of advanced medical imaging has increased signif cantly in recent year s for patients with abdominal pain.
- In one-third of patients, a cause will not be found in the ED .
- The vast majority of patients with undifferentiated abdominal pain will impro ve spontaneously.
- Elderly patients are at high risk for serious and surgical pathologies.

Pathophysiology

- Abdominal pain is often approached anatomically , using the approximate location of the pain as a star ting point.
- As inner vation of organs and their capsules relates to their embr yologic development or spinal ner ve level, several organs ma y present with pain in similar areas.
- Symptoms ma y range from h ypotension or shock, to nausea and vomiting , diarrhea, diffuse pain, anxiety, or localized pain depending on the organ affected.
- To differentiate the cause, it is essential to piece together elements from the histor y and physical examination to establish a pain patter n representative of more specif c pathologies.
- Visceral pain is often described as deep, dull, and poor ly localized.
  - It may be caused b y distention, inf ammation, or ischemic insults to organs.
  - Pain is often accompanied b y anxiety, diaphoresis, or a feeling of impending doom.
    - The localization of pain is poor as pain f bers enter the spinal cord at multiple le vels.

- ▶ Foregut organs, including the stomach, duodenum, liver, gallbladder, and pancreas produce upper abdominal pain.
    - ▶ Midgut organs, including the small bow el, appendix, and proximal colon produce periumbilical pain.
    - ▶ Hindgut str uctures, including the distal colon and the genitourinar y system cause low er abdominal pain.
- Parietal pain or somatic abdominal pain is produced b y ischemia, inf ammation, or stretching of the parietal peritoneum.
    - Localization of pain is specif c to the side and der matome le vel of the pain, unlike visceral pain.
    - Pain is usually shar p, constant, and specif c to the area of the organ in question.
- Referred pain is felt in an area distinct from the origin of pain.
    - It results from sharing of afferent neurons from different locations in the body .
    - Pain in non-abdominal areas ma y be refer red to the abdomen. Examples include pneumonia, glaucoma, and myocardial infarction.
    - Pain derived from abdominal processes ma y be felt in other areas such as the pelvis or thorax. An example is biliar y disease refer red to the right shoulder .
    - No one description of abdominal pain can be def nitively cor related with a specif c cause.

Undifferentiated Abdominal P ain

- Those patients who are assessed to ha ve nonsurgical abdominal examinations and are diagnosed with "undifferentiated abdominal pain" or abdominal pain NYD include approximately one-third of all cases of abdominal pain in the ED .
- The vast majority of patients lea ving the ED will ha ve complete resolution of pain within 2 w eeks of discharge.
- Analgesia should be given at the ear liest in patients with abdominal pain, including in those patients without a conf rmed diagnosis.
- Withholding pain medication before diagnosis w as based on antiquated methods, yet up to 76% of ph ysicians fail to give appropriate analgesia for these patients prior to getting a surgical e valuation.
- Many studies show impro ved diagnostic accuracy when the patient is given analgesics.
- Those who receive opioids tend to ha ve more se vere disease and are associated with higher mor tality, but no causal link has been established and analgesia should be vie wed as an impor tant aspect of care in the ED .
- Hemodynamically unstable patients need to ha ve ear ly aggressive suppor tive and surgical care prior to def nitive diagnosis or imaging in man y cases.

Abdominal Aor tic Aneur ysm

- Five percent of patients abo ve 65 year s ha ve an abdominal aor tic aneur ysm (AAA).

- It is associated with atherosclerosis, smoking, hypertension, and family history.
- Ruptured AAA has a very high mortality. Early ED identifcation may decrease mortality from 75%–35%.
- Patients with AAA may present with severe abdominal, fank or back pain. This may be accompanied by radiation to the groin or thigh.
  - Occasionally, it may present with only syncope as the presenting complaint.
  - Syncope followed by abdominal pain or hypotension should be presumed to represent AAA until proven otherwise.
- Clinical examination may show hypotension, diffuse abdominal tenderness, pulsatile abdominal mass, abdominal bruits, abdominal or fank ecchymosis, or absent distal pulses.
- The triad of abdominal pain, hypotension, and a palpable pulsatile mass are seen in approximately one-half of patients with ruptured AAA.
- Clinical examination is of limited value and ultrasound (US) examination is recommended in the ED to rule in and rule out the disease.
  - US is 98% sensitive for detecting AAA.
  - US is much less reliable for detecting rupture.
  - A 5-cm AAA associated with abdominal pain is at imminent risk for rupture.
- Management:
  - Patients with hemodynamic instability and a history of AAA or have ED US confrmation of AAA need immediate surgical consultation and operative intervention.
  - Hypertensive patients with AAA should be treated with labetolol or esmolol when an expanding but unruptured AAA is associated with elevated blood pressure.
  - Analgesia should be given judiciously to avoid hypotension.
  - Angiography or CT may be used in stable patients after consultation with surgeon.
- Despite rapid management, patients have a high mortality from ruptured AAA, and 50% of those who survive to reach the operating room will die.

Appendicitis

- Appendicitis is the most common surgical cause of abdominal pain in adults.
- Appendicitis remains a diffcult diagnosis, and missed diagnosis is one of the most common reasons for malpractice.
- It is challenging to diagnose in some cases, especially in pregnancy and elderly patients.
  - Only 20% of elderly patients present with classic symptoms of anorexia, fever, right lower quadrant (RLQ) pain, and leukocytosis.
- Typically presents as a poorly differentiated pain localizing to the periumbilical area, later localizing to the RLQ with peritoneal irritation. It is often associated with anorexia, fever, and nausea.
- Clinical examination may reveal tenderness in the RLQ, but may extend to anywhere along the length of the appendix.

- Examination ma y evolve to include peritoneal ir ritation with localized tender ness, and diffuse rigidity with increased ir ritation or perforation.
- The appendix ma y be located in se veral locations with relation to the cecum and may also extend past the midline and result in left low er quadrant (LLQ) pain.
- Laborator y f ndings:
  - High white blood cell (WBC) count ma y indicate a greater lik elihood of appendicitis, but nor mal WBC is ver y common and does not exclude disease.
  - C-reactive protein (CRP) ma y similar ly be ele vated in acute appendicitis, but a normal CRP does not exclude disease.
- Imaging:
  - In typical presentations, patients ma y proceed to surger y without imaging .
  - CT or US imaging can help clarify the diagnosis.
  - Abdominal US is often chosen as f rst line to limit radiation in facilities with operators experienced in graded compression US.
  - CT scanning with or without contrast can be used to more reliably exclude the diagnosis in cases not conf rmed by US.
  - Plain f lms are rarely helpful and not indicated.
- Serial examination should be done within 12 hour s or ear lier if symptoms e volve as longer dela ys may result in an increase in perforation.
- Management:
  - Generally, operative inter vention with appendectom y is the accepted standard of care although antibiotics ma y be successful in eliminating some cases of appendicitis.
  - Antibiotics are used if there are signs of peritoneal ir ritation.
  - PiP-Taz 3.375 g IV or Cefoxitin 2 g IV Q6H are acceptable choices preoperatively.
- It is generally acceptable to operate within 12 hour s of diagnosis.

