**Neuroanesthesia**

CO2 is the most potent cerebral vasodilator  
Hyperventilation 🡪 decreased PaCO2 🡪 decreased CBV and CBF  
Goal: end tidal CO2 25-30mm Hg = PaCO2 30-35mm Hg  
Hypothermia: each 1 degree C drop in T reduces cerebral metabolic rate of oxygen (CMRO2) by 7%  
Reducing CMRO2 helps protect again ischemic injury  
Lowering the head increases arterial blood flow and increases ICP by impairing venous outflow

**Inhilational agents**- reduce cerebral metabolism (except nitrous oxide) by suppressing neuronal activity  
- disturb cerebral autoregulation and cause cerebral vasodilation which increases CBV and can increase ICP  
- cause cerebral vasodilation  
- may increase ICP  
- if given > 2 hours, increase CSF volume  
- affect intra-op EP monitoring  
- nitrous oxide  
 - potent vasodilator  
 - markedly increases cerebral blood flow  
 - contributes to post-operative nausea and vomiting  
 - solubility is ~34 x that of nitrogen  
 - when it comes out of solution, it can convert pneumocephalus t0 tension pneumocephalus  
 - risk of tension pneumocephalus may be reduced by filling cavity with fluid and turning off nitrous 10 minutes before dural closure  
 - may aggravate air embolism  
- halogenated agents (eg. Isoflurane)  
 - produce isoelectric EEG  
 - provide some degree of cerebral protection  
 - may increase CBF and ICP  
 - isofluorane  
 - Desflurane: cerebral vasodilator, increase CBF and ICP, decreases CMRO2  compensatory vasoconstriction  
 - Sevoflurane: mildly increases CBP and ICP, reduces CMRO2, mild neg inotrope (CO not as well maintained)

**Intravenous anesthetic agents**- propofol: decreases MAP and ICP  
- barbiturates: reduce CMRO2; produce dose-dependent EEG suppression; minimally affect EPs; most are anticonvulsant; myocardial   
 suppression and peripheral vasodilation can cause hypotension and compromise CPP; sodium thiopental (most common agent  
- etomidate (anesthetic and amnestic but not analgesic); may produce myoclonus which may be confused with seizures  
- ketamine (dissociative anesthesia)  
- Propofol: may be used for induction and continuous infusion during total IV anesthesia (TIVA); causes dose dependent decrease in MAP and   
 ICP  
  
**Narcotics**- slow EEG, but will not produce isoelectric tracing  
- dose-dependent respiratory depression (can cause hypoventilation which can increase ICP)  
- morphine: does not significantly cross the BBB  
- fentanyl: crosses the BBB; reduces cerebral metabolism/CBV/ICP  
- remifentanil  
- increase CSF absorption  
- dose-dependent respiratory depression

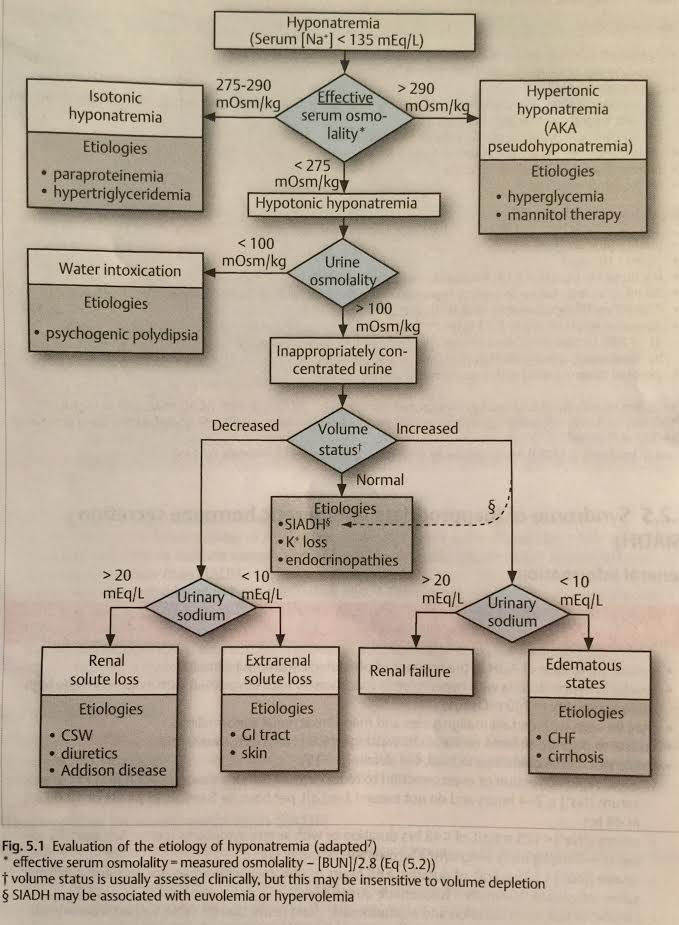
**Miscellaneous**  
- Dexmedetomidine (Precedex)  
 - sedation without losing neuro exam  
 - alpha-2-adrenergic receptor agonist  
  
