Objectives

- Hyperkalemia
- Acute Kidney Injury
- Hypertensive Emergency
Hyperkalemia
Hyperkalemia

- **Frequency**
  - 2.6% of emergency visits and 3.5% of hospital admissions in Canada
  - US studies up to 8% of hospitalized patients
  - 7.7-73% in pts with CKD

- **Definition** (KI 2016;89:546-554)
  - Mild: 5.1-6.5 mmol/l
  - Moderate: 6-8 mmol/l
  - Severe: ≥ 6.5 mmol/l
Hyperkalemia DDx

- Pseudohyperkalemia
  - Blood drawing technique
  - Cooling of specimen, deterioration due to length of storage
  - Thrombocytosis
  - High WBC (<120,000 microL) from ALL
  - Hereditary (familial) forms of pseudohyperkalemia (rare)
Major Causes of Hyperkalemia: Increased K release from Cells

- Metabolic acidosis
- Insulin deficiency, hyperglycemia, hyperosmolality
- Increased tissue catabolism
- Beta blockers
- Exercise
- Hyperkalemic periodic paralysis
Major Causes of Hyperkalemia: Increased K release from Cells

- Overdose of digitalis
- Red cell transfusion
- Succinylcholine
- Arginine HCl
- Activators of ATP-dependent K channels:
  - Calcineurin inhibitors
  - Diazoxide
  - Minoxidil
  - Volatile anesthetics
Major Causes of Hyperkalemia: Reduced Urinary Potassium Excretion

- Reduced aldosterone secretion
- Reduced response to aldosterone
- Reduced distal sodium or water delivery
- Acute and chronic kidney disease
- Selective impairment in K secretion
- Gordon’s syndrome
- Ureterojejunostomy
Symptoms of Hyperkalemia

- Usually does not occur until $\geq 7\text{mEq/L}$
  - Lower if acute hyperkalemia
- Severe muscle weakness and paralysis
- Cardiac manifestations
- Reduced urinary acid excretion
Why it Happens

- Hyperkalemia impairs neuromuscular transmission
- Elevated extracellular potassium decreases the ratio, partially depolarizing the cell membranes
- Initially increases the membrane excitability
- Persistent depolarization decreases membrane excitability
Paralysis

- Ascending paralysis
  - Legs to trunk
  - Usually without cranial nerve weakness or loss of sphincter tone
  - Rare respiratory weakness
- Resolves with correction of hyperkalemia
Cardiac Manifestations

- ECG changes
  - Tall peaked T waves
  - Shortened QT interval
  - Lengthening of the PR interval
  - Increased QRS duration
  - Sine wave pattern

- Conduction abnormalities
  - RBBB, LBBB, Bifascicular block
  - Sinus bradycardia, slow idioventricular rhythms
  - Ventricular tachycardia, Vib
  - Asystole
Cardiac Manifestations

- Progression and severity of ECG changes do not correlate well with serum potassium level
- May actually have normal ECG despite elevated potassium
- More likely with rapid onset of hyperkalemia
- Don’t use ECG for monitoring
ECG Changes

ECG showing peaked T waves in hyperkalemia

A tall peaked and symmetrical T wave is the first change seen on the electrocardiogram (ECG) in a patient with hyperkalemia.
## ECG Changes in Hyperkalemia

<table>
<thead>
<tr>
<th>Approximate serum potassium (mmol/L)</th>
<th>ECG change</th>
</tr>
</thead>
<tbody>
<tr>
<td>~4</td>
<td>Normal</td>
</tr>
<tr>
<td>6–7</td>
<td>Peaked T waves</td>
</tr>
<tr>
<td>7–8</td>
<td>Flattened P wave, prolonged PR interval, depressed ST segment, peaked T wave</td>
</tr>
<tr>
<td>8–9</td>
<td>Atrial standstill, prolonged QRS duration, further peaking T waves</td>
</tr>
<tr>
<td>&gt;9</td>
<td>Sine-wave pattern</td>
</tr>
</tbody>
</table>

### Dyskalemia Flashcards
Differential Diagnosis of Peaked T waves

- **Ischemic**
  - Hyperacute phase of MI
  - Acute transient transmural ischemia (Prinzmetal’s angina)
  - Chronic (evolving) phase of MI