Bowel Obstr uction

- Bowel obstr uction is the second most common cause for surgical inter vention in the elder ly.
- It may occur in small or large intestine.
  - Large-bowel obstr uction may be caused b y neoplasm, diver ticulitis, or volvulus.
  - Small-bowel obstr uction is most commonly caused b y adhesions, her nias, or neoplasms.
- Causes ma y include extrinsic, intrinsic, or intraluminal processes.
  - Accumulation of gastric, biliar y, pancreatic secretions, and oral intak e.
  - Distention of bow els and perforation ma y occur.
  - Prior surger y may lead to adhesions, causing mechanical obstr uction.
- Typically, pain is described as diffuse, poor ly localized cramping and is moderate to se vere.

- ■ Symptoms usually include:
  - ● Nausea and vomiting .
  - ● Bloating and inability to pass gas or stool.
  - ● Abdominal distention.
- ■ Fever, general abdominal tender ness, peritoneal signs, and increased or high-pitched bowel sounds may be seen on examination.
- ■ Plain radiographs may show obstruction and remains one of the few clinical settings where plain films are used in workup of abdominal pain.
- ■ If obstruction is seen, most patients will require CT evaluation.

Diverticulitis

- ■ Typically in patients older than age 50.
- ■ Colonic diverticuli may become obstructed by fecal matter, resulting in bacterial growth and subsequent inflammation and distension.
- ■ Occurs in ~30% of patients with diverticulosis.
  - ● Usually more common in sigmoid colon, thus presenting with left-sided pain.
  - ● May occur anywhere throughout the colon.
  - ● May occur in younger individuals who have more severe disease.
- ■ Typical presentation is fever, LLQ pain, and elevated WBC in a patient older than age 50.
  - ● Patients may experience diarrhea or constipation.
  - ● Nausea and vomiting may occur.
  - ● Fifty percent of patients will have heme-positive stools.
  - ● Patients may have toxic appearance if perforation has occurred.
- ■ Typically, pain is deep, unremitting, and may progress to severe diffuse pain if the patient has a perforation.
- ■ Palpation of a mass in the LLQ may be appreciated, and tenderness on rectal examination is common. A rigid abdomen with guarding may be present following perforation.
- ■ Imaging:
  - ● CT is the test of choice for diverticulitis and the workup of undifferentiated abdominal pain in the elderly.
  - ● Simple uncomplicated diverticulitis may be differentiated from abscessed or perforated diverticulitis on CT.
- ■ Management:
  - ● Inpatient treatment involves analgesia, bowel rest, IV antibiotics, and surgical consultation.
  - ● Outpatient treatment is appropriate for simple uncomplicated diverticulitis.
    - ▶ Opioid analgesia.
    - ▶ Bowel rest with clear fluids for 48 hours.
    - ▶ Metronidazole 500 mg po TID plus Ciprofloxacin 500 mg po TID is an appropriate regimen for 10 days.
    - ▶ Moxifloxacin 400 mg po OD for 10 days alternative.

Ectopic Pregnancy

- Ectopic pregnancy is the leading cause of pregnancy-related death.
- Ectopic implantation most commonly occur s in the distal ampulla of the fallopian tube.
- Forty percent are missed on the f rst ED visit.
- Two percent of all pregnancies are ectopic and the incidence is rising .
  - Risk increased with prior ectopic, IUD, PID, prior tube surger y, and assisted reproduction.
  - Over 50% of cases occur without an y risk factor s.
- Abdominal pain, missed menstr ual period, and vaginal bleeding is the classic presentation, but is not reliably seen.
- All females of child-bearing age who present with abdominal pain need a pregnancy test.
- Pregnancy in a patient who has had prior tubal ligation should be considered ectopic until pro ven otherwise.
- Clinical examination is not reliable in excluding ectopic pregnancy .
  - Patients ma y be hemodynamically unstable or stable following r upture.
  - Uterine size is often the same as intrauterine pregnancies.
- Serum quantitative HCG testing ma y show failure to double in 48 hour s.
  - An increase of <50% in 48 hour s indicates an abnor mal pregnancy.
  - Transvaginal US should show an intrauterine pregnancy (IUP) b y the time the HCG is >1,500 mIU/mL.
  - Transabdominal US should show an IUP b y the time the HCG is >5,000.
  - HCG <1,000 does *not* preclude US utility .
  - Thirty percent of ectopics are seen with HCG readings below 1,000.
- US should be done in the ED to assess for free f uid and IUP.
- Patients with free f uid and no IUP should obtain emergent surgical consultation for operative inter vention.
- Management:
  - Medical management:
    - Methotrexate is used only in reliable patients who are hemodynamically stable with small ectopic pregnancy (<4 cm), HCG < 5,000 and no free f uid on US.
    - Contraindications include hepatic or renal dysfunction, blood dyscrasia, impaired immune function, and breastfeeding .
    - Methotrexate should be offered after consultation with g ynecology.
- Surgical management:
  - Surgical treatment with laparotom y and salpingostom y or salpingectom y is the treatment of choice in unstable patients.
- Pearls:
  - Twenty percent of women with abdominal pain and bleeding in the f rst trimester will ha ve an ectopic pregnancy .
  - Twenty percent of r uptured ectopic pregnancies will ha ve normal vital signs despite class IV shock.

Epiploic Appendigitis

- Epiploic appendigitis is much more commonly diagnosed with the increased use of CT and US scanning in abdominal pain.
- Typically seen in young adults and may be seen in the LLQ or less commonly in the RLQ.
- Diarrhea may be seen in 25% of cases.
- The colon typically has approximately 50–100 fatty appendinges, which may become torsed, resulting in necrosed tissue and surrounding inflammation.
- Examination may reveal focal tenderness, usually in the LLQ.
- Guarding and rebound tenderness may occur with local peritoneal irritation.
- Management:
  - This disease process is self-limiting and does not require antibiotics for treatment.
  - Nonsteroidal anti-inflammatory drugs (NSAIDs) may reduce the pain of epiploic appendigitis.
  - Opioid analgesics should be provided for pain control.

