

INTRAOPERATIVE HYPERTENSION MANAGEMENT

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Hypertension occurs commonly during anaesthesia and is usually promptly and appropriately treated by anaesthetists. However, its recognition is dependent on correctly functioning and calibrated monitors. If it is not diagnosed and/or promptly corrected, it has the potential to cause significant morbidity and even mortality. When treatment is necessary, therapy should be individualized for the patient.

Intra operative hypertension often occurs in conjunction with one of the following events: during the induction of anesthesia; intraoperatively as associated with acute pain-induced sympathetic stimulation leading to vasoconstriction; in the early post anesthesia period, associated with pain induced sympathetic stimulation, hypothermia, hypoxia, or intravascular volume overload from excessive intraoperative fluid therapy; and in the 24 to 48 hours after postoperatively as fluid is mobilized from the extra vascular space. In addition, blood pressure elevation secondary to discontinuation of long-term antihypertensive medication may occur postoperatively.

Hypertensive events occur most commonly with carotid surgery, abdominal aortic surgery, peripheral vascular procedures, and intraperitoneal, or intrathoracic surgery. At least 25% of patients undergoing noncardiac surgery have hypertension prior to their surgical procedure; elevated blood pressures (systolic ≥ 170 mm Hg, diastolic ≥ 110 mm Hg) have been associated with complications such as myocardial ischemia.

BLOOD PRESSURE RESPONSE DURING SURGERY:

During surgery, patients with and without preexisting hypertension are likely to develop blood pressure elevations and tachycardia during the induction of anesthesia. Sympathetic activation during the induction of anesthesia can cause the blood pressure to rise by 20 to 30 mmHg and the heart rate to increase by 15 to 20 beats per minute in normotensive individuals. These responses may be more pronounced in patients with untreated hypertension in whom the systolic blood pressure can increase by 90 mmHg and heart rate by 40 beats per minute. Blood pressure and heart rate slowly increase as patients recover from the effects of anesthesia during the immediate postoperative period. Hypertensive individuals in particular may experience significant increases in these parameters

PERIOPERATIVE RISKS ASSOCIATED WITH HYPERTENSION

The mean arterial pressure tends to fall as the period of anesthesia progresses due to a variety of factors, including direct effects of the anesthetic, inhibition of the sympathetic nervous system, and loss of the baroreceptor reflex control of arterial pressure. These changes can result in episodes of intraoperative hypotension. Patients with preexisting hypertension are more likely to experience intraoperative blood pressure lability (either hypotension or hypertension) which may lead to myocardial ischemia.

PREOPERATIVE HYPERTENSION:

Preoperative hypertension increased the odds ratio for postoperative mortality to 3.8 times that of normotensives.

PATHOGENESIS OF HYPERTENSION Hypertension is usually caused by an increase in sympathetic activity. The factors determining blood pressure are cardiac output & systemic vascular resistance. $BP = CO \times SVR$. The majority of patients have an increase in SVR. Majority of hypertension is idiopathic {95%}, only 5% is secondary hypertension.

Perioperative risks depends on:

1. Level of hypertension
2. Duration of treatment
3. Degree of end organ damage
4. Type of surgery
5. Cause of hypertension

1.LEVEL OF BP: The “Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure” provides a new guideline for hypertension prevention and management.

CATEGORY	SBP	DBP
HIGH NORMAL	130-139	85-89
HT MILD STAGE I	140-159	90-99
MOD STAGE II	160-179	100-109
SEVERE STAGE III	≥ 180	≥ 110

The BP readings must be on two separate occasions after initial evaluation & exclude white coat HT. According to JNC7, systolic HT is a more important risk factor than diastolic HT because of LVH. Severe hypertension is associated with increased risk of BP liability, myocardial ischemia or infarction, pulmonary edema, arrhythmias, renal failure & neurologic damage.

2. DURATION OF TREATMENT:

Ideally BP should be controlled over weeks as rapid control might affect auto regulation of organs. Control might be lost with irregular drug intake.