- **Nonischemic Causes**
  - Normal variants
  - Hyperkalemia
  - Acute hemopericardium
  - Cerebrovascular hemorrhage
  - LVH
  - LBBB
  - Acute pericarditis
Treatment of Hyperkalemia

- Urgency based on cause and presence or absence of symptoms/signs
- Marked tissue breakdown treat aggressively even if mild potassium level
- Indications to treat:
  - Hyperkalemia and ECG changes
  - Serum K 6.5-7meq/l
  - Less degrees of hyperkalemia with rapidly increasing serum potassium
Treatment of Hyperkalemia

- Treatment Approaches
  - Antagonizing the membrane actions of potassium
  - Driving extracellular potassium into the membrane
  - Removing excess potassium from the body
  - Measure serum potassium 1-2 hr after initiation of therapy
Calcium

- Antagonizes the membrane actions of hyperkalemia
- Begins within minutes, relatively short lived (30-60 minutes)
- Calcium Chloride vs. gluconate
  - CaCl $3 \times$ the concentration of elemental calcium
  - CaCl 5-10ml of 10% solution
  - Ca gluconate 10ml of 10% solution
Calcium and Digoxin

- Use calcium for same indication in treating hyperkalemia (ie widening QRS or loss of P waves)
- Use a dilute solution of 10mL of 10% of calcium gluconate in 100mL of 5% D5W over 20-30 minutes
- With digoxin toxicity-digoxin-specific antibody fragments
Promoting Uptake of Potassium by Cells

- Transiently lower serum potassium by pushing potassium into skeletal muscle
- Insulin
- Beta agonists
- Sodium bicarbonate
Promoting Uptake of Potassium by Cells: Insulin

- 10 U of regular insulin IV, 25g of D50
- 20 U 1 hour infusion
- Lowers potassium by 1 mmol/l in 1 hr
- Check glucose 1 hr after giving insulin
- Effect begins in 10-20 minutes
- Peaks at 30 to 60 minutes
- Lasts 4-6 hours
Promoting Uptake of Potassium by Cells: beta agonists

- Albuterol 10-20mg in 4 mL saline by nebulization over 10 minutes
  - 0.5mg IV infusion
- Terbutaline SC
- Peak effect 30 minutes (IV), 90 minutes (nebulization)
- Lowers serum potassium by 0.5 to 1.5meq/L
- Additive effect with insulin
- Avoid with active coronary disease
Promoting Uptake of Potassium by Cells: Sodium Bicarbonate

- Studies done in 1959, NaHCO3 infusion lowered by 2-3 mmol/l
- Beneficial in patients with metabolic acidosis
  - 2 mmol/l drop in K for a 10 mmol/l increase in serum bicarbonate
- Little effect in hemodialysis patients
- Not recommended as solitary therapy
Potassium Removal

- Diuretics
  - Loop or thiazide diuretics
- Fludrocortisone
- Cation exchange resins
- Dialysis
Cation Exchange Resins: Kayexalate

- Sodium Polystyrene Sulfonate
- First introduced in the 1950s
- Exchanges sodium for calcium, magnesium, ammonium, potassium
- Decreased K by 0.7-1.1 meq/l (N Engl J Med 1961;264:115-119)
- Onset of action 2-4hr
- Most effective in rectum
  - PO with sorbitol (15-30g, may repeat in 4-6hr)
  - Retention enema (50g in 150mL of water)
Cation Exchange Resins: Kayexalate

- Concern for bowel obstruction/fatal colonic perforation
  - Resin swells when it comes into contact with water
- Black Box Warning to avoid giving with sorbitol
- Retrospective study of 2194/123,391 treated with SPS in sorbitol
  - 0.14% developed colonic necrosis
  - Did not differ significantly from pts not receiving the drug (0.07%)
  - Number needed to harm 1395

Am J Kidney Dis 2012;60:409-416
Cation Exchange Resins: Kayexalate

- Do not use in:
  - Postoperative patients
  - Patients with an ileus or receiving opiates
  - Patients with large or small bowel obstruction
Cation Exchange Resins: Novel Agents: Patiromer