GI Bleeds/Peptic Ulcer Disease

- Most peptic ulcers are now knows to be caused by *H. pylori* infection.
- It is classically described as burning epigastric pain but may be sharp, dull or aching. The pain may be relieved by eating and awaken the patient from sleep.
  - Rapid change in typical pain may represent perforation. Will usually have associated hemodynamic instability.
  - New onset of back pain in a patient with peptic ulcer disease (PUD) may represent ulceration into the pancreas with associated pancreatitis.
- Pearl: 50% of elderly patients with PUD present with acute abdomen.
- Nausea, coffee ground emesis, or vomiting blood may be associated or independent of pain.
- Melena stools may be seen in upper GI bleeds, and stools with bright red blood may also occur in larger bleeds or those with rapid passage of blood through the GI tract.
- Investigations:
  - CBC, liver function testing, lipase, and blood type for cross-match should be obtained early.
  - Radiographs may show free air below the diaphragm.
  - CT may help clarify the diagnosis in patients with peritonitis of unclear etiology.
- Management:
  - Patients should be kept NPO.
  - Supportive care should include aggressive fluid management and early blood administration in unstable patients.
  - Proton pump inhibitors such as pantoprazole 80 mg bolus followed by 8 mg/hr drip helps raise the gastric pH rapidly.

- Broad-spectr um antibiotics are recommended when complicated b y perforation.
- Admission and surgical consultation should be obtained for patients with hemorrhage, perforation, or obstr uction.

Hernias

- Hernias result from a defect in the abdominal w all. Abdominal contents, including fat, peritoneum, omentum, or abdominal organs, may pass through the defect transiently or become incarcerated or strangulated.
- Pain is usually localized to the area of the her nia.
  - Most commonly her nias are in the inguinal area.
  - There may be a histor y of hea vy lifting.
  - Femoral her nias are more commonly seen in females.
  - Incisional, abdominal w all, and periumbilical her nias are also possible.
- Fever and tachycardia may be present with ir reducible her nias.
- Hernias that are not incarcerated ma y be reproduced b y increasing intra-abdominal pressure.
- Investigations:
  - WBC may be ele vated, but is not reliable f nding.
  - Plain f lms may show perforation if the incarceration has resulted in perforation.
  - US or CT ma y be useful in conf rming the diagnosis.
- Management:
  - Reduction may be attempted manually , but often requires emergent surgical intervention.
  - In cases where there is question of dela yed presentation where necrotic bowel may be present, manual reduction is not advisable.
  - Patients should be k ept NPO and IV h ydration star ted pending surgical consultation.
  - Opioid analgesia should be offered.

Inf ammatory Bowel Disease

- Inf ammatory bowel disease (IBD) is a chronic disease characterized b y intermittent exacerbation of abdominal pain.
- It is often subdivided into Crohn' s, ulcerative colitis, and indeter minate colitis.
- Clinical features of the disease are often related to the anatomic distribution and severity of disease.
  - Abdominal pain is often se vere. It ma y be crampy, colicky, or unrelenting .
  - Bloody diar rhea, weight loss, and anorexia are commonly seen but symptoms are highly individualized.
  - Fever and toxicity ma y be present in cases complicated b y abscess.
  - Extraintestinal manifestations include ar thritis, episcleritis, uveitis, erythema nodosum, hepatobiliar y disease, renal colic, and thromboembolic complications.

- The course is unpredictable, but f are ups and remissions are highly individualized.
- Examination may reveal diffuse or localized tenderness depending on the individual.
- Laboratory tests may include CBC, electrolytes, CRP, and occasionally type and cross-match.
- CT imaging may be indicated to assess surgical pathology if complications are suspected.
- Management:
  - Analgesia in these patients is challenging as patients with chronic pain require high doses of opioids.
    - ▶ Many patients are inappropriately labeled as drug seekers.
    - ▶ Patients with pseudo-addiction may normalize when pain is appropriately treated.
    - ▶ Chronic pain results in complex psychosocial adaptation and patients may be challenging for many physicians.
    - ▶ Parental opioids are normally needed for pain in IBD.
  - Consultation with gastroenterology or surgery is indicated for patients who have significant complications, treatment failure, or peritoneal signs.
  - Mainstays of treatment include steroids, antibiotics for abscesses, immunosuppressive drugs, 5-ASA preparations, immune modulators, and surgical intervention.
  - Hydration, analgesia, and serial examination should be done within 12 hours or earlier if symptoms evolve as longer delays may result in an increase in perforation.
- Complications may include abscess, f ssure, f stulas, rectal prolapsed, malnutrition, and malabsorption.

Mesenteric Ischemia

- Mesenteric ischemia is usually divided into arterial or venous disease.
  - Arterial mesenteric ischemia is divided into low f ow states and occlusive disease.
  - Occlusive disease is subdivided into thrombotic or embolic.
- Pain may be gradual onset (low f ow) or acute (embolism).
- Pain tends to be poorly localized and dull but may be out of proportion to clinical f ndings.
- Pearl: Pain out of proportion to examination is a very worrisome f nding.
- The patient may have significant nausea and vomiting.
- Patients may feel significant anxiety, impending doom, or appear relatively well, making the diagnosis very diff cult.
- Patients later in presentation may have a toxic appearance with hypotension and instability.
- Serum lactate rises late in the disease process and is a poor prognostic sign.
- Radiography or CT may show pneumatosis intestinalis or portal vein gas.
  - May get false reassurance of alternate diagnosis if ileus or obstruction is found.

- Angiography, CT, or MRI ma y be used, but none is completely reliable for detecting mesenteric ischemia.
- Despite aggressive treatment, surgical inter vention to remo ve necrotic bow el, and suppor tive care, sur vival is 50% when diagnosed within 24 hour s.

Ovarian Torsion

- Ovarian or adnexal tor sion occur s when the o vary or fallopian tube twists and cuts off its blood supply .
- Early diagnosis and surgical treatment are essential if fer tility is to be preser ved.
- It can occur at an y age but primarily in reproductive year s, with 20% occur ring during pregnancy.
- Pain is typically sudden onset and located in the low er abdomen and pelvis. It localizes to the affected side, and is shar p and se vere in nature.
- Radiation of the pain ma y be to the back, pelvis, or groin.
- Late presentations ma y be associated with fe ver, peritonitis, or sepsis.
- The o vary will remain viable for approximately 6 hour s from the time of tor sion.
- Ninety-f ve percent of tor sion occur s in abnor mal adnexa (examples include tumors or cysts).
- US with color f ow assessment of blood f ow is the test of choice for diagnosis.
- Management:
  - Consult g ynecolog y for surgical management.

