Vesalius SCALpel™ : Anesthesia

Risk

ASA (American Society of Anesthesiologists) physiologic status grading
- I no systemic disease
- II mild/moderate, controlled
- III severe, poorly controlled (eg insulin dependent diabetes)
- IV life-threatening (eg intubated, end stage renal disease, class IV heart failure)
- V moribund
- VI organ donor
- subheading E = emergency
- 8w pre-op smoking cessation necessary for benefit

ACC/AHA clinical evaluation for non-cardiac surgery
- major risk (need cardiology consult):
  - unstable coronary syndrome: recent MI with ischemic risk
  - unstable/severe angina
  - decompensated CHF
  - significant arrhythmia
    - AV block
    - symptomatic ventricular arrhythmia
    - supraventricular arrhythmia with uncontrolled ventricular rate
    - severe valve disease
  - dobutamine provocative stress test to further stratify cardiac risk
- intermediate
  - mild angina, hx MI (4-6w post MI, base on risk stratification)
  - compensated/hisory of CHF
  - diabetes
  - renal disease
- minor risk
  - age
  - abnormal EKG
  - non-sinus arrhythmia
  - low functional capacity
  - hx stroke, incontrolled hypertension

surgical risk factors
- high risk/5% risk of MI
  - emergency major surgery in elderly
  - long procedure
  - large fluid shifts and/or major blood loss
  - peripheral vascular procedures
- intermediate/< 5%
  - carotid endarterectomy
  - head and neck surgery

intraperitoneal/thoracic
orthopedic
prostate

low risk/ < 1%
endoscopy
superficial
cataract
breast

Beta blockade pre/periop

decreases heart rate, ischemia
selective beta 1 block less bronchoconstriction
target heart rate 55-70
contraindicated with hi-grade conduction disease, AS, low ejection fraction
bradycardia most common adverse event
calcium channel blockers, NTG do not have same protective effect
AF w rapid vent response: beta block or Ca channel block
(SVT adenosine)

Preemptive local anesthesia:

prevent afferent pain excitation of CNS
dramatic change post-op pain profile
port site benefit, no benefit intraperitoneal
all local anesthetics block Na currents in nerve fibers
lidocaine
onset 2m
1% (10mg/cc), 3-5mg (0.5cc)/kg (7 with epi), 300mg max, redose 2h
no epi in digits, penis, nose, ears: risk of ischemia
IV for ventricular arrhythmias
side effects: bradycardia, decreased contractility, A-V block, ventricular arrhythmia, arrest, decreased respirations (respiratory acidosis, hypoxia), vasovagal, metallic taste, tremor, seizure
marcaine/bupivicaine
onset 5m, duration 2-4h, 2.5mg (1cc)/kg (max) of 0.25%,(3 w epi)
seizure, heart block, myocardial depression (more cardiotoxic than lidocaine)
local anesthetic CNS toxicity
dose dependent
tinnitus, dizziness, numbness tongue, circumoral, muscle twitch, visual disturbance, seizure, unconsciousness, coma
respiratory acidosis enhances CNS drug level, exacerbates seizure threashold
benzodiazepines increase CNS neuronal discharge threshold, protect against seizure
Rx: supportive measures, ABCs, assist ventilation w O_2
atropine for bradycardia, ephedrine for hypotension

Epidural

avoids higher dose narcotics, less GI dysfunction, mental status changes
useful in chest trauma, chest surgery, ileus, pancreatitis, intractable angina
contraindications: coagulopathy, bacteremia/sepsis (fever, elevated WBC alone not contraindication), local infection, hypovolemin, hemodynamic instability, cord hematoma/compression
epidural block sympathetic 2 levels above sensation, high (T3) causes bradycardia

Sedatives/benzodiazepines

diazepam: several active metabolites, mild sedative and amnestic
effects far beyond half life
medazolam/versed: rapid onset, short duration (5-10m), few metabolites, incremental dose
CNS depression, supress seizures
only benzodiazapene that can be used as infusion (the rest IV push)
lorazepam/ativan: slower onset, less prolonged effect, long term ICU sedation
flumazenil/romazicon: reverse sedative and psychomotor effects of benzodiazepines
little effect on respiratory depression

