AMERICAN HEALTH CONSULTANTS®

Rapid Reference Card Enclosed

Log on to www.EMRonline.com, your source for emergency medicine information

The Practical Journal for Emergency Physicians

Emergency Medicine Reports

Volume 23, Number 22 October 21, 2002

The appropriate management of pain and anxiety in the emergency department (ED) is an important component of emergency care in all age groups. Administering medications for analgesia and sedation can facilitate interventional procedures and minimize patient pain and suffering. The use of such medicines, or procedural sedation, is an integral part of the practice of emergency medicine. Procedural sedation is performed regularly by other non-anesthesiologists, including gastroenterologists, radiologists, and cardiologists.[1]

For successful and safe procedural sedation, appropriate drugs and dosages must be chosen and administered in the proper setting on appropriate patients. Patient evaluation should be performed before, during, and after their use.

Part I of this article discussed the variety of agents available to the practicing emergency physician for procedural sedation. Part II will provide a pragmatic overview of the logistics of procedural sedation. Starting with choosing appropriate patients, this article will summarize the steps of a successful sedation, including preparation, monitoring, documentation, and post-procedure care. Specific scenarios in which sedation is employed will be reviewed, as will the use of topical agents in the ED.

—The Editor

Procedural Sedation

Part II: Specific Scenarios, Topical Agents, and Establishment of Procedural Sedation Policy within the Emergency Department

Authors: **Jonathan Glauser, MD, FACEP**, Department of Emergency Medicine, Cleveland Clinic Foundation, Cleveland, OH; Faculty, Emergency Medicine Residency, MetroHealth Medical Center, Cleveland, OH; **Brian Cullison, MD**, Resident, Department of Emergency Medicine, MetroHealth Medical Center, Cleveland, OH.

Peer Reviewer: **Sandra M. Schneider, MD, FACEP**, Professor and Chair, Department of Emergency Medicine, Strong Memorial Hospital, University of Rochester, NY.

Patient Selection

The emergency physician determines whether a patient is appropriate for procedural sedation. This determination is based on the intended procedure, the patient's medical status as defined by the history and physical exam, and by the level of sedation/analgesia required to complete the procedure. Recent food intake is not a contraindication for administering procedural sedation and analgesia, but should be considered in choosing the depth of sedation. The American Society of Anesthesiologists (ASA) recommends fasting six hours for solids and two hours for liquids,[1] but the literature does not show a change in outcomes or adverse events with fasting for procedural sedation.[2,3] Ranitidine and metoclopramide can be given 30-60 minutes prior to sedation to

increase gastric pH and reduce gastric volume.[4] NPO guidelines by age are summarized in Table 1.

In complicated patients, or patients with severe systemic disease in which the sedation/analgesia and the complexity of the procedures would reduce the patient's reserve, the physician may find an anesthesiology consult helpful in determining if the ED or operating room is best for the required procedure. Likewise, the physician should use caution in patients with significant oxygenation or anatomic airway/ventilation abnormalities as noted by history or physical exam (see below). Relative contraindications include hemodynamic instability, respiratory depression, and significant underlying medical problems.

In children, patients with severe systemic disease (ASA III or IV), infants younger than 3 months of age, premature infants younger than 60 post-conceptual weeks of age, and children with underlying respiratory airway disease, neurological conditions, central nervous system (CNS) injury, multiple trauma, or liver/kidney disease are at increased risk for sedation complications and require consultation with an anesthesiologist.[5-7] This discussion applies only to patients classified as ASA I or II. (*See Table 2.*)

Table 1. NPO Status for Children

CHILDREN < 6 MONTHS OLD	
2 hours fast clear liquids	
4 hours fast milk, solids	
CHILDREN 6 MONTHS - 3 YEARS	
3 hours fast clear liquids	
6 hours fast milk, solids	
CHILDREN > 3 MONTHS OLD	
3 hours fast clear liquids	
6-8 hours fast milk, solids	

Preparation

To enhance ED flow, the emergency physician should communicate clearly with the consulting physicians and nursing staff, so that all procedures on a given patient requiring procedural sedation and analgesia can be performed at the same time, and adequate nursing staff are available for monitoring the patient. For deep sedation, the person responsible for monitoring the patient should not be supervising, directing, or performing any part of the procedure, and should remain at the head of the patient at all times.

As part of the procedure ledger, a pre-procedure history and physical examination is performed on the patient and documented. Pertinent aspects include medical history, particularly cardiac or pulmonary disease, sleep apnea, renal or hepatic disease, and prior CNS disease. (*See Table 3.*) Alcohol, tobacco, or illicit drug use; previous problems with sedative or analgesic agents; current medications; allergies; pregnancy status; and last oral intake must be documented. A history of prior sedation/analgesia, including the adequacy of pain control for those procedures, can be helpful. Along with vital signs and oxygen saturation, the examination may include auscultation of the heart and lungs and examination of the airway for potential problems. (*See Table 4.*) Baseline ambulation status should be documented.[8]

Pertinent laboratory evaluation and/or radiographs should be considered. Currently, there is no literature to support the need for specific laboratory testing before procedural sedation and analgesia. Laboratory testing should be driven by the patient's comorbid status.

Finally, discussion should take place with the patient or legal guardian about the risks, benefits, and alternatives to sedation as well as the planned procedure, and to obtain informed consent. (*See Table 5*) Written and verbal post-procedure and sedation discharge instructions are reviewed with the patient and/or responsible adult prior to the procedure.[4,6,9] Documentation and appropriate signatures are obtained before any medication is administered. The patient must have a responsible adult with whom to be

Emergency Medicine Reports™ (ISSN 0746-2506) is published biweekly by American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President/Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Specialty Editor: Shelly Morrow
Marketing Manager: Schandale Kornegay
GST Registration No.: R128870672

Periodicals postage paid at Atlanta, GA. **POSTMASTER:** Send address changes to ***Emergency Medicine Reports***, P.O. Box 740059, Atlanta, GA 30374.

Back issues: $29. Missing issues will be fulfilled by customer service free of charge when contacted within one month of the missing issue's date.

Multiple copy prices: One to nine additional copies, $323 each; 10 to 20 additional copies, $287 each.

Accreditation

Emergency Medicine Reports™ continuing education materials are sponsored and supervised by American Health Consultants. American Health Consultants designates this continuing education activity for up to 52 hours in Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit that he/she actually spent in the educational activity.

This CME activity was planned and produced in accordance with the ACCME Essentials.

Emergency Medicine Reports™ also is approved by the American College of Emergency Physicians for 60 hours of ACEP Category 1 credit and has been approved for 52 Category 2B credit hours by the American Osteopathic Association. Emergency Medicine Reports has been reviewed by the American Academy of Family Physicians as having educational content acceptable for Prescribed credit hours. This volume has been approved for up to 60 Prescribed credit hours. Term of approval covers issues published within one year from the beginning distribution date of 1/02. Credit may be claimed for one year from the date of this issue.

American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

THOMSON
AMERICAN HEALTH CONSULTANTS

Subscriber Information

Customer Service: 1-800-688-2421

Customer Service E-Mail: customerservice@ahcpub.com
Editorial E-Mail: shelly.morrow@ahcpub.com

World Wide Web page: http://www.ahcpub.com

Subscription Prices

1 year *with* 60 ACEP/60 AMA/60 AAFP Category 1/Prescribed credits (52 AOA Category 2B credits): $524
1 year *without* credit: $379
Resident's rate $189

All prices U.S. only.
U.S. possessions and Canada, add $30 plus applicable GST. Other international orders, add $30.

For Customer Service and CME questions,

Please call our customer service department at **(800) 688-2421.** For editorial questions or comments, please contact **Shelly Morrow,** Specialty Editor, at shelly.morrow@ahcpub.com or (404) 262-5514.

Statement of Financial Disclosure

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Glauser, Cullision (authors), and Schneider (peer reviewer) report no relationships with companies related to the field of study covered by this CME program. Dr. Bosker (editor) is on the speaker's bureau for Pfizer, Aventis, Bayer, and Hoffman-La Roche. Dr. Bosker also acknowledges that he receives or has received royalties, honoraria, commissions, and other compensation relating to the sale of textbooks, consultation services, reprints of articles, and other written materials to the following pharmaceutical companies: Pfizer, Genentech, Aventis, Ortho-McNeil, Pharmacia, Hoffman-La Roche, and Bayer.

Table 2. American Society of Anesthesiologists Classifications

CLASS	PATIENT STATUS
I	Normally healthy patient. The pathologic process for which the procedure is to be performed is localized and not a systemic disturbance.
II	Mild systemic disease under control (e.g., asthma)
III	Severe systemic disease from any cause
IV	Severe systemic disease that is a constant life threat, not always correctable by the operative procedure
V	Moribund patient who is not expected to survive without the operation

Table 3. Relevant History and Physical

1. Patient age
2. History of abnormalities of major organ systems, including heart, lungs, kidneys, or airway (e.g., sleep apnea, snoring, stridor)
3. Pregnancy test only in women who are unable to ensure (based on history) that they are not pregnant
4. Current medications
5. History of any adverse or allergic drug reactions with anesthesia or sedation/analgesia
6. History of prior sedation/analgesia, including adequacy of pain control for those procedures
7. History of tobacco, alcohol, or substance use or abuse
8. Vital signs (weight, heart rate, blood pressure, respiratory rate)
9. Cardiopulmonary examination
10. Airway examination *(See Table 4.)*
11. Laboratory evaluation based on the patient's medical condition and the effect the results of such evaluation will have on the plan for procedural sedation/analgesia

discharged home once the procedure is finished.[7]

Obtaining and preparing the appropriate equipment for the sedation and for managing the airway is paramount prior to beginning the procedure. A nurse or respiratory therapist dedicated to the sedation, and who is skilled in the use of airways, bag and mask, and monitors, and knowledgeable about the pharmacology of sedatives and analgesics, should be at the patient's bedside throughout the procedure and recovery period. Oxygen delivery equipment (bag-valve mask, nasal cannula, and or/face mask) must be available at the bedside. Age-appropriate resuscitation equipment, including oxygen, intubation equipment, suction, emergency cart, and defibrillator likewise are immediately available at all times.[10]

A respiratory therapist, if available at the particular institution, should be notified of all conscious sedation procedures and ensure availability of all necessary airway and oxygen equipment at the bedside. The therapist provides an additional set of hands for assistance with any needed airway management. Capnometry is recommended for patients with the potential for decreased hypoxic ventilatory drive.[11]

Drug reversal agents, such as flumazenil for benzodiazepines and naloxone for narcotics, sometimes are used to aid in the recovery process, and must be available for immediate use. In pediatric patients, premeasured doses should be prepared at the bedside.

Monitoring

Patients who are at risk for respiratory depression and/or airway compromise, including those who have undergone deep sedation, require a high level of monitoring. This entails the presence of a physician, nurse, and respiratory therapist during procedures performed upon such patients. Intravenous (IV) access with immediate bedside availability of reversal agents, intubation, suction, and airway and oxygen equipment must be present. With the exception of those patients who have received dissociative agents, patients who are unable to follow commands are potentially at risk for airway compromise.[5,11]

Intraprocedural monitoring is documented on a monitoring record throughout the procedure. Cardiorespiratory monitoring should be performed on all patients. This includes, but is not limited to, noninvasive blood pressure, electrocardiogram (ECG) monitoring, pulse oximetry, and vital sign observation. During deep sedation, vital signs are recorded every five minutes. For other conscious sedation this should be no less frequently than every 15 minutes for adequate safety. The patient should not be left unattended at any time during the procedure.[5]

The Bispectral index (BIS monitor) is an innovation used commonly in the operating room and other settings, and increasingly in conscious sedation. Through EEG waveform monitoring, the monitor documents the patient's level of sedation and gives an analog value 0 (no brain activity) to 100 (awake).[12]

Vital signs and level of consciousness should be assessed and recorded at a minimum before the beginning of the procedure, after administration of sedative/analgesic drugs, and at regular intervals during a procedural sedation, and at the end of the procedure. The Modified Ramsay Score is a convenient, accepted nomenclature for documenting sedation. (*See Table 6.*) For normal conscious sedation cases, a patient does not remain below level 4 for longer than 15 minutes, and does not reach level 5 or 6. For deep sedation, patients normally do not remain at level 6 for longer than 15 minutes.

Pulse oximetry is used to assess oxygenation continuously and quantitatively. In general, oxygen saturation should be maintained with oxygen at greater than 95%. In patients with chronic hypoxemia, a reasonable goal is to maintain saturation at or above their baseline.[1,2] Capnography can be used to monitor ventilation.[3,9]

Electrocardiographic monitoring should be considered for use on those patients who have or are at increased risk for cardiac disease. This may include, but not be limited to, those with a history of congestive heart failure, dysrhythmias, diabetes, coronary artery disease, peripheral vascular disease, age over 50, and a smoking history of more than 20 packs per year.[1,11]

In certain circumstances, one of the above monitoring require-

ments may be suspended temporarily if performance of that requirement would result in interference with the procedure (i.e., computed tomography [CT]/magnetic resonance imaging [MRI] scans). The requirement for monitoring heart rate and oxygen saturation by pulse oximetry, however, never should be suspended.

Specific Scenarios

With most agents, slow, careful titration is the key to safe and effective procedural sedation and analgesia. The physician aims to balance sedation and analgesia, having a goal of a patient who is drowsy and falls asleep when not stimulated and who experiences no pain. Cautious titration of the analgesic (i.e., morphine) and sedative (i.e., midazolam) to achieve this balance is the key to safety, efficacy, and, ultimately, patient satisfaction with the experience.

In titrating the medications, it is important to wait 2-3 minutes for the medication to equilibrate. Further, injections should be given slowly over 1-2 minutes to decrease the risk of sudden drop in mental status or respiratory effort.

Sedation for the Ventilated Patient. Once intubated, many patients will require sedation to tolerate mechanical ventilation and to maintain an artificial airway. Inadequate sedation has been shown to be a significant risk factor for unplanned extubation.[13,14]

Benzodiazepines are the most commonly used sedative agents in the intensive care unit (ICU) setting.[15,16] Midazolam and lorazepam are the most frequently used in the pediatric ICU.[17] Lorazepam is given in a continuous infusion at 0.025-0.1 mg/kg/hour, up to 2 mg/hour in children. The loading dose is 0.05- 0.1 mg/kg. The propylene glycol vehicle may cause acidosis with extended use. Duration of effect is 10-20 hours.

Midazolam is given as a continuous infusion at 0.05-0.2 mg/kg/hour in children following a loading dose of 0.02-0.2 mg/kg. In adults, the typical infusion is 0.02-0.10 mg/kg/hour, or approximately 1-7 mg/hr. Metabolism may be affected by hepatic dysfunction.[18] Long-term infusion (more than 100 hours) may cause a transient encephalopathy, with choreoathetosis, dystonic posturing, and decreased alertness.

Propofol may be given in a pediatric dose of 0.025-0.130 mg/kg/min.[19] At all times, the patient must be monitored for respiratory depression. Even after continuous infusion, recovery from sedation is rapid, on the order of 3-6 minutes.[20] In patients older than 16 years, an infusion rate of 50 mcg/kg/min or 3 mg/kg/hour has been shown to acceptably sedate more than 50% of patients.[21] With initiation of any increase in infusion, a bolus of 1.0-2.5 mg/kg generally is administered. Propofol can induce hypotension and mild myocardial depression.[22]

Barbiturates infrequently are used in this role, due to their relatively long duration of action and potential for global CNS and myocardial depression.[23] Ketamine in a dose of 0.5-1.0 mg/kg/hour has been used in children,[24] but due to potential side effects, including emergence phenomena and potential to decrease seizure threshold, either benzodiazepines or propofol is preferred.

Pediatrics

Procedural sedation in pediatric patients is similar to the adult population, though the physician must take into consideration the different pharmokinetics in infants and children. As a generalization, the younger and smaller the child, the longer the clearance time and drug half-life due to decreased metabolizing capacity and protein-binding capacity.

The trauma involved with placing IV lines in infants or small children often makes non-IV forms of sedation, such as oral, nasal, rectal, or intramuscular (IM), more attractive. As examples, rectal methohexital for imaging procedures (e.g., CT and MRI) in pediatrics has taken on an increasing role due to its ease of administration and rates of success.[25-27] For oral delivery of fentanyl, as noted in part I of this article, oral transmucosal fentanyl citrate (OTFC) is available as a raspberry-flavored lozenge that can cause conscious sedation in an efficacious and safe manner for diagnostic or therapeutic procedures.[28]

It is critically important that dosages be calculated using a weight obtained in the ED and that equipment and resuscitation medications be size and age appropriate before commencing.