Pancreatitis

- Pancreatitis typically presents as se vere dull epigastric pain, which ma y radiate through to the back. Se vere cases ma y be associated with hemodynamic instability .
- Patients typically ha ve local tender ness in the epigastric area.
- Investigations:
  - Workup should include electrolytes, lipase, and contrast abdominal CT .
    - ▶ Lipase ma y be ele vated in alcoholic patients in absence of pancreatitis.
  - CT can be used to assess complications such as pseudocyst, abscess, and can grade se verity, which helps with prognostication.
  - If a biliar y cause is suspected, US ma y be perfor med to assess mechanical common bile duct obstr uction. ERCP or MRCP ma y be needed in some cases.
- Management in volves aggressive suppor tive care.
- IV f uids.
- Analgesia.
- Keep patient NPO .
- Medical ver sus surgical consultation will depend on the cause of pancreatitis.

Pelvic Inf ammatory Disease

- Pelvic inf ammator y disease is an infection of the female reproductive tract caused b y ascending sexually transmitted infection from the vagina or cer vix.

- It is the single most common serious infection in women of child-bearing age and affects one million Americans yearly.
- Two-thirds of cases go unrecognized, and may lead to serious sequelae including peritonitis, chronic pelvic pain, infertility, and increased risk for ectopic pregnancy.
- Up to 15% of affected women will become infertile after infection.
- PID is commonly caused by *Chlamydia trachomatis and Neisseria gonorrheae,* with many patients suffering from polymicrobial infections.
- Lower abdominal or pelvic pain is the most common presentation. Pain may be associated with vaginal discharge, bleeding, dysuria, or dyspareunia.
- CDC criteria for PID includes:
  - Major criteria (should all be present):
    - ▶ Cervical motion tenderness.
    - ▶ Lower abdominal tenderness.
    - ▶ Adnexal tenderness.
  - Minor criteria (may help to enhance specificity):
    - ▶ Temperature >38 °C.
    - ▶ Abnormal vaginal or cervical discharge.
    - ▶ Elevated ESR or CRP.
    - ▶ Documented gonorrhea or *Chlamydia* infection.
    - ▶ Used in daily practice.
- US may be used to assess tubo-ovarian abscess.
  - It may also show alternate diagnosis such as appendicitis or ovarian torsion.
  - Does not exclude disease, as PID is a clinical diagnosis.
- If no other cause of lower abdominal pain or tenderness is found, it is advisable to treat as PID in the absence of formal criteria as missed disease can result in serious consequences and complications.
- Management:
  - Patients need to be hospitalized if the patient is not responding to oral therapy, cannot tolerate oral treatment, has fever, there is evidence of tubo-ovarian abscess, or other surgical diagnosis cannot be excluded.
  - Intravenous antibiotic regimens may include doxycycline 100 mg IV/po BID plus cefoxitin 2 g IV Q6H.
  - Outpatient regimens may include ceftriaxone 250 mg IM plus doxycycline 100 mg po BID for 14 days plus metronidazole 500 mg po BID for 14 days (CDC guidelines 2010).
  - Supportive therapy should include intravenous fuids, analgesia, and anti-nausea medications.
  - Opioid analgesia and NSAIDs may be used to treat associated pelvic pain.

Testicular Torsion

- The incidence of torsion is 1 in 4,000 and often occurs in young males.
- Torsion of the testis results from twisting of the testes when there is abnormal fxation of the testis within the tunica vaginalis.

- Typically, pain is acute low er abdominal, pelvic, groin, or testicular pain. The pain may be constant or inter mittent.
  - Associated nausea and vomiting is common.
  - Abdominal pain is the presenting complaint in 20%.
- It is essential to examine the testes in all males with abdominal pain.
  - The testis ma y be f rm, tender, and at a higher and transver se lie.
- Laboratory and urine testing are not helpful.
- Doppler US ma y be used if the diagnosis is in doubt.
- Management:
  - Emergent surger y is essential to preser ve viability of the testis.
  - Surgical exploration is gold standard for diagnosis and dela y for testing should be a voided.
  - Manual detor sion should be attempted preoperatively if there is an y delay to the OR.
    - ▶ Detorsion of the affected testis is perfor med by rotating the testis in a manner similar to opening a book when vie wing the testes from the patient's feet. Relief of pain is a positive end point.
  - Eighty to 100% testicle viability if OR within 6 hour s, but drops to 20% at 10 hours.

Volvulus

- Volvulus ma y occur in the stomach, cecum, colon, or sigmoid.
- Commonly volvulus ma y be associated with chronic constipation.
- Volvulus typically presents as cramp y lower abdominal pain.
- Physical examination typically re veals diffuse tender ness and abdominal distension.
- Laboratory testing should include CBC, electrolytes, renal function, and lactate.
- Imaging:
  - Plain f lms ma y be diagnostic.
  - CT is often necessar y to elucidate an y underlying volvulus ma y occur in the stomach, cecum, colon, or sigmoid.
- Management:
  - Volvulus is a surgical disease and requires ear ly inter vention.
  - Suppor tive care and f uid replacement should be prompt.
  - Broad-spectr um antibiotic administration such as Pip-T az 3.375 mg IV Q6H should be given to patients.

Abdominal Pain in Women of Child-Bearing Age

- Abdominal pain in women of child-bearing age range from benign to life-threatening etiologies including ectopic pregnancy , PID, adnexal tor sion, ovarian cyst, endometriosis, f broids, appendicitis, renal colic, and biliar y disease.
- *All* women of child-bearing age need a pregnancy test.

- History must include menstrual history, sexual history, and any genitourinary symptoms.
- Examination should routinely include pelvic examination.
- Urinalysis with HCG is essential to direct the further care.
  - Positive HCG testing should be followed with quantitative testing.
- CBC is routinely obtained, but may not be helpful in diagnosis.
- A blood type/cross-match should be available for all women of child-bearing age who are pregnant and experiencing abdominal pain or are hemodynamically unstable.
- US is generally the imaging test of choice in pregnancy to avoid radiation.
- Analgesia should be given to treat painful conditions, keeping in mind potential that patients may be pregnant.
- PID is the most common serious infection among reproductive age women.
  - Majority of PID goes unrecognized.
  - Must be vigilant in looking for PID as complications are serious and may include infertility or increased risk of ectopic pregnancy.

