Supportive and Preventative Medicine
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Conflict of Interest Disclosures

- None
Learning Objectives

1. Identify the importance of key components of intensive care medicine that can be applied to all critically ill patients.
2. Recommend therapeutic options to prevent stress-related mucosal disease.
3. Recommend therapeutic options to prevent venous thromboembolism in a critically ill patient.
4. Discuss therapeutic options for patients with heparin-induced thrombocytopenia.
5. Discuss medications that can be used to provide comfort to a critically ill patient at the end of life.
Agenda

1. FAST-HUG mnemonic
2. Stress ulcer prophylaxis
3. Deep vein thrombosis prophylaxis
4. Heparin-induced thrombocytopenia
5. End of life considerations
FAST-HUG Mnemonic

- Feeding
- Analgesia
- Sedation
- Thromboembolic prophylaxis
- Head of bed elevation
- Ulcer prophylaxis
- Glycemic control
## Elements of the FAST-HUG Mnemonic

<table>
<thead>
<tr>
<th>Element</th>
<th>Considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>F</strong>eeding</td>
<td>Initiate oral or enteral feeding as soon as possible</td>
</tr>
<tr>
<td><strong>A</strong>nalgesia and <strong>S</strong>edation</td>
<td>Pain and sedation should be assessed and reassessed with a validated tool; maintain light levels of sedation and if appropriate, execute sedative interruption</td>
</tr>
<tr>
<td><strong>T</strong>hromboembolic prophylaxis</td>
<td>Initiate appropriate prophylaxis considering VTE and bleeding risks</td>
</tr>
<tr>
<td><strong>H</strong>ead of bed elevation</td>
<td>Ensure patient position periodically throughout the day</td>
</tr>
<tr>
<td><strong>U</strong>lcer prophylaxis</td>
<td>Consider acid-suppressive medications in patients at risk and discontinue when risk factors are no longer present</td>
</tr>
<tr>
<td><strong>G</strong>lycemic control</td>
<td>Continuous insulin infusions to maintain blood glucose between 140 and 180 mg/dL when blood glucose levels are &gt; 150 mg/dL</td>
</tr>
</tbody>
</table>
Patient Case # 1

- A 68-year-old man is intubated and admitted to the ICU for pneumonia
- His current medications include:
  - Antimicrobials: ceftriaxone 1 g IV daily and vancomycin 1250 mg IV q12h
  - Analgesia and sedation: fentanyl infusion at 50 mcg/h and midazolam infusion at 1 mg/h titrated to a Richmond Agitation Sedation Scale (RASS) of 0 to -1
  - Glucose control: regular insulin infusion at 1.5 units/h titrated to maintain blood glucose 140–180 mg/dL
  - DVT prophylaxis: heparin 5000 units SC q8h
- Currently, the patient’s head is 30 degrees above the bed, his RASS is documented as -4, he is on minimal ventilator settings, and he has a NGT
Patient Case #1

Which are the best recommendations to make to the team?

A. Initiate enteral nutrition by NGT, add SUP, and discontinue fentanyl and midazolam infusions

B. Initiate enteral nutrition by NGT, discontinue DVT prophylaxis, and transition insulin infusion to sliding scale

C. Transition insulin infusion to sliding scale, add SUP, and discontinue fentanyl and midazolam infusions

D. Discontinue fentanyl and midazolam infusions, discontinue DVT prophylaxis, and add SUP
Agenda

✓ FAST-HUG mnemonic

2. Stress ulcer prophylaxis

3. Deep vein thrombosis prophylaxis

4. Heparin-induced thrombocytopenia

5. End of life considerations
Epidemiology of Stress-Related Mucosal Disease (SRMD)

- Endoscopic evidence of superficial mucosal damage occurs in 75%–100% of patients within 1–2 days after ICU admission

- Mortality from stress-related bleeding ranges from 50% to 70% in the critically ill, with a 20% mortality rate attributable to SRMD
# Stress Ulcers vs. Peptic Ulcers

