Hypertension

Hypertension is the most common circulatory derangement in adults in the US, affecting approximately 25% of adults. Hypertension is a significant risk factor for the development of ischemic heart disease, CHF and CVA, arterial aneurysm and ESRD.

ANESTHETIC CONSIDERATIONS:

1. Tight blood pressure regulation within 20% of patient's baseline to prevent end organ hypoperfusion secondary to altered renal and cerebral autogregulation
2. Concomitant diseases including CAD, CVD, DM, CHF – LVH, diastolic dysfunction, CRD
3. Anticipate exaggerated blood pressure responses (both hyper and hypotension) due to decreased vascular compliance
4. Perioperative optimization of anti hypertensive medications and continuation of beta blockers and alpha blockade
5. May have increased risk of postoperative myocardial reinfarction and incidence of neurologic complications in patients undergoing carotid endarterectomy (no evidence that isolated systolic or diastolic hypertension alone increases perioperative risk)

ANESTHETIC GOALS:

- Maintenance of blood pressure within 20% of patient's baseline
- Vigilant monitoring for end organ dysfunction
- Anticipation of extreme blood pressure variability

PriorityResuscitationHistoryExamLabsPfts(etc)Regional

PREOPERATIVE:
(Emergent, Urgent, Elective)

HISTORY

HPI
Past medical history including CAD/angina, CHF, DM, CVD, CRD
Allergies
Medications and possible interactions
Past anesthetic/surgical history
Functional capacity

PHYSICAL

Airway exam
Focused cardiorespiratory exam (rule out CHF; caution with diastolic dysfunction in LVH)

INVESTIGATIONS

Labs (CBCd, lytes, BUN, Cr INR, PTT, type and screen + crossmatch)
ECG – LVH, ischemic changes

OPTIMIZATION

- Ensure perioperative continuation of beta blockers, alpha blockers
- ACE inhibitors – discontinuation vs continuation (discontinue if high risk of blood loss/perioperative fluid shifts); can have resistant hypotension which can be effectively treated with vasopressin)
- Rule out active end organ dysfunction / hypertensive emergencies

ROOM PREPARATION/SETUP

- standard emergency drugs
- standard machine check (including suction, gas line supply, emergency O2 supply, inhalational agents, CO2 absorbant, circuit leak test)
- standard CAS monitors, consider 5 lead ECG +/- art line

INDUCTION:

- balanced induction with anticipation of hypotension with induction followed by hypertension upon direct laryngoscopy
- consider local anesthetics, beta blockade, vasodilator use or potent fast acting opioid to blunt hypertensive response to laryngoscopy in high risk patients
- limit laryngoscopy to <15s if possible

MAINTENANCE:

- caution with high sympathetic blocks
- volatile anesthetic agents are powerful antihypertensives and can produce dose dependent decreases in blood pressure (reflecting decreased SVR and myocardial depression)

EMERGENCE:

- anticipate postoperative hypertension
- adequate analgesia postoperatively to prevent post operative hypertension

Pathophysiology:

- Essential HTN: accounts for >95% of all cases, usually familial/inherited biochemical abnormailities including increased SNS activity, overproduction of Na retaining hormoenes and vasoconstrictors, high sodium intake, increased rennin secretion, deficiencies of endogenous vasodilators such as PG and NO, presence of DM/Oobesity. Final common pathway in pathophysiology of essential HTN is Na and water retention.
- Secondary HTN: has a demonstratable cause, accounts for <5% of all cases of systemic HTN. Includes: renovascular disease (RAS), hyperaldosteronism, coarctation of aorta, pheochromocytoma, cushings syndrome, renal parenchymal disease, PIH, drug induced

Final common pathway includes development of concentric LVH with subsequent increase in myocardial oxygen demand, myocardial ischemia, systolic dysfunction (secondary to myocardial ischemia), diastolic dysfunction (secondary to LVH) , possible atrial fibrillation secondary to LA enlargement

Predisposing and/or Associated Factors:

1. Familial predisposition
2. Diabetes
3. OSA (30% of HTN patients have OSA)
4. Dyslipidemia (strong association – 40% of patients with HTN have dyslipidemia)
5. Smoking (independent risk factor for CAD)
6. ETOH

Treatment:

Essential HTN:

1. Lifestyle modification – weight loss (most effective), moderation of ETOH intake (moderate ETOH decreases overall cardiovascular risk but excessive ETOH can cause resistance to antiHTN drugs), increased physical activity, maintenance of dietary Ca and K, moderation in Na intake (small effect, most prominent in patients with low rennin activity – elderly and African Americans)
2. Pharmacologic therapy:
    a. initial therapy – thiazide
    b. if CHF – ACE-I, aldosterone antagonist, B blocker, diuretic
    c. if CAD – ACE I, B blocker, CCB, diuretic
    d. Diabetes – ACEI, ARB, B blocker, CCB, Diuretic
    e. CKD – ACEI or ARB

Secondary HTN:

1. Surgical therapy: correction of RAS (angioplasty or direct repair), adrenalectomy (adrenal adenoma or phenochromocytoma)
2. Pharmacologic therapy: ACE-I and/or diuretics

Pathophysiology

- Systemic hypertension: systemic blood pressure greater than 140/90 mmHg or more on at least 2 occasions measured at least 1 to 2 weeks apart
- Prehypertension: SBP120-39/80-89
- Stage 1 HTN: 140-159/90-99
- Stage 2 HTN: >160/>100
- Hypertensive Crises (typically SBP>180/DBP>120)
    - Hypertensive Urgency: severely elevated BP without evidence of end organ damage; may still have headache, epistaxis or anxiety
    - Hypertensive Emergency: evidence of acute or ongoing target organ damange (encephalopathy [rare unless DBP>150], ICH, acute LV failure with pulmonary edema, UA, dissecting aortic aneurysm, MI, eclampsia, MAHA, renal insufficiency); requires prompt pharmacologic intervention to lower systemic blood pressure. Goal is to decrease MAP by 20% in 1st 60 min, then more gradually. Goal to 160/110 without hypoperfusion of end organs. (initial therapy: nitroprusside – watch for lactic acidosis secondary to cyanide toxicity)

References:

Stoeltings Anesthesia and Coexisting Disease, Chapter 5