**Paralytics for intubation**- Succinylcholine (depolarizing agent)  
 - 1-1.5 mg/kg (20mg/cc) – 3.5-5 cc for a 70kg male  
 - onset 60-90 seconds  
 - duration 3-10 minutes (may repeat x 1)  
 - may transiently increase ICP  
- Rocuronium (non-depolarizing)  
- Vecuronium (not approved for rapid sequence intubation)

**Anesthesia and IOM**  
- total intravenous anesthesia (TIVA) is ideal  
- if inhalational anesthesia is used, use < 1 MAC (minimal alveolar concentration), ideally < 0.5 MAC  
- SSEPs can be affected by hyper- and hypo-thermia and changes in BP  
- All volatile agents produce dose-dependent reduction in SSEP peak amplitude and increase peak latency  
- non-depolarizing agents have little effect on IOM  
- propofol has mild effect  
- benzodiazepines have mild-to-moderate effect  
- SSEP can be affected by hyper/hypothermia and BP changes  
- AED do not affect SSEP  
  
**Malignant hyperthermia**

- hypermetabolic state of skeletal muscle by block of Ca re-entry into sarcoplasmic reticulum  
- multifactorial genetic predisposition  
- total body O2 consumption increases 2-3 times  
- 50% had previous anesthesia without MH  
- associated with halogenated agents and succinylcholine  
- 30% mortality  
- presentation:  
 - fever > 1050  
 - increase in ETCO2  
 - muscle rigidity (may involve masseters which leads to difficulty intubating)  
 - tachycardia  
 - brown urine  
- progression: DIC, metabolic acidosis, decreasing PO2, pulmonary edema, elevated T (1130F), limb rigidity, rhabdomyolysis  
- terminal: hypotension, bradycardia, cardiac arrest  
- treatment:   
 - eliminate offending agents (stop OR, d/c agent, change tubing on anesthesia machine)   
 - **dantrolene** 2.5 mg/kg IV (up to 10 mg/kg)  
 - hyperventilation with 100% O2  
 - cooling blanket  
 - bicarbonate 1-2mEq/kg for acidosis  
 - IV insulin and glucose to lower potassium  
 - procainamide for arrhythmias  
 - diuresis  
- prevention:   
 - identify at risk patients (muscle biopsy)  
 - family history  
 - pts with heavy musculature, Duchenne MD, scoliosis  
 - pts with masseter spasm following succinylcholine  
 - in pts at risk avoid succinylcholine and use non-halogenated anesthetics   
 - prophylactic oral dantrolene: 4-8mg/kg/day for 1-2 days (last dose 2 hours before anesthesia)  
- any relative with syndrome puts patient at risk  
- may be inherited as an AD disorder  
- prophylactic oral dantrolene (4-8 mg/kg/day) may be given for 1-2 days before anesthesia  
- only reliable test: 4 cm viable muscle biopsy

**Sodium homeostasis and osmolality**  
  
normal serum osmolality: 285-295 mOsm/L [> 320 mOsm/L: risk of renal failure]  
  