<table>
<thead>
<tr>
<th>Stress Ulcers</th>
<th>Peptic Ulcers</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute</td>
<td>Chronic</td>
</tr>
<tr>
<td>Multiple lesions</td>
<td>Single lesion</td>
</tr>
<tr>
<td>Superficial</td>
<td>Deep</td>
</tr>
<tr>
<td>Proximal stomach bulb</td>
<td>Duodenal</td>
</tr>
<tr>
<td>Occur in specific clinical setting</td>
<td>Occur at anytime</td>
</tr>
</tbody>
</table>
Pathophysiology of SRMD

Gut Mucosal Ischemia

Impaired Defense Mechanisms:
- $\uparrow$ H$^+$ back diffusion
- $\downarrow$ Mucous/bicarbonate barrier
- $\downarrow$ Prostaglandin production
- $\downarrow$ GI motility

Reperfusion injury
- Inflammation
- Free radical formation

Acid secretion

Stress Ulceration

Gastrointestinal Bleed

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## Bleeding Definitions

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Incidence in ICU Patients</th>
<th>Definition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Endoscopically evident mucosal damage</td>
<td>75%–100%</td>
<td>Superficial lesions identified on endoscopy</td>
</tr>
<tr>
<td>Occult bleeding</td>
<td>15%–50%</td>
<td>Presence of guaiac-positive stools or nasogastric aspirate</td>
</tr>
<tr>
<td>Overt or clinically evident bleeding</td>
<td>5%–25%</td>
<td>Appearance of coffee grounds in nasogastric aspirate, hematemesis, melena, or hematochezia</td>
</tr>
<tr>
<td>Clinically significant bleeding</td>
<td>1%–5%</td>
<td>Bleeding with hemodynamic instability and/or blood transfusion</td>
</tr>
</tbody>
</table>
# Bleeding Definitions

<table>
<thead>
<tr>
<th>Clinically Evident Bleeding</th>
<th>Clinically Significant Bleeding</th>
</tr>
</thead>
<tbody>
<tr>
<td>Presence of “coffee grounds” in NG aspirate</td>
<td>Bleeding + one of the following:</td>
</tr>
<tr>
<td>Guaiac-positive NG aspirate</td>
<td>▪ Decrease in systolic blood pressure &gt;20 mm Hg within 24 h of bleed</td>
</tr>
<tr>
<td>Guaiac-positive stools</td>
<td>▪ Orthostatic increase in pulse rate of &gt;20 beats/min and decrease in systolic blood pressure &gt;10 mm Hg</td>
</tr>
<tr>
<td>Hematemesis</td>
<td>▪ Decrease in hemoglobin of at least 2 g/dL in 24 h AND subsequent transfusion after which hemoglobin did not increase by at least the number of units transfused minus 2 g/dL</td>
</tr>
<tr>
<td>Melena</td>
<td></td>
</tr>
<tr>
<td>Hematochezia</td>
<td></td>
</tr>
</tbody>
</table>
Decisions Surrounding Stress Ulcer Prophylaxis

Who needs stress ulcer prophylaxis?

Which agent should be used?  
What dose?  
What route of administration?

When should prophylaxis be discontinued?
Risk Factors for Stress-Related Bleeding

- 2,252 patients enrolled with moderate to severe illness admitted to surgical and medical ICUs
- Results:
  - 3.7% of patients had clinically significant bleeding with ≥ 1 risk factor
  - 0.1% of patients had clinically significant bleeding with no risk factors