Sedation for Non-painful Conditions: Pediatric CT and MRI Scans

Relative immobility is essential during painless procedures such as radiographic imaging that may, nonetheless, produce anxiety. The goals for these procedures are hypnosis and immobility to facilitate the procedure at hand while ensuring patient safety. Ages of 7-8 years have been cited as old enough for children to comply with instructions and hold still.[29] However, with more rapid multisectional helical CT examinations, it has been reported that only 8% of those children age 1 year or younger required any sedation at all to undergo body CT.[30] As with other agents, only ASA Class I and II patients should be considered for sedation for procedures to manage nonlife-threatening problems. (*See Table 2.*)

In general, the practitioner has a choice of the following agents: pentobarbital or other short-acting barbiturate, chloral hydrate, a benzodiazepine such as midazolam, or propofol. Of these, pentobarbital and chloral hydrate are used most frequently.

Pentobarbital is an oxybarbiturate analog that induces sleep within 1-2 minutes of IV administration. Sedation is achieved

Table 4. Pre-procedural Examination of the Airway

INDICATORS OF POTENTIAL PROBLEMATIC AIRWAY:
• Decreased mouth opening
• Micrognathia
• Retrognathia
• Significant malocclusion
• Dentures
• Macroglossia
• Nonvisible uvula
• Decreased neck flexibility
• Advanced rheumatoid arthritis
• Dysmorphic facial features
– Pierre Robin syndrome
– Trisomy 21
– < 3 cm hyoid-mental distance (adult)
• Tracheal deviation

Table 5. Pre-sedation Assessment on the Day of the Procedure

1. Documentation of any changes in history and physical
2. Time and nature of last oral intake
3. Vital signs, including heart rate, blood pressure, respiratory rate, and temperature
4. Baseline ambulation status
5. Patient or legal guardian must be informed about the risks, benefits, and alternatives to the proposed procedural sedation/analgesia.

Table 6. Modified Ramsay Score for Procedural Sedation

1 = Anxious
2 = Awake, tranquil
3 = Drowsy, responds easily to verbal commands
4 = Asleep, brisk response to tactile or loud auditory stimulus
5 = Asleep, minimal response to tactile or loud auditory stimulus
6 = Asleep, no response

reliably by IV administration of pentobarbital, a short-acting barbiturate with no analgesic effect. Two to 6 mg/kg are infused slowly in increments of one-half to one-quarter of the total dose, titrated to desired response.[31] Up to 9 mg/kg total IV have been recommended for radiographic procedures.[32] Alternatively, it may be given IM at a dose of 5-6 mg/kg, although an additional dose of 1-3 mg/kg may be required 15% of the time. Time to sedation typically is 2-5 minutes via this route, with time to recovery after IV pentobarbital of approximately 55 minutes.[33] Oral pentobarbital (Nembutal) may be used, in a dose of 4-6 mg/kg, mixed with cherry syrup, in infants undergoing CT or MRI. In one study, it was better tolerated by patients than was chloral hydrate.[34]

Chloral hydrate 35-75 mg/kg may be given orally as an alternative, up to a maximum dosage of 100 mg/kg or 1 gram total in children younger than 1 year.[32] It has been used for many years for light sedation for radiographic studies.[35,36] Its absorption is erratic, and it has slow onset. Repeat dosing frequently is necessary, and its use has led to respiratory depression and death.[37,38] Onset to sedation is 30-105 minutes, with recovery time of 60-120 minutes typically.[33] Since discharge before the patient is completely awake is ill-advised, and because of delayed onset of action, chloral hydrate is considered to be a poor choice in the busy ED setting.[39]

Benzodiazepines such as midazolam have been advocated by some for sedation with immobility; however, it has been shown to be unreliable for this purpose. In one report, children ages 6 months to 6 years requiring head CT scans were given either pentobarbital 2.5 mg/kg IV dose, followed by two 1.25 mg/kg doses at 1-minute intervals (total dose of 5 mg/kg over 3.5 minutes), or midazolam in a 0.1 mg/kg IV dose followed by two 0.05 mg/kg doses. Each midazolam dose was given over 2 minutes, followed by a 2-minute wait, for a total dose of 0.2 mg/kg over 10 minutes. Three of 26 children in the midazolam group had good sedation, while 28 of 29 in the pentobarbital group had good sedation.[40] Diazepam 0.2 mg/kg and alprazolam 0.5 mg/23 kg also have been used for sedation for magnetic resonance imaging.[41] When immobilization is necessary, a short-acting barbiturate or propofol is more likely to be efficacious.[39]

Sedation can be performed on a patient with a full stomach in emergent situations. It generally is recommended that age-specific fasting guidelines be adhered to whenever possible. These are summarized in Table 1.[42,43]

Topical Agents

While not producing sedation per se, there are several topical agents that may aid in the performance of certain procedures, including venipuncture, lumbar puncture,[44] or laceration repair. Nerve blocks and tissue adhesives are considerations for laceration repair that are beyond the scope of this discussion.

Eutectic mixture of local anesthetics (EMLA) cream consists of an emulsification of highly concentrated anesthetics lidocaine and prilocaine in a ratio of 1:1 by weight (lidocaine 2.5% and prilocaine 2.5%).[45,46] The high concentration and small droplet size promote anesthetic penetration of intact skin. Ideally, patients can be identified in triage or registration who may require IV placement, to allow 60 minutes of application time. Total dosage recommended is 2.5 grams, applied 60 minutes prior to the planned procedure. The emulsion must be used on intact skin, not left for more than 2 hours, and not applied to large areas due to risk of lidocaine and prilocaine toxicity.

Depth of anesthesia ranges from 3 mm after 60 minutes to 5 mm after 90-120 minutes of the cream's application under an occlusive dressing.[47] Predisposition to methemoglobinemia is a contraindication to prilocaine use. The time frame involved for adequate absorption limits its utility in EDs. Also, it is not sterile and is not used for anesthesia of lacerations or open wounds. Local reactions such as erythema and blanching may occur, but the incidence of severe or systemic reactions is very low. Most commonly, EMLA cream is used in pediatrics for topical anesthesia, applied approximately 1 hour prior to arterial puncture, lumbar puncture, vascular cannulation, or venipuncture.

Tetracaine, adrenaline, and cocaine (TAC) solutions or gels generally contain 0.5% tetracaine, 0.05% adrenaline, and 4-11% cocaine. It is a controlled substance due to the cocaine content. The mixture provides local anesthesia 20-30 minutes after instillation into an open wound, and lasts 45-60 minutes.[47,48] Until recently, TAC was the most commonly used topical anesthetic in the United States. However it is 10-20 times more expensive than lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5% (LET) (below), and case reports of seizures, respiratory arrest, and death from absorption of cocaine systemically from incorrect application have led to more limited use.

Toxicity from rapid absorption makes the use of these solutions dangerous on mucous membranes and large abrasions. Total dose recommended is 1-3 mL. TAC should not be used on digits and other areas with end-arteriolar blood supply. It must be

Table 7. Topical Anesthesia

MEDICATION	RECOMMENDED DOSAGE	ROUTE OF ADMINISTRATION	ONSET	DURATION	ADDITIONAL INSTRUCTIONS	PRECAUTIONS/ CONTRAINDICATIONS
EMLA CREAM (2.5% LIDOCAINE, 2.5% PRILOCAINE)						
	2.5 gm	Topically (venipuncture and venous cannulation, lumbar puncture, arterial puncture)	½ - 1 hour	1 hour after removal of occlusive dressing	Provides dermal/topical anesthesia. Dermal application may cause a transient, local blanching followed by transient, local redness or erythema. Apply cream 2 in x 2 in area in a thick layer at the site of procedures. Place an occlusive (i.e., OpSite, transparent) dressing over site. Apply at least ½ hour before IV puncture. After 1-1½ hr, wipe off cream, clean the area with an antiseptic solution, and prepare for vein puncture.	Do not use in children younger than 1 month of age. Do not apply to broken or inflamed skin. Contra-indicated in patients with a known history of sensitivity to local anesthe-tics. Use with care in patients with conditions or therapy associated with methemoglobinemia (prilocaine). Avoid inadvertent trauma to the treated area by scratching, rubbing, or exposure to extreme hot or cold temperatures until complete sensation has returned.
TAC (TETRACAINE/ADRENALINE/COCAINE)						
	Total dose of TAC should be limited to 1.5 mL/10 kg (Concentration: 0.5% tetracaine 0.05% adrenaline, 4-11% cocaine)	Topically (suture of lacerations)	10-20 min	45-60 min	Soak gauze pad in solution and place directly over wound for 5-10 min. Anesthesia can be judged by the appearance of blanching at the wound site.	Do not use on mucous membranes or areas with end-arterial circulation (such as fingers, toes, nose, and penis).
TETRACAINE (AMETHOCAIME) CREAM						
	4% cream	Topically under occlusion	40 min	4 hours	Apply under occlusion for 40 min in children undergoing IV placement. Vasodilation has been noted.	
IONTOPHORESIS						
	2% lidocaine with epinephrine 0.6-1 mL	Electrode well over intact skin	10 min	10 min without epinephrine, 60 min with epinephrine		Tingling, itching, burning of skin over both electrodes may occur. Do not use in patients with pacemakers or over metal indwelling catheter ports, or fingers, nose, toes, or penis.
LET (LIDOCAINE 5%, EPINEPHRINE 0.1%, TETRACAINE 0.5%)						
	1-3 mL		20-30 min	45-60 min	May need to supplement with injected buffered lidocaine before suturing.	Effective in 75-98% of facial and scalp lacerations, only 40-60% of extremity wounds.

Table 8. Post-sedation and Analgesia Discharge Criteria

- Return to baseline verbal skills
  - –Can understand and follow directions
  - –Can verbalize, including correct diction
- Return to baseline muscular control function
  - –If an infant, can sit unattended
  - –If a child or adult, can walk unassisted
- Return to baseline mental status
- Patient or responsible person with patient can understand procedural sedation and/or analgesia ED discharge instructions
- Transportation home can be arranged

Table 9. Adverse Events as a Result of Sedation and Analgesia

- Death
- Cardiopulmonary arrest
- Airway compromise during procedural sedation
- Prolonged sedation
- New neurologic deficit
- Significant hypoxemia (saturation < 90% for 1 minute in otherwise healthy patient
- Aspiration
- Significant hypotension
- Significant bradycardia
- Significant tachycardia
- Admissions to the hospital as a direct result of intravenous conscious sedation

refrigerated. TAC does provide good hemostasis, does not distort wound edges, and can be applied painlessly. Because of necessary drug control measures , as well as the risk of seizures and death associated with the cocaine component of TAC, some prefer other topical agents.[50,51]

Lidocaine 4%, epinephrine 0.1%, and tetracaine 0.5% (LET) also may be used topically, and has been found to be as effective as TAC in local anesthesia for laceration repair. Dripped onto a wound or taped on with gauze, LET produces maximum anesthesia after 20 minutes. As with TAC, it must be made freshly and refrigerated. Greater anesthesia is achieved in scalp and facial wounds than extremity or truncal wounds. Physicians should avoid using LET on mucous membranes, the nose, penis, fingers, toes, and the ear. As a gel (with methylcellulose powder) it may be used cautiously near the eyes, nose, or mouth. It is less expensive than TAC, and requires fewer drug control measures.[52,53]

Tetracaine 4% cream applied under occlusion for 40 minutes in children provided complete anesthesia in 62%, and acceptable anesthesia in 85% of recipients.[54] It did not affect rates of successful venipuncture. It provides anesthesia for up to 4 hours and induces some vasodilation.[55]

Iontophoresis entails using a 9-volt battery to generate an electric current that draws ionized 2% lidocaine and epinephrine from an electrode well through intact skin. The unit's current of 4 mA achieves an anesthetic depth of 5-7 mm by 10 minutes. Total dose is 0.6-1.0 mL, with onset of 12-20 minutes and duration of 30 minutes.[56]

Children age 7-18 years reported less pain during IV placement after 11 minutes of iontophoresis of 2% lidocaine with epinephrine compared with placebo.[57] Dermal anesthesia with iontophoresis of lidocaine has been found to be more effective than that achieved with lidocaine/prilocaine.[58] Iontophoresis is contraindicated in fingers, toes, nose, and penis because of the epinephrine component. (*See Table 7 for a summary of the dosages and use of these agents.*)[59]

Post-Procedure Care

If a recovery area is used, it should be equipped with appropriate monitoring and resuscitation equipment. As with the procedure, an individual capable of monitoring patients and able to recognize complications of procedural sedation/analgesia should be present. The physician performing the procedure or his/her physician designee should be available immediately for consultation until the patient is discharged.

Patients who still are sedated at the end of the procedure or those who received narcotic or benzodiazepine reversal are recovered within the ED prior to discharge home. The duration of all agents used, especially including reversal agents, must be taken into consideration before discharging the patient. Due to the relatively brief half-life of existing reversal agents, patients usually are observed for up to 2 hours after administration of naloxone or flumazenil.

To ensure safe recovery, vital signs and level of consciousness continually are assessed until the patient's mental status has returned to baseline and the patient completely awakens from the sedation. Recovery should take place in the presence of an individual capable of monitoring patients and recognizing complications. Vital signs are monitored until they are within 20% of the patient's normal range. Assessment for pain, wound drainage, any nausea and vomiting, bladder distention, or compromised neurovascular status should be carried out prior to discharging the patient. No patient should be discharged or sent to a medically unsupervised setting (x-ray, clinics, etc.) until his or her mental status has returned to the presedation state. If the patient is transferred prior to the return to presedation mental status, the patient should be accompanied by a nurse.

A standard set of discharge criteria help to ensure safe discharge of the patient. (*See Table 8.*) Discharge criteria, including level of consciousness criteria, should be met for at least a 30-minute period prior to the patient's discharge home. The patient should be able to ambulate with assistance consistent with age and prior ambulatory status. The post-procedure and sedation discharge instructions are reviewed once again with the patient and/or responsible party prior to the patient's leaving the ED.

Discharge Instructions

Discharge instructions include general warnings regarding decreased mental acuity following sedation, and specific activi-

Figure 1. Conscious Sedation: Sample Discharge Instructions Form

Emergency Department
Hospital Name
Hospital Address
Hospital City, State, Zip

GENERAL INFORMATION: You or your child were give medications to induce sleepiness so the doctors could perform a procedure on you with minimal discomfort. The anesthetics, sedatives, or pain-killers which were given to you in the emergency department may still be active for the next 24 hours. You or your child may feel a little drowsy from them.

The medications you or your child received:

_____
_____
_____

INSTRUCTIONS:

Activity
Adult: Rest at home today, progressing to regular activities as tolerated. Do not drive a car, operate dangerous machinery or power tools for 24 hours. This includes sewing machines and blenders, as well as heavy equipment. Do not drink alcohol or take unprescribed medication for 24 hours. Do not make any important decisions. Be careful climbing stairs.

Child: Restrict activity to quiet play, watching TV, coloring. Do not let child play unattended for 24 hours due to possible dizziness or drowsiness. Do not let your child ride bicycles or play on swingsets for the next 24 hours.

You or your child may feel sleepy. It is fine to sleep.

Diet
Adult: Clear liquids are best tolerated at first. If you are not nauseated, you can progress your diet to solid foods as tolerated.

Child: Start with fluids, then advance to appropriate diet for child's age, as tolerated. Fluids are more important for the first 24 hours.

CONTACT YOUR DOCTOR OR RETURN TO THE EMERGENCY DEPARTMENT IF:
1. There is vomiting more than 24 hours.
2. There are signs of infection such as fever or chills.
3. You or your child have pain unrelieved by acetaminophen or other prescribed pain medication.
4. There are any complications from the procedure performed.

ties to avoid. (*See Figure 1.*) The patient is warned that the anesthetics used still may be active for 24 hours after the procedure.