- Nonabsorbable synthetic polymer
- Smooth spherical beads
- Most effective in rectum
- Taken orally, increases fecal K in a dose-related fashion
  - 15 to 30 g/day increased daily fecal K by 15 to 20 mmols
- Adverse effects: hypomagnesemia, binds to PO medications
Cation Exchange Resins: Novel Agents: Patiromer

- Studies have been done in chronic outpatient setting in patients with CHF and spironolactone or CKD and RAAS blockers
- Dose 4.2g or 8.4g bid
- Dropped K+ 0.4-1mmol/L

Crit Care Med 2008;36(12)3246-3251;
Cation Exchange Resins: Novel Agents: ZS-9

- Sodium zirconium cyclosilicate
- Highly selective cation exchanger that binds K in exchange for sodium and hydrogen
- Works throughout the GI tract
- Suggests it will be effective in the management of acute hyperkalemia
  - 10g of ZS-9 in pts with K≥6mmol/l lowered K by 0.4 mmol/l at 1hr, 0.6mmol/l at 2hr, 0.7 mmol at 4hr

Cation Exchange Resins: Novel Agents: ZS-9

- Of note, has not been approved by the FDA yet
Hyperkalemia in Hemodialysis Patients

- 0.85 meq/L reduction with insulin & glucose
- 0.3 meq/L with albuterol
- No change with sodium bicarbonate
- 1.3 meq/L reduction with hemodialysis

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Dose</th>
<th>Route of administration</th>
<th>Onset of action</th>
<th>Duration of effect</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calcium</td>
<td>10 ml CaCl, 30 ml Ca gluc</td>
<td>IV</td>
<td>1-3 min</td>
<td>30-60 min</td>
</tr>
<tr>
<td>NaHCO3</td>
<td>50-250mL</td>
<td>IV</td>
<td>5-10 min</td>
<td>~2 hr</td>
</tr>
<tr>
<td>Insulin</td>
<td>10u Regular</td>
<td>IV</td>
<td>30 min</td>
<td>4-6 hr</td>
</tr>
<tr>
<td>Beta-2-agonist</td>
<td>10-20mg neb, 0.5mg/100mL D5W</td>
<td>Neb or IV</td>
<td>30 min</td>
<td>2-4 hr</td>
</tr>
<tr>
<td>Diuretics</td>
<td>40mg IV lasix</td>
<td>IV</td>
<td>Varies</td>
<td>Varies</td>
</tr>
<tr>
<td>Fludrocortisone</td>
<td>0.1 mg</td>
<td>PO</td>
<td>n/a</td>
<td>n/a</td>
</tr>
<tr>
<td>Cation exchange resins</td>
<td>20-50g</td>
<td>PO or PR</td>
<td>1-2 hr</td>
<td>4-6hr</td>
</tr>
<tr>
<td>Dialysis</td>
<td>n/a</td>
<td></td>
<td>Minutes</td>
<td>Duration of HD</td>
</tr>
</tbody>
</table>

Adapted from Nat Rev Nephrol 2014;10(11):63-662
Acute Kidney Injury
Acute Kidney Injury

- Important because increased risk for short and long term risk of death in AKI
- AKI diagnosis does not take into account the metabolic, endocrine, or immunological functions of the kidney
- Creatinine rise lags behind injury event
Acute Kidney Injury

- Diagnosis:
  - Increase in serum creatinine
  - Decrease in urine output

- Diagnostic criteria
  - RIFLE criteria 2004
  - AKIN criteria 2007
  - KDIGO 2012
KDIGO guidelines

- Increase in serum creatinine by ≥0.3mg/dl within 48hr
- Increase in serum creatinine to ≥1.5 times baseline
- Urine volume <0.5ml/kg/hr for 6hr
# KDIGO guideline

<table>
<thead>
<tr>
<th>AKI Stage</th>
<th>Serum creatinine criteria</th>
<th>Urine output criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>AKI I</td>
<td>Increase in Cr by $\geq 0.3$ mg/dl or $\geq 1.5 \times$ baseline</td>
<td>UOP $&lt; 0.5$ ml/kg/hr for 6-12 hr</td>
</tr>
<tr>
<td>AKI II</td>
<td>Increase in Cr by 2-2.9x baseline</td>
<td>UOP $&lt; 0.5$ ml/kg/hr for $\geq 12$ hr</td>
</tr>
<tr>
<td>AKI III</td>
<td>Increase in Cr by $\geq 3 \times$ from baseline or Cr $\geq 4$ or treatment with RRT</td>
<td>UOP $&lt; 0.3$ ml/kg/hr $\geq 24$ hr or anuria for $\geq 12$ hr</td>
</tr>
</tbody>
</table>