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Multivariate Risk Ratio</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Respiratory failure</td>
<td>15.6</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Coagulopathy</td>
<td>4.3</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Hypotension</td>
<td>3.7</td>
<td>0.08</td>
</tr>
<tr>
<td>Sepsis</td>
<td>2.0</td>
<td>0.17</td>
</tr>
<tr>
<td>Hepatic failure</td>
<td>1.6</td>
<td>0.27</td>
</tr>
<tr>
<td>Renal failure</td>
<td>1.6</td>
<td>0.26</td>
</tr>
<tr>
<td>Enteral feeding</td>
<td>1.0</td>
<td>0.99</td>
</tr>
<tr>
<td>Corticosteroids</td>
<td>1.5</td>
<td>0.26</td>
</tr>
<tr>
<td>Transplant</td>
<td>1.5</td>
<td>0.42</td>
</tr>
<tr>
<td>Anticoagulant therapy</td>
<td>1.1</td>
<td>0.88</td>
</tr>
</tbody>
</table>
Risk Factors for Stress-Related Bleeding: Mechanically Ventilated

- Acute kidney injury
- Age (50 years or older)
- Male sex
- Acute respiratory failure
- Myocardial infarction
- Neurologic injury
- Sepsis
- Shock
- Acute or chronic hepatic failure
- Coagulopathy
### SRMD and Gastric pH Monitoring

<table>
<thead>
<tr>
<th>Gastric pH</th>
<th>Physiologic Activity</th>
</tr>
</thead>
<tbody>
<tr>
<td>≥3.5</td>
<td>Decreased incidence of stress-induced bleeding</td>
</tr>
<tr>
<td>≥4.5</td>
<td>Pepsin inactivation</td>
</tr>
<tr>
<td>=5.0</td>
<td>99.9% acid neutralization</td>
</tr>
<tr>
<td>&lt;7</td>
<td>Alterations in coagulation and platelet aggregation</td>
</tr>
<tr>
<td>≥7</td>
<td>Potential decrease in incidence of rebleeding</td>
</tr>
<tr>
<td>≥8</td>
<td>Pepsin destruction</td>
</tr>
</tbody>
</table>

Rationale for level of acid suppression based on *in vitro* and animal studies
Pharmacologic Therapy for Preventing Stress Ulcers

- Antacids
- Sucralfate
- Histamine-2 Receptor Antagonists (H2RA)
  - Cimetidine
  - Famotidine
  - Nizatidine
  - Ranitidine
- Proton Pump Inhibitors (PPI)
  - DEXLANSOPRAZOLE
  - Esomeprazole
  - Lansoprazole
  - Omeprazole
  - Pantoprazole
  - Rabeprazole

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**Sucralfate vs. H₂RAs**

- Randomized, double-blind study of 1200 mechanically ventilated ICU patients
- Sucralfate 1 g NG/OG q6h vs ranitidine 50 mg IV q8h
- Clinically significant bleeding (transfusion or hypotension):

![Bar chart showing percent of patients with clinically significant bleeding]

- Sucralfate: 3.8%
- Ranitidine: 1.7%

*p = 0.02*
H$_2$RAs vs. PPIs

Meta-analyses favor PPIs over H2RAs to prevent clinically important bleeding rates

- Randomized, double-blind, double-dummy, non-inferiority trial including 359 mechanically ventilated ICU patients
  - IV cimetidine 300 mg IV x1 then 50 mg/h (titrated to pH) vs. oral omeprazole 40 mg daily
  - Clinically-significant bleeding (bloody gastric lavage):
    - Cimetidine=5.5% vs. Omeprazole=3.9%
  - Any bleeding:
    - Cimetidine=32% vs. Omeprazole=19% (p=0.005)

- Randomized trial including 67 mechanically ventilated patients
  - Ranitidine 50 mg IV x1 then 150 mg/24h or 50 mg IV q8h vs. oral simplified omeprazole solution (SOS) 20 mg daily
  - Clinically significant bleeding (transfusion or hypotension):
    - Ranitidine=31% vs. SOS=6% (p=0.013)