**Hyponatremia**  
- serum Na < 135 mEq/L  
- common etiologies:   
 - SIADH (hypotonic hyponatremia)  
 - serum osm< 275 mOsm/L  
 - inappropriately high urinary concentration  
 - euvolemia or hypervolemia  
 - etiologies: tumor (small-cell cancer, lymphoma), infection, hypothyroid, adrenal insufficiency, stress, AIP, …  
 - cerebral salt wasting  
 - inappropriate natriuresis with volume depletion  
 - treated with volume replacement and sodium  
 - symptoms may be exacerbated by fluid restriction  
 - extracellular fluid volume depletion due to renal sodium loss   
 - urinary Na > 20 mEq/L  
 - renal failure  
 - pseudohyponatremia  
- work-up:   
 - serum Na  
 - serum osmolarity  
 - urine osmolarity  
 - clinical assessment of volume status  
 - urinary Na  
 - TSH (r/o hypothyroidism)  
- risk of overly rapid correction: osmotic demyelination (central pontine myelinolysis)

**Evaluation of hyponatremia**

****  
  
- Na < 135 mEq/L  
- measure serum osmolality to r/o pseudohyponatremia  
- volume status differentiates SIADH and CSW  
 - weight/edema/dry mucous membranes/loss of skin turgor/orthostatic hypotension  
- urinary Na  
- determine duration of hyponatremia

**Syndrome of inappropriate antidiuretic hormone secretion (SIADH)**- most common cause of euvolemic hyponatremia  
- the associated hyponatremia is a result of excess water, not a deficiency of Na+  
- hyponatremia and hypo-osmolality resulting from inappropriate, continued secretion or action of the hormone ADH despite normal or   
 increased plasma volume, which results in impaired water excretion  
- release of ADH in the absence of physiologic (osmotic) stimuli  
- ADH promotes reabsorption of water from the collecting duct; does not affect Na reabsorption; causes arteriolar vasoconstriction (↑BP)  
- excess water retention in the face of hyponatremia  
- rapid and complete recovery when drug-induced SIADH if offending agent is withdrawn  
- results in hyponatremia with hypervolemia (occasionally euvolemia) with inappropriately high urine osmolality (>100 mOsm/L)  
- serum osmolality < 275 mOsm/kg  
- urine osmolality > 100 mOsm/kg  
- increased volume status or euvolemia  
- urinary Na > 40 mEq/L with normal Na intake  
- normal thyroid and adrenal function  
- may be seen with certain malignancies and many intracranial abnormalities (CXR to r/o lung etiology)  
- critical to distinguish from cerebral salt wasting which produces hypovolemia  
- hallmarks:   
 - hyponatremia with hypo-osmolality  
 - inappropriately elevated urine osmolality (>100 mOsm/kg) – continued renal excretion of Na  
 - decreased serum osmolality in a euvolemic patient  
 - absence of clinical evidence of volume depletion (normal skin turgor, and blood pressure)  
 - absence of other causes of hyponatremia (adrenal insufficiency, hypothyroidism, cardiac failure, pituitary insufficiency, renal disease   
 with salt wasting, hepatic disease, drugs that impair renal water excretion)  
 - correction of hyponatremia by fluid restriction  
- treatment:  
 - avoid rapid correction of hyponatremia  
 - do not exceed 1 mEq/L per hour, or 12 mEq/L per 24 hours, or 18 mEq/L in 48 hours of correction  
 - check Na Q4H  
 - if refractory, **demeclocycline** (tetracycline abx that antagonizes ADH) 300-600 mg PO BID  
- if hyponatremia is severe (< 125 mEq/L) and the patient is symptomatic (lethargy, coma, seizures), initiate aggressive treatment:   
 - 3% NaCl and furosemide 20 mg IV QD  
- drugs that stimulate arginine vasopressin release and influence SIADH:  
 - acetylcholine  
 - antineoplastic agents: cyclophosphamide, vincristine, vinblastine  
 - barbiturates  
 - bromocriptine  
 - dibenzazepines (carbamazepine, oxcarbazepine)  
 - Haldol  
 - TCA (amitriptyline)  
 - SSRI (sertraline, fluoxetine, paroxetine)