Activity for the patient is minimized during the day following sedation, progressing to regular activities as tolerated. Instructions for avoiding driving cars or operating dangerous machinery or power tools for 24 hours should be given. As examples, instructions can include avoiding sewing machines, blenders, and heavy equipment. Patients are advised not to drink alcohol or take any other sedatives for one day, and not to make important decisions for one day following anesthetics. Caution is advised when climbing or descending stairs. For adults, a diet consisting of clear liquids is best tolerated at first. If the patient has no nausea 6 hours after the procedure, the diet can be advanced as tolerated to solid foods.

For pediatric patients, a similar but more age-appropriate set of instructions is utilized. Parents are advised that their children may feel sleepy, and that sleeping, rest, or quiet play (coloring, watching TV) are recommended activities after sedation. The child's diet likewise is started with fluids, and advanced as tolerated to the appropriate diet for the child's age.

Warnings included in discharge instructions request the patient to return or contact the doctor if the patient has persistent vomiting for more than 24 hours, experiences signs of infection such as fever or chills, or if there are any complications from the procedure performed. In children, if the patient has pain unrelieved by acetaminophen or other prescribed pain medication, the patient is warned to return. Table 8 summarizes discharge criteria, and Figure 1 is a sample discharge instruction form.

Quality Assurance and Complications

Adverse events in the ED as a result of sedation and analgesia are multifold, and should be documented both for the patient record and as a part of quality assessment and improvement activities. (*See Table 9.*) An established quality assurance process

Table 10. Model Quality Assurance Pathway

1. Each procedural area should identify an individual responsible for adverse event reporting and follow-up.
2. Each procedural area should determine a system for assuring compliance with documentation requirements.
3. Monthly summary reports should be made to the quality management authority or equivalent. Among specific events to be reported include:
   a. Total number of sedation cases per month per physician and whether the cases were moderate or deep sedation;
   b. Recovery phase > 3 hrs;
   c. Admissions as a direct result of procedural sedation;
   d. Emergency resuscitation of any type (drugs, airway, or cardiopulmonary support) initiated due to procedural sedation;
   e. Procedure cancellation due to adverse events related to sedation/analgesia;
   f. For moderate sedation cases, any prolonged airway intervention;
   g. For moderate sedation cases, any need to rescue the patient from slipping into deep sedation; or
   h. For deep sedation cases, any need to rescue the patient from slipping into general anesthesia.
4. The monthly reports should be reviewed by the hospital quality management board and the quality review officer of the department from which the report originated.

helps maintain a high degree of safety and should be an integral part of the ED's conscious sedation policy. (*See Table 10.*)

Conclusion

Procedural sedation enables the emergency physician to perform therapeutic interventions in a safe and humane fashion. As well, sedation enables diagnostic testing to be done accurately and in a controlled setting. As with other interventions, proper use of sedative agents requires screening, preparation, and an ongoing quality monitoring process.

References

1. American Society of Anesthesiologists. Practice guidelines for sedation and analgesia by non-anesthesiologists. *Anesthesiology* 1996; 84:459-471.
2. American College of Emergency Physicians. Clinical Policy for procedural sedation and analgesia in the emergency department. *Ann Emerg Med* May 1998;31:663-677.
3. Innes G, Murphy M, Nijssen-Jordan C, et al. Procedural sedation and analgesia in the emergency department. Canadian consensus guidelines. *J Emerg Med* 1999;17:145-156.
4. Sacchetti A, Schafermeyer R, Gerardi M, et al. Pediatric analgesia and sedation. *Ann Emerg Med* 1994:23:237-250.
5. Holzmann, R, Cullen D, Eichhorn J, et al. Guidelines for sedation by nonanesthesiologist during diagnostic and therapeutic procedures. *J Clin Anesth* 1994;6:265-276.
6. American College of Emergency Physicians. Use of Pediatric Sedation and Analgesia. *Ann Emerg Med* 1997;29:834-835.
7. Ostman PL, White PF. Outpatient anesthesia. In: Miller. *Anesthesia*, 4th Edition. New York: Churchill Livingstone;1994.
8. Rose J, Koenig K. Procedural sedation and analgesia. *Critical Decisions in Emergency Medicine* 1996;10:9-15.
9. American Academy of Pediatrics: Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. *Pediatrics* 1992;89:1110-1115.
10. Ward KR, Yealy DM. Systemic analgesia and sedation for procedures. In: Roberts. *Clinical Procedures in Emergency Medicine*, 3rd Edition, Philadelphia: W.B. Saunders Co.;1998.
11. Gross JB. et al. Guidelines for sedation and analgesia by nonanesthesiologists. *Anesthesiology* 1996;84:459.
12. Glass P, Johansen A. The Bispectral Index monitor. *Surg Serv Mgmt* 1998;4:50.
13. Boulain T. Unplanned extubations in the adult intensive care unit: A prospective multicenter study. *Am J Respir Crit Care Med* 1998; 157:1131-1137.
14. Chevron V. Unplanned extubation: Risk factors of development and predictive criteria for rein. *Crit Care Med* 1998;26:1049-1053.
15. Burns AM, Shelly MP, Park GR. The use of sedative agents in critically ill patients. *Drugs* 1992;43:507-515.
16. Hansen-Flaschen JH, Brazinsky S, Basile C, et al. Use of sedating drugs and neuromuscular blocking agents in patients requiring mechanical ventilation for respiratory failure. *JAMA* 1991; 266:2870.
17. Reves JG, Fragen RJ, Vinik HR, et al. Midazolam: Pharmacology and uses. *Anesthesiology* 1985;63:310.
18. Primak LK, Lowrie L. Paralyzation and sedation of the ventilated trauma patient. *Resp Care Clin N Am* 2001;7:97-126.
19. Sacchetti A, Schafermeyer R, Gerardi M, et al. Pediatric analgesia and sedation. *Ann Emerg Med* 1994;23:237-250.
20. Lund N, Papadakos PJ. Barbiturates, neuroleptics, and propofol for sedation. *Crit Care Clin* 1995;11:875.
21. Norreslet J, Wahlgreen C. Propofol infusion for sedation of children. *Crit Care Med* 1990;18:890.
22. Puttick RM, Diedericks J, Sear JW, et al. Effects of graded infusion rates of propofol on regional and global left ventricular function in the dog. *Br J Anesth* 1992;69:375.
23. Mirski MA, Muffelman B, Ulatowski JA, et al. Sedation for the critically ill neurologic patient. *Crit Care Med* 1995;23:2038.
24. Tobias JD, Martin LD, Wetzel RC. Ketamine by continuous infusion for sedation in the pediatric intensive care unit. *Crit Care Med* 1990;18:819.
25. Pomeranz ES, Chudnofsky CR, Deegan TJ, et al. Rectal methohexital sedation for computed tomography imaging of stable pediatric emergency department patients. *Pediatrics* 2000;105:1110.
26. Zink BJ, Darfler K, Salluzzo RF, et al. The efficacy and safety of methohexital in the emergency department. *Ann Emerg Med* 1991;20:1293.
27. Bono JV, Rella JG, Zink BJ, et al. Methohexital for orthopaedic procedures in the emergency department. *Orthop Rev* 1993;22:833.
28. Schutzmann SA, Liebelt, E, Wisk M, et al. Comparison of oral transmucosal fentanyl citrate and intramuscular meperidine, promethazine, and chlorpromazine for conscious sedation of children

undergoing laceration repair. *Ann Emerg Med* 1996;28:385.
29. Lawson GR. Sedation of children for magnetic resonance imaging. *Arch Dis Child* 2000;82:150-153.
30. Pappas JN, Donnelly LF, Frush DP. Reduced frequency of sedation of young children with multisection helical CT. *Radiology* 2000; 215:897-899.
31. Kennedy RM, Luhmann JD. Pharmacological management of pain and anxiety during emergency procedures in children. *Pediatric Drugs* 2001;3:337-354.
32. Kaye RD, Sane SS, Towbin RB. Pediatric Intervention: An update—Part I. *Jour Vasc Interv Radiol* 2000;11:683-697.
33. Strain JD, Harvey LA, Foley LC, et al. Intravenously administered pentobarbital sodium for sedation in pediatric CT. *Radiology* 1986; 161:105-108.
34. Chung T, Hoffer FA, Connor L, et al. The use of oral pentobarbital sodium (Nembutal) versus chloral hydrate in infants undergoing CT and MR imaging—A pilot study. *Pediatr Radiol* 2000;30:332-335.
35. Thompson JR, Scneider S, Ashwal S, et al. The choice of sedation for computed tomography in children: A prospective evaluation. *Radiology* 1982;143:475-479.
36. Keeter S, Benator RM, Weinberg SM, et al. Sedation in pediatric CT: National survey of current practice. *Radiology* 1990;175: 745-752.
37. Binder LS, Leake LA. Chloral hydrate for emergency pediatric procedural sedation: A new look at an old drug. *Am J Emerg Med* 1991;9:530-534.
38. Greenberg SB, Faerber EN, Aspinall CL, et al. High dose chloral hydrate sedation for children undergoing CT. *J Comput Assist Tomog* 1991;15:467-469
39. Proudfoot J. Pediatric procedural sedation and analgesia: Keeping it simple and safe. *Pediatr Emerg Med Reports* 2002;7:13-24.
40. Moro-Sutherland DM et al. Comparison of intravenous midazolam with pentobarbital for sedation for head computed tomography imaging. *Acad Emerg Med* 2000;7:1370-1375.
41. Bluemke DA, Breiter SN. Sedation procedures in MR imaging: Safety, effectiveness, and nursing effect on examinations. *Radiology* 2000;216:645-652.
42. Cote CJ. Sedation for the pediatric patient. *Pediatr Clin North Am* 1994;41:31-58.
43. Cameron ML, Sponseller PD, Rossberg MI. Pediatric analgesia and sedation for the management of orthopedic conditions. *Am J Orthopedics* 2000;29:665-672.
44. Ljungman G, Gordh T, Sorensen S, et al. Lumbar puncture in pediatric oncology: Conscious sedation vs. general anesthesia. *Med Pediatr Oncol* 2001;36:372-379.
45. Gajraj NM, Pennant JH, Watcha MF. Eutectic mixture of local anesthetics (EMLA) cream. *Anesth Analg* 1994;78:574-583.
46. Maunuksela EL, Korpela R. Double-blind evaluation of a lignocaine-prilocaine cream (EMLA) in children. *Br J Anaesth* 1986; 58:1242-1245.
47. Bjerring P, Arendt-Nielsen L. Depth and duration of skin analgesia to needle insertion after topical application of EMLA cream. *Br J Anaesth* 1990;64:173-177.
48. Ernst AA, Marvez E, Nick TG, et al. Lidocaine adrenaline tetracaine gel versus tetracaine adrenaline cocaine gel for topical anesthesia in linear scalp and facial lacerations in children aged 5 to 17 years. *Pediatrics* 1995;95:255-258.
49. Schilling CG, Bank DE, Borchert BA, et al. Tetracaine, epinephrine (adrenalin), and cocaine (TAC) versus lidocaine, epinephrine, and tetracaine (LET) for anesthesia of lacerations in children. *Ann Emerg Med* 1996;28:396-398.
50. Dailey RH. Fatality secondary to misuse of TAC solution. *Ann Emerg Med* 1988;17:159-160.
51. Wehner D, Hamilton GC. Seizures following topical application of local anesthetics to burn patients. *Ann Emerg Med* 1984;13: 456-458.
52. Schilling CG, Bank DE, Borchert BA, et al. Tetracaine, epinephrine, and cocaine (TAC) versus lidocaine, epinephrine, and tetracaine (LET) for anesthesia of lacerations in children. *Ann Emerg Med* 1995;25:203-208.

53. Smith GA, Strausbaugh SD, Harbeck-Weber C, et al. New non-cocaine-containing topical anesthetics compared with tetracaine-adrenaline-cocaine during repair of lacerations. *Pediatrics* 1997;100:825-830.
54. Lawson RA, Smart NG, Gudgeon AC, et al. Evaluation of an amethocaine (tetracaine) gel preparation for percutaneous analgesia before venous cannulation in children. *Br J Anaesth* 1995;75: 282-285.
55. Van Kan HJM, Egberts ACG, Rijnvos WPM, et al. Tetracaine versus lidocaine-prilocaine for preventing venipuncture-induced pain in children. *Am J Health Syst Pharm* 1997;54:388-392.
56. Kennedy RM, Luhmann JD. The "Ouchless Emergency Department": Advances in decreaseing distress during painful procedures in the emergency department. *Ped Clin N Am* 1999;46:1215-1247.
57. Zempsky WT, Anand KJS, Sullivan KM, et al. Lidocaine iontophoresis for topical anesthesiabefore intravenous line placement in children. *J Pediatr* 1998;132:1061-1063.
58. Irsfeld S, Klement W, Lipfert P. Dermal anesthesia: Comparison of EMLA cream with iontophoretic local anesthesia. *Br J Anaesth* 1993;71:375-378.
59. Committee on Drugs, American Academy of Pediatrics. Guidelines for monitoring and management of pediatric patients during and after sedation for diagnostic and therapeutic procedures. *Pediatrics* 1992;89:1110-1115.

Physician CME Questions

81. Which of the following should be documented prior to procedural sedation?
    A. Alcohol and tobacco use
    B. Vital signs and oxygen saturation
    C. Baseline ambulation status
    D. Cardiac or pulmonary disease
    E. All of the above

82. Suggested laboratory testing for patients undergoing procedural sedation includes:
    A. CBC and serum electrolytes on every patient.
    B. chest radiography prior to any sedation.
    C. testing driven by the patient's premorbid status.
    D. routine screening for liver and renal disease.
    E. toxicologic screening to ensure no untoward drug interactions.

83. Monitoring the patient for procedural sedation entails which of the following?
    A. The patient should not be left unattended for more than two minutes at a time.
    B. Pulse oximetry and ECG monitoring is mandatory.
    C. Suction, airway, and oxygen equipment should be available within the emergency department.
    D. For patients with chronic hypoxemia, it is reasonable to maintain saturation within 5-7% of the patient's baseline.
    E. All of the above

84. For sedation of the ventilated patient, which of the following is true?
    A. Benzodiazepines are the most commonly used in the ICU setting.
    B. Propofol may be used in the pediatric as well as the adult setting.
    C. The recovery time is shorter with propofol use as compared to benzodiazepenes.
    D. Ketamine has the potential to cause emergence phenomena and to lower the seizure threshold.
    E. All of the above are true.

85. The two most commonly employed agents for pediatric sedation for computed tomography (CT) and magnetic resonance imaging (MRI) are:
    A. ketamine and midazolam.
    B. pentobarbital and chloral hydrate.
    C. morphine sulfate and meperidine.
    D. fentanyl and midazolam.

86. Which of the following statements is true of tetracaine, adrenaline, and cocaine (TAC)?
    A. It has been associated with seizures, respiratory arrest, and death.
    B. It is safe to use on mucous membranes and on large abrasions for comfort and hemostasis.
    C. It is less expensive than lidocaine-epinephrine-tetracaine (LET).
    D. The total dose recommended is 7-10 cc.
    E. It may be stored at room temperature.

87. Post-procedure care following sedation entails:
    A. immediate radiography in the x-ray suite while the patient is sedated to assure adequate reduction while the patient is cooperative for imaging, if an orthopedic procedure was performed.
    B. attendance by a friend or family member if the patient is asleep to warn health care personnel if any problems develop.
    C. observation for at least two hours after administration of reversal agents such as flumazenil or naloxone.
    D. vital signs and level of consciousness monitoring until the patient's oxygen saturation stays above 90% for at least 5 minutes.
    E. All of the above

88. A patient may be discharged home after procedural sedation:
    A. immediately if he wakes up quickly after naloxone administration.
    B. with a responsible adult if the patient is asleep but oxygenating well, provided that the guardian is reliable and will return if the patient is still drowsy in six hours.
    C. to drive his own car so long as the emergency physician documents that he is awake.
    D. in the company of a responsible adult, with transportation arranged.
    E. with advice to avoid heavy alcohol intake for at least four hours.