Workbook Page 1-117; Table 6
H₂RAs vs. PPIs

- Meta-analyses favor PPIs over H₂RAs to prevent clinically important bleeding rates
  - The studies that were included lacked methodological quality with unexpectedly high baseline bleeding rates, a disproportionate number of risk factors between patient groups, inconsistent definitions of bleeding, and different routes and dosing of agents
Potential Complications of PPIs

- Interstitial nephritis
- Hypomagnesemia (≥ 3 months of therapy)
- Neurologic effects with high-dose intravenous omeprazole (hearing and vision disturbances)
- Hypophosphatemia and metabolic alkalosis when administered with sodium bicarbonate
- Increased risk of fractures (hip, waist, and spine)
- *C. difficile* infection (definitive cause-effect relationship is not well established)
- Risk of nosocomial pneumonia
Infectious Complications

- Increases in gastric pH promote bacterial overgrowth, and PPIs have a greater propensity to maintain a sustained higher pH

- Pneumonia
  - Meta-analyses demonstrate lower pneumonia rates with sucralfate compared to H$_2$RAs alone or combined with antacids; no differences when H$_2$RAs were compared with PPIs
  - An observational study of cardiac surgical patients detected a higher rate of pneumonia with PPIs than with H$_2$RAs
    - RR 1.19 (95% CI 1.03–1.38) after propensity score adjustment
Infectious Complications

- **C. difficile** infection (CDI)
  - A cohort study observed incremental increases in the risk of nosocomial CDI as the level of acid suppression increased
  - Pharmacoepidemiologic cohort study of acid suppressive therapy in critically ill patients (ICD-9 coded CDI):
    - OR = 1.29 (95% CI, 1.04-1.59) by propensity and covariate adjustment
    - OR = 1.31 (95% CI, 1.04-1.64) by matching
  - Many published trials have different definitions of CDI, unclear association of antisecretory therapy initiation and CDI diagnosis, and variable infection control practices
Guideline Recommendations

- The Surviving Sepsis Guidelines published in 2012 recommend PPIs over H₂RAs for SUP (weak/low quality evidence [Grade 2D])

- SCCM/ACCM Guidelines on Stress Ulcer Prophylaxis – *to be released soon*
Patient Case # 2

- A 72-year-old woman is intubated and admitted to the ICU for pneumonia

- She is currently receiving fluid boluses, norepinephrine and vasopressin infusions, and appropriate antimicrobial agents

- Her WBC is $20 \times 10^3$ cells/mm$^3$, platelet 45,000/mm$^3$, BUN 70 mg/dL, SCr 4.5 mg/dL (baseline 0.9 mg/dL), potassium 4.5 mEq/L, INR 1.4, AST 30 IU/mL, and AST 46 IU/mL

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Patient Case #2

Which best reflects this patient’s number of risk factors for stress-related bleeding?

A. One
B. Two
C. Four
D. Five
Patient Case #2

Which would be most appropriate for preventing stress-related bleeding?

A. Sucralfate 1 g four times daily by NGT

A. Magnesium hydroxide 30 mL q4h by NGT

A. Pantoprazole 40 mg intravenously daily

A. Famotidine 20 mg intravenously daily
Patient Case # 2

- One week later, the patient’s respiratory status has greatly improved
- She is extubated, off sedation and vasopressors, and working with physical therapy
- Her medications include ceftriaxone, heparin SC, and SUP
- Laboratory values are as follows: WBC 6 x 10^3 cells/mm^3, platelet 256,000/mm^3, BUN 10 mg/dL, SCr 1.1 mg/dL, potassium 4.0 mEq/L, INR 0.8, AST 15 IU/mL, and ALT 10 IU/mL
Patient Case #2

Which would be the most appropriate recommendation to make regarding this patient’s SUP regimen?