3.DEGREE OF END ORGAN DAMAGE:

Uncontrolled hypertension is a minor risk factor in ACC/AHA 2007 guidelines. The risk of morbidity & mortality is more if there is a history of recent MI in < 6 months, recent CCF, recent stroke, higher creatinine levels. Autoregulation of cerebral & renal blood flow is shifted to the right in patients with untreated or uncontrolled hypertension. The general rule is

- a. 25% decrease in MAP reaches the lower limit of autoregulation

- b. 55% decrease in MAP reaches symptomatic brain hypoperfusion. Patients who already have CVD/ carotid artery disease may suffer watershed ischemia from relative hypoperfusion
- c. Treatment of HT significantly reduces the risk of stroke
- 4. **TYPE OF SURGERY:** Major surgeries like aortic, carotid, CABG, craniotomies are associated with increased risk.
- 5. **CAUSE OF HT:** Diagnosing the cause & treatment varies with the cause of HT

PREOP CAUSES HT	INTRAOP CAUSES HT	POSTOP CAUSES HT	
FAMILIAL	INADEQUATE PREMEDICATION	PAIN	
OBESITY	INADEQUATE ANALGESIA	EMERGENCE DELIRIUM	
SMOKING	DL SCOPY/ INTUBATION	HYPERCARBIA	
AGE	LIGHT PLANE	HYPOVENTILATION	
RECENT /UNDETECT SHT	POSITION	ETT INTOLERANCE	
ANXIETY	HYPERCARBIA	HYPERVOLEMIA	
PAIN	KINKING ETT	HYPOTHERMIA& SHIVERING	
EMERGENCY	HIGH CUFF PRESSURE	ANTI HT DRUGS WITHDRAWAL	
TRAUMA	TT PROLONGED	VASOPRESSORS IN EXCESS	
RENAL	HYPOXIA	FULL BLADDER	
TUMOR/HEAD INJURY	ENDOBONCHIAL		
PIH	PIH		
ENDOCRINE	PHEOCHROMOCYTOMA		
	THYROID STROM		
	MALIGNANT HYPERPYREXIA		
	ACC		
	MEASUREMENT ERROR		

INTRAOPERATIVE & POSTOPERATIVE HYPERTENSION

Acute elevations in blood pressure (>20%) in the intraoperative period are typically considered hypertensive emergencies. Postoperative hypertension (arbitrarily defined as systolic BP \geq 190 mm Hg and/or diastolic BP 100 mm Hg on 2 consecutive readings following surgery) may have significant adverse sequelae in both cardiac and noncardiac patients. Hypertension, and hypertensive crises, are very common in the early postoperative period and are related to increased sympathetic tone and vascular resistance. Postoperative hypertension often begins ~10–20 minutes after surgery and may last up to 4 hours. If left untreated, patients are at increased risk for bleeding, cerebrovascular events, and myocardial infarctions. The transient nature of postoperative hypertension, and the unique clinical factors present in the postoperative period, requires that this clinical syndrome be given particular attention.

CAUSES:

PATIENT	SURGICAL	ANESTHESIA
HYPERTENSIVE	EXCESS STIMULATION	INADEQUATE DEPTH
LABILE / WHITECOAT HT	ACC	INADEQUATE ANALGESIA
HEAD INJURY / ICT	CAROTID ENDARTERECTOMY	AWARENESS / HYPERVOLEMIA
PIH	GAS INSUFFLATION SURGERY	HYPOXIA – LARYNGO/BRONCHOSPASM
THYROID STORM / PHEOCHROMOCYTOMA	DISTENSION OF URINARY BLADDER	HYPERCAPNIA – HYPOVENTILATION, NA LIME EXHAUSTION
AUTONOMIC HYPERREFLEXIA	POSITION	AIRWAY PROBLEMS/ HYPOTHEMIA
		DRUGS / INADVERTENT/ OVER DOSE

IMPORTANCE OF INTRAOPERATIVE HYPERTENSION MANAGEMENT:

1. Risk factor CVS disease. Intraop HT leads to an increase in myocardial O₂ demand.
2. Causes end organ damage – intracranial he, stroke, sub endocardial ischemia, pulmonary edema,
3. Chronic HT –shift to right in cerebral /renal autoregulation
4. Chronic HT – severe hypotension after regional techniques

The approach to the treatment of perioperative hypertension is considerably different than the treatment of chronic hypertension. The initial approach to treatment is prevention. Hypertension that occurs in relation to tracheal intubation, surgical incision, and emergence from anesthesia may be treated with short-acting β -blockers, angiotensin-converting enzyme (ACE) inhibitors, calcium channel blockers, or vasodilators. Postoperative situations that may result in a hypertensive emergency include rebound hypertension after withdrawal of antihypertensive medications, hypertension resulting in bleeding from vascular surgery suture lines, hypertension associated with head trauma, and hypertension caused by acute catecholamine excess (eg, pheochromocytoma). An initial approach is to reverse precipitating factors (pain, hypervolemia, hypoxia, hypercarbia, and hypothermia).

