## Perioperative Implications

### Preoperative Preparation

- Assess total and ionized $Ca^{2+}$ levels. No intervention for $Ca^{2+}$ level <12 mg/dL.
- Reduce serum total calcium to <14 mg/dL.
- For higher $Ca^{2+}$ levels use saline hydration, furosemide (rapid action), phosphate repletion, and consider calcitonin (acts in 1–2 h), mithramycin (acts in 6–12 h), cinacalcet, bisphosphonates, glucocorticoids, or hemodialysis.
- Consider $H_2$-receptor antagonists, nonparticulate antacids, and metoclopramide.

### Monitoring

- Routine; pay attention to changes in $QT_c$ interval ($QT_c$ by itself poorly correlated with ionized $Ca^{2+}$, but changes correlate).
- Intraop calcium and PTH level.

### Airway

- Possibility of pathologic fractures requires careful positioning for laryngoscopy.

### Preinduction/Induction

- No preferred agents or techniques.
- Avoid ketamine in pts with psychosis due to hypercalcemia.
- Hypovolemia can lead to hemodynamic instability if usual dose of induction agents is given.
- Minimally invasive procedures can be performed using local or regional anesthesia.

### Maintenance

- No preferred agents or techniques. Possibility of pathologic fractures requires careful positioning and padding of pressure points.
- Weakness may warrant smaller dose of nondepolarizers.

### Extubation

- Airway edema, surgical site hematoma, or recurrent laryngeal nerve injury may cause airway compromise.

### Adjuvants

- Response to NM blockers may be unpredictable if $Ca^{2+}$ level elevated.

## Anticipated Problems/Concerns

- Cardiac arrhythmias due to hypercalcemia
- Postop airway compromise secondary to bleeding or recurrent laryngeal nerve injury
- Pneumothorax secondary to surgical procedure
- Fluid overload and lyte abnormalities from too aggressive hydration

# Hypertension

Lee A. Fleisher

## Risk

- In USA about 77.9 million (1:3) adults have high BP.
- Incidence of Htn increases with advancing age. Half of people 60–69 y and three-quarters of people >70 y are affected.
- There is a continuous relationship between BP and the risk of CVD, including MI, heart failure, stroke, and kidney disease. For people 40–70 y, an increase of 20 mm Hg in systolic pressure or of 10 mm Hg in diastolic pressure doubles the risk of CVD across the entire range of BPs.

## Perioperative Risks

- BPs of up to 180/100 mm Hg are not independently associated with an increased risk of periop complications. Limited data suggest that BPs greater than this may be associated with an increased risk of such complications.
- In cardiac surgery, high preop pulse pressures have been associated with a threefold increase in periop mortality, an increased incidence of renal impairment, and reduced long-term survival.
- Isolated systolic Htn (>180 mm Hg, or marked increase to >200 mm Hg) has been associated with increased risk in noncardiac surgery in some studies.
- Intraop CV lability, especially hypotension, poses risks that may precipitate myocardial ischemia or predispose a pt to stroke.

## Worry About

- Markedly elevated BP (>180/110 mm Hg)
- Possible second-degree Htn
- Myocardial ischemia and MI
- CVA

## Overview

- Approx 95% of people with elevated BP have essential Htn: in 5% of people, an underlying cause for Htn can be identified.
- The aim of the long-term medical management of Htn is to reduce the burden of CV morbidity and mortality associated with chronically raised BP.
- The primary concern of the anesthetist in managing a hypertensive pt through the periop period is to prevent or curtail myocardial ischemia and labile BP that have been associated with anesthesia and surgery in Htn pts.
- Target-organ damage associated with Htn may of itself increase periop risk.
  - Ischemic heart disease
  - Heart failure
  - Cerebrovascular disease
  - Renal impairment
  - Peripheral vascular and aortic disease
- Recent JNC 8 recommendations for blood pressure treatment advocate lower blood pressure goals than previously

## Etiology

- Essential Htn appears to be a complex, mulitfactorial condition; a single cause has not been identified. Factors that play a role in the development of essential Htn include genetics, race (increased prevalence and severity in African Americans), age, sedentary lifestyle, obesity (in particular visceral obesity), sodium intake, alcohol intake, childhood influences (birth weight, BP tracking). Htn is part of the constellation of disorders that constitute the metabolic syndrome.
- Secondary Htn is found in approx 5% of people with raised BP. Identifiable causes of Htn incl sleep apnea, drug-induced Htn, chronic renal disease, renovascular disease, primary aldosteronism, Cushing syndrome, chronic steroid treatment, pheochromocytoma, and thyroid/parathyroid disease.
- Many pts who are found to have elevated BP at presentation for surgery will be found to not to be hypertensive when the BP is rechecked in a less stressful setting.

## Usual Treatment

- Lifestyle modification should be encouraged in all pts with elevated BP.
- In the general population above age 60, the current goal of pharmacologic treatment is to establish a goal of <150 mm Hg for systolic BP and <90 mm Hg for diastolic BP.
- BP reduction is more important than the choice of drug in the primary prevention of CV complications. There is evidence to support ACEIs, ARBs, calcium channel blockers, and thiazide diuretics as first-line therapy. Combination therapy is frequently required to achieve and sustain long-term BP control.
- Specific classes of antihypertensive drugs may provide better secondary prevention in pts with compelling indications for BP control based upon race.