A. SUP should be continued until hospital discharge
B. SUP should be continued until ICU discharge
C. SUP should be discontinued now
D. SUP should be continued until the patient is off antimicrobials

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Agenda

✓ FAST-HUG mnemonic

✓ Stress ulcer prophylaxis

3. Deep vein thrombosis prophylaxis

4. Heparin-induced thrombocytopenia

5. End of life considerations
## Risk Factors for Venous Thromboembolism (VTE)

### General Medical
- Advanced age
- Malignancy
- Recent surgery / trauma
- Previous VTE
- Pregnancy
- Obesity
- Hormonal therapy
- Erythropoiesis-stimulating agents with hemoglobin $\geq 12$ g/dL
- Inherited or acquired hemophilia

### ICU-Acquired
- Immobility
- Stroke
- Trauma
- Mechanical ventilation
- Central venous lines
- Sepsis
- Cardiac or respiratory failure
- End-stage renal failure
- Vasopressor use
- Platelet transfusion
Prevention of VTE

- Low-dose unfractionated heparin or low-molecular-weight heparin should initiated over no prophylaxis.

- Mechanical VTE prophylaxis should be used in patients who are bleeding or at high risk for bleeding.

- Once bleeding risk is no longer present, initiate pharmacologic VTE prophylaxis.
## Agents Used for VTE Prophylaxis

<table>
<thead>
<tr>
<th>Agent</th>
<th>Dose in Patients with Normal Renal Function</th>
<th>Dose in Patients with Renal Impairment (estimated CrCl 20-30 mL/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Enoxaparin</td>
<td>40 mg SC daily</td>
<td>30 mg SC daily</td>
</tr>
<tr>
<td>Dalteparin</td>
<td>5000 units SC daily</td>
<td><em>Specific dosage adjustments have not been recommended; accumulation was not observed in critically ill patients with severe renal insufficiency. No adjustment needed for CrCl &gt; 20 mL/min</em></td>
</tr>
<tr>
<td>LDUH</td>
<td>5000 units SC every 8–12 h: choosing between q8h and q12 h should be based on the patient’s risk of thrombosis and bleeding</td>
<td></td>
</tr>
<tr>
<td>Fondaparinux</td>
<td>2.5 mg SC once daily for patients ≥ 50 kg</td>
<td><em>Contraindicated; however, doses of 2.5 mg SC q48 h have been used</em></td>
</tr>
</tbody>
</table>
Considerations in the Critically Ill

- Reduction in bioavailability with concomitant use of vasopressors or significant edema

- Renal impairment
  - 138 critically ill patients with CrCl < 30 mL/min receiving prophylactic dalteparin had no evidence of accumulation or an increased risk of bleeding

- Bleeding
  - Patients at high risk of bleeding are often excluded from studies
**Novel Oral Anticoagulants**

- No studies to date conducted in ICU patients for VTE prophylaxis

- Rivaroxaban 10 mg po daily (35-day regimen) was compared with enoxaparin 40 mg SC daily x 10 days, followed by placebo in acutely ill, hospitalized patients

- Rates of composite end point (asymptomatic or symptomatic VTE, pulmonary embolism, or death):
  - Day 10: 2.7% in both the rivaroxaban and enoxaparin groups, p=0.003; met criteria for noninferiority

- Clinically relevant bleeding
  - Day 10: 2.8% in the rivaroxaban group vs. 1.2% in the enoxaparin group, p<0.001

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Patient Case # 3

- A 93-year-old bedbound man (weight 45 kg) is admitted with a COPD exacerbation requiring mechanical ventilation
- He has a history of diabetes mellitus and heart failure
- His laboratory values are all within normal limits except for a BUN of 35 mg/dL and a SCr of 2.8 mg/dL (baseline 0.5)
Patient Case #3

Which would be the most appropriate recommendation for VTE prophylaxis in this patient?

A. Intermittent pneumatic compression device
B. Enoxaparin 30 mg SC once daily
C. Heparin 5000 units SC q12h
D. Fondaparinux 2.5 mg SC daily

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Agenda

- FAST-HUG mnemonic
- Stress ulcer prophylaxis
- Deep vein thrombosis prophylaxis
- Heparin-induced thrombocytopenia
- End of life considerations
Heparin-Induced Thrombocytopenia (HIT)

- A severe, immune-mediated reaction potentially leading to life-threatening complications
Frequency of HIT

- Thrombocytopenia is one of the most common laboratory abnormalities found among hospitalized patients.