Causes of spurious hypertension	Drug related causes
Calibration drift of invasive device Sphygmomanometer cuff herniation Calibration error of non-invasive device	Vasopressor administration: inadvertent, by anaesthetist, by a surgeon IV adrenaline with local anaesthetic
In all cases the error was detected by the use of a faulty sphygmomanometer to auscultate the blood pressure	Anaesthetic failure: failure to deliver volatile agent failure to deliver nitrous oxide Associated with epidural injection , interscalene blocks , cervical plexus block All due to malpositioning of the vaporiser.

Equipment related causes	Level of identification of cause using the algorithm
Ventilation problem: Stuck valve Hypoventilation Soda lime exhaustion Endobronchial intubation	V1 ventilation V2 vaporiser E Equipment R Review/Recheck D Drugs Unidentified*

PERIOPERATIVE RISK REDUCTION STRATEGY:

The GOAL is prevention of end organ damage. So preservation of vital organs perfusion by maintaining the lowest safety blood pressure.

1. Pre operative evaluation: history/examine/ adequacy of BP control/end organ damage

2. Methods to reduce perioperative risk: vigilant monitoring methods individualized

- a. Adequate control of BP,
- b. Anti hypertensives to attenuate hemodynamic responses,
- c. Hydration
- d. choice of agents with minimal hemodynamic changes
- e. analgesics,
- f. avoid hypoxia,hypercarbia
- g. laryngoscopy; limit time /iv opioids/ volatiles/topical& iv lidocaine/blockers
- h. well balanced anesthesia
- i. parenteral medications: $\alpha\beta$ blockers,, α blockers, vasodilators,CCB
- j. techniques: regional/ general/RA+GA/LA/TIVA

3. Postoperative care: Avoid hypothermia, hypoxia, hypercarbia,

Good Analgesics, maintain normovolemia

INTRAOPERATIVE HYPERTENSION: PNEUMOPERITONEUM

The triggering factor for catecholamine release in absence of major surgical stimuli or visceral dissection is due to mechanical effect of pneumoperitoneum.

Pneumoperitoneum : intra abdominal pressure > 15mmHg leads to \uparrow SVR, \downarrow venous return, \downarrow myocardial performance, \downarrow in cardiac output. \uparrow in SVR may increase myocardial oxygen demand, \uparrow CBF&ICT \downarrow HBF,RBF,Urine output,femoral blood flow - \uparrow DVT

Head down position - \uparrow central blood volume& pressure , \uparrow cardiac output

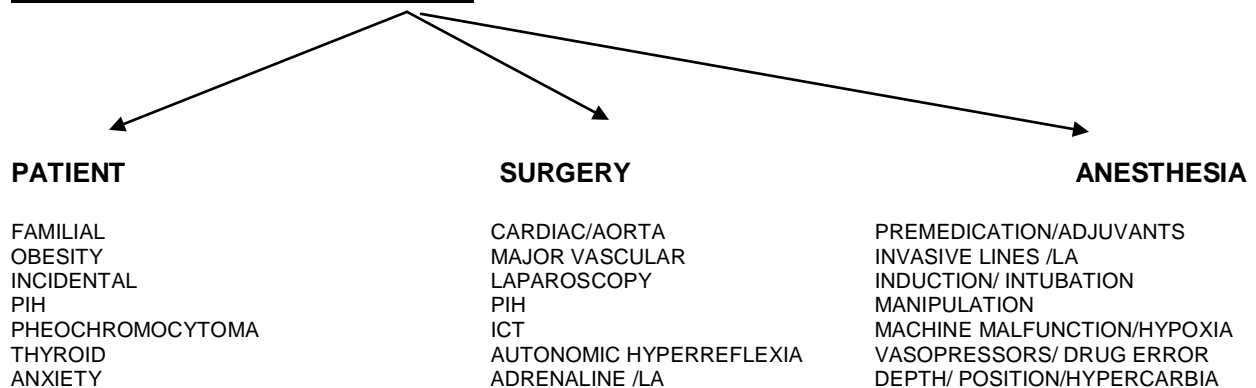
Treatment: pneumoperitoneum stimulates sympathetic response, \uparrow BP,HR.