## Assessment Points

| System | Effect | Assessment by Hx | PE | Test |
|---|---|---|---|---|
| CV | CAD | MI, angina, previous CABG or PCI | | ECG |
| | LVH/LVF | Dyspnea, orthopnea | Displaced apex beat<br>$S_3$, basal crepitations | CXR, ECHO |
| | Peripheral vascular/aortic disease | Claudication/rest pain | Rales<br>Pulses<br>Ankle brachial pressure index | Doppler<br>Angiography/CT angiography/MR angiography |
| METAB | Metabolic syndrome | | Central obesity | Fasting blood glucose<br>Triglycerides<br>HDL cholesterol |
| RENAL | Renal impairment | | | Creatinine<br>Estimated creatinine clearance<br>Microalbumin urine test |
| CNS | TIA/CVA | Hx of TIA/CVA | Neurologic signs<br>Carotid bruit | Doppler<br>CT/MRI<br>Angiography/CT angiography/MR angiography |

**Key References:** Lapage KG, Wouters PF: The patient with hypertension undergoing surgery, *Curr Opin Anaesthesiol* 29(3):397–402, 2016; James PA, Oparil S, Carter BL, et al.: 2014 evidence-based guideline for the management of high blood pressure in adults: report from the panel members appointed to the Eighth Joint National Committee (JNC 8), *J Am Med Assoc* 311(5):507–520, 2014.

## Perioperative Implications

### Preinduction/Induction/Maintenance

- There is no clear evidence to support deferring surgery or for acute management of BP in pts presenting with moderate Htn in the absence of CAD.
- Severe Htn (>180/110 mm Hg) confirmed on multiple readings should be controlled prior to surgery if the delay necessary to achieve this will not compromise the pt (especially if the pt has evidence of target-organ damage).
- Consider withholding ACEIs and ARBs for 12 h before surgery, as they may be associated with an increased incidence of intraop hypotension. If they are held, it is critical to restart them as soon as possible.
- Maintain treatment with other antihypertensive medications (in particular beta-blockers) unless the pt is hypotensive or has evidence of postural hypotension.
- Maintain euvolemia, especially in pts taking vasodilating drugs such as ACEIs or ARBs.

### Monitoring

- Standard monitoring.
- Frequent BP readings should be taken at times of potential CV instability, such as induction, in order to detect sudden changes in BP.
- Consider direct arterial pressure monitoring if surgery is proceeding in the face of severe Htn or a large fluid shift.
- Consider dynamic (e.g., pulse pressure variation) or static (CVP) monitoring if significant hypovolemia is suspected.

### General Anesthesia

- Pts may develop profound hypotension at induction and Htn at intubation.
- Consider a fluid preload prior to induction if relative hypovolemia is suspected.
- Consider preparing a short-acting vasopressor prior to induction.
- Consider the use of opiates or short-acting vasoactive drugs to control the response to intubation in pts with significant CVD.
- Aim to keep intraop BP within 20% of best estimate of preop BP with appropriate use of fluids and vasoactive drugs.
- No anesthetic maintenance technique has been demonstrated to be superior in this setting.

### Regional Anesthesia

- Risk of hypotension with neuroaxial blockade.
- Consider a fluid preload prior to neuroaxial blockade.
- Take BP readings every 1–2 min immediately after neuroaxial blockade if using noninvasive monitoring.
- As with general anesthesia, aim to keep intraop BP within 20% of best estimate of preop BP.

### Postoperative Period

- Resume normal antihypertensive treatment as soon as possible.
- If the pt is not on appropriate CVD prevention, make appropriate medical referrals to rectify this if possible.
- In some cases, parenteral treatment of BP may be required if the pt cannot take oral medications.
- Consider parenteral beta blockade if a pt who is chronically treated with a beta-blocker is unable to resume this treatment.

## Anticipated Problems/Concerns

- In pts with preexisting CVD, poorly controlled BP in the postop period may precipitate myocardial ischemia and cardiac complications.

# Hypertension, Uncontrolled With Cardiomyopathy

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## Risk

- 1.5 billion worldwide in 2014
- 70 million people in USA; approximately 1:3 people
- USA highest prevalence: African American
- Male = female

## Perioperative Risks

- Increased risk of MI and stroke
- Increased risk of CHF, ventricular hypertrophy, coronary artery disease, and atrial fibrillation
- Increased risk of cerebral hypoperfusion due to right shift of the cerebral blood flow autoregulation curve
- Increased risk of renal failure
- Increased blood loss
- Prolonged hospitalizations

## Overview

- Eighth Joint National Commission Hypertension Guidelines:
  - BP goal <150/90 mm Hg—anyone >60 y who does not have DM or CKD.
  - BP goal <140/90 mm Hg—anyone <60 y without major comorbidities and in pts >60 y with DM, CKD, or both.
  - Possibility of masked hypovolemia.
  - Silent myocardial ischemia may occur from supply-demand mismatches, even in absence of CAD.
  - May be forerunner of renal failure and/or stroke.
  - CHF may be presenting sign.
  - May develop LVH ± strain pattern on ECG.
  - May require >6 wk of treatment for regression of LVH.

## Etiology

- Idiopathic with genetic predisposition (>90%) and with up to 50% of global population
- Secondary hypertension due to thyroid, renal, and adrenal abnormalities
- Substance abuse (alcohol, cocaine, amphetamines)
- Valvular heart pathology (e.g., aortic insufficiency)
- High peripheral resistance is accelerated with time

## Usual Treatment

- Preload optimization (diuretics, venodilators)
- Afterload optimization (ACE inhibitors, angiotensin receptor blockers, CCBs, $alpha_1$-blockers, beta-blockers with $alpha_1$ activity, $alpha_2$ adrenomimetics, direct vasodilators, and sodium nitroprusside for emergencies)
- Drugs with negative inotropic effect (beta-blockers, calcium channel blockers)
- Atherosclerosis prophylaxis (statins)
- Surgical correction of secondary forms of hypertension