Acute Kidney Injury: Misdiagnosis causes

- Administration of a drug that interferes with tubular secretion of creatinine
- Reduced production of creatinine
- Ingestion of substances which lead to increased generation of creatinine independent of renal function
- Interference with analytical measurement of creatinine
- Fluid resuscitation or overload
- Extrinsic creatinine administration as a buffer in medications
AKI—Common Causes

- Sepsis
- Congestive heart failure
- Hemodynamic instability
- Hypovolemia
- Exposure to nephrotoxic agents
- Obstruction
- Acute parenchymal and glomerular diseases
AKI—ED work up

- Chemistries—including phosphorus
- Urinalysis—dipstick, microscopy
- Renal ultrasound
- If history indicates:
  - CK, myoglobin
  - Cardiac work-up
  - Cultures
  - ANA, anti-GBM, ANCA —esp pulmonary-renal syndrome patients
Complications requiring immediate attention

- Hyperkalemia
- Fluid overload/volume depletion
- Severe metabolic acidosis
- Signs of uremia
Management: Fluid assessment: Volume Depletion

- Fluid resuscitation
- Crystalloid
  - Avoid potassium containing solutions
  - Avoid sodium bicarbonate containing with acidosis
- 1-3 L
- Target physiologic endpoints
Management: Fluid assessment: Volume Overload

- Cardiorenal syndrome, AKI on CKD
- Diuretics
  - IV loop diuretics
- Dialysis
Management: Metabolic acidosis

- IV Bicarbonate
  - Not volume overloaded
  - Non-anion gap acidosis related to diarrhea
  - Severe organic acidosis while awaiting HD
  - Rhabdomyolysis
- Goal Serum bicarbonate between 20-22 mEq/L, pH >7.2
Management: Metabolic acidosis

- Dialysis
  - Volume overload, severe oligo-anuric AKI with pH <7.1
  - AKI and organic acidosis (lactic or ketoacidosis), pH <7.1
Management: Uremic symptoms

- Pericarditis
- Neuropathy
- Unexplained decline in mental status
- Dialysis
Future Diagnostic Tools

- AKI biomarkers
  - Help predict AKI and etiology of AKI
  - Not quite ready for prime time
  - Mainly used currently to look for AKI after CV surgery
Hypertensive Emergencies
Hypertension

- Very common - occurs in about 1/3 adults
- New guidelines (JNC 8) - have loosened up BP control to <150/90 in pt over 60yrs old
- Concerns that the new JNC-8 guidelines may increase risk for more presentations of hypertensive
- ~1-2% of patients with HTN will present with hypertensive emergency

Hypertensive Crisis

- Hypertensive Urgency
- Hypertensive Emergency
- Both with >180/>120
- Differentiated by end organ damage
  - Cerebrovascular
  - Cardiovascular
  - Pulmonary
  - Renovascular systems
Hypertensive Urgency

- Acute increase in BP
- Not associated with end organ damage
- Can have headache, epistaxis, shortness of breath
- Usually occurs in noncompliant patients or patients lost to follow up

Hypertensive Urgency

- Risk of therapy: hypotension and organ hypoperfusion
- Optimal treatment:
  - Close outpatient follow up
  - No studies have shown acutely lowering BP in the ED has benefits in short term risk reduction
  - 2013 clinical policy of American College of Emergency Physicians—discouraged initiation of BP meds in asymptomatic patients

Hypertensive Emergencies

- Severe HTN and end organ damage
- 1920s, 80% 1 year mortality
- 1-year mortality 10% in 1998
- Represent up to ¼ of all ED visits

Arch Intern Med 1928;4:264-78
Hypertensive Emergencies Evaluation

- Patient’s medication list
- Over the counter medications
- Illicit substances
- Family history
Hypertensive Emergencies