**Cerebral salt wasting**  
- renal loss of Na due to intracranial disease  
- there is also usually hypovolemia  
- treatment with fluid restriction could exacerbate condition (especially in the setting of SAH)  
- laboratory tests may be identical with SIADH (serum and urine osmolalities and electrolytes)  
- treatment: hydrate patient with 0.9% NaCl (3% NaCl in severe cases)  
- do not give furosemide

**Hypernatremia**- serum Na > 150 mEq/L  
- most often seen in the setting of DI  
- calculate free water deficit and replace   
- due to low levels of ADH  
- high output of dilute urine (SG < 1.003)  
- normal or high serum osmolality  
- danger of severe dehydration if not managed carefully  
- central/neurogenic versus nephrogenic DI  
 - central/neurogenic DI: subnormal levels of ADH caused by hypothalamic-pituitary axis dysfunction  
 - nephrogenic DI: resistance of the kidney to normal or supra-normal levels of ADH

**Diabetes insipidus**  
- diagnosis: urine output > 250 cc/hr, above normal Na level, dilute urine (SpGr < 1.003), normal adrenal function  
- treatment: encourage patients to drink when thirsty, ddAVP 0.1 mg PO BID (0.1-0.8 mg QD)  
 - ddAVP doses: 0.1 mg PO BID, 2-4 mcg IV/SC BID, 10-40 mcg intranasal divided BID

**General neurocritical care**  
  
**Blood pressure control:**- contributes to CPP  
- decrease when working around aneurysm  
- increase to enhance collateral circulation during temporary clipping  
- MAP = [2DBP + SBP] / 3  
  
**Nicardipine** (calcium channel blocker)  
- does not require an arterial line  
- does not raise ICP or reduce heart rate  
- start at 5 mg/hr IV  
- increase by 2.5 mg/hr every 5-15 minutes up to a maximum of 15 mg/hr

**Labetalol:** 100 mg po BID (up to 2400 mg/day)  
**Enalapril (Vasotec)**: 1.25 mg IV over 5 minutes (increase up to 5 mg Q6H prn)  
**Propranolol:** used to counteract tachycardia with vasodilators, 1-10 mg slow IVP, then 3 mg/hr  
  
**Plasma expanders in shock**:  
Crystalloids: normal saline (less tendency to promote cerebral edema)  
Colloids: repeated administration may increase PT/PTT  
Blood products: expensive with risk of transmissible diseases or tranfusion reaction  
  
**Dopamine**: start with 2-5 µg/kg/min and titrate up to > 10  
**Dobutamine:** 2.5-10 µg/kg/min  
**Phenylephrine** (Neo-Synephrine): pure alpha sympathomimetic; useful in hypotension associated with tachycardia  
 - avoid in spinal cord injury  
 - 100-180 µg/min  
**Levophed**: 8-12 µg/min; direct beta stimulation

**Prophylaxis for stress ulcers**: Omeprazole 20-40 mg PO QD, Pantoprazole 40 mg PO QD  
 - Lansoprazole (Prevacid) 15-30 mg PO QD   
 - found NOT to have an effect on drugs metabolized by P450 (phenytoin, warfarin, prednisone)

**Sedatives, Paralytics, Analgesics**

**SEDATIVES:**

**Remifentanil**  
- potency similar to fentanyl  
- lowers ICP  
 **Fentanyl**  
- 100 x more potent than morphine  
- lowers ICP  
- may persist longer than respiratory depression   
- 25-100 µg IVP

**Propofol**  
- sedative hypnotic  
- start at 5-10 µg/kg/min  
- side effect: Propofol infusion syndrome (metabolic acidosis, hyperkalemia, hepatomegaly, myocardial failure, rhabdomyolysis)

**Dexmedetomidine (Precedex)**  
- sedative and analgesic  
- reduces the risk of respiratory depression and amount or narcotic analgesic required  
- load with 1µg/kg IV over 10 minutes  
- maintenance: 0.2-1.0 µg/kg/hour  
- may cause significant bradycardia