89. As part of a quality assurance program for procedural sedation in the emergency department, which of the following should be done?
    A. The emergency department should identify an individual responsible for adverse event reporting and follow-up.
    B. The emergency department should determine a system for assuring compliance with documentation requirements.
    C. Monthly reports should be reviewed by the hospital quality management board.
    D. Adverse events to be reported include recovery of more than three hours, admissions as a result of procedural sedation, and emergency resuscitations as a result of procedural sedation.
    E. All of the above

90. Discharge instructions should include information about limiting activities for 24 hours after sedation, avoiding operating heavy machinery, avoiding making important decisions for one day, and consuming a diet of clear liquids at first.
    A. True
    B. False

In Future Issues: Hand Emergencies

Emergency Medicine Reports

CME Objectives

To help physicians:

- quickly recognize or increase index of suspicion for specific conditions;
- understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- be educated about how to correctly perform necessary diagnostic tests;
- take a meaningful patient history that will reveal the most important details about the particular medical problem discussed;
- apply state-of-the-art therapeutic techniques (including the implications of pharmaceutical therapy discussed) to patients with the particular medical problems discussed;
- understand the differential diagnosis of the entity discussed;
- understand both likely and rare complications that may occur;
- and provide patients with any necessary discharge instructions.

AMERICAN HEALTH CONSULTANTS®

Earn bonus CME credits with this special supplement!

GERIATRIC

Your Guide to Caring for Elderly Patients in the Emergency Department

Emergency Medicine Reports™

Volume 1, No. 3 October 2002

Malignancy in any age group is a harbinger to potential complications. Complications of malignancy cross all age groups, although many features are common in both adult and geriatric patients. Cancer occurs more frequently in patients older than age 65, and ranks second to cardiovascular disease in mortality rate for that age group.[1,2] Fifty percent of all cancers occur in patients older than age 65, and it is predicted that this figure will increase to 60% by the year 2010.[2] Advanced age not only predisposes to cancer, but complications also can affect the elderly in unique ways compared to pediatric and adult populations. For example, the risk of thromboembolism is higher in the elderly; in the presence of malignancy, that risk is further increased in the older patient. Elders reached 13.1%[3] of the U.S. population in the year 2000, and with advances in surgery, chemotherapy, radiation, and critical care, more elderly patients are seeking emergency department (ED) care for malignancy-related complications.

—The Editor

Malignancy Complications in the Elderly

Author: **Fitzgerald Alcindor, MD, FACEP,** Assistant Director, Department of Emergency Medicine, North Shore University Hospital at Forest Hills, Queens, NY; Emergency Medicine Instructor, New York University School of Medicine Emergency Medicine Residency Program at North Shore University Hospital at Manhasset.

Peer Reviewer: **Verena T. Valley, MD,** Associate Professor, Department of Emergency Medicine, University of Mississippi Medical Center, Jackson.

Introduction

Malignancy complications in the elderly may present as subtly as altered mental status or as dramatically as seizures or frank bleeding. Emergency physicians should aggressively approach and manage elderly patients with an acute or an established malignancy-related complication; there is a 17% increase in cancer mortality in patients older than age 55, yet cancer mortality has declined in the younger population in recent years.[1,2]

This article will review malignancy complications in the elderly from an organ system approach, outlining the physiological changes of aging and how they are related to the consequences of cancer when relevant. *(See partial list in Table 1.)* As many complications are similar in both adults and the elderly, features of disease, the differential diagnosis, and management strategies that are unique to the elderly will be emphasized. It should be noted that, although older age predisposes patients to cancer, cancer is not an inevitable consequence of normal aging.[2]

Neurologic Complications in the Elderly

Brain function in the healthy older adult remains mostly unchanged with age.[6] However, brain weight decreases with age, and functions such as memory, attention, and language variably decline in later years.[7] Neurological manifestations of malignancy occur in 15-20% of cancer patients.[8] Altered mental status is the most frequent neurological presentation and presents a challenge to the emergency physician, as the differential diagnosis is broad. The etiologies include cerebral herniation, central nervous system (CNS) infections, increased intracranial pressure, and toxic/metabolic disorders. Spinal cord compression (SCC) and seizures are common complications and warrant further examination.

Spinal Cord Compression. SCC is the second most common neurologic complication of malignancy second to brain metastases. It is found in 5% of cancer patients at autopsy.[9] The lifetime risk of this potentially devastating event for a cancer patient is 1%.[9] Most patients with SCC are known to have cancer. However, in 10% of SCC patients, it is the first sign of the presence of a neoplasm. Every year, 18,000-20,000 cases of SCC occur in the United States.[10] The compression may be epidural, intramedullary, or extradural. The tumor initially spreads hematogeneously and leads to compression as its bulk

increases. Anterior compression, the most common type, is secondary to posterior extension of a vertebral body mass into the epidural space (25%) or vertebral body collapse (75%), leading to vascular compromise of the spinal cord (edema, ischemia, and necrosis).[11] SCC occurs most frequently with neoplasm of the breast (20.6%), neoplasm of the lung (17%), and lymphoma.[3] The onset of cord impingement typically occurs earlier in lung carcinoma (within 3 months of diagnosis in 87% of patients) than in breast cancer (19 years after the initial diagnosis).[3]

Cord compression most commonly affects the thoracic spine (70%), followed by the lumbosacral region (20%) and the cervical spine (10%).[12] SCC usually is a gradual process, but it also may be sudden. The most significant prognostic sign is the patient's premorbid neurologic status. Patients who are ambulatory are likely to fare better than those with prior neurologic impairment.[11] The most common initial symptom is localized or radicular pain (70-95%). The pain of cord compression typically is dull, persistent, and not relieved by rest, distinguishing it from the pain of a herniated disc. Movement, sneezing, and neck flexion tend to worsen the discomfort. The second most common complaint is weakness, which is seen in 80% of patients. Patients may complain of difficulty climbing stairs or rising from chairs. The physician should observe the patient walking. Paresthesias, ataxia, and later urinary frequency, urgency, urinary retention, impotence, and constipation are common. The emergency physician can pinpoint the level of dysfunction by obtaining a detailed history and performing a careful neurologic exam.

The differential diagnosis of back pain in the elderly ED patient includes a search for mechanical/degenerative, visceral, vascular, neurogenic, psychogenic, infectious, traumatic, metabolic, inflammatory, and neoplastic causes. In general, low back pain is benign in the majority of patients; it has a serious etiology, such as a malignancy, in 5% of patients ages 20 to 55 years, and in 20% of patients older than age 55.[13]

Plain spine radiographs are diagnostic of vertebral metastases in 72-83% of patients, and should be obtained as the initial study.[11] However, they may be normal in up 28% of cases, especially in patients with lymphoma. Computed tomography (CT) and magnetic resonance imaging (MRI) are more sensitive for detecting posterior lesions than plain radiographs. CT is more sensitive than MRI for assessing vertebral stability and bone destruction. MRI has replaced myelography and is very useful for planning radiotherapy and surgery.[11] Obtain a complete blood count (CBC), metabolic profile, erythrocyte sedimentation rate (ESR), and urinalysis to further define the etiology of low back pain in the elderly.

Patients with late signs such as localized weakness, bowel or bladder incontinence, and ataxia require emergent management and portend a poor prognosis. The emergency physician should administer a loading dose of dexamethasone of 10 mg IV. The patient's clinical situation will dictate surgery, radiation therapy, or chemotherapy. Surgical candidates are individuals with spinal instability, unknown malignancy type, deteriorating neurologic function during radiotherapy, recurrent cord compression, or compression due to bone fragments. Nonsurgical candidates are eligible for emergent radiotherapy. A radiation therapy consult should be obtained in the ED. Chemotherapy is applied to patients who are not eligible for surgery or radiotherapy. Recent studies have shown that 80% of ambulatory patients retain the ability to walk, vs. 20-60% of patients who were paretic at the time of treatment.[4,5]

An area of controversy in the literature involves anterior cord compression management via laminectomy.[16,17] Currently, laminectomy is applied to posterior lesions, while other techniques are used for anterior decompression and spinal stabilization.[18]

Another area of controversy has been the role of high-dose

Table 1. Malignancy Complications in the Elderly

NEUROLOGICAL
- Spinal cord compression
- Seizure
- Meningitis/encephalitis/abcess

CARDIOPULMONARY
- Superior vena cava syndrome
- Pulmonary embolism
- Cardiac tamponade

MUSCULOSKELETAL
- Pathologic fractures

PAIN

HEMATOLOGIC
- Thrombosis
- Bleeding

GASTROINTESTINAL
- Bowel obstruction
- Perforated viscus
- Bleeding

RENAL/METABOLIC
- Hypercalcemia
- Hyperuricemia

Geriatric Emergency Medicine Reports™ (ISSN 1527-9146) is published by American Health Consultants, 3525 Piedmont Road, N.E., Six Piedmont Center, Suite 400, Atlanta, GA 30305. Telephone: (800) 688-2421 or (404) 262-7436.

Vice President and Group Publisher: Brenda Mooney
Editorial Group Head: Valerie Loner
Managing Editor: Allison Mechem
Specialty Editor: Shelly Morrow
Marketing Manager: Schandale Kornegay

GST Registration No.: R128870672

Subscriber Information

Customer Service: 1-800-688-2421
Customer Service E-Mail Address: customerservice@ahcpub.com
Editorial E-Mail Address: allison.mechem@ahcpub.com
World-Wide Web page: http://www.ahcpub.com

Accreditation

Geriatric Emergency Medicine Reports™ continuing education materials are sponsored and supervised by American Health Consultants. American Health Consultants designates this continuing education activity for up to 2.5 hours in Category 1 credit toward the AMA Physician's Recognition Award. Each physician should claim only those hours of credit the he/she actually spent in the educational activity.

American Health Consultants (AHC) is accredited by the Accreditation Council for Continuing Medical Education (ACCME) to provide continuing medical education for physicians.

This CME activity was planned and produced in accordance with the ACCME Essentials.

This is an educational publication designed to present scientific information and opinion to health professionals, to stimulate thought, and further investigation. It does not provide advice regarding medical diagnosis or treatment for any individual case. It is not intended for use by the layman. Opinions expressed are not necessarily those of this publication. Mention of products or services does not constitute endorsement. Clinical, legal, tax, and other comments are offered for general guidance only; professional counsel should be sought for specific situations.

THOMSON
AMERICAN HEALTH CONSULTANTS

For Editorial Questions & Comments
Please call **Allison Mechem,** Managing Editor, at (404) 262-5589 between 8 a.m. and 4 p.m. ET, Monday-Friday.

Statement of Financial Disclosure

In order to reveal any potential bias in this publication, and in accordance with Accreditation Council for Continuing Medical Education guidelines, we disclose that Drs. Alcindor (author) and Valley (peer reviewer and board member) report no financial relationships with companies having ties to this field of study. Drs. Meldon (editor-in-chief), Gerson, Kalbfleische, LaMantia, Ma, Miller, Osgood, Strange, and Woolard (board members) report no financial relationships with companies having ties to this field of study. Dr. Lewis is a researcher and serves on speaker's bureaus for Roche, Merck, and Bayer. Dr. Bosker is on the speaker's bureau for Pfizer, Rhone-Poulenc Rorer, and Parke-Davis. Dr. Bosker also acknowledges that he receives royalties, commissions, and other compensation relating to the sale of textbooks, reprints of articles, and other written materials to the following pharmaceutical companies: Pfizer, Genentech, Aventis, Pharmacia, and Bayer.

corticosteroids in the management of cord compression. Corticosteroids clearly are needed in the management of cord and nerve root compression, and animal model studies have demonstrated improvement in function.[18-20] However, several clinical trials have not shown an advantage of high-dose corticosteroids over the standard dose.[18]

Seizures. The most common brain tumors are metastatic lesions to the brain (20-40%).[2] Seizures in cancer patients mainly are associated with cranial metastases and less commonly are seen with primary brain tumors. Seizure, especially if focal, in any patient older than age 40 signals a mass lesion and possible malignancy until proven otherwise.[21]

Patients may present with a headache, in status epilepticus, or postictally. A history of cancer or epilepsy may not be readily available to the emergency physician. Issues of noncompliance, recent chemotherapy, and radiation should be raised during the initial evaluation. As in all emergencies, airway, breathing, and circulation should be addressed. Give oxygen, obtain a dextrose stick, and administer thiamine 100 mg IV. Look for papilledema and signs of head trauma. Draw a CBC, electrolytes, coagulation profile, anticonvulsant levels, and blood cultures as appropriate. The physician should obtain a contrast head CT to rule out any masses or abcesses after a non-contrast head CT has been performed.

ED management in these patients is similar to that of other patients with seizures. Rapid stabilization with benzodiazepines such as diazepam (0.2 mg/kg IV at 2 mg/min) or lorazepam (0.1 mg/kg IV at 1-2 mg/min) may be necessary. A longer-acting anticonvulsant such as phenytoin (15 mg/kg in the elderly) or fosphenytoin (15-20 PE/kg at 100-150 mg PE/min) also can be administered. The physician should provide a bolus for non-therapeutic anticonvulsant levels and adjust maintenance dosage as needed. Indications for admission include new diagnosis of cancer, status epilepticus or recurrent seizures, hemodynamic instability, and brain metastases with significant edema and CNS infections. Patients also should be referred for radiotherapy to decrease the size of metastases.

Cardiac Complications

With aging, the cardiovascular system undergoes atherosclerotic changes that result in arrhythmias, myocardial ischemia/infarction, and loss of distensibility of the heart muscle. The older heart cannot tolerate major fluid loads, especially postsurgically.[22] Cancer complications, such as superior vena cava syndrome (SVCS) and pericardial tamponade, when superimposed on these expected physiologic changes, can result in significant morbidity.

Superior Vena Cava Syndrome. SVCS occurs when there is obstruction of the thin-walled, low-pressure superior vena cava. Pressures in the superior vena cava can reach 200-500 cm H_2O in SVCS and can translate into a dramatic clinical presentation.[18] The increased pressure is a gradual process but may occur rapidly depending on the presence or absence of tributaries to the superior vena cava. Kinking of a central venous catheter may cause rapid onset of SVCS.[23] Currently, malignancy is the etiology in 85-95% of cases.[24] Bronchogenic carcinoma of the lung alone accounts for 75%, lymphoma 15%, and metastatic lesions make up 7%.[18,23] It is surprising that Hodgkin's lymphoma rarely causes SVCS although it commonly affects the mediastinum. Breast cancer is the most frequent metastatic disease that causes SVCS (11%).[18] Nonmalignant causes are the culprit in SVCS in 10-25% of cases today.[18] These include central venous catheters, including Swan-Ganz catheters, and total parenteral nutrition lines, pacemakers, LeVeen shunts, and substernal goiter.

The diagnosis of SVCS often is straightforward due to typical symptoms such as dyspnea (63%), facial and head swelling (50%), cough (24%), arm swelling (18%), chest pain (15%), and dysphagia (9%).[18] Physicians should search for distention of the upper body: neck (66%), facial edema (46%), plethora (19%), conjunctival suffusion, and cyanosis (9%). Stoke's sign (tightness of the shirt collar) also may be observed. Headache, altered mental status, vertigo, stupor, and syncope may occur due to cerebral edema.[23] In the differential diagnosis, physicians should consider congestive heart failure (CHF), pericardial tamponade, and nephrotic syndrome. The physiology of aging differentiates little in the pathophysiology of SVCS in adults and the elderly with cancer.