History

- Signs and symptoms
  - Confusion, seizures
  - Weakness
  - Chest pain
  - Shortness of breath
  - Urine output
  - Targeted organ damage
Hypertensive Emergencies
Physical Evaluation

- Confirm BP with appropriate sized cuff
- Neurologic exam
- Ophthalmologic exam
- Pulmonary exam
- Cardiac exam
- Volume status

![Image of normal and papilledema optic discs with grades 1 to 4]
Hypertensive Emergencies Lab Evaluation

- CT
- ECG
- Troponin
- CXR
- BMP
- Urinalysis
- CBC with manual differential
Causes of Hypertensive Crises

- Essential Hypertension
- Endocrine
  - Pheochromocytoma
  - Cushing syndrome
  - Renin-secreting tumor
  - Primary hyperaldosteronism
- Renovascular disease
  - Renal artery stenosis
  - Polyarteritis nodosa
  - Takayasu arteritis
Causes of Hypertensive Crises

- Drugs
  - Cocaine
  - Phencyclidine
  - Sympathomimetics
  - Antihypertensive medication withdrawal
  - Amphetamines
  - Lead intoxication

- Tyramine reaction with use of MOA inhibitors
- Serotonin syndrome
Causes of Hypertensive Crises

- Central nervous system
  - Cerebral edema
  - Cerebral hemorrhage
  - Brain tumor
  - Spinal cord injury
- Coarctation of the aorta
- Pain
- Burns
Goals of Therapy

- Mean arterial pressure reduction by ~25% in the first hour
- Avoidance of precipitously reducing blood pressure
  - Except with aortic dissection
- Goals of therapy vary on patient history
Hypertensive Encephalopathy

- Also called PRES
  - Posterior reversible encephalopathy syndrome
- Cerebral edema occurs when cerebral regulatory system overwhelmed
- Symptoms:
  - Acute delirium, lethargy, confusion, seizures, severe headache
- Rule out stroke, infection
- More common in women

Mayo Clin Proc 2010;85:427
PRES

- Unfortunately, not always reversible
- Not always confined to the white matter or posterior regions of the brain
- Other common causes: eclampsia, immunosuppressive therapies

UpToDate Reversible posterior leukoencephalopathy syndrome
PRES Treatment

- Seizure control: phenytoin
- Blood pressure reduction:
  - MAP reduced by ~20-25% in first hour
  - IV nitroprusside
  - IV labetalol
  - IV nicardipine
  - IV enalapril
- Avoid centrally acting agents
Pulmonary Edema

- Fluctuations in blood pressure lead to volume overload and flash pulmonary edema
- Usually in patients with CHF or CKD/ESRD
- Noninvasive ventilation, intubation
Pulmonary Edema

- **Treatment**
  - Preload reduction
    - Nitrates
    - Nitroprusside
  - Afterload reduction
    - ACE inhibitors
    - Nitroprusside
  - Loop diuretics
Cardiac Issues

- Myocardial Ischemia
  - IV β-blocker, ASA, NTG
  - Caution with hydralazine, nitroprusside
- Aortic dissection
  - Sudden onset chest pain with radiation to the back
  - Mortality increases 1-2% every hour during first 24hrs
Aortic Dissection

- Type A
  - Aortic arch only
- Type B
  - Descending aorta
Aortic Dissection-PE

- Measure BP in both arms
- Assess pulses
  - ~19% of type A, 9% with type B have no pulse deficits
- Diastolic murmur
  - 44% of type A
- CXR
  - Widened mediastinum
  - 15% have normal CXR

Emerg Med Clin N Am 2015;33:539-551
Aortic Dissection- Treatment

- β-blockers
  - Reduces shearing force
  - Decreases HR, prevents reflex tachycardia
  - IV esmolol

- Nitroprusside

- Vascular surgery evaluation
Acute renal failure

- May be direct cause or an effect of the HTN
  - Need to know previous labs
- Volume status assessment
  - Diuretics
- Calcium channel blockers
- Fenoldopam
- Scleroderma renal crisis
  - ACE I
Sympathomimetic Crisis: Causes

- Drugs
  - Cocaine, phencyclidine, amphetamine
- Pheochromocytoma
- Interaction of MAO with SSRIs
- Tyramine reaction
- Abrupt cessation of sympatholytic medications
- Alcohol withdrawal
Sympathomimetic Crisis: Treatment