**PARALYTICS**- paralyzed patients may still be conscious!!!- simultaneous use of sedation is required

**Succinylcholine**- ultra-short acting (duration = 5-10 minutes)  
- 0.6-1.1 mg/kg, may repeat x 1  
- depolarizing agent  
- may increase potassium  
- linked to malignant hyperthermia

**Rocuronium**- short-acting (duration = 20-35 minutes)  
- 0.6-1 mg/kg

**ANALGESICS**

**I. Non-opioid pain medication** **Nonsteroidal anti-inflammatory drugs** - aspirin  
 - ibuprofen  
 - ketorolac (Toradol) (IV): 30 mg IV/IM Q6H  
 **Acetaminophen**

**II. Opioid pain mediation  
 Agonists** - tramadol (Ultram): 50-100 mg Q4-6H prn  
 - hydrocodone (Vicodin): 5/500, 7.5/500, 10/500  
 - hydromorphone (Dilaudid) **Partial agonists  
 Mixed agonist/antagonists  
III. Adjuvant drugs** (to be used with above analgesics)  
 **Tricyclic antidepressants  
 Anticonvulsants  
 Caffeine  
 Hydroxyzine  
 Corticosteroids**

**Endocrinology**

- under normal conditions: zona fasciculata of the adrenal cortex secretes 15-25 mg/day of cortisol (aka hydrocortisone) and 1.5-4 mg/day of   
 corticosterone

corticotropin releasing hormone (CRH) in hypothalamus  
 ↓  
 adrenocorticotrophic hormone (ACTH) in pituitary  
 ↓  
 cortisol by the adrenal glands  
  
- primary adrenocortical insufficiency (**Addison’s disease**)  
 - glucocorticoids and mineralocorticoids must be replaced

- secondary adrenal insufficiency  
 - caused by deficient ACTH released by the pituitary  
 - mineralocorticoids are normal  
 - glucocorticoids need to be replaced  
 - hydrocortisone: 20 mg PO QAM and 10 mg PO QPM  
 - prednisone: 5 mg PO QAM and 2.5 mg PO QPM

- chronic steroid administration suppresses the hypothalamic-pituitary-adrenal axis (HPA axis) and leads to adrenal atrophy  
 - if exogenous steroids are abruptly stopped or acute illness occurs, adrenal insufficiency may occur  
 - **Addisonian crisis** may occur  
 - recovery of adrenal cortex lags behind the pituitary (ACTH levels increase before cortisol levels do)  
 - after a month or more of steroids, the HPA axis may be depressed for as long as one year  
 - measuring morning plasma cortisol may evaluate the degree of recovery of basal adrenocortical function, but does not assess   
 adequacy of stress response  
 - OK to d/c steroids without taper if used for less than 5-7 days

- symptoms of steroid withdrawal  
 - fatigue  
 - anorexia  
 - nausea  
 - orthostatic dizziness  
 - exacerbation of underlying condition for which steroids were used  
 - hypotension  
 - hypoglycemia  
 - Addisonian crisis  
  
- if withdrawal occurs, backtrack and continue steroids at previous dose  
- then, after 2-4 weeks, a morning cortisol level is drawn (prior to AM dose) until the 8AM cortisol is > 10 µg/100 mL (indicating return of   
 baseline adrenal function)  
- when baseline adrenal function returns:  
 - daily steroids are stopped, but stress doses must still be given when needed  
 - during physiologic stress, the normal adrenal gland produces 250-300 mg hydrocortisone per day  
 - monthly cosyntropin stimulation tests are performed until normal  
 - aka ACTH stimulation test  
 - measures how well the adrenal glands respond to ACTH (normally produced by the pituitary)  
 1. Blood is drawn  
 2. Injection of ACTH is given (into shoulder IM)  
 3. Blood is drawn again after 30 or 60 minutes to check for cortisol levels   
 - a normal response is cortisol level higher than 18-20 µg/dL