Chest radiography is a useful diagnostic tool in SVCS; it reveals a mass in most cases. In one study, a normal chest x-ray was found in only 16% of patients.[24] Chest radiographic findings include superior mediastinal widening (64% of cases), pleural effusion (26%), right hilar mass (12%), bilateral diffuse infiltrates (7%), cardiomegaly (6%), calcified paratracheal nodes (5%), and anterior mediastinal mass (3%).[18] CT of the chest details the surrounding structures such as superior vena cava tributaries, the bronchi, and the spinal cord. At this time, MRI can delineate the vascular anatomy and give clues to etiology.[23] Contrast venography is controversial in the diagnostic approach to SVCS because it is thought to increase bleeding at the puncture site in the setting of venous hypertension.[18] Radionuclide Technetium-99m venography can show patency and flow pattern, although the images are not as well defined as contrast venography. The key to the diagnosis of the underlying cancer is mediastinoscopy or bronchoscopic cytology, although SVCS is diagnosed 50% of the time by history, physical exam, or chest radiography before the underlying malignancy is discovered.[18] In the presence of a pleural effusion, thoracentesis is diagnostic 71% of the time. Other diagnostic modalities include supraclavicular lymph node biopsy and bone marrow biopsy.[24]

Treatment of SVCS includes symptomatic relief and treatment of the causative malignancy. Institute temporizing measures such as oxygen and head-of-bed elevation in the ED. Diuretic therapy and reduced salt intake can be palliative, but may risk further thrombosis associated with dehydration. Such issues are not relevant to the ED management of SVCS. Some clinicians advocate steroids for respiratory compromise, but there is no proven benefit.[25] Combination chemotherapy and/or radiation therapy is considered the treatment of choice of small cell carcinoma of the lung.[26] Therapy involves a multidisciplinary approach including the patient, family, primary care physician, surgeon, radiation oncologist, and oncologist. However, recurrence of SVCS has been reported after initial treatment.[26] For unknown reasons, some small studies of small cell carcinoma have found SVCS to be a good prognostic sign.[27] Radiation therapy is the treatment of choice for SVCS

induced by non-small cell carcinoma of the lung, and is the initial treatment if the diagnosis is not yet established and the patient's condition is worsening. Chemotherapy is the first line of treatment for small-cell carcinoma of the lungs.[23] The average survival rate for malignancy-induced SVCS is five months vs. nine years for non-malignant causes.[28] Catheter-induced SVCS can be managed with streptokinase, urokinase, or recombinant tissue-type plasminogen activator.[29,30] Heparin and coumadin may help reduce the extent of thrombosis. Removal of the catheter, along with anticoagulation, is another treatment choice. Percutaneous transluminal angioplasty has achieved a 93% patency rate for catheter-induced SVCS.[31]

The prognosis for SVCS depends on the tumor type and is better for lymphoma than bronchogenic carcinoma. The overall prognosis for patients with SVCS caused by non-small cell carcinoma of the lung is poor, with a survival of 17% at one year and 2% at two years.[18] Other complications include recurrent obstruction, thrombosis, esophageal invasion, cardiac tamponade, vocal cord paralysis, and spinal cord compression. Rarely, bleeding, pulmonary edema, and stent migration have occurred.[23]

Cardiac Tamponade. Cardiac tamponade is a potentially lethal complication of malignancy due to malignant pericardial effusion. It can be associated with radiation-induced pericarditis and fibrosis. Impaired cardiac filling leads to decreased cardiac output. The fluid accumulation within the pericardial sac progresses to diastolic dysfunction and ultimately to cardiac decompensation. The elderly patient is particularly prone to cardiac tamponade, especially in the presence of underlying cardiac disease. Bronchogenic carcinoma, breast cancer, lymphoma, leukemia, melanoma, and sarcomas are the most common causes.[21] The typical history includes dyspnea and substernal chest pain, which can be confused for a variety of entities including CHF, pneumonia, acute coronary syndrome, or pulmonary embolism. Additional complaints of hiccups, hoarseness, nausea, vomiting, and epigastric pain often can be elicited. The patient may appear confused, pale, or diaphoretic, and the initial impression may be one of myocardial infarction. The physical exam may reveal tachypnea, Beck's triad (muffled heart sounds, distended neck veins, and hypotension), facial swelling, ascites, and hepatic enlargement. Muffled heart sounds may not always be easy to auscultate in the noisy ED setting. Pulsus paradoxicus is an important finding that typifies cardiac tamponade, but is not pathognomonic. Physicians should keep in mind other causes of pulsus paradoxicus, such as chronic obstructive pulmonary disease, asthma, pulmonary embolism, restrictive cardiomyopathy, infarction of the right ventricle, and cardiogenic shock. Electrical alternans on the electrocardiogram (ECG), is specific for tamponade, but is not seen often. Additional electrocardiographic findings may include sinus tachycardia, non-specific ST-T wave changes, and ST elevation. Chest x-ray in malignant cardiac tamponade shows cardiomegaly, mediastinal enlargement, and a prominent pulmonary hilum.

The definitive diagnosis of cardiac tamponade is via emergent echocardiography. In addition to pericardial fluid, bedside echocardiography may show diffuse hypokinesis and diastolic collapse of the right atrium, right ventricle, and ventricular outflow tract. Central venous pressure measurements show equalized pressures throughout the cardiac chamber. Emergent management includes rapid isotonic fluid infusion, close monitoring, oxygenation, and pericardiocentesis. For non-malignant cardiac tamponade, removal of 50-100 mL of fluid can yield dramatic hemodynamic improvement until more definitive measures such as a pericardial window or pericardiectomy can be performed in the operating room. In malignant cardiac tamponade, much more fluid needs to be removed for clinically significant improvement. The outlook for any patient with malignancy-induced cardiac tamponade is poor.

Gastrointestinal Complications

Gastrointestinal (GI) complications of malignancy in the elderly such as abdominal pain, obstruction, bleeding, perforation, and infection may manifest themselves similarly to non-malignant disorders. Several factors may confound the diagnosis in the elderly, including: difficulty in obtaining a history, memory deficits, alteration in mental status, abnormal hearing and speech, and altered perception of pain. Abdominal pain is the most common complaint and is considered the best signal to acute gastrointestinal pathology in the elderly cancer patient. Evaluation demands the same broad differential as other common nonmalignant conditions. Constipation is a frequent cause of abdominal pain and may be due to chemotherapeutic agents such as vincristine, narcotics, anticholinergics, iron, low-fiber diet, immobilization, dehydration, and electrolyte abnormalities.[32] Tumor extension into the nerve supply of the gut may lead to intestinal pseudoobstruction, commonly termed Ogilvie's syndrome. Right upper quadrant pain may be the initial presentation of metastatic lesions to the liver. The pain of liver metastases may radiate to the shoulder and cause diaphragmatic irritation. It also may be the occult manifestation of venoocclusive disease in the postsurgical bone marrow transplant patient or occur after intense chemotherapy (4% to 22%).[33] Such patients are jaundiced, have hepatomegaly, ascites, neutropenia, thrombocytopenia, and may display signs of encephalopathy. Hepatic vein obstruction (the Budd-Chiari syndrome), secondary to hepatoma, adrenal carcinoma, renal cell carcinoma, or thrombosis, may have a similar presentation. Lymphoma causing splenomegaly may be the cause of left upper quadrant pain and left shoulder pain (Kehr's sign). Rectus sheath hematoma in a thrombocytopenic patient or a patient on warfarin may elicit abdominal wall pain. Back pain may be a manifestation of enlarged retroperitoneal adenopathy, retroperitoneal hematoma, or pancreatitis due to cytotoxic medications (i.e., L-asparaginase, 6-mercaptopurine, and corticosteroids). Corticosteroids are known culprits to delays in the diagnosis of abdominal complications. Physical findings such as fever and leukocytosis are likely to be masked, and these patients are at a higher risk of localized perforations. In summary, abdominal pain in the elder cancer patient should elicit a broad differential diagnosis, as the etiologies are protean.

Intestinal obstruction occurs in 59-100% of patients with recurrent malignant disease, and is the indication for laparotomy in 33% of cancer patients.[18] Colorectal, ovarian, and gastric malignancies account for two-thirds of cancer-related intestinal obstruction. The sites of obstruction include small bowel

(59%), colorectal (29%), both sites simultaneously (5%), and duodenum and stomach together (7%).[34] The presentation of bowel obstruction does not differentiate malignant from non-malignant causes of obstruction, crampy abdominal pain that is initially intermittent, then becomes more constant as the obstruction progresses, and is associated with nausea, vomiting (usually bilious), abdominal distention, and anorexia. The patient may look ill, dehydrated, and even cachectic. There may not be an abdominal scar as a remnant of prior surgery. The initial examination may reveal distention, localized tenderness (becoming diffuse with time), hyperactive bowel sounds. A mass may not be palpable. Important physical examination keys include searching for a mass, fecal impaction, or tenderness. Obtain a CBC, electrolytes, coagulation profile, urinalysis, a chest radiograph, and abdominal series. The patient should be hydrated with isotonic saline and have a nasogastric tube inserted. Colonoscopic decompression is needed when cecal distention is 12-14 cm. Conservative management with a nasogastric tube, bowel rest, hydration, serial abdominal exams (preferably by the same physician), and abdominal radiographs is generally indicated, since the majority of partial small bowel obstructions will relieve with this approach.[18] However, in a few studies, conservative management in cancer patients yielded a 12-29% resolution rate within 3-9 days and a recurrence rate of 32-45% necessitating operative intervention.[35,36] Surgery is indicated to relieve the symptoms and prevent bowel strangulation or gangrene if conservative management fails after three or more days, or if leukocytosis, fever, increased pain, distention, or peritonitis occur. Surgery is effective in 55-96% of cases. The recurrence rate of obstruction after surgical intervention is 9-33%. Morbidity and mortality rates are 31% and 16%, respectively. The mortality rate for emergency laparotomy is highest (38-54%) in patients ages 70 and older, whereas only 3-7% of the older surgical candidates die after an elective procedure.[35,36] The mortality difference is most reflective of pre-morbid conditions. The extent of the operation and related treatment regimens should not depend on age alone, but rather should take into account the patient's physiology, health status, and wishes.[37]

GI perforation is the indication for laparotomy in 33% of cancer patients. Lymphoma causes about 50% of GI perforations. The patient typically presents with abdominal pain, distention, fever, and peritoneal signs. Free air under the diaphragm is visualized about 50-75% of the time on an upright chest radiograph. The flat and upright abdominal films, along with a left lateral decubitus view, increase the sensitivity for free air detection. Sensitivity also can be increased by instilling 200 cc of air via a nasogastric tube prior to obtaining the radiograph. A CT scan of the abdomen should be obtained to confirm the diagnosis in equivocal cases when clinical suspicion is high. The absence of free air on plain films does not preclude perforation. Intravenous hydration, treatment of shock, repletion of electrolytes, broad-spectrum antibiotics, and immediate surgical consultation are the necessary ED steps.

Postoperative hemorrhage is the most common cause (67%) of bleeding in the cancer patient. According to endoscopic studies, malignancy is the culprit in 12-27% of GI bleeds in cancer patients, while gastritis, ulcer disease, and esophagitis (44-76%); and esophagitis and varices (11-17%) account for the rest.[38] Other uncommon causes are Mallory-Weiss tears from vomiting due to chemotherapy, hemobilia due to hepatobiliary tumors or instrumentation, aortoduodenal fistula from a perforated metastastic tumor, and iatrogenic liver and spleen injuries after thoracentesis and percutaneous needle biopsy.[39] Emergency management should include a rapid assessment of the ABCs (airway, breathing, and circulation) followed by intravenous fluid, packed red blood cells, and H_2 antagonists as indicated. Coagulopathies should be corrected and any causative drugs such as aspirin, warfarin, heparin, nonsteroidal antiinflammatory agents, and alcohol discontinued. A surgical or gastroenterology consult for endoscopy should be obtained. A radionuclide-labeled erythrocyte scan or angiogram may be helpful if the endoscopy is inconclusive or if bleeding continues. Angiography is both diagnostic and therapeutic in that the bleeding site may be identified and gelfoam embolization may be performed. If the endoscopic work-up is inconclusive, an abdominopelvic CT to search for a retroperitoneal hematoma should be considered. Patients who remain unstable may require operative management.

Other GI complications of malignancy include abdominal abcesses and radiation enteritis. Abcesses are a common postoperative event and respond well to percutaneous drainage or surgery if there is no improvement with intravenous antibiotics within 24 hours. Anorectal infections are common and are managed in the standard fashion. Perirectal lesions that fail to heal need a biopsy to rule out carcinoma. Radiation enteropathy is an entity characterized by nausea, vomiting, intermittent crampy abdominal pain, weight loss, and watery or bloody diarrhea in a patient with a history of abdominal radiation.[18] Treatment is supportive.

Renal/Metabolic Complications

With aging, there is a decrease in renal function and mass, glomerular filtration rate, and creatinine clearance, thereby affecting drug excretion and making the kidneys more susceptible to ischemia from anesthesia and major surgery.[22] There also is decreased volume of distribution of water-soluble drugs.[2]

Hypercalcemia. Hypercalcemia represents the most frequent metabolic complication found in cancer patients. It is encountered in 20% of cancer patients.[40] It most commonly is associated with multiple myeloma and breast cancer (40%), and is frequently seen in non-small cell carcinoma of the lung. It is an uncommon presentation in colon, prostate, and small cell carcinoma. The pathophysiology involves osteolysis from bony metastases and tumor production of parathyroid-like hormones, with the net effect being an elevated calcium level. Prostaglandins and other peptides also have been implicated. Any patient with an acute altered mental status may well be suffering from carcinoma-induced hypercalcemia. Other symptoms of hypercalcemia are vague and include anorexia, nausea, vomiting, constipation, fatigue, weakness, abdominal pain, polyuria, polydypsia, and memory loss. The clinical presentation is not directly related to the serum level. Many of these symptoms are so common in the elderly emergency patient that even the astute emergency physician may fail to

associate them with hypercalcemia.

In addition to a serum calcium level, laboratory analysis should include electrolytes and renal function testing to look for renal insufficiency. An ECG should be obtained with emphasis on a shortened QT syndrome, dysrhythmias, and digitalis toxicity. Management includes ECG monitoring, saline hydration to correct dehydration and increase renal blood flow, and treatment of associated hypokalemia or hypomagnesemia. Furosemide 1-3 mg/kg may further enhance renal excretion of calcium after adequate urine output has been achieved. Avoid fluid overload in the frail elderly with poor cardiopulmonary reserve. To avoid hypovolemia and maintain fluid balance, the use of furosemide is limited to the patient who has been appropriately rehydrated (e.g., urine output of 100 mL/hr).

Acute therapy directed toward calcium excretion is now the standard of care. Bisphosphonates inhibit bone resorption by osteoclasts and have shown clinical efficacy. Examples include oral etidronate, clodronate, and intravenous pamidronate, as well as galium nitrate, an inhibitor of bone resorption (for resistant hypercalcemia).[41,42] These drugs are not used in the ED. Calcitonin rapidly (onset of action 2-4 hours) increases calcium excretion by inhibiting bone resorption, however its peak action is at 48 hours and falls off afterwards. It is being used in combination with pamidronate and galium nitrate. Its role in the ED setting is not established. Plicamycin (mithramycin) is cytotoxic to osteoclasts; however, its 48-hour onset of action and associated thrombocytopenia and hepatic and renal toxicity preclude its use in the initial management of hypercalcemia. Aspirin and indomethacin act as inhibitors of prostaglandins to lower the serum calcium level. Corticosteroids are reserved for steroid-responsive malignancies such as breast carcinoma, myeloma, and lymphoma after oncologic consultation has been obtained. The ultimate treatment of malignancy-related hypercalcemia is the treatment of the underlying malignancy.

Most patients with serum calcium levels greater than 12 mg/dL do not respond to intravenous hydration and diuretics alone and often require treatment with a bisphosphonate or gallium nitrate. Calcitonin 8 U/kg may be injected IM every six hours for calcium level of 15 mg/dL associated with coma or cardiac arrythmias. Marked hypercalcemia associated with renal insufficiency requires hemodialysis. The patient should be admitted if the serum calcium is greater than 12 mg/dL, and is accompanied by nausea, vomiting, dehydration, altered mental status, renal insufficiency, cardiac arrhythmia, obstipation, and ileus, or if the patient lives alone or has difficulty accessing medical care.[18]

Hyperuricemia. Hyperuricemia is a malignancy complication found mostly after chemotherapy or radiation therapy of lymphomas and leukemias and myeloproliferative disorders. It also may be encountered with hyperparathyroidism, sarcoidosis, psoriasis, renal failure, and gout. Hyperuricemia also can occur as a chronic sequela of thiazides, ethacrynic acid, and furosemide. The underlying mechanism is either excess production or decreased excretion of uric acid, especially in the presence of underlying renal impairment, as may be present in the elderly. It may be an incidental finding, or it may be seen in an acutely oliguric patient after chemotherapy. Hyperuricemia may be asymptomatic. Affected patients are prone to acute renal failure secondary to intrarenal obstruction. Ultrasound or CT scans are useful to assess for obstructive uropathy. Patients also are at risk for acute hyperuricemic nephropathy, uric acid nephrolithiasis, and gouty nephropathy.

