Sedation-Analgesia Recovery
The Recovery Phase

- From termination of procedure or test until discharge criteria are met
- The period immediately post-procedure is the most dangerous time. Stimuli have ceased and sedation-analgesia is unopposed by pain.
- All hospital areas that practice sedation-analgesia must have a post-procedure recovery area with all equipment and monitoring as required for SA available.
- The procedure room may also be a recovery area.
Post-procedural Monitoring I

- Continuous attendance by someone with current privileges in sedation-analgesia must occur until discharge.
- Continuous monitoring and documentation q 15 minutes of:
  - BP, HR and rhythm
  - SpO₂ and oxygen supplementation
  - RR
  - level of consciousness (I-IV)
The frequency of VS must adjust to patient status. Documentation q5 min is resumed and the rescue procedure is initiated if any of the following are noted:

- Decreased level of consciousness (Level 3)
- Respiratory depression (RR < 10)
- SpO₂ <90% despite supplemental oxygen
In transporting patient from procedure room to recovery area:

- Administer supplemental O₂
- Privileged SA provider will accompany patient
- Report shall be given to the accepting nursing staff and between attendings:
  - Patient history, procedure, sedation administered and any complications or side effects
Patient Transportation II

- If patient is transferred to another area the **same standards** of care and monitoring apply until discharge criteria is meet.
- Patient may be admitted to a monitored hospital unit with the same standards and availability of equipment and personnel.
Discharge Criteria I

- Patient is awakened by verbal commands
- Patient is appropriately oriented
- Recovery of protective reflexes
- VS are stable: pre-sedation baseline
- PAR score of 9-10 or equivalent to pre-sedation
- $\text{SaO}_2 > 94\%$ or not less than baseline value on room air
- A “patient discharge to floor/home” order is required by a licensed independent practitioner to discontinue monitored care
Discharge Criteria II: Outpatients

- Able to tolerate PO intake and ambulate independently or at pre-procedure status
- Discharge patient in the care of a responsible adult
- Verbal and written information regarding activity level, medications, sign/symptoms of complications and course of action in the case of complications or an emergency
- This must be documented on the chart
Discharge Criteria III: Variants

- Any case requiring unexpected hospital admission or upgrade to a special monitored unit (PARU, ICU, stepdown) requires documentation of events and a written plan by the attending responsible for the sedation.

- This increased level of needed care must be documented in the Outcomes Evaluation Tool sent to the Quality Office for each sedation case.
Respiratory failure/arrest

- Excessive sedation may cause respiratory failure/arrest
- Monitoring hallmarks include: decreased respiratory rate, decreased tidal volume, hypoxia, cyanosis, cardiac arrhythmias, restlessness, agitation and even seizures
- CO₂ retention can potentiate the respiratory depression of sedative drugs
- Treatment includes: OXYGEN, stimulation, assisted ventilation (mask/intubation), consideration of reversal agents, initiation of the rescue protocol
SA Airway Complications I

Aspiration

- Nausea, vomiting and decreased protective airway reflexes can occur during SA
- Pulmonary aspiration of gastric contents leads to a chemical pneumonitis
- Loose teeth may cause foreign body aspiration
- Treatment is supportive: supplemental O2, intubation-ventilation if required and patient placement in a monitored environment
- The risk may be decreased with administration of a non-particulate antacid (e.g. Bicitra) and gastric emptying therapy (metaclopramide) drugs immediately prior to sedation/analgesia or acid reducing drugs (proton pump inhibitors, ranitidine, etc.) six hours before
SA Airway Complications II

**Airway Obstruction**

- Sedation causes muscle relaxation, including oropharyngeal muscles, leading to airway collapse and lack of effective ventilation (inadequate tidal volume)

- Although the patient frequently “appears” to be breathing while chest and abdominal muscles contract there is little or no air movement. Monitor respiratory rate AND quality. If in doubt, look and feel (hand in front of mouth and nose to feel quantity of air movement)

- Treatment: halt procedure, lighten sedation, jaw thrust, oral/nasal airway, consider positive pressure ventilation
Laryngospasm

- Stimulation of the lower airway with secretions/foreign bodies can cause the vocal cords to close, allowing no ventilation. Patient may again “appear” to breathe due to abdominal/chest muscle contraction with no effective volume exchange.
- Treatment includes positive pressure ventilation and initiation of the rescue procedure.
Negative Pressure Pulmonary Edema

- During upper airway soft tissue obstruction or laryngospasm, the patient attempts to inspire with increasing force. This may expose the lung to excessive negative pressure forces, drawing fluid into the alveoli. Clinically, the patient presents with acute hypoxemia and non-cardiogenic pulmonary edema.

- Treatment includes: supplemental O2, fluid restriction, diuretics, and possibly mechanical ventilation with PEEP.
**Hypotension**

- 30% drop in baseline BP
- Causes include
  - Excessive sedation
  - Cardiac: pump/rhythm failure
  - Hypovolemia
  - Acidosis: respiratory/cardiac
  - Sepsis related to procedure
  - Drug reaction
- In healthy adults, initial treatment is volume resuscitation 10ml/kg balanced salt solution over 10 minutes
- Consult supervising MD
SA Cardiovascular Complications

- **Hypertension**
  - 30% increase in baseline BP
  - Causes include
    - Pain: sympathetic discharge
    - Respiratory depression: CO\textsubscript{2} retention
    - Fluid overload
    - Urinary retention
    - Drug related (LA with epi)
    - Anti-hypertensive drug withdrawal (NPO)
  
  - Treatment best addresses underlying etiology
  - If needed or if BP excessive (>200mmHg)
    pharmacologic control of BP
  - Consult supervising MD
SA Neurological Complications I

- Pressure on nerves due to positioning may cause neurologic deficits most commonly in the distribution of ulnar, median and peroneal nerves. Elderly, obese and diabetic patients are at higher risk of nerve injury.
- Any pre-existing neurologic deficits must be documented in the pre-sedation evaluation.
- Eye protective reflexes are decreased. Eyes must be protected and clear from foreign bodies to avoid corneal abrasions and blindness due to pressure.
Sedation-analgesia can cause nausea and vomiting. Patients at risk include those with:

- History of nausea, motion sickness or chemotherapy
- Younger patients and female patients
- Procedures on gonadal organs, pharynx, or vestibuloculcular system
Nausea and Vomiting

- Treatment may be therapeutic or prophylactic
  - Single therapy most effective - 5 HT3 blockers like granisetron (Kytril) 0.1mg or ondansetron (Zofran) 4 mg
  - Other antiemetics: phenothiazines (promethazine = phenergan, initial dose 12.5 mg), anticholinergics, butyrophenones, benzamides (Tigan), steroids
  - Gastric antiacids/prokinetics: H2 receptor blockers, proton pump inhibitors, metoclopramide, cisapride