- Serologically proven HIT occurs in 1-3% of patients with heparin exposure.

- Higher risk in cardiac or orthopedic surgical patients receiving LDUH (1-5%) than in medical patients (0.1-1%).
Clinical Diagnosis of HIT

- Absolute platelet count $< 150,000/mm^3$, or relative platelet decline of $\geq 50\%$ from baseline

<table>
<thead>
<tr>
<th></th>
<th>Time course after heparin exposure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Typical</td>
<td>5-10 days</td>
</tr>
<tr>
<td>Rapid</td>
<td>Within 24 h</td>
</tr>
<tr>
<td>Delayed</td>
<td>Up to 3 weeks after cessation of therapy</td>
</tr>
</tbody>
</table>

- With or without thrombosis
Laboratory Testing

**Antigen Assay**
- Detects the presence of HIT antibodies reactive against PF4/heparin or PF4/polyvinyl sulfonate
- Positive result: > 0.40 optical density
- High sensitivity, moderate specificity

**Functional Assays**
- Heparin-induced platelet aggregation and C14 serotonin release assay
- High sensitivity and specificity
- Technically challenging and not readily available

Should not wait until laboratory results come back to initiate treatment
Treatment of HIT

- Immediately discontinue all sources of heparin
- Initiate alternative anticoagulation with a parenteral direct thrombin inhibitor or factor Xa inhibitor
- May begin warfarin once the platelet count has recovered and is within normal limits (at least 150,000/mm³) and after at least 5 days of therapy with an alternative anticoagulant OR conservative warfarin dosing may begin once platelet count is recovering
- Reverse with vitamin K if a patient is on warfarin at the time of HIT diagnosis

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# Parenteral Agents for the Treatment of HIT

<table>
<thead>
<tr>
<th></th>
<th>Argatroban</th>
<th>Desirudin</th>
<th>Bivalirudin</th>
<th>Fondaparinux</th>
</tr>
</thead>
<tbody>
<tr>
<td>FDA approved for the treatment of HIT</td>
<td>Yes</td>
<td>No</td>
<td>Percutaneous coronary intervention with HIT</td>
<td>No</td>
</tr>
<tr>
<td>Mechanism of action</td>
<td>Direct thrombin inhibitor</td>
<td>Direct thrombin inhibitor</td>
<td>Direct thrombin inhibitor</td>
<td>Factor Xa inhibitor</td>
</tr>
<tr>
<td>Elimination half-life</td>
<td>40–50 min</td>
<td>120 min</td>
<td>25 min</td>
<td>17–20 h</td>
</tr>
<tr>
<td>Elimination</td>
<td>Hepatobiliary</td>
<td>Renal</td>
<td>80% enzymatic; 20% renal</td>
<td>Renal</td>
</tr>
</tbody>
</table>
Parenteral Agents for the Treatment of HIT

- Studies have demonstrated lower argatroban and bivalirudin dosing requirements in the critically ill
  - Argatroban studies:
    - Mean dose in critically ill patients: 0.24 ± 0.16 mcg/kg/min
    - Mean dose in critically ill patients with multiple organ dysfunction: 0.22 ± 0.15 mcg/kg/min
    - Severe liver impairment: 0.5 mcg/kg/min
  - Bivalirudin studies:
    - Dose reductions to 0.05–0.1 mg/kg/h, depending on renal function
Patient Case # 4

- A 55-year-old man with a medical history of a DVT 2 months ago secondary to trauma is admitted to the ICU for acute respiratory failure from influenza virus.
- Pertinent laboratory values include platelet count 250,000/mm³, BUN 13 mg/dL, SCr 0.9 mg/dL, INR 1.2, AST 22 IU/mL, and AST 11 IU/mL.
- Medications include piperacillin/tazobactam, vancomycin, regular insulin infusion, famotidine, and a heparin infusion.
- Five days later it is noted that his platelet count has decreased to 112,000/mm³, and his BUN and SCr have increased to 45 mg/dL and 2.7 mg/dL, respectively.
- Heparin-PF4 immunoassay; results will not come back for 48 h.
Patient Case #4

Which would be the best course of action in this patient?