The key to management is to keep patients well hydrated (aim for daily urine output of 2 liters), ideally before the start of chemotherapy. Oral allopurinol 300-900 mg ideally should be started 48-72 hours before cancer therapy. Withdrawal of offending drugs should be instituted. Alkalinization of the urine to a pH above 7 is an additional step. Give colchicine 0.6 mg PO bid if a history of gouty arthritis is present. Further alkalinization can be obtained with acetozalamide 1 gram IV, a carbonic anhydrase inhibitor. Oliguria can be ameliorated with 12.5 gm of 20% mannitol. If the patient is still oliguric, dialysis is a last resort.[18]

The ED management of hyperuricemia is the same regardless of the etiology. Aggressive hydration is paramount, with careful attention to fluid overload in the frail elderly patient. The best treatment remains treatment of the underlying malignancy and prevention of this complication.

Tumor Lysis Syndrome. Tumor lysis syndrome is a constellation of malignancy-induced metabolic abnormalities resulting from the accumulation of intracellular products in the bloodstream. Hyperuricemia, hyperkalemia, hyperphosphotemia, and hypocalcemia are the end products. Such patients are at risk for dangerous arrhythmias (hyperkalemia), acute renal failure (hyperphosphotemia), muscle cramps, and tetany (hypocalcemia). The tumors most commonly responsible are bulky, highly proliferative malignancies that are very responsive to cytotoxic regimens. They include lymhomas, leukemias, and solid tumors.[43,44] This syndrome also has been linked to potent myelosuppressive agents and other agents such as methotrexate, tamoxifen, interferon alpha, and cladribine. Early diagnosis and aggressive management are key. Hydrate patients at risk prior to initiating chemotherapy. Check and correct electrolytes, uric acid, phosphorus, and calcium as often as the clinical situation dictates for 3-4 days after receiving chemotherapy. ED management involves hydration, ECG monitoring, treating the presenting electrolyte abnormalities, and consultation with the patient's oncologist.

Lactic Acidosis. Lactic acidosis is a rare metabolic complication of malignancy. It generally is associated with leukemia and lymphoma.[45,46] Its onset usually signifies progression of a hematologic malignancy. Patients with solid tumors usually have hepatic metastases once lactic acidosis occurs. The clinical presentation may vary from vague symptoms such as tachycardia, weakness, nausea, and stupor to hyperventilation, hypotension, and elevated lactic acid. Differential diagnosis includes sepsis, mesenteric ischemia, and other causes of lactic acidosis. The prognosis is poor for patients with lactic acid levels greater than 4 mEq/L, and the outcome depends on treatment of the underlying cancer. Sodium bicarbonate therapy does not appear to affect survival.[18]

Hypoglycemia. Hypoglycemia in cancer is usually the result of insulin-producing islet cell tumors. The symptoms are the same as those seen in hypoglycemia of other etiologies: weakness, dizziness, diaphoresis, and nausea. Patients also may present with seizures, coma, and varying neurologic

deficits. Malignancy-induced hypoglycemia may be derived from the secretion of insulin-like substances, excessive tumor utilization of glucose (by large tumors), decreased gluconeogenesis, or lack of counter-regulatory hormonal action such as growth hormone.[47] Anti-tumor therapy should be aimed at the underlying malignancy, although little efficacy has been shown in halting the development of hypoglycemia. Mild hypoglycemia may be approached by increasing the frequency of meals; corticosteroids and intravenous glucose infusions may provide symptomatic relief in more severe cases.

Vascular/Hematologic Complications

It should be emphasized that hematopoiesis, unlike most organ systems in the elderly, essentially remains the same as one ages. The volume of packed red cells, total leukocytes, neutrophils, lymphocytes, platelets, and the iron turnover rate in the healthy older person is no different from that of the young adult .[48] However, the incidence of anemia increases in the elderly patient with concurrent illness, implying that illness causes a dysregulation of hematopoiesis.[49]

Thrombosis. Malignancy and older age are known risk factors for thrombosis. Other risk factors that are common to older patients include immobility, tobacco use, diabetes, and CHF. Mucin-producing tumors of the gastrointestinal tract and carcinomas of the lung, breast, ovary, and primary brain are particularly prone to thrombosis. Because of its prevalence, lung cancer is the tumor most frequently associated with thromboembolic disease. Malignancy creates a hypercoagulable state. The classic pathogenetic factors still play a critical role today, including: abnormalities in blood flow, blood composition, and blood vessel wall, as advocated by Virchow. Thromboembolism has been mentioned as one of the earliest signs of occult malignancy.[50] The mediators of the hypercoagulable state include tumor cell procoagulants, procoagulant activities induced by host response to tumor, platelet aggregation, and endothelial cell activity. The presence of fibrin in the vicinity of a tumor is felt to be a nidus for tumor growth, metastasis, and a barrier to host humoral defenses. Thrombocytosis is encountered 30-60% of cancer patients, yet the absolute platelet count is not the underlying mechanism for thrombosis. It is thought to be induced by increased platelet reactivity via clonal abnormalities or tumor cell-platelet interactions.[51]

Certainly, surgery accompanied by immobilization is a major setting for thrombosis. The hypercoagulable state induced by malignancy also is maintained by the antineoplastic agents used to treat the various malignancies. For example, myocardial ischemia/infarction secondary to vasospasm and/or thrombosis has been observed with the vinca alkaloids, 5-fuorouracil, cisplastin, etoposide, and bleomycin in combination with vinblastine or cisplastin.[52] Cerebral vascular accidents can occur due to cisplastin and L-asparaginase. L-asparaginase has been implicated in dural sinus thrombosis, and has been linked to deep venous thrombosis (DVT). The underlying mechanism is depletion of plasma asparagines leading to inhibition of protein synthesis (primarily a reduction of antithrombin III). High-dose chemotherapy after bone marrow transplant increases the risk of thrombosis. After bone marrow transplant, low levels of proteins C and S and antithrombin III have been observed in one study.[53] However, it is uncertain whether a low protein C level can be used as a reliable marker for possible venoocclusive disease. Other agents implicated include tamoxifen (antiestrogen with weak estrogenic properties) and corticosteroids.[53] Several studies have shown a higher risk for occult malignancy in patients with idiopathic DVT compared with patients with secondary DVT (DVT due to known risk factors/etiologies).[50,54]

Thrombosis occurs in 15% of cancer patients. The diagnosis of thromboembolic disease in the elderly cancer patient is the same as for the general population. Knowledge of the risk factors for thrombosis is essential. A thorough physical exam focusing on edema, venous cords, varicose veins, and tenderness is in order. The diagnostic approach also is the same as for noncancer patients. Although ascending venography is considered the criterion-standard and is reliable for both proximal and distal thrombosis, its use is limited because of patient discomfort, lack of portability, risk of contrast reaction, and precipitation of thrombosis. For these reasons, noninvasive tests are advocated as initial studies. Impedance plethysmography, Doppler ultrasound, real-time (B-mode) ultrasonography, and duplex scanning have a higher sensitivity for proximal than for distal DVT.[55] If suspicion remains high, subsequent venography or a repeat non-invasive test (e.g., duplex scan) in 2-3 days may be indicated. MRI has shown similar accuracy to venous Doppler and venography in the assessment of DVT, but it is not widely used.[55]

Upper extremity thrombosis due to venous obstruction is seen more frequently in the cancer patient as a result of vascular injuries from intravenous catheters and the caustic effects of chemotherapy. Forty percent of Hickman catheters ultimately lead to venous thrombosis.[56] Subclavian thrombosis is seen in 16% of completely implanted catheters and is manifested clinically by swelling and pain of the upper extremity, neck, shoulder, and chest wall. A triple-lumen catheter carries a higher risk of occlusion than a double-lumen catheter. Left-sided catheters tend to cause more complications, and a higher rate of venous obstruction has been observed when a catheter is placed above the level of the third thoracic vertebra.[56] Large mediastinal tumors or bulky axillary lymphadenopathy also can cause venous obstruction. Diagnosis of upper extremity thrombosis can be made with either non-invasive studies or venography. A rare entity, the Trousseau syndrome, should be considered when a patient presents with recurrent migratory thrombosis of the superficial veins of the chest wall or the arms.[52]

Pulmonary embolism is another complication in elderly cancer patients. It is the complication of a DVT in 70% of patients. Again, a targeted history examining risk factors is indicated. Importantly, signs and symptoms may be nonspecific and a high level of suspicion is required. The work-up is the same as that used for the general population. Although pulmonary angiogram is the standard criterion, it usually is obtained if noninvasive tests and a ventilation/perfusion (V/Q) scan are nondiagnostic and clinical suspicion remains high. Spiral CT is another tool that can demonstrate a pulmonary embolism (PE) and is gaining popularity.[51]

Arterial thrombosis is much less common than its venous counterpart. It is primarily due to nonbacterial thrombotic endocarditis (NBTE) and is related to mucin-producing adenocarcinomas. The presenting signs are a direct result of arterial

embolization, which can lead to end-organ failure. The brain, heart, spleen, and kidneys are the sites most commonly affected.[51] Arterial thrombosis in the central nervous system can cause transient ischemic attacks, headaches, visual changes, and seizures, while peripheral arterial thrombosis results in digital ischemia or infarction.

The therapy for venoocclusive disease in the elderly cancer patient differs little from that used for the general population. Heparin, followed by warfarin, can be used. Low-molecular weight heparin currently is gaining wider use. Surgical patients with cancer need DVT prophylaxis. Those patients who have in-dwelling catheters may receive a 1 mg daily warfarin dose. An inferior vana caval filter (e.g., Greenfield filter) may be indicated in those with recurrent thrombosis while on warfarin, or if there is a contraindication to anticoagulation. One author has noted that resistance to anticoagulation is a sign of occult malignancy.[50]

Acral erythrodysplasia (painful erythema of the palms and soles) may result from standard or high-dose arabinoside, hydroxyurea, and prolonged use of 5-fluorouracil or doxorubicin.

Cancer patients also may complain of intense burning of the hands and feet, warmth, and erythema of the skin, especially when exposed to heat. This spectrum of signs and symptoms, called erythromelalgia, is reported to be very responsive to aspirin. It may due to a paraneoplastic syndrome in solid tumors, essential thrombocythenia, or polycythemia vera.[57]

Hyperviscosity Syndrome. Impairment of blood flow, massive leukocytosis, and delay in the clearance of clotting factors in cancer patients may predispose them to the hyperviscosity syndrome. The pathology extends to all organs. It is characterized by a variety of symptoms ranging from altered mental status, vertigo, headache, visual disturbances, seizures, bleeding, and cardiopulmonary dysfunction, to renal impairment and signs of thrombosis. This syndrome is most commonly secondary to Waldenstrom macroglobulinemia, followed by multiple myeloma.[40] Various types of leukemias, cryoglobulinemia, polycythemia vera, and sickle cell disease account as other causes. The diagnostic approach should include electrolytes and coagulation profiles. A bedside test of relative serum viscosity has been described.[58] Management includes hydration, diuresis, and emergent plasmapheresis and phlebotomy for severe cases, followed by initiation of chemotherapy.

Thrombocytopenia. Malignancy-induced thrombocytopenia is the most common reason for platelet transfusion. Bone marrow replacement by malignant cells, chemotherapy, radiation therapy, and infections (i.e., bacterial, viral, fungal, protozoal infections) are common etiologies. Infection-induced thrombocytopenia, however, usually does not cause major bleeding.[55] Leukemia patients are at particular risk for thrombocytopenia and associated bleeding. There is increasing support for prophylactic platelet transfusion in leukemic patients with a platelet counts of 5000-10,000.[59]

Cancer patients also are at increased risk for heparin-induced thrombocytopenia, which typically occurs during the first several days of heparin administration. This occurs in patients on both full-dose heparin and from heparin flushes of venous access. The etiology is heparin-dependent IgG antibodies. Drug-induced thrombocytopenia should be followed by discontinuation of the responsible agent. Platelet counts usually begin to improve within 2-3 days. For patients requiring prolonged anticoagulation, warfarin should be instituted. Severe cases of thrombocytopenia associated with bleeding can further be managed by the use of intravenous IgG or Rh immune globulin (anti-D).[60]

Musculoskeletal Complications

Osteopenia in the elderly predisposes them to fractures of the long bones. The cancer-afflicted elderly patient is at further risk for fracture and spinal instability due to local metastases and tumor-induced osteopenia. Pathologic fractures can occur from seemingly trivial trauma. Long bones, especially the subtrochanteric region, are common sites of occurrence. The management of pathologic fractures includes rapid immobilization, analgesia with opioid narcotics, intravenous hydration, and prompt orthopedic and oncologic consultations.

Infectious Complications

The decline in the immune competence of the elderly predisposes them to a higher rate of infection.[1] This section will concentrate on fever in the neutropenic cancer patient.

Fever is a common presentation in the elderly cancer patient. Its presence may be a marker to an underlying infection. Infection is a leading cause of death in the cancer patient (50-75%), and has a mortality rate of 20-50% if not promptly treated.[63] Other etiologies include the underlying malignancy, cytokines, transfusion, thromboembolism, atelectasis, or drug therapy. One helpful distinction is that tumor-induced fever does not usually produce chills or rigors and the patient does not appear toxic, as compared to infectious etiologies of fever.[63]

A key parameter in the assessment of cancer patients with fever is the absolute neutrophil count (ANC), with neutropenia defined as an ANC less than 500/mm^3. The duration of neutropenia along with certain risk factors dictates the likelihood of an infectious complication. High-risk groups include those with neutropenia of more than seven days duration, underlying malignancy, chemotherapy, altered cellular and humoral immunity, recent bone marrow transplantation, and the elderly and those with an indwelling catheter.[64] Prolonged neutropenia is associated with potentially fatal infections from resistant bacteria, *Candida* species, and *Asperigillus* species. Bacteria, such as the coagulase-negative staphalococci, have become the dominant etiologies in the United States, secondary to increased indwelling catheter use.[64] *Staphylococcus aureus* and Streptococci and Enterococci account for one-half of gram-positive isolates.[64] Nonimmunized patients who are splenectomized because of Hodgkin's disease are prone to *Streptococcus pneumoniae, Neisseria meningitidis*, and *Haemophilus influenzae*.

The greatest risk for the neutropenic febrile patient is gram-negative organisms, such as *Pseudomonas aeruginosa,* which can cause pneumonia and bacteremia. Neutropenic transplant patients on chemotherapy also are vulnerable to viruses, such as respiratory syncytial virus, adenovirus, parainfluenza virus, cytomegalovirus, and herpes simplex virus, as well as *Pneumocystis carinii*, mycobacteria, and fungi.

The evaluation of a febrile neutropenic patient takes on the same urgency as that of meningitis. Rapid history targeting risk factors and a thorough physical exam focusing on catheter site

infection, and searching all sites for infection are necessary. In the majority of neutropenic patients, a site of infection is not found. The clinical evaluation should be followed by hematologic and coagulation, blood, urine, sputum, nasal, and pharyngeal, studies; and gentle rectal swabs; chest radiography; intravenous hydration; and prompt antibiotic therapy. Traditionally, broad spectrum antibiotic combination was advocated, but recent studies have evaluated monotherapy with ceftazidime alone.[65,66] A recent study showed that carefully selected low-risk hospitalized neutropenic patients on chemotherapy fare well on oral ciprofloxacin and amoxicillin-clavulinate.[67]

Pain as a Complication of Cancer

Pain is a frequent companion to cancer. Nonmetastatic disease is associated with pain 15% of the time, whereas pain is seen in 74% of patients with metastases.[1] Direct tumor invasion, such as in breast and prostate cancers, accounts for pain in 50% of patients with metastases, while nerve compression is the mechanism in the remainder. Cancer-related therapy causes pain in 25% of patients.

There are several issues related to pain management in the elderly. Normal aging is not significantly associated with impairment of pain sensation. The clinician's lack of experience in pain assessment, the patient's reluctance to report pain, and the cost of outpatient pain medications have contributed to the poor management of pain in the elderly cancer patient.[61] Fear of addiction by patients and their families also is an important concern, but in one study of 12,000 patients treated with narcotics, only 0.1% became addicted.[61] Management includes proper pain assessment (location, characteristics, radiation, co-related factors, and implications of the pain on the patient's activities of daily living). Pain may result in sleep disorders, depression, and fear of cancer recurrence. A low-dose antidepressant and proper pain medications are in order. The physician should reassess the patient's disease process and initiate the proper studies based on the physical exam. For example, increased low back pain may mean a collapsed vertebra with impending spinal cord compression, in which case an MRI is indicated. The absence of neurologic deficits does not preclude impending spinal cord compression.