1. Increase the depth
2. Propofol / NTG infusion
3. Esmolol

Alternate methods: 1. Gasless laparoscopy [apneumotic]
2. Helium laparoscopy

Clinical situation	Mechanism of action	Drug of choice
Severe acute hypertension	Nitric oxide donor	Sodium nitroprusside
Hypertension plus ischaemia	Nitric oxide donor	Nitroglycerine infusion
Hypertension plus tachycardia and ischaemia	β -Blocker	Esmolol, bolus or infusion
	α - and β -blocker	Labetalol, bolus or infusion
Hypertension plus heart failure	ACE inhibitor, inodilator, vasodilator	ACE inhibitor
		Dobutamine, dopexamine
Hypertension without cardiac complication	Vasodilator	Hydralazine, phentolamine
		Nifedipine, nicardipine
Hypertension phaeochromocytoma	Vasodilator, α - and β -blocker	Phentolamine
		Labetalol, doxazo, terazo, prazo

INTRAOPERATIVE HYPERTENSION:



PHARMACOTHERAPY:

The agent of choice in any particular situation will depend on the clinical presentation. The ideal agent for treatment of hypertensive emergencies should be rapid acting, predictable and easily titrated, safe, inexpensive, and convenient. Preferred agents include labetalol, esmolol, nicardipine, and fenoldopam. Clonidine & ACE inhibitors are long acting and poorly titratable; however, these agents may be useful in the management of hypertensive urgencies. ACE inhibitors are contraindicated in pregnancy. A review of agents used in the management of perioperative hypertension, preferred conditions, and dosing is presented

<u>Agent</u>	<u>Comment</u>
Enalaprilat	Intravenous intermittent: 0.625–1.25 mg (lower dose if hyponatremia, possible volume depletion, concomitant diuretic therapy, or renal failure) over 5 min, then double at 4- to 6-h intervals until desired response, a single maximal dose of 1.25–5 mg (doses \geq 1.25 mg have not been of additional benefit, but doses \leq 5 mg have been given), toxicity, or a cumulative dose of 20 mg within a 24-h period; contraindicated in 2nd and 3rd trimester of pregnancy
Esmolol	Intravenous infusion: 250–500 μ g/kg/min for 1 min, followed by a 50–100 μ g/kg/min infusion for 4 min, then titrate using same sequence (ie, with bolus before each rate increase) until desired response, a maximal dose of 300 μ g/kg/min, or toxicity
Fenoldopam	Intravenous intermittent: 0.1 μ g/kg/min initially, then titrate in 0.1 μ g/kg/min increments every 15 min until desired response, a maximal dose of 1.6 μ g/kg/min, or toxicity Hydralazine Intravenous intermittent: 3–20 mg (the lower end of the dosing range is preferred in the immediate perioperative period and in patients with renal failure) slow IV push every 20–60 min
Labetalol	Intravenous intermittent: 20 mg over 2 min, then double at 10 min intervals until desired response, a single maximal dose of 80 mg, toxicity, or a cumulative dose of 300 mg/d Intravenous infusion: 2 mg/min initially, then titrate in 2 mg increments every 10 min until response, toxicity, or a cumulative dose of 300 mg/24-h Nicardipine Intravenous infusion: 5 mg/h initially, then titrate dose by 2.5 mg/h increments every 5–15 min until desired response, a maximal dose of 15 mg/h, or toxicity
Nitroglycerin	Intravenous infusion: 5 μ g/min initially, then titrate in 5 μ g/min increments (may use 10 to 20 μ g/min increments when doses $>$ 20 μ g/min) every 3–5 min until desired response or toxicity; no absolute dosing limit, but the risk of hypotension increases with doses $>$ 200 μ g/min; therefore, alternative therapy should be considered
Nitroprusside	Intravenous infusion: 0.25–0.5 μ g/kg/min initially, then titrate dose every 1–2 min until desired response, a maximal dose of 10 μ g/kg/min (limit to duration $<$ 10 min), or toxicity.