Abdominal Pain in the Elderly

- Age in years roughly equals the probability of admission.
- Elderly patients are at higher risk for inadequate analgesia.
- These patients are also at higher risk for adverse reactions and interactions with opioids, and smaller doses should be used while titrating accordingly.
- High risk for serious pathology such as AAA, ischemic bowel, myocardial infarction, and perforated bowel.
- Most common diagnoses are biliary disease, undifferentiated abdominal pain, appendicitis bowel obstruction, and diverticulitis. Delayed diagnosis is very common.
- Mortality is markedly higher and patients may have more rapid progression of disease in the elderly population.
  - Overall mortality is ~10%.
- Diagnosis is generally more difficult with increasing age.
  - Presenting complaints and physical examination accuracy are much less reliable with advancing age.
- Laboratory testing is usually indicated, but may be misleading.
  - Normal WBC is seen in many patients with advanced surgical diagnosis.
  - Serum lactate and blood gases may be useful in identifying underappreciated serious pathology, but generally only appears late in processes such as mesenteric ischemia.
  - Lipase and liver function testing may also be inaccurate but can be useful.
- Advanced imaging is generally indicated for elderly patients with abdominal pain complaints.
  - ED US is an important consideration for patients with possible AAA.
- Serial examination and admission should be the norm for elderly patients without clear diagnosis.

Abdominal Pain in Immunocompromised and HIV

- Patients with altered immunity may develop unusual conditions including drug-induced pancreatitis, antiretroviral-induced lactic acidosis, AIDS-cholangiopathy, bacterial colitis, typhilitis, or other opportunistic infections.
- Abdominal pain is related to immune dysfunction in ~65% of AIDS patients.
  - Patients may have the usual pathologies seen by other populations as well.
- Causes may include lymphoma, CMV esophagitis/gastritis/enteritis, colitis, sclerosing cholangitis, and cryptosporidial infection.
- Clinical examination alone is rarely diagnostic in this group.
- Correlation of CD4 count may be helpful when considering opportunistic infections.
- Advanced imaging is usually indicated in patients with HIV and abdominal pain as serious pathology is common and may not be clinically apparent.

Summary

- Abdominal pain is a common presentation to the ED with a variety of causes.
- Several life-threatening causes need emergent investigations and specific treatment.

Suggested Reading

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Pediatric Pain Management

Suzan Schneeweiss

Introduction

- There is a lack of recognition and treatment of pain in children.
- Pain relief for adult patients in the emergency department (ED) is 73% versus 53% for children.
  - Younger preverbal children generally receive less analgesics than older children.
- Medical procedures in the ED are often painful, unexpected, and heightened by stress and anxiety.
- Use of analgesic agents allows child to be more comfortable and improves cooperation during diagnostic evaluation.
- Appropriate management of pain reduces negative long-term effects of pain which include:
  - Conditioned anxiety response.
  - Increased response to pain with subsequent procedures.
  - Diminished analgesic response at subsequent visit.
  - "Blood-injection-injury phobia," which affects up to 25% of adult population.

Pain Assessment

- See Chapter 13.
- Need to consider the following with a careful pain assessment:
  - Age of child.
  - Developmental level.
  - Cognitive and communication skills.
  - Previous pain experiences.
  - Associated beliefs.
- Pain history: location, intensity, quality, duration, frequency, duration, and aggravating and relieving factors.
- Pain assessment and documentation improves administration of analgesics.

Three Main Methods Are Currently Used to Measure Pain Intensity

- Self-report – considered gold standard.

- Behavioral measures
  - Crying, facial expressions, body postures, and mo vements.
  - Generally used with neonates,   infants, and younger children where communication is diff cult.
- Physiologic measures
  - Heart rate, blood pressure, respiration, oxygen saturation, palmer sw eating, and sometimes neuroendocrine responses.
  - Similar ph ysiological responses occur during stress,   which result in diff culty distinguishing stress ver sus pain responses.

Developmental Issues

- Children ha ve pain words b y 18–24 months (e.g ., "hur t," "ow ," "ouch").
- Word "pain" appear s much later in children' s vocabularies.
- Children can repor t degree of pain b y 3–4 years.
- Children >6 years can pro vide detailed descriptions of pain intensity   , quality, and location.

Pain Scales (See Chapter 13)

FLACC (F ace, Legs, Activity , Cr y, Consolability)

- For infants and children ages 2 months–7 year s and cognitively impaired.
- See Table 22.1 .

TABLE 22.1: FLACC scale for pain assessment

Category	0	1	2
Face	No par ticular expression or smile	Occasional grimace or frown, withdrawn, disinterested	Frequent to constant quivering chin, clenched ja w
Legs	Normal position or relaxed	Uneasy, restless, tense	Kicking, or legs dra wn up
Activity	Lying quietly, normal position, moves easily	Squirming, shifting back and forth, tense	Arching, rigid or jer king
Cry	No cr y (awake or asleep)	Moans or whimper s; occasional complaint	Crying steadily, screams or sobs, frequent complaints
Consolability	Content, relaxed	Reassured b y occasional touching, hugging , or being talk ed to, distractible	Diff cult to console or comfor t

Each of the f ve categories is scored from 0–2,   which results in a total score of 0–10.
From Mer kel SI, Voepel-Lewis T, Shayevitz JR, Malyvia S. The FLA CC: a beha vioral scale for scoring postoperative pain in young children. P ediatr Nurs 1997;23(3):293–297.

Pain Word Scale

- For children ages 3–7 years and older children unable to use numerical rating scale.
- "None," "a little, " "medium, " and "a lot. "

Faces Pain Scale-Revised (FPS-R)

- For ages 5–12 years (Figure 22.1).
  - Point to the face that shows how much you hurt. Score the chosen face 0, 2, 4, 6, 8, or 10, so "0" = no pain and "10" = very much pain.

0	1	2	3	4	5
No hurt	Hurts a little bit	Hurts a little more	Hurts even more	Hurts a whole lot	Hurts worst
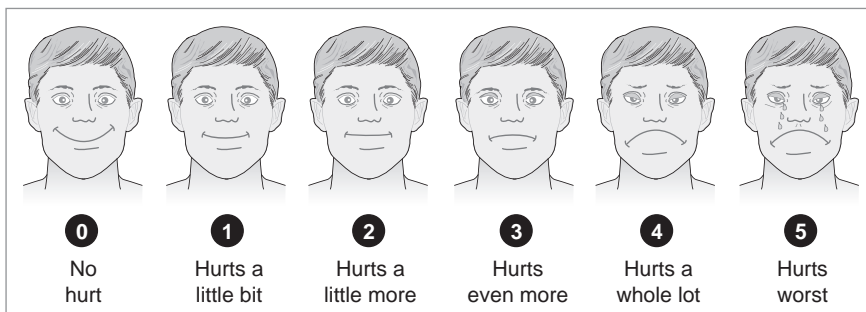
FIGURE 22.1: Faces scale for pain assessment.

Numerical Rating Scale

- For children >7 years.
- Numerical scales from 0–10 or 0–100.