One nonpharmacologic way of approaching pain is to treat the cancer itself. Examples include cancers that are responsive to chemotherapy or radiation (e.g., bone metastases). An exception is pancreatic cancer, which is not responsive to chemotherapy, but is helped by a celiac plexus block, for example. However, the toxicities of the treatments must be taken into account in the management of cancer pain.

The World Health Organization recently made some recommendations on managing pain: Pain relief must be a priority for the elderly cancer patient, and it must continue until relief is achieved.[62] Additional modalities of pain control involve nerve blocks with chemicals or neuroablative methods, such as chordotomy (ablation of pain pathways in the spinal cord).

Toxicity of Chemotherapy

Unfortunately, the same chemotherapeutic agents used in the fight against cancer also are cytotoxic to normally dividing cells. *(See Table 2.)* The bone marrow is the most susceptible because its precursor cells have the most rapid turn over.[32,68]

Table 2. Effects of Antineoplastic Agents on Hypercoagulable State of Cancer

AGENT	ORGAN/SYSTEM	EFFECTS
Cisplastin	Heart	MI/Ischemia
	Kidney	Insufficiency
	Hematologic	Hemolytic anemia
	CNS	Stroke
L-Asparaginase	Hematologic	DVT
	CNS	Dural sinus thrombosis
5-Fluorouracil	Heart	MI/Ischemia
	Skin	Acral erythrodysplasia
Vinca alkaloids	Heart	MI/Ischemia Cardiomyopathy

Bone marrow suppression results in anemia, thrombocytopenia, and neutropenia. The agents least likely to suppress it include asparaginase, bleomycin, and vincristine. Chemotherapy may induce malignant cells to release procoagulant substances, along with patient risk factors, further increase the risk of thrombosis. Chemotherapy also causes thrombocytopenia, which may lead to bleeding complications that may result in thrombosis or disseminated intravascular coagulation. Hemolysis may be caused by melphalan, fluorouracil, and cisplatin.

Gastrointestinal toxicity such as nausea and vomiting, diarrhea, and constipation are common. Extreme cases lead to dehydration, electrolyte imbalance, weakness, and weight loss. Acute nausea and vomiting usually take place within 24 hours after chemotherapy. The mechanism is unknown. The most emetogenic agents are cisplastin, cyclophosphamide, cytarabine, dacarbazine, actinomycin D, mechlorethamine, and streptozotocin. Serotonin, dopamine, and histamine blockers are helpful. The most effective therapy involves the 5-HT3 (serotonin) antagonists. Diarrhea and constipation are most commonly associated with fluorouracil and idarubicin.[32] The causative agent must be stopped.

The mouth and throat also are susceptible to necrotic ulcers, mucositis, thrush, and herpes simplex in neutropenic patients on high-dose chemotherapy. Intensive oral hygiene is important to prevent hematogenous bacterial seeding. Oropharyngeal candidiasis may spread to the esophagus and vocal cords, causing dysphagia and dysphonia. Effective therapy for Candida and herpes simplex infections include flucanozole and acyclovir, respectively.

The antitumor antibiotics, anthracyclines (daunorubicin, doxorubicin, epirubicin, and idarubicin), and anthraquinones (mithramycin, mitomycin C, and Actinomycin D) are responsible for a dose-related cardiomyopathy and transient benign arrhythmias. The risk is increased in the elderly with underlying heart disease or prior mediastinal radiotherapy.[32] Cyclophosphamide in high doses (>120 mg/kg) may result in fatal hemorrhagic myocardial necrosis within the first few days of treatment.[32] A baseline echocardiogram often is obtained in this case to determine the patient's ejection fraction prior to initiation of these therapies. These regimens are stopped if the ejection fraction is less than 50% or if it falls by 10% between treatments. The newer epirubicin and idarubicin are less toxic then the other anthracyclines. As with other

dilated cardiomyopathies, treatment with angiotensin-converting enzyme (ACE) inhibitors may be indicated.

Asymptomatic elevation of liver enzymes is the most common liver abnormality secondary to chemotherapy. More serious entities involve hepatitis, cholestatic jaundice, and venoocclusive disease (which may occur with cyclophosphamide after bone marrow transplantation).[32,33]

Neurological manifestations of chemotherapy include peripheral neuropathy (paresthesias, weakness, loss of deep tendon reflexes), which can occur following treatment with vincristine. The elderly are prone to vinca alkaloid-induced autonomic neuropathy (vincristine, paclitaxel, and cisplatin) characterized by postural hypotension, severe abdominal pain, constipation, and paralytic ileus occurring within three days of treatment.[56] Symptoms improve after discontinuing the agents. Cerebellar dysfunction may be seen with high-dose cytarabine and cisplatin. Alternate-day dosing of cytarabine has reduced this side effect. Intrathecal methotrexate has been linked to a stroke-like syndrome when used in combination with craniospinal irradiation for acute lymphocytic leukemia and non-Hodgkin's lymphoma. Lower dosage appears to decrease the incidence of this complication.

Pulmonary complications of chemotherapy include pulmonary fibrosis and pulmonary edema. Pulmonary fibrosis following busulfan occurs within 3-4 years after treatment. Management includes steroids. Cyclophophamide also is associated with a pneumonitis leading to fibrosis. Melphalan (an alkylating agent) and carmustine (a nitrosurea) also have been linked to pulmonary fibrosis. Carmustine has been associated with pulmonary embolism, Wegener's granulomatosis, adult respiratory distress syndrome, and pneumothorax.[69] Five to eight percent of patients treated with methotrexate may develop pulmonary edema, pulmonary fibrosis, and hypersensitivity reactions. Headache, dyspnea, cough, and fever may be the presenting complaints.[69] Continuous administration of cytarabine may result in a capillary leak syndrome and noncardiogenic pulmonary edema in 20% of patients within 2-20 days.[32] Older patients are especially susceptible to hypersensitivity reactions and direct toxicity of antitumor antibiotics such as bleomycin, mitomycin, and doxorubicin. Steroids may be helpful.[69]

Renal toxicity is seen with cisplatin, mitomycin, methotrexate, and ifosfamide. This risk increases with the concurrent use of other nephrotoxic drugs. High-dose cyclophosphamide can lead to the syndrome of inappropriate secretion of antidiuretic hormone.[32]

Ocular toxicity is seen with busulfan, chlorambucil, cisplatin, cyclophosphamide, doxorubicin, methotrexate, and vincristine. Cataracts, diplopia, papilledema, conjunctivitis, and blurred vision are some of the ophtholmologic symptoms associated with common antineoplastic agents.[32,68]

Drug- and dose-dependent alopecia is the most frequent dermatologic manifestation of chemotherapy. Onset occurs within 7-10 days of treatment, and regrowth may begin within 1-2 months after completing treatment. This side effect carries its own psychologic sequelae. Treatment has not been shown to be effective.[32,68]

A paradoxidal effect of antineoplastic therapy is the occurrence of secondary malignancies. This is true concerning the anthracyclines, methotrexate, cytarabine, and cisplastin, among others. This potentially fatal side effect is dose and time-dependent. The most common secondary malignancies are acute myeloid leukemia and high-grade non-Hodgkin's lymphoma. Myeolodysplastic syndrome is a risk factor. Older patients who have survived Hodgkin's lymphoma are at greater risk.[70,71]

Conclusion

As the elderly population of the United States grows, the incidence of cancer is expected to rise almost proportionately.[1] Malignancy complications in the elderly affect every organ system, and the symptoms often masquerade as common disorders. This is a further insult to the progressive systemic decline associated with normal aging. Altered mental status, seizures, chest pain, dyspnea, fever, hypercalcemia, abdominal pain, bleeding, fractures, thromboembolic disease, and various infections are all daily encounters in the ED. However, these common presentations become more challenging when they are accompanied by underlying malignancy in the elderly. As malignancy complications in the elderly become more commonplace in the ED, every emergency physician must consider occult malignancy in his or her differential diagnosis when approaching the elderly patient.

References

1. Kennedy BJ. Aging and cancer. In: Balducci L, Lyman GH, Ershler WB, eds. *Comprehensive Geriatric Oncology.* Australia: Harwood Academic Publishers; 1998:2.
2. Balducci L, Extermann M. Cancer And Age. In: Haskell CM, ed. *Cancer Treatment.* 5th ed. Philadelphia: W.B. Saunders Company; 2001:435-451.
3. McNamara RM. Demographics, aging, and emergency medical care. In: Sanders AB, ed. *Emergency Care of the Elder Person.* St. Louis, MO: Beverly Cracom Publications; 1996:3-9.
4. Balducci L, Extermann M. Cancer and aging—An evolving panorama. *Hematol Oncol Clin North Am* 2000;14:11.
5. Fox SA, Roetzheim RG, Kington RS. Barriers to cancer prevention in the older person. In: Balducci L, Lyman GH, Ershler WB. *Comprehensive Geriatric Oncology.* London: Harwood Academy; 1998:351-362.
6. Kenney RA. Physiology of aging. *Clin Geriatr Med* 1985;1:37-59.
7. Evans, R. Physiology of aging. In: Sanders AB, ed. *Emergency Care of the Elder Person*. St. Louis, MO: Beverly Cracom Publications; 1996:11-28.
8. Posner J. Neurologic complications of systemic cancer. *Med Clin North Am* 1979;63:783.
9. Posner JB. Management of central nervous system metastases. *Semin Oncol* 1977;4:81.
10. Siegal T, Siegal T. Current considerations in the management of neoplastic spinal cord compression. *Spine* 1989;14:223.
11. Byrne TN. Current concepts: Spinal cord compression from epidural metastases. *N Eng J Med* 1992;327:614.
12. Cloustin PD, DeAngelis LM, Posner JD. The spectrum of neurological disease in patients with systemic cancer. *Ann Neurol* 1992;31:268.
13. Deyo RA, Rainville J, Kent DL. What can the history and physical examination tell us about low back pain? *JAMA* 1992;268:760.
14. Maranzano E, Latini P. Effectiveness of radiation therapy without surgery in metastatic spinal cord compression: Final results from a prospective trial. *Int J Radiat Oncol Biol Phys* 1995;32:959.
15. Sioutos PJ, Arbit E, Meshulan CF, et al. Spinal metastases from solid tumors. *Cancer* 1995;76:1453.
16. Landmann C, Hunig R, Gratzl O. The role of laminectomy in the combined

treatment of metastatic spinal cord compression. *Int J Radiat Oncol Biol Phys* 1992;24:627.
17. Young RF, Post EM, King GA. Treatment of spinal epidural metastases. Randomized prospective comparison of laminectomy and radiotherapy. *J Neurosurg* 1980;53:741.
18. Yahalom J. Oncologic emergencies. In: DeVita VT, Hellamnn S, Rosenberg SA, eds. *Cancer: Principles and Practice of Oncology.* 5th ed. Philadelphia:Lippincott-Raven Publishers;1997:2469-2475.
19. Ushio Y, Posner R, Posner JB, et al. Experimental spinal cord compression by epidural neoplasms. *Neurology* 1977;27:422.
20. Delatre JY, Arbit E, Thaler HT, et al. A dose response study of dexamethasone in a model of spinal cord compression caused by tumor. *J Neurosurg* 1989;70:920.
21. McCachren SS, Silberman HR. Approach to oncologic emergencies in the elderly. *Clin Geriat Med* 1987;3:575-586.
22. Kemeny MM, Busch-Devereaux E, Merriam LT, et al. Cancer surgery in the elderly. *Hematol Oncol Clin North Am* 2000;14:169-192.
23. Nishikubo C, Sadeghi A, Haskell CM. Superior vena cava syndrome. In: Haskell CM, ed. *Cancer Treatment.* 5th ed. Philadelphia: W.B. Saunders Co.; 2001:299-301.
24. Parish JM, Marsche RF, Dines DE, et al. Etiologic considerations in superior vena cava syndrome. *Mayo Clin Proc* 1981;56:407.
25. Lokich JJ, Goodman RL. Superior vena cava syndrome. *JAMA* 1975; 231:58.
26. Maddox AM, Valdivieso M, Lukeman J, et al. Superior vena cava obstruction in small cell bronchogenic carcinoma. *Cancer* 1983;52:2165.
27. Wurschmidt F, Bunemann H, Heilmann HP. Small cell lung cancer with and without superior vena cava syndrome: A multivariate analysis of prognostic factors in 408 cases. *Int J Radiat Oncol Biol Phys* 1995;33:77.
28. Mahajan V, Strimlan V, Van Ordstrand HS, et al. Benign superior vena cava syndrome. *Chest* 1975;68:32.
29. Fine DG, Shepherd RF, Welch TJ. Thrombolytic therapy for superior vena cava syndrome (Letter). *Lancet* 1989;1:1200.
30. Grenberg S, Kosinski R, Daniels J. Treatment of superior vena cava syndrome with recombinant tissue-type plasminogen activator. *Chest* 1991; 99:1298.
31. Hennequin LM, Fade O, Fays JG, et al. Superior vena cava stent placement: Results with the Wallstent endoprosthesis. *Radiology* 1995;196:353.
32. Lowenthal RM, Eaton K. Toxicity of chemotherapy. *Hematol Oncol Clin North Am* 1996;10:967-990.
33. Ayash LJ, Hunt M, Antman K, et al. Hepatic venoocclusive disease in autologous bone marrow transplantation of solid tumors and lymphomas. *J Clin Oncol* 1990;8:1699.
34. Tang E, Davis J, Silberman H. Bowel obstruction in cancer patients. *Arch Surg* 1995;130:832.
35. Gallick HL, Weaver DW, Sachs RJ, et al. Intestinal obstruction in cancer patients. An assessment of risk factors and outcome. *Am Surg* 1986;52:434.
36. Butler JA, Cameron BL, Morrow M, et al. Small bowel obstruction in patients with a prior history of cancer. *Am Surg* 1991;162:624.
37. Sial SH, Catalano MF. Gastrointestinal disorders in the elderly. *Gastroenterol Clin North Am* 2001;30:567.
38. Shivshanker K, Chu DZJ, Stroehlein JR, et al. Gastrointestinal hemorrhage in the cancer patient. *Gastrointest Endos* 1983;29:273.
39. Birkett DH. Gastrointestinal tract bleeding. *Surg Clin North Am* 1911;71: 1259.
40. Pimentel L. Medical complications of oncologic disease. *Emerg Med Clin North Am* 1993;11:407.
41. Bertheault-Cvitkovic F, Armand J-P, Tubiana-Hulin M, et al. Randomized, double-blind comparison of pamidronate versus galium nitrate for acute control of cancer-related hypercalcemia. *Proc 9th EORT/NCI Symposium on New Drugs in Cancer Therapy* 1996:140.
42. Body JJ, Dumon JC. Treatment of tumour-induced hypercalcemia with the bisphosphonate pamidronate: Dose-response relationship and influence of tumour type. *Ann Oncol* 1994;5:359.
43. Arrambide K, Toto RD. Tumor lysis syndrome. *Semin Nephrol* 1993; 13:273.
44. Fleming DR, Doukas MA. Acute tumor lysis syndrome in hematologic malignancies. *Leuk Lymph* 1992;8:315.
45. Madias NE. Lactic acidosis. *Kidney Int* 1986;29:752.
46. Sculier JP, Nicaise C, Klastersky J. Lactic acidosis: A metabolic complication of extensive metastatic cancer. *Eur J Clin Oncol* 1983;19:597.
47. Macaulay VM. Insulin-like growth factors and cancer. *Br J Cancer* 1992; 65:311.
48. Lipschitz DA, Udupa KB, Milton KY, et al. Effect of age on hematopoiesis in man. *Blood* 1984;63:502.
49. Baraldi-Junkins CA, Beck AC, Rothstein G. Hematopoiesis and cytokines —Relevance to cancer and aging. *Hematol Oncol Clin North Am* 2000;14: 45-61.
50. Wright IS. The pathogenesis and treatment of venous thrombosis. *Circulation* 1952;5:161.
51. Green Kb, Silverstein RL. Hypercoagulability in cancer. *Hematol Oncol Clin Am* 1996;10:499.
52. Rickles FR, Edwards RL. Hemostatic alterations in cancer patients. *Cancer Metastasis Rev* 1992;1:241.
53. Lipton A, Harvey HA, Hamilton RW. Venous thrombosis as a side effect of tamoxifen treatment. *Cancer Treat Rep* 1984;68:887.
54. Prandoni P, Lensing AWA, Buller HR, et al. Deep-vein thrombosis and the subsequent incidence of symptomatic malignant disease. *N Eng J Med* 1992;327:1128.
55. Goad KE, Gralnick HR. Coagulation disorders in cancer. *Hematol Oncol Clin North Am* 1996;10:457.
56. Eastridge BJ, Lefor AT. Complications of indwelling venous access devices in cancer patients. *J Clin Oncol* 1995;13:233.
57. Luzzatto G, Schafe AI. The prethrombotic state in cancer. *Semin Oncol* 1990;17:147.
58. Block KJ, Maki DG. Hyperviscosity syndrome associated with immunoglobulin abnormalities. *Semin Hematol* 1974;10:113.
59. Gmur J, Burger J, Schanz U, et al. Safety of stringent prophylactic platelet transfusion policy for patient with acute leukemia. *Lancet* 1991;338: 1223-1226.
60. Hackett T, Kelton JG, Powers P. Drug-induced platelet destruction. *Semin Thromb Hemost* 1982;8:116-137.
61. Porter J, Jick J. Addiction is rare in patients treated with narcotics. *N Eng J Med* 1980;302:123.
62. World Health Organization. Cancer pain relief and palliative care:Report of a WHO Expert Committee. Geneva, Switzerland: World Health Organization: 1990 Technical Report Series 804.
63. Emmanouilides C, Glaspy J. Opportunistic infections in oncologic patients. *Hematol Oncol Clin North Am* 1996;10:841.
64. Pizzo PA. Fever in immunocompromised patients. *N Eng J Med* 1999;341: 893-899.
65. Hughes WT, Armstrong D, Bodey GP, et al. 1997 Guidelines for the use of antimicrobial agents in neutropenic patients with unexplained fever. *Clin Infect Dis* 1997;25:551-573.
66. Freifeld AG, Walsh T, Marshall D, et al. Monotherapy for fever and neutropenia in cancer patients: A randomized comparison of ceftazidime versus imipenem. *J Clin Oncol* 1995;13:165-176.
67. Freifeld A, Marchigiani D, Walsh T, et al. A double-blind comparison of