Total intravenous anesthesia (TIVA)

propofol/dexmedetomidine: sedative/hypnotic for induction and maintenance
extremely short half life, effects stop almost immediately after IV stopped
dexmedetomidine: little respiratory depression, rare allergic reaction
propofol: causes respiratory depression
ideal transition from long-term sedation in preparation for extubation
less post-op nausea and vomiting than inhalational
IV Rx for post-op N/V
dose-related hypotension, more in elderly (16-30% decrease MAP 1st 10m)
peripheral vasodilatation, myocardial depression
bradycardia 4% with opoids or long term beta blockers
transient, moderated by slow administration
caution with movement disorders (Parkinson's)
propofol infusion syndrome:
children, pts w acute neurologic illness, sepsis susceptible
rare, with prolonged hi dose
systemic cytokine catechol and glucocorticoid activation
cardiac and peripheral neuromuscular dysfunction
rapid, marked bradycardia to asystole
rhabdomyolysis, renal failure
limit dose to 5mg/kg/h, less than 48h
etomidate: minimal cardiovascular effects v other rapid onset induction agents
ketamine
IV rapis dissociative state, useful for induction
sympathetic effect: 25% increase BP, tachycardia

bronchodilatation
myocardial depression
contraindicated in head injury: increased cerebral blood flow and O2 consumption and metabolic rate
contraindicated with increased ICP
with max adrenergic tone in traumatic hypovolemia, ketamine can drop BP
halucinogen, emergence delerium

droperidol/inapsine
neuroleptic, antiemetic, antianxiety
contraindications: prolonged Q-T (enhances), not used with other CNS depressants

methohexital & thiopental
significant cardiovascular depression

opoids
demerol poor choice: metabolite normeperidine causes convulsions
long half life
lowers seizure threshold
cumulative after multiple doses, esp with renal failure
occasional use to decrease shivering
contraindicated with MAO inhibitors
morphine
1-2h half life
IV .08-.12mg/kg over 10-15m loading dose
maintenance 4-6mg/h
affects central and peripheral mu, kappa and delta receptors
arteriolar and venous dilatation (release of histamine from mast and other cells)
increases venous capacitance > arterial, relieve CHF
least lipophilic of opoids, slow onset, long duration
more effective than fentanyl and demerol which are lipid soluble and rapidly absorbed
more rostral spread in epidural, spinal
no incompatibility with local
cirrhosis, renal disease prolong half life
MS & phenothiazine can ppt pheo crisis
fentanyl/sublimaze: does not release histamine, choice in pts with bronchospasm
rapid onset and clearance, decreased nausea and vomiting
addiction: compulsive maladaptive behavior
naloxone/narcan: reverses opiod sedation and respiratory depression
tolerance: need for increasing doses, can occur without dependence
dependence: withdrawal symptoms (after tolerance develops)

MAC

minimal alveolar concentration of anesthesia at 1 atm. that prevents movement in 50% of pts in responsive to pain
threshold increased by chronic alcohol abuse, hyperthermia, cocaine
threshold decreased by hypoxia, acute intoxication, hypothermia

beta blockade makes hard to evaluate

Muscle relaxants

both kinds of muscle relaxants most common OR cause of anaphylactic reaction (latex 2nd)
- signs of anaphylaxis: bronchospasm, angioedema, cardiovascular collapse
- (antibiotics, contrast most common causes of anaphylaxis outside OR)

depolarizing
- succinyl choline (1mg/kg): 2 Ach molecules linked end to end
- mimics acetylcholine
- only depolarizing agent still in use
- short-acting depolarization at myoneural junction
  - fasciculation
  - irreversible
  - 5-7m return of spontaneous respiration
- side effects: bradycardia, hypotension, dysrrhythmias, hyperkalemia, myalgia, increased intracranial pressure, malignant hyperthermia
- increases K by 1mEq within minutes, can be profound with burns and maj trauma
- contraindicated in ophthalmologic surgery (increased intraocular pressure) and closed head injury (increses intracranial pressure)
- para/quadriplegics and burns have upregulated muscle endplate receptors: standard dose of succ can cause massive depolarization, acute life-threatening hyperkalemia
- contraindicated in end-stage liver disease (impaired metabolism prolongs duration), renal insufficiency (decreased excretion) make pt susceptible to rapid changes in K, arrhythmias

non-depolarizing
- longer onset than succ
- competitive inhibition of Ach
  - reversed by acetylcholinesterase inhibitors
- short acting: mivacuronium (histamine release)
- intermediate: roncuronium, cisatrecuronium, vecuronium
  - usual agents for intubation
  - effects last 30-45m
- long acting: pavulon, curare
- neostigmine: anticholinesterase, decreased breakdown of ach, reverse block