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Steroid | Dose (mg) | Route | Dosing | Mineralocorticoid potency |
| Cortisone | 25 | PO, IM | 2/3 in AM, 1/3 in PM | 2+ |
| Hydrocortisone aka cortisol | 20 | PO | 2/3 in AM, 1/3 in PM | 2+ |
| Prednisone | 5 | PO | Divided BID-TID | 1+ |
| Methylprednisone | 4 | PO, IV, IM |  | 0 |
| Dexamethasone | 0.75 | PO, IV | Divided BID-QID | 0 |

🞴*Use of cortisone and cortisol chronically for conditions other than primary adrenocortical insufficiency (Addison’s disease) may lead to salt   
 and fluid retention, hypertension, and hypokalemia due to mineralocorticoid activity*  
Medrol Dosepak: 21 tabs of 4mg methylprednisolone – tapers dosage from 24 mg/d to 4 mg/d over six days

**Hypocortisolism (adrenal insufficiency)**- assess 8AM serum cortisol  
- Addisonian crisis (aka adrenal crisis)  
 - mental status changes, muscle weakness, postural hypotension or shock, hyperthermia (1050F)  
 - hyponatremia, hyperkalemia, hypoglycemia  
 - give fluids and,   
 - for glucocorticoid emergency: hydrocortisone 100 mg IV STAT and then 50 mg IV Q6H  
 - for mineralocorticoid emergency: fludrocortisone 0.2 mg PO QD  
 - methylprednisolone is NOT recommended for emergency treatment  
  
**Hypothyroidism**  
- plasma TSH will determine primary hypothyroidism (high TSH) from secondary hypothyroidism (low TSH)  
- replacement: Levothyroxine 75 µg PO QD  
- myxedema coma: emergency of hypothyroidism (50% mortality)  
 - altered mental status, unresponsiveness, hypotension, bradycardia, hyponatremia, hypoglycemia, hypothermia  
 - treatment: IV fluids for hypotension, hydrocortisone 300-400 mg IV over 24 hours, levothyroxine 500 µg IV

**Pituitary hormones**  
- adenohypophysis (evagination of oropharynx – Rathke’s pouch)  
 1. ACTH (aka corticotropin) – CRH from hypothalamus stimulates release  
 2. Prolactin – under inhibitory control from the hypothalamus (prolactin inhibitory factor)  
 3. Growth hormone – GHRH from hypothalamus stimulates release  
 - insulin-like growth factor-1 (IGF-1) secreted by the liver in response to GH is responsible for most of GH’s effects  
 4. Thyroid stimulating hormone (aka thyrotropin) – TRH from hypothalamus stimulates release and somatostatin inhibits  
 5. Gonadotropins (FSH and LH) – GnRH from hypothalamus stimulates release  
 6. Propiomelanocortin (POMC)

- neurohypophysis (evagination of floor of third ventricle)  
 1. ADH (antidiuretic hormone)  
 2. Oxytocin (milk letdown reflex in breastfeeding and uterine contraction during labor)

**Hematology**

**Cryoprecipitate**

**Prothrombin complex concentrate** (PCC) [Kcentra and others]  
- contains clotting factors II, VII, IX, and X, with protein C & S to prevent thrombosis  
- dose: 25 IU/kg  
  
Heparin reversal: 1mg protamine sulfate for every 100U of heparin administered

Fondaparinux: use in patients with HIT  
Argatroban: use in patients with HIT

Dabigatran (Pradaxa)  
 - direct thrombin inhibitor  
 - reversal: Praxbind  
  
Rivaroxaban (Xarelto)  
 - factor Xa inhibitor  
  
Apixaban (Eliquis)  
 - factor Xa inhibitor

Acute warfarin reversal for emergent neurosurgical procedures:   
 - PCC (25-100 units/kg over 10-60 minutes)  
 - 2 U FFP (15 mL/kg)   
 - Vitamin K 10-20 mg IV

Herbal supplements that affect platelet function:   
- Fish oil  
- Garlic  
- Ginkgo  
- Ginseng