AGENT	CONDITIONS	DOSE
[ACE INHIBITOR] ENALAPRIL	CCF	1.25 mg IV /5mts /6hrs titrated by increments of 1.25 mg at 12-24 hrs intervals to maximum of 5mg /6hrs
[LOOP DIURETICS] FRUSEMIDE	CCF /FLUID OVERLOAD	10-40 mg IV watch urine output/volume load
β - BLOCKERS ESMOLOL	SHT /TACHYCARDIA/ ANXIETY	200-500 μ g /kg in 1 mt then 50-100 μ g /kg/mt with maximum of 300 μ g/kg/mt
α - β BLOCKERS LABETALOL	ACUTEMI/PREECLAMPSIA/AORTIC DISSECTION/STROKE	20mg bolus then 20-80mg bolus or infusion of 1-2 mg/mt.maximum 300mg/hr
DOPAMINE 1 AGONIST/FENOLDAPAM	ACUTE MI/ PUL EDEMA/RENAL FAILURE/ STROKE/SYMPATHETIC CRISIS	0.1 μ g/kg/mt increments 0.05 to maximum 1.6 μ g/kg/mt
NICARDIPINE DIHYDROPYRIDIN CCB	ACUTEMI/PREECLAMPSIA/ECLAMPSIA/STROKE/ SYMPATHETIC CRISIS/COCAINE OVER DOSE	5mg/hr increasing 2.5mg /hr to maximum of 15mg/hr
III GEN CCBCLEVIDIPINE	CCF/MI	Selective arteriolar dilator 1-2mts ultra short action time
HYDRALAZINE	PIH	12.5mg
GTN SNP	ANTI HYPERTENSIVE/ANTIANGINAL SNP-Emergency not responding to other drugs	5 μ g/min initially, then titrate in 5 μ g/min increments (may use 10 to 20 μ g/min increments when doses >20 μ g/min) every 3–5 min until dosing limit, [SNP 0.1 μ g/kg/mt]

TREATMENT STRATEGY:

SWIFT CHECK- COMPLETE COVER ABCD

CONFIRM THE HYPERTENSION

ASSESS DEPTH / IF SOS DEEPEN

RULE OUT VASOPRESSORS CAUSE

STOP SURGICAL STIMULUS & WATCH & CORRECT THE CAUSES [PNEUMOPERITONIUM]

DELIVERY OF DRUGS& ANESTHESIA RECHECK

APPROPRIATE DOSE OF OPIOIDS/ANALGESICS

ANTIHYPERTENSIVES CONSIDER

CONCLUSION

The goal of controlling perioperative hypertension is to protect organ function, and is currently recommended based on the assumption that the risk of complications will be reduced and outcomes improved. In general, the treatment goal should be based on the patient's preoperative BP. A conservative target would be approximately 10% above that baseline; however, a more aggressive approach to lowering BP may be warranted for patients at very high risk of bleeding or with severe heart failure who would benefit from afterload reduction. Careful monitoring of patient response to therapy, and adjustment of treatment, are paramount to safe and effective treatment of perioperative hypertension.

The choice of agent in specific cases should be determined by the clinical situation, the patient's characteristics, the setting of care, and the experience of the clinicians. A wide selection of available intravenous antihypertensive agents has provided the clinician with the ability to optimize therapy based on the specific needs of the situation and condition of the patient.

Early recognition of the cause with vigilant monitoring & therapeutic intervention alters the hemodynamic variation & reduces morbidity & mortality.

POINTS TO PONDER:

1. PREOP EVALUATION & PREPARATION
2. DRUGS REGULAR TRT
3. PROPER COUNSELLING
4. ANXIOLYSIS/ ADEQUATE PREMEDICATION
5. VIGILANT MONITORING
6. ADEQUATE HYDRATION
7. ADEQUATE ANALGESIA
8. POSITION
9. GASLESS METHODS
10. LESS VASOPRESSORS
11. INVASIVE LINES UNDER ANESTHESIA
12. MACHINES PROPERLY FUNCTIONING /SODA LIME/ CONNECTIONS
13. ADJUST THE DEPTH OF ANESTHESIA
14. INTERACTION
15. ANTI HYPERTENSIVES IN EMERGENCY