Nonpharmacologic Measures for Pain Management

- See Chapter 13.
- Neonates and Infants:
  - Nonnutritive sucking .
  - Skin to skin contact with the mother (kangaroo care).
  - Rocking and holding the infant.
  - Swaddling the infant.
- Toddlers and preschoolers
  - Active distraction: blowing bubbles, providing toys with lots of colors or toys that light up, distracting conservations.
  - Passive distraction: reading age-appropriate book to child, singing songs, and practicing "blowing out birthday candles. "
- Child
  - Active techniques: blowing bubbles, singing songs, using squeeze balls, relaxation breathing , and playing with electronic devices.
  - Passive distraction: watching videos, listening to music on headphones, reading a book to the child, or telling them a story.

- Adolescent
  - Ensure private setting .
  - Allow to choose method of distraction,   presence of friends/family .
  - Active distraction: striking con versation, using squeeze balls, and pla ying with electronic de vices.
  - Passive distraction: w atching videos, training them to breathe deeply (in from the nose, count to 5 and out through the mouth),   and listening to music.

Pharmacological Measure for P ain Management

Guidelines for P ain Management

- Prevent pain whene ver possible; low er analgesic requirements with pretreatment for painful procedures.
- Adequate assessment of pain is k ey to appropriate management of pain.
- Give analgesics regular ly.
- Use least in vasive route; a void IM injections whene ver possible.
- Use WHO pain analgesic ladder (see   Figure 22.2).
  - Match analgesic to pain se verity.
  - Use more than one class of analgesics to promote better pain relief and reduce opioid requirement.
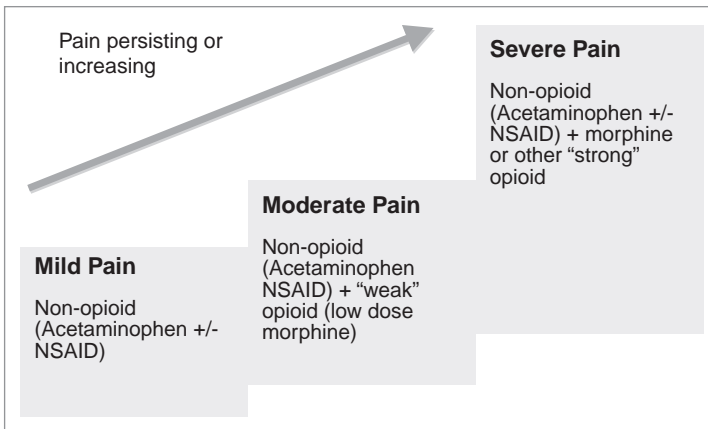
FIGURE 22.2: WHO pain analgesic ladder .

Non-opioid Analgesics

- See Table 22.2 .

Acetaminophen

- Do not exceed maximum daily recommended dose of 75 mg/kg/da y or 4 g/da y if >12 year s.
- Avoid using combination of opioid-acetaminophen (e.g   ., Tylenol #3) products as it is diff cult to titrate.

TABLE 22.2: Analgesics for mild pain in pediatric patients

Analgesic	Dosage	Advantages	Disadvantages

Acetaminophen
 Drops: 80 mg/mL
 Suspension: 32 mg/mL
 Chewtab: 80 mg
 Suppositories: 120, 325, 650 mg, respectively
	10–15 mg/kg q4–6 hr po
	 10–20 mg/kg PR
	 Dose limit:
	  75 mg/kg/day or
	  4 g/d if >12 yrs, whichever is less
		Well tolerated
		 Safe
			Liver toxicity if overdosed

Ibuprophen Drops: 40 mg/mL Suspension: 20 mg/mL	5–10 mg/kg po q6–8 hr Dose limit: 40 mg/kg/d or 2,400 mg/d adult	Longer duration of action	Gastrointestinal (GI) irritation Increased risk of bleeding after tonsillectomy
Naproxen Suspension: 25 mg/mL Tablets: 125, 250, 375 mg, respectively	10–20 mg/kg/d po divided bid Dose limit 1 g/d	Long duration of action Oral suspension	GI irritation

GI, gastrointestinal.

Nonsteroidal Anti-inflammatory Drugs
- Nonsteroidal anti-inflammatory drugs (NSAIDs) have a ceiling effect after which there is no additional analgesic benefit when maximum dose of an NSAID is attained.
- All NSAIDs offer same degree of analgesic effect (e.g., no advantage of IV ketorolac vs. po ibuprophen).
- Controversy regarding use of NSAIDs for fractures.
  - Affect bone healing in animal models, but this has not been shown in humans.
- Avoid NSAIDs for children with suspected or known inflammatory bowel disease and renal disease.

Sucrose (24% Solution)
- Effective method of procedural analgesia in neonates and young infants <6 months.
- Combine with other nonpharmacologic methods and pharmacologic measures.
- *Indications:* procedural pain including venipuncture, heel sticks, catheter insertion, LP, suturing, dressing changes, injections, and IV starts.
- One to 2 mL dripped onto anterior portion of tongue 2 minutes prior to procedure (maximum daily dose: 4 times in 24 hours).
- Increase efficacy when used with pacifier.

Opioid Analgesics
- See Table 22.3.
- Opioids are the most widely used agents for moderate to severe pain.

TABLE 22.3: Analgesics for moderate to severe pain in pediatric patients

Analgesic	Dosage	Advantages	Disadvantages
Morphine	**Intermittent dosing:** 0.2–0.4 mg/kg po/PR q4 hr child >50 kg 10–20 mg q3–4 hr 0.05–0.1 mg/kg IV/subcut q2–4 hr Dose limit: 15 mg/dose IV/subcut **Continuous infusion:** 0.1–0.2 mg/kg loading dose 0.01–0.04 mg/kg/hr IV/subcut infusion	Oral morphine more palatable than oral codeine, less GI effects Rapid onset Potent, analgesic	Respiratory depression Hypotension
Codeine	0.5–1 mg/kg po/IM q4–6 hr Maximum 1.5 mg/kg/dose 6 mg/kg/d (120 mg) Usual adult dose: 30–60 mg/dose	Potent analgesic No renal or hepatic toxicity Low addiction potential	Nausea Constipation Variability in metabolism to morphine (consider oral morphine)
Fentanyl	0.5–2.0 mcg/kg IV 1.4 mcg/kg Intranasal	Potent analgesic Less hypotension	Respiratory depression Apnea, chest wall rigidity
Hydromorphone	Intermittent IV: 0.015–0.02 mg/kg/ dose q2–4 hr Dose limit: 0.2–0.6 mg/dose IV Continuous infusion: 4–8 mcg/kg/hr IV Children ≤50 kg: 0.04–0.08 mg/kg/ dose po q3–4 hr prn Children >50 kg: 2–4 mg/ dose po q3–4 hr prn	Potent analgesic Alternative to morphine	Respiratory depression Hypotension

GI, gastrointestinal.