Heparin dose: bolus = 80 U/kg/hour, maintenance = 18 U/kg/hour

**Coma**

**GCS** (T – designation for intubated patient)

- eye opening: 4 – spontaneous, 3 – to voice, 2 – to stimulation, 1 – no eye opening  
- motor: 6 – spontaneous, 5 – localizing, 4 – withdraws, 3 – decorticate, 2 – decerebrate, 1 – none   
- speech: 5 – oriented, 4 – confused, 3 – inappropriate, 2 – incomprehensible, 1 – none

**Approach to a comatose patient:**- cardiovascular stabilization (HR, BP) and airway

- STAT labs: glucose, ABG, BMP, CBC, coags  
- tox screen, ammonia, AED levels  
- neuro exam  
- emergency meds:   
 - **glucose** (25 mL of D50 IVP)  
 - **naloxone** 0.4 mg IVP  
 - **flumazenil** 0.2 mg IVP (up to 3 mg)  
 - **thiamine** 50-100 mg IVP

- STAT HCT  
- if meningitis is suspected, HCT then LP  
- if expeditious HCT is not possible, use small gauge needle (<22) to do LP

- 🞴do not miss vascular injury or status epilepticus leading to coma

- in pontine hemorrhage, pinpoint pupils appear because the loss of sympathetics leaves the parasympathetics unopposed  
- Kernohan’s phenomenon: contralateral cerebral peduncle may be compressed against the tentorial edge causing bilateral hemiplegia  
- anoxic coma: myoclonus is common  
 - poor prognosis if unreactive pupils and no motor response  
 - if present a few hours after cardiac arrest, there is an 80% risk of death of permanent vegetative state  
 - if present at 3 days, the risk is 100% of death or permanent vegetative state

**Brain death and organ donation**  
- Uniform determination of death act, 1980 (UDDA)

- Clinical criteria:  
 - absence of brainstem reflexes  
 - fixed pupils   
 - they are usually midpoint (4-6 mm)  
 - they may be dilated because the cervical sympathetic pathways remain intact  
 - absent corneal reflex  
 - absent oculocephalic “doll’s eyes” reflex – do not perform is C-spine is not cleared  
 - absent oculovestibular reflex (cold water calorics) – instill 60 cc ice water   
 - absent oropharyngeal (gag) reflex  
 - absent cough to bronchial suctioning  
 - no motor function (no response to deep CENTRAL pain)  
 - absence of complicating conditions  
 - use twitch monitor to r/o neuromuscular blockade   
 - neuromuscular blockade does not affect pupils because the iris lacks nicotinic receptors  
- core temperature > 360 C (96.80 F)  
- SBP > 100 mm Hg  
- no drugs that could simulate brain death  
- EtOH < 0.08%

- apnea test: PaCO2 > 60 mm Hg or PaCO2> 20 mm Hg over baseline  
 - CO2 is the most potent stimulus for respirations  
 - prior to test, bring the PaCO2 to 35-40 mm Hg (to shorten the test time and reduce risk of hypoxemia)  
 - pre-oxygenate for > 10 minutes and disconnect from ventilator, nasal cannula O2 during test  
 - may take 6 minutes to reach appropriate level of hypercapnia  
 - abort test if:   
 - SBP drops below 90 mm Hg  
 - O2 saturation drops < 80% for > 30 seconds  
 - significant cardiac arrhythmias occur  
 - the patient breathes  
- if patient is coming out of pentobarbital coma, wait until level is < 10 µg/mL  
- confirmation of brain death using ancillary confirmatory tests (EEG, angiography, cerebral radionuclide angiogram) is not required  
- a second exam may or may not be required based on local State law