General anesthesia

loss of consciousness

shorter acting agents are less lipid-soluble
- isoflurane, desflurane, sevoflurane (less airway irritation)

N_2O decreases necessary dose of second agent decreasitg toxicity and facilitating emergence
- contraindications: small bowel obstruction, pneumothorax

diffuses into closed spaces faster than N diffuses out
halothane and enflurane replaced by newer agents with less side effects
halothane sensitizes myocardium to catechols, rare fulmanent hepatitis (middle aged obese women)
enflurane: nephrotoxic fluoride ion, brain toxicity, seizure

Pneumoperitoneum/laparoscopic surgery

increase mean arterial pressure (sympathetic response), systemic vascular resistance, afterload, decrease cardiac filling (decreased cardiac index), tachycardia
CO_2 hypercarbia, increased $ETCO_2$: increase minute ventilation
air or CO_2 embolus put pt in left lateral decubitus
bone is the largest CO_2 reservoir
rapid pneumoperitoneum can cause vasovagal effect: atropine
increased pressure causes decreased femoral venous flow, decreased renal flow and GFR
IVC flow is not affected until pressure of 20mm
reverse Trendelenberg: decreased venous return decreases cardiac output
load pts with fluid (1-2L) to prevent asystole (especially obese)
effects accentuated with beta blockade, ace inhibitors, renin-angiotensin block
CO_2 absorption increases cerebral blood flow, cerebral pressure (risk in head injury)
lung: FRC & compliance decreased
heat does not trigger visceral pain
intraperitoneal local little effect, beneficial @ port sites

Malignant hyperthermia

autosomal dominant, several mutations
muscle membrane disorder
ryantodine receptor (calcium release channel) abnormality
abnormal function of calcium release channel in sarcoplasmic reticulum
massive build-up of calcium in myoplasm
succinylcholine: generalized skeletal muscle contraction
potent halogenated inhalational agents trigger: halothane, isofluorane, devofluorane, desfluorane
change integrity of myolemma
other triggers: infectious disease, neuroleptics, severe exercise in heat
not triggered by local or spinal, propofol or narcotics
manifestations:
early sign masseter contraction
abrupt increase end tidal CO_2, hot anesthesia cannister early
signs in order of occurrence: increased ET CO_2, then tachycardia, then PVCs
violent sustained muscle contraction
unexpected cardiac arrest or arrhythmia (PVCs) after induction (increased Ca^{++}, K^{+})
increased temp, arrhythmia (tachycardia), acidosis, muscle rigidity, increase creatinine kinase, myogolbinuria

mixed respiratory and metabolic acidosis
similar to heat stroke, neuroleptic malignant syndrome
confirm with muscle biopsy, caffeine-halothane contraction test
aggressive Rx with dantrolene, reduces mortality from 70-5%
1-2mg/kg doses IV push repeated up to total 10mg/kg
dantrolene causes muscle relaxation beyond the myoneural junction, possibly interfering with the release of Ca^{++} from the sarcoplasmic reticulum
dantrolene causes muscle weakness, GI upset
aggressive cooling
pretreat with dantrolene if suspect

Antiemetics

ondansteron: serotonin receptor antagonist, non-sedating, few side effects
droperidol/butyrophenone: effective, cheap; Torsades, Q-T prolongation, need EKG monitor, anxiety
metoclopramide/reglan: limited prophylactic antiemetic, short half life, contraindicated in Parkinsons (acts on dopamine receptors)
promethazine: very sedating, effective

Hypothermia

shivering leads to increased O_2 consumption
L shift oxyhemoglobin dissociation curve
inhibits coagulation

Latex allergy

immediate massive vasodilation, bronchoconstriction
Rx: IV epi, steroid, antihistamine, D/C anesthesia, large volume reusucitation

Aspiration: lung injury pH < 2.5, volume > 0.5cc/kg
no food or cloudy drink 6-8h pre-op
no water 2h pre-op

pulse ox

measures only on pulsatile flow
NTG causes metHb
CO poisoning: need to measure direct arterial O_2 saturation