- ■ They have no ceiling effect.
    - ● Increasing dose generates more analgesic effect (titrate the dose to pain intensity), but also increases risk of adverse effects.
    - ● Therefore, appropriate monitoring is necessary.
- ■ Treating pain with opioid analgesics does not lead to psychological dependence or addiction.

Codeine
- ■ Weak opiate; good analgesic for moderate pain.
- ■ Combined tablets with acetaminophen limits flexibility in dosing, therefore best prescribed separately in pediatrics.
- ■ Analgesic effect dependent on metabolism to morphine.
    - ● Codeine is metabolized to morphine by cytochrome p450 enzyme CYP2D6.
    - ● Genetic variability of enzyme activity with a significant number of poor metabolizers or non-metabolizers, and extensive metabolizers – patients in whom this medication will not work well.
    - ● Less reliable and efficacious than morphine.

Morphine
- Gold standard.
- Half-life of mor phine varies with age.
  - Nine hours in preter m neonates, 6.5 hour s in ter m neonates, and 2 hour s in older infants and children.
- Delayed renal clearance of mor phine metabolites ma y contribute to the analgesic, respirator y depressant, and rarely con vulsant effects of mor phine in the neonate.
- Induces histamine release (cautious use in patients with asthma and hypovolemia).

Hydromorphone
- Derivative of mor phine (f ve to se ven times more potent than mor phine).
- Causes less sedation, pruritus, and nausea than mor phine.
- Useful alter native to parenteral mor phine.

Fentanyl
- Eighty to 100 times more potent than mor phine.
- Few cardiovascular effects other than bradycardia; ideal in patients with congenital hear t disease and trauma (neurosurgical) victims.
- Ideal for procedures as has a shor t duration of action (<60 minutes).
- Chest w all and glottic rigidity are complications to be monitored carefully .
  - Generally tends to occur with high (>5 mcg/kg) bolus but ma y occur e ven with low doses (1–2 mcg/kg).
  - Treated with naloxone or neuromuscular blockade and controlled mechanical ventilation.
- *Intranasal fentanyl* allows painless administration of analgesia, equivalent to IV morphine for pain.
  - Onset of action 4 minutes.
  - No serious adver se effects.
  - Equivalent to IV mor phine for pain.
  - Patient positioning – sitting at 45-degree angle; use nasal atomizer de vice for improved dr ug deliver y.
  - Maximum volume for nasal administration, generally 1 mL.

Management of Procedural P ain
- Ensure child-center approach (listen to needs of child and family) (see Chapter 13).
- Use parents for positive assistants rather than negative restraint.
- Active par ticipation of child and family as opposed to passive recipients.
- Use least in vasive equipment where possible.
- Use appropriate combination of nonphar macologic and phar macologic interventions.

- Sedation alone does not pro vide pain relief.
- Ensure that procedures are car ried out to maximize patient safety .
- The following should be addressed prior to an y procedure:
    - Explanation of need and impor tance of procedure.
    - An accurate description of the procedure.
    - Expectation of the intensity and duration of pain the child ma y experience.
    - Possible need for repeated procedural attempts.
    - Measures used to alle viate pain.
    - Parental guidance as to how child ma y react to pain.

Local Anesthesia

- Needle injection, venipuncture, and IV cannulation are common procedures in the pediatric ED, but may cause signif cant pain and anxiety .
- Consider the following strategies:

Strategies to Reduce P ain with Injection

- Use a small, long needle (30 G).
- Consider distraction techniques.
- Inject slowly.
- Buffered solution: add 1 mL $NaHCO_3$ to 9 mL lidocaine solution.
    - Neutralizes pH of lidocaine without affecting analgesic proper ties.
    - Stable at room temperature for 1 w eek.
- J-tip syringe
    - Sterile, single-use, needle-less syringe that deliver s medication under high pressure from a compressed carbon dioxide gas car tridge.
    - Used for injection of buffered lidocaine for intra venous cannulation.
    - Medication penetrates to depth of 5–8 mm in 0.2 seconds.
    - Found to be more effective than EMLA® and Maxilene® in reducing pain with IV cannulation.

Topical Anesthetics for Intact Skin

- See Table 22.4 .
- Can be used safely in neonates.
- Be cautions with the use of EMLA® in neonates as it can result in methemoglobinemia. Risk factor s include reduced le vel of methemoglobin reductase, prolonged exposure/repeat use, and using other methemoglobin-inducing agents concur rently.
    - Can cause methemoglobinemia.
- EMLA®is less effective than oral glucose solution for venipuncture/heel prick.

TABLE 22.4: Comparison of different topical analgesic agents

	EMLA™	AMETOP™	MAXILENE™
Pharmacology	Eutectic mixture of two amide-type local anesthetics, lidocaine, and prilocaine	4% tetracaine, ester anesthetic	4% liposome encapsulated lidocaine
Onset of action	60 min with vaso-occlusive dressing to intact skin	30–45 min with vaso-occlusive dressing to intact skin	30 min with or without vaso-occlusive dressing
Duration of action	1–2 hrs	Up to 4–6 hrs	1–2 hrs
Contraindications	Hypersensitivity to amide type anesthetics Congenital or idiopathic methemoglobinemia Children 6–12 mo receiving sulphonamides	Hypersensitivity to ester-type local anesthetics, PABA Patients with low plasma cholinesterase	Hypersensitivity to amide-type local anesthetics Avoid near mucous membranes
Adverse effects	Local reactions: whitening or erythema, slight edema, burning, or itching	Local reactions: slight erythema, slight edema, pruritus Contact sensitization	Local reactions: irritation, itching, rash Systemic effects if serum concentration in toxic range

PABA, para-aminobenzoic acid.

Summary

- Pediatric pain assessment and management requires special consideration.
- Appropriate weight-based dosing is important for adequate pain management.