empirical oral and intravenous antibiotic therapy for low-risk febrile patients with neutropenia during cancer chemotherapy. *N Eng J Med* 1999; 341:305-311.
68. Dorr RT, VonHoff DD. *Cancer Chemotherapy Handbook.* 2nd ed. Norwalk, CT: Appleton and Lange;1994.
69. Kreisman H, Wolkove N. Pulmonary toxicity of antineoplastic therapy. *Semin Oncol* 1992;19:508.
70. Travis LB, Curtis RE, Stovall M, et al. Risk of leukemia following treatment for non-Hodgkin's lymphoma. *J Natl Can Inst* 1994;86:1450.
71. van Leeuwen FE, Chorus AM, van den Belt-Dusebout AW, et al. Leukemia risk following Hodgkin's disease: Relation to cumulative dose of alkylating agents, treatment with teniposide combinations, number of episodes of chemotherapy and bone marrow damage. *J Clin Oncol* 1994;12:1063.

Physician CME Questions

To earn CME credit for this issue of Geriatric Emergency Medicine Reports, *please refer to the enclosed Scantron form for directions on taking the test and submitting your answers.*

1. Which of the following is true about the elderly?
   A. Memory, attention, and language are unaffected by age.
   B. Altered mental status is the most frequent neurological presentation in elderly cancer patients.
   C. Cancer is an inevitable consequence of normal aging.
   D. Cancer ranks ahead of cardiovascular disease in mortality in patients older than age 65.
   E. Eighty percent of all cancers occur in patients older than age 65.

2. The most common location of spinal cord compression is:
   A. the sacral region.
   B. the cervical and lumbar levels, equally.
   C. the lumbar region alone.
   D. the thoracic region.
   E. the cervical region alone.

3. Which of the following is true about superior vena cava syndrome (SVCS)?
   A. It is most commonly associated with Hodgkins' lymphoma.
   B. SVCS is best diagnosed by laborarory tests.
   C. SVCS may present with altered mental status.
   D. There is no role for thrombolytics in the management of SVCS.
   E. A salt-free diet markedly relieves the symptoms of SVCS.

4. A characteristic of cardiac tamponade in the elderly is:
   A. It can be confused with congestive heart failure.
   B. Pulsus paradoxicus is pathognonomonic.
   C. Diuretics and nitrates constitute the best treatment.
   D. Left ventricular pressure is markedly higher than right ventricular pressure.

5. Which of the following is true of neutropenic fever in the elderly on chemotherapy?
   A. Neutropenia does not occur in the elderly, as they normally are neutropenic.
   B. It may be the marker of an underlying infection.
   C. A duration of more than 10 days portends a good prognosis for the elderly.
   D. Neutropenia is defined as an absolute neutrophil count of $1200/mm^3$.
   E. Most causes of neutropenia are discovered in the ED.

6. Which of the following is true about pain management in the elderly cancer patient?
   A. Pain sensation is markedly lessened as one ages.
   B. Cancer patients on narcotics have a high rate of proven addiction.
   C. The clinician's lack of experience in pain assessment may contribute negatively to its management.
   D. The presence of pain is a sign of the end of life for the elderly.
   E. The absence of neurological signs precludes the diagnosis of impending spinal cord compression.

7. Which of the following is true of hypercalcemia in the elderly cancer patient?
   A. Serum calcium level is not directly related to the symptoms.
   B. Patients are usually very well hydrated on presentation.
   C. Furosemide is the first line of treatment.
   D. ECG changes include prolonged QT and sine wave.
   E. Hypercalcemic symptoms preclude treatment-induced fluid overload in the frail elderly.

8. Which of the following is true of abdominal pain in the elderly cancer patient?
   A. Signs and symptoms may be masked in the patient on corticosteroids.
   B. Right upper quadrant pain may be due to hepatic metastases.
   C. The most common site of bowel obstruction is the small bowel.
   D. Cecal distention of 12-14 cm on x-rays is an indication for colonoscopic decompression.
   E. All of the above

9. Management of pulmonary fibrosis following busulfan includes steroids.
   A. True
   B. False

10. Drug- and dose-dependent alopecia is the most frequent dermatologic manifestation of chemotherapy.
    A. True
    B. False

CME Objectives

Upon completing this program, participants will be able to:

- Quickly recognize or increase index of suspicion for specific conditions in the elderly patient;
- Understand the epidemiology, etiology, pathophysiology, and clinical features of the entity discussed;
- Perform necessary diagnostic tests correctly and take a meaningful patient history that will reveal the most important details about the particular medical problem discussed;
- Apply state-of-the-art therapeutic techniques (including the implications of the pharmaceutical therapy discussed) to patients with the particular medical problem discussed; and
- Provide patients with any necessary discharge instructions.

Emergency Medicine Reports
The Practical Journal for Emergency Physicians

Procedural Sedation, Part II

NPO Status for Children

CHILDREN < 6 MONTHS OLD
2 hours fast clear liquids
4 hours fast milk, solids

CHILDREN 6 MONTHS - 3 YEARS
3 hours fast clear liquids
6 hours fast milk, solids

CHILDREN > 3 MONTHS OLD
3 hours fast clear liquids
6-8 hours fast milk, solids

American Society of Anesthesiologists Classifcations

CLASS	PATIENT STATUS
I	Normally healthy patient. The pathologic process for which the procedure is to be performed is localized and not a systemic disturbance.
II	Mild systemic disease under control (e.g., asthma)
III	Severe systemic disease from any cause
IV	Severe systemic disease that is a constant life threat, not always correctable by the operative procedure
V	Moribund patient who is not expected to survive without the operation

Relevant History and Physical

1. Patient age
2. History of abnormalities of major organ systems, including heart, lungs, kidneys, or airway (e.g., sleep apnea, snoring, stridor)
3. Pregnancy test only in women who are unable to ensure (based on history) that they are not pregnant
4. Current medications
5. History of any adverse or allergic drug reactions with anesthesia or sedation/analgesia
6. History of prior sedation/analgesia, including adequacy of pain control for those procedures
7. History of tobacco, alcohol, or substance use or abuse
8. Vital signs (weight, heart rate, blood pressure, respiratory rate)
9. Cardiopulmonary examination
10. Airway examination
11. Laboratory evaluation based on the patient's medical condition and the effect the results of such evaluation will have on the plan for procedural sedation/analgesia

Adverse Events as a Result of Sedation and Analgesia

- Death
- Cardiopulmonary arrest
- Airway compromise during procedural sedation
- Prolonged sedation
- New neurologic deficit
- Significant hypoxemia (saturation < 90% for 1 minute in otherwise healthy patient
- Aspiration
- Significant hypotension
- Significant bradycardia
- Significant tachycardia
- Admissions to the hospital as a direct result of intravenous conscious sedation

Pre-procedural Examination of the Airway

INDICATORS OF POTENTIAL PROBLEMATIC AIRWAY:
- Decreased mouth opening
- Micrognathia
- Retrognathia
- Significant malocclusion
- Dentures
- Macroglossia
- Nonvisible uvula
- Decreased neck flexibility
- Advanced rheumatoid arthritis
- Dysmorphic facial features
  - – Pierre Robin syndrome
  - – Trisomy 21
  - – < 3 cm hyoid-mental distance (adult)
- Tracheal deviation

Pre-sedation Assessment on the Day of the Procedure

1. Documentation of any changes in history and physical
2. Time and nature of last oral intake
3. Vital signs, including heart rate, blood pressure, respiratory rate, and temperature
4. Baseline ambulation status
5. Patient or legal guardian must be informed about the risks, benefits, and alternatives to the proposed procedural sedation/analgesia.

Modified Ramsay Score for Procedural Sedation

1 = Anxious
2 = Awake, tranquil
3 = Drowsy, responds easily to verbal commands
4 = Asleep, brisk response to tactile or loud auditory stimulus
5 = Asleep, minimal response to tactile or loud auditory stimulus
6 = Asleep, no response

Post-sedation and Analgesia Discharge Criteria

- Return to baseline verbal skills
  - –Can understand and follow directions
  - –Can verbalize, including correct diction
- Return to baseline muscular control function
  - –If an infant, can sit unattended
  - –If a child or adult, can walk unassisted
- Return to baseline mental status
- Patient or responsible person with patient can understand procedural sedation and/or analgesia ED discharge instructions
- Transportation home can be arranged

Model Quality Assurance Pathway

1. Each procedural area should identify an individual responsible for adverse event reporting and follow-up.
2. Each procedural area should determine a system for assuring compliance with documentation requirements.
3. Monthly summary reports should be made to the quality management authority or equivalent. Among specific events to be reported include:
   a. Total number of sedation cases per month per physician and whether the cases were moderate or deep sedation;
   b. Recovery phase > 3 hrs;
   c. Admissions as a direct result of procedural sedation;
   d. Emergency resuscitation of any type (drugs, airway, or cardiopulmonary support) initiated due to procedural sedation;
   e. Procedure cancellation due to adverse events related to sedation/analgesia;
   f. For moderate sedation cases, any prolonged airway intervention;
   g. For moderate sedation cases, any need to rescue the patient from slipping into deep sedation; or
   h. For deep sedation cases, any need to rescue the patient from slipping into general anesthesia.
4. The monthly reports should be reviewed by the hospital quality management board and the quality review officer of the department from which the report originated.

Topical Anesthesia

MEDICATION	RECOMMENDED DOSAGE	ROUTE OF ADMINISTRATION	ONSET	DURATION	ADDITIONAL INSTRUCTIONS	PRECAUTIONS/ CONTRAINDICATIONS
EMLA CREAM (2.5% LIDOCAINE, 2.5% PRILOCAINE)						
	2.5 gm	Topically (venipuncture and venous cannulation, lumbar puncture, arterial puncture)	½ - 1 hour	1 hour after removal of occlusive dressing	Provides dermal/topical anesthesia. Dermal application may cause a transient, local blanching followed by transient, local redness or erythema. Apply cream 2 in x 2 in area in a thick layer at the site of procedures. Place an occlusive (i.e., OpSite, transparent) dressing over site. Apply at least ½ hour before IV puncture. After 1-1½ hr, wipe off cream, clean the area with an antiseptic solution, and prepare for vein puncture.	Do not use in children younger than 1 month of age. Do not apply to broken or inflamed skin. Contra-indicated in patients with a known history of sensitivity to local anesthe-tics. Use with care in patients with conditions or therapy associated with methemoglobinemia (prilocaine). Avoid inadvertent trauma to the treated area by scratching, rubbing, or exposure to extreme hot or cold temperatures until complete sensation has returned.
TAC (TETRACAINE/ADRENALINE/COCAINE)						
	Total dose of TAC should be limited to 1.5 mL/10 kg (Concentration: 0.5% tetracaine 0.05% adrenaline, 4-11% cocaine)	Topically (suture of lacerations)	10-20 min	45-60 min	Soak gauze pad in solution and place directly over wound for 5-10 min. Anesthesia can be judged by the appearance of blanching at the wound site.	Do not use on mucous membranes or areas with end-arterial circulation (such as fingers, toes, nose, and penis).
TETRACAINE (AMETHOCAIME) CREAM						
	4% cream	Topically under occlusion	40 min	4 hours	Apply under occlusion for 40 min in children undergoing IV placement. Vasodilation has been noted.	
IONTOPHORESIS						
	2% lidocaine with epinephrine 0.6-1 mL	Electrode well over intact skin	10 min	10 min without epinephrine, 60 min with epinephrine		Tingling, itching, burning of skin over both electrodes may occur. Do not use in patients with pacemakers or over metal indwelling catheter ports, or fingers, nose, toes, or penis.
LET (LIDOCAINE 5%, EPINEPHRINE 0.1%, TETRACAINE 0.5%)						
	1-3 mL		20-30 min	45-60 min	May need to supplement with injected buffered lidocaine before suturing.	Effective in 75-98% of facial and scalp lacerations, only 40-60% of extremity wounds.

Emergency Department
Hospital Name
Hospital Address
Hospital City, State, Zip

GENERAL INFORMATION: You or your child were give medications to induce sleepiness so the doctors could perform a procedure on you with minimal discomfort. The anesthetics, sedatives, or pain-killers which were given to you in the emergency department may still be active for the next 24 hours. You or your child may feel a little drowsy from them.

The medications you or your child received:
_____
_____
_____

INSTRUCTIONS:

Activity
Adult: Rest at home today, progressing to regular activities as tolerated. Do not drive a car, operate dangerous machinery or power tools for 24 hours. This includes sewing machines and blenders, as well as heavy equipment. Do not drink alcohol or take unpre-scribed medication for 24 hours. Do not make any important decisions. Be careful climbing stairs.

Child: Restrict activity to quiet play, watching TV, coloring. Do not let child play unattended for 24 hours due to possible dizziness or drowsiness. Do not let your child ride bicycles or play on swingsets for the next 24 hours.

You or your child may feel sleepy. It is fine to sleep.

Diet
Adult: Clear liquids are best tolerated at first. If you are not nauseated, you can progress your diet to solid foods as tolerated.

Child: Start with fluids, then advance to appropriate diet for child's age, as tolerated. Fluids are more important for the first 24 hours.

CONTACT YOUR DOCTOR OR RETURN TO THE EMERGENCY DEPARTMENT IF:
1. There is vomiting more than 24 hours.
2. There are signs of infection such as fever or chills.
3. You or your child have pain unrelieved by acetaminophen or other prescribed pain medication.
4. There are any complications from the procedure performed.

Supplement to *Emergency Medicine Reports*, October 21, 2002: "Procedural Sedation. Part II: Specific Scenarios, Topical Agents, and Establishment of Procedural Sedation Policy within the Emergency Department." *Authors:* **Jonathan Glauser, MD, FACEP,** Department of Emergency Medicine, Cleveland Clinic Foundation, Cleveland, OH; Faculty, Emergency Medicine Residency, MetroHealth Medical Center, Cleveland, OH; **Brian Cullison, MD,** Resident, Department of Emergency Medicine, MetroHealth Medical Center, Cleveland, OH. *Emergency Medicine Reports' "Rapid Access Guidelines."* **Editor-in-Chief:** Gideon Bosker, MD, FACEP. **Vice President and Group Publisher:** Brenda Mooney. **Editorial Group Head:** Valerie Loner. **Specialty Editor:** Shelly Morrow. For customer service, call: **1-800-688-2421.**