**ICP management**  
- keep ICP < 20 mm Hg and CPP > 60 mm Hg  
- for elevated ICP:  
 - make sure that there is not a mass lesion that needs to be surgically addressed (HCT STAT)  
 - avoid hypoxemia and hypotension/hypertension  
 - aggressive control of fever  
 - positioning: HOB 30-450, keep head midline to avoid kinking of jugular veins  
 - light sedation: codeine 30-60 mg IM Q4H prn, lorazepam (Ativan) 1-2 mg IV Q4-6H prn  
 - make sure EVD is draining   
 - osmotic therapy  
 - mannitol: 0.25-1 g/kg (over 15 minutes) and furosemide 10-20 mg IV  
 - mannitol can be given as long as osmolarity is < 320 mOsm/L (risk = renal failure)  
 - can be given at 0.25-0.50 g/kg IV Q6H prn  
 - hypertonic saline: continuous 3% NaCl or 20 cc bolus of 23.4% NaCl  
 - hold osmotic therapy if serum osmolarity is > 320 mOsm/L  
 - hyperventilation to PaCO2 = 30-35 mm Hg (use acutely)  
 - STEROIDS HAVE NOT BEEN FOUND TO BE USEFUL – not useful in cytotoxic edema seen following trauma  
 - if ICP continues to remain elevated and HCT does not demonstrate any surgical pathology, obtain EEG to r/o subclinical status  
- high dose barbiturate therapy  
 - pentobarbital  
 - loading dose: 10 mg/kg IV over 30 minutes  
 - then 5 mg/kg Q1H x 3 doses  
 - maintenance: 1mg/kg/hr  
 - EEG to monitor   
 - aim for level 3-5 mg% (burst suppression on EEG)  
 - check level after loading dose and then daily  
 - if ICP < 20 mm Hg, continue treatment for 48 hours, then taper dose   
 - if ICP rises, backtrack  
 - will take 2 days off pentobarb for neuro exam to return  
 - level needs to be < 10 µg/mL (1 mg%) to perform a brain death exam  
- hypothermia – target temperature 32-350 (Level III evidence) [Arctic sun surface cooling device]  
  
Iodinated contrast with allergies or renal insufficiency  
- prednisone 50 mg PO at 24 hrs, 12 hrs, and 2 hrs before study  
- IV methylprednisolone can also be used (25 mg)  
- diphenhydramine 50 mg one hour before study  
- if emergency: hydrocortisone 100 mg IV, then scan within 2 hours  
  
Hounsfield units  
-1000 = air  
0 = water  
+5 = CSF  
+1000 = bone

**Multiple endocrine neoplasia**  
- usually (but not always) inherited – AD   
- MEN 1: parathyroid, pancreatic, pituitary (3Ps)  
- MEN2a: medullary thyroid cancer, pheochromocytoma, parathyroid  
- MEN 2b: medullary thyroid cancer, pheochromocytoma, neuromas

**Work-up for altered mental status**- **m**etabolic: Na, glucose, endocrine (Addison)  
- **i**nfectious: systemic (UTI, bacteremia), CNS (meningitis)  
- **n**eurologic: HCP/ICP, hemorrhage, seizure  
- **t**oxicology: EtOH, illicit drugs  
- **s**ystemic: hypoxia (PE, pneumonia), liver failure

**Propofol infusion syndrome**  
- normal dose is 5-10 mcg/kg/min for sedation  
- symptoms: bradycardia, hyperkalemia, metabolic acidosis, hepatomegaly, lipemia, myocardial failure, rhabdomyolysis (lactic acid), renal   
 failure, death   
- treatment: stop propofol gtt, supportive measures (in some cases, hemodialysis)  
- treat hyperkalemia with glucose and insulin

**Stevens-Johnson Syndrome**- fever, swelling, red/purplish painful rash that spreads and blisters with shedding of the skin  
- involves mucous membranes (mouth, eyes, nose)  
- causes: Phenytoin, acetazolamide, carbamazepine, ethosuximide, lamotrigine, allopurinol, penicillins  
- can be seen in HIV, HSV  
- treatment: stop offending agent, fluid replacement, transfer to burn center

**Neuroleptic malignant syndrome**  
- fever, sweating, hemodynamic instability, stupor, muscular rigidity, autonomic dysfunction  
- most cases develop within 2 weeks of initiating drug treatment with neuroleptic or antipsychotic drugs  
- causes: haldol, metoclopramide, abrupt discontinuation of levodopa  
- can occur in PD if drugs are abruptly discontinued  
- treatment: stop drug immediately, treat fever aggressively, muscle relaxant, dopaminergic drugs may be useful  
- anesthesia may be a risk to patients that have experienced NMS