A. Discontinue heparin infusion, and initiate argatroban continuous infusion at 0.5 mcg/kg/min

B. Do nothing because the patient has several other reasons to be thrombocytopenic

C. Discontinue heparin infusion, and initiate fondaparinux at 10 mg SC daily

D. Do nothing until the heparin-PF4 immunoassay results
Agenda

✓ FAST-HUG mnemonic
✓ Stress ulcer prophylaxis
✓ Deep vein thrombosis prophylaxis
✓ Heparin-induced thrombocytopenia

5. End of life considerations
General Considerations

- Prevent any uncomfortable or unnecessary procedures, tests, or treatments
- Minimize or discontinue routine vital sign checks, patient weights, cardiac or other electronic monitoring, fingersticks, and intermittent pneumatic compression devices
- Consider discontinuing routine blood draws, radiologic imaging, and other diagnostic procedures
- Consider discontinuing all medications not necessary for patient comfort
Symptom Management

- Pain
  - Opioids are the mainstay of treatment
  - Morphine most commonly used
  - Administer opioid as an IV bolus dose and begin an IV continuous infusion, adjusting rates to maintain comfort
  - Tolerance may develop over time
  - Evidence demonstrates that pain can be improved with correct dosing and titration without causing respiratory depression or hastening death
Symptom Management

- **Anxiety/agitation/delirium**
  - Frequent reorientation to the environment and reduction in noise and other bothersome or stimulating environmental factors
  - IV haloperidol may be used without electrocardiographic monitoring
  - Benzodiazepines (midazolam and lorazepam)

- **Fever**
  - Acetaminophen, nonsteroidal anti-inflammatory drug, or dexamethasone
Symptom Management

- **Nausea and vomiting**
  - Underlying causes including drugs and conditions should be treated or eliminated, if possible.
  - Agents to consider include haloperidol, metoclopramide, ondansetron, dexamethasone, or lorazepam (use of more than one agent may be necessary for symptom relief).

- **Cough**
  - Non-opioid antitussives
  - Opioid – more than one has not shown to be additive
Symptom Management

- Secretions
  - The mainstay of treatment includes anticholinergic and antimuscarinic medications
  - Glycopyrrolate (0.1 mg IV q4h) or atropine (1% ophthalmic solution 2 drops sublingually q4h as needed)
  - Scopolamine patch is more gradual in onset (12 h)
Patient Case # 1

- An 88-year-old woman is admitted to the ICU for decompensated heart failure and acute kidney injury.
- This is her 4th admission to the ICU in the past 5 months.
- In speaking with the patient, you find that she wishes not to be resuscitated or intubated but only to be comfortable.
- All family members agree that they do not want to see her suffer any longer.
- It is decided to initiate a morphine infusion at 2 mg/h; titration parameters include giving a bolus dose equivalent to the current rate and increasing the infusion by 25% for comfort.
Patient Case #2

The nurse taking care of the patient believes that the titration parameters are too aggressive. Which would be the most appropriate change in titration parameters?

A. Change the parameters to increase the morphine infusion only when the patient shows signs of discomfort, such as an increase in blood pressure or heart rate
B. Discontinue titration parameters and keep the morphine infusion at the current rate
C. Discontinue titration parameters and keep the morphine infusion at the current rate and add a midazolam infusion at 2 mg/h
D. Do not change the titration parameters at this time

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