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23 Renal and Biliary Colic

Andrew Worster and Rahim Valani

Renal Colic

- Renal colic occurs when calculi are formed in the renal collecting system.
  - Risk factors include family history, bowel disease, increased sodium or oxalate intake, dietary habits, gout, obesity, immobilization, and low urine output.
  - The urinary tract migration of calculi causes local tissue irritation, bleeding and increased tension to the ureteral wall, and submucosal edema.
  - These factors cause local prostaglandin secretion that, in turn, causes smooth muscle spasm and vasodilatation.
  - The latter that causes diuresis, which again increases the tension to the ureteral wall and renal pelvis. As the stone migrates through the urinary tract, the character and location of the pain may change from muscle spasm-type flank pain to ipsilateral groin and genital discomfort and symptoms of cystitis.
- If the stone is large enough, it will cause partial or complete obstruction of the ureter and, eventually, urinary tract and renal capsule distension (hydronephrosis).
- In the industrialized world, urolithiasis typically affects young, healthy adults.
  - Seventy percent are between 20 and 50 years of age with a peak incidence in males at 30 years and two peaks in women at 35 and 55 years.
  - It also affects 1–2% of the Western pediatric population.
  - White populations > Black irrespective of geographical region.
  - Men > women in White and Asian populations.
  - Women > men in Black and Hispanic populations.
- Diagnosis:
  - Urinalysis may reveal hematuria.
  - Computed tomography (CT) is the imaging of choice.
    - Advantages of CT include no intravenous contrast, visualization of radiolucent calculi, visualization of pathology outside the urinary tract, and shorter examination time.
  - Ultrasound (US) should be considered as the first choice for imaging test in patients with suspected ureteric colic for whom there is concern over the potentially harmful cumulative long-term effect of radiation.

- ▶ US has been repor ted to o verestimate stone size and, overall, might not be as accurate as CT although the differences betw een the two ma y not be clinically signif cant.
- Plain radiographs of the kidne ys, ureter s, and bladder (KUB) ha ve little value in the diagnosis of acute ureteric colic.
- Management:
  - Most (70%) stones 5 mm or less in diameter and half of those from 5.1 to 10.1 mm can be expected to pass spontaneously .
    - ▶ The length of time from symptom onset to spontaneous stone passage can vary from hour s to da ys and patients ma y remain symptomatic throughout.
  - Extracorporeal shock w ave lithotripsy (ESWL) and obser vation with analgesia with or without adjuvant medications to facilitate stone passage, that is, medical expulsive therap y (MET) are the nonin vasive management options for patients with a ne wly diagnosed ureteral stone smaller than 10 mm diameter .
  - Nonsteroidal anti-inf ammatory drugs (NSAIDs) such as k etorolac and cyclooxygenase-2 (CO X-2) inhibitor s, which interfere with prostaglandin synthesis and release can reduce the associated pain of acute ureteric colic by blocking the mechanism of action.
  - Opioids ha ve long ser ved as the analgesic of choice b y these patients and their attending emergency ph ysicians.
  - The combination of opioids and NSAIDs has been found to ha ve an additive analgesic effect greater than either medication alone.
  - Calcium channel block ers, alpha-adrenergic block ers, beta-adrenergic blockers, prostaglandin-synthesis inhibitor s, glycer yl trinitrate, and steroids have all been used as MET .
  - The use of antimuscarinics for the treatment of pain from acute ureteric colic secondar y to ureteral smooth muscle spasm might be theoretically sound but has no basis in clinical e vidence. There is also no e vidence suppor ting diuretics, high-volume f uid therapy, or antimuscarinic agents.
- Prolonged duration of symptoms is considered a complication of ureteric colic and, like infection and renal function impair ment is an indication for in vasive, def nitive therapy with ureteroscop y, percutaneous nephrolithotom y, or open surger y.
- Patients older than 60 year s are more lik ely to suffer infection or be admitted to a hospital.
- PEARL: Alw ays consider abdominal aor tic aneur ysm in elder ly patients who ha ve symptoms consistent with nephrolithiasis.

Biliary Colic

- Bile is produced b y the hepatocyte cells in the liver and stored in the gallbladder .
- Contraction of the gallbladder is stimulated with the presence of food through vagal impulses and cholecystokinin.
- Bile is needed for the digestion of lipids.
- Gallstones occur when one of the components of bile reaches a super saturation level that results in precipitation.

- Most patients with gallstones are asymptomatic.
- Types of stones include the following:
  - Cholesterol stones.
    - ▶ Is the most common type of gallstone.
  - Pigmented stones (about 20% of cases).
    - ▶ Black stones--usually in elder ly patients or those with diseases in volved with intra vascular hemolysis.
    - ▶ Brown stones--seen with infection.
- Patients can be symptomatic without stones due to biliar y sludge or gallbladder dyskinesia.
- Risk factor s for cholesterol gallstones include the following:
  - Increase age
  - Female gender
  - Obesity
  - Pregnancy
  - Rapid weight loss
  - Cystic f brosis
  - Medications--oral contraceptives, ceftriaxone, estrogens, and total parenteral nutrition.
- Patient presents with var ying pain, usually located in the right upper quadrant or epigastrium.
  - Usually post-prandial.
  - Associated nausea and vomiting is common.
  - If patient is febrile, consider acute cholecystitis or cholangitis.
- Investigations:
  - Bloodwork.
    - ▶ There are no clinical tests that can identify gallstones or biliar y colic.
    - ▶ Always check liver transaminases (check for hepatitis), alkaline phosphatase (to deter mine if there are common bile duct stones), and lipase (for gallstone pancreatitis).
- Radiological in vestigations.
  - X-ray.
    - ▶ Limited utility as only 10–15% of stones ha ve a high concentration of calcium to be seen on plain radiograph y.
    - ▶ The stone must ha ve at least 4% calcium b y weight to be radio-opaque.
  - US--has a high specif city (>98%) and sensitivity (>95%), which mak es it the imaging of choice.
    - ▶ Presence of thick ened gallbladder w all (>5 mm), pericholecystic f uid, and positive sonographic Mur phy's sign are signs of acute cholecystitis.
  - Oral cholecystograph y--now replaced by US.
  - CT--useful if suspecting other etiolog y for patient's pain. Although not an ideal test, it can detect cer tain gallstones but is super seded b y US.

- Magnetic resonance cholangio-pancreatogram--limited utility due to expense and better tests such as US to determine the presence of stones in the gallbladder.
- Useful for looking the hepatic, cystic, and common bile ducts, and choledocholithiasis.
  - Management:
    - Fluid and electrolyte correction.
    - Anti-emetic such as Metoclopramide 10 mg IV or Dimenhydrinate 50 mg IV.
    - Anti-spasmodic--Glycopyrrolate 0.2 mg IV push that can be given every 10 minutes up to three doses.
    - Analgesia--Morphine 2–5 mg IV titrated for pain relief.
    - Anti-inflammatory agents--Ketorolac 30 mg IV.
    - Antibiotics are suspected or proven acute cholecystitis.
    - Definitive management is cholecystectomy.
    - Limited utility of oral bile acids.
  - Complications of gallstones include the following:
    - Acute cholecystitis
    - Cholangitis
    - Choledocholithiasis
    - Pancreatitis
    - Gallstone ileus
    - Gallbladder empyema
    - Mirizzi syndrome--external compression of the common bile duct. The obstruction is due to gallstone in the cystic duct or Hartmann's pouch.

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