- DO NOT use β-blockers as solitary therapy
- Phenoxybenzmine
- Phentolamine
- Nitroprusside
- Labetalol
- Cocaine-benzodiazepines
- Clonidine withdrawal—clonidine
Pharmacologic Agents for Hypertensive Emergencies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Dose (IV)</th>
<th>Onset of action (min)</th>
<th>Duration of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin</td>
<td>5-100mcg/min</td>
<td>2-5</td>
<td>5-30 min</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>0.25-10mcg/kg/min</td>
<td>Immediate</td>
<td>1-2 min</td>
</tr>
<tr>
<td>Fenoldopam</td>
<td>0.1-0.3 mcg/kg/min</td>
<td>&lt;5</td>
<td>30 min</td>
</tr>
<tr>
<td>Enalaprilat</td>
<td>1.25-5mg q6h</td>
<td>15-30</td>
<td>6-12hr</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>10-20 mg IV</td>
<td>10-20min</td>
<td>1-4h</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>5-15mg/h</td>
<td>5-10</td>
<td>15-30 min</td>
</tr>
</tbody>
</table>

Adapted from Hypertension 2003;42:1206-52
## Pharmacologic Agents for Hypertensive Emergencies

<table>
<thead>
<tr>
<th>Drug</th>
<th>Adverse Effects</th>
<th>Indications</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitroglycerin</td>
<td>Headaches, vomiting, methemoglobinemia, caution in RV infarct</td>
<td>Cardiac ischemia, flash pulmonary edema, caution with recent use of phosphodiesterase inhibitors</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Nausea, vomiting, muscle twitching, sweats, thiocyanate &amp; cyandine toxicity</td>
<td>Most HTN emergencies, caution with high intracranial pressure or azotemia</td>
</tr>
<tr>
<td>Fenoldopam</td>
<td>Tachycardia, HA, flushing, nausea</td>
<td>Best for HTN nephropathy emergencies</td>
</tr>
</tbody>
</table>

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# Pharmacologic Agents for Hypertensive Emergencies

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<tbody>
<tr>
<td>Enalaprilat</td>
<td>Significant reductions in BP in high renin states</td>
<td>Acute LV failure, flash pulmonary edema, avoid in AMI</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Tachycardia, flushing, HA, vomiting, worsening angina</td>
<td>Eclampsia, caution given erratic response</td>
</tr>
<tr>
<td>Nicardipine</td>
<td>Tachycardia, HA, flushing, local phlebitis</td>
<td>Most HTN emergencies, caution of coronary steal with cardiac ischemia</td>
</tr>
</tbody>
</table>

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Pharmacologic Agents for Hypertensive Emergencies

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<th>Drug</th>
<th>Dose (IV)</th>
<th>Onset of action (min)</th>
<th>Duration of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Clevidipine</td>
<td>1-2mg/h, titrate q5-10min</td>
<td>2-4</td>
<td>5-15 min</td>
</tr>
<tr>
<td>Esmolol</td>
<td>250-500mcg/kg/min iv bolus, 50-100mcg/kg/min infusion, repeat bolus after 5min or increase to 300mcg/min</td>
<td>1-2</td>
<td>10-30 min</td>
</tr>
</tbody>
</table>

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Pharmacologic Agents for Hypertensive Emergencies

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<thead>
<tr>
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<th>Indications</th>
</tr>
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<tbody>
<tr>
<td>Clevidipine</td>
<td>HA, nausea, vomiting, hypotension, rebound HTN, reflex tachycardia</td>
<td>Postop HTN, HTN emergency in renal dysfunction or acute HF</td>
</tr>
<tr>
<td>Esmolol</td>
<td>Hypotension, nausea, asthma exacerbation, first degree heart block, heart failure</td>
<td>Aortic dissection, perioperative</td>
</tr>
</tbody>
</table>

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Pharmacologic Agents for Hypertensive Emergencies

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<th>Duration of action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Labetalol</td>
<td>20-80mg IV bolus q10min, 0.5-2 mg/min IV infusion</td>
<td>5-10</td>
<td>3-6 h</td>
</tr>
<tr>
<td>Phentolamine</td>
<td>5-15 mg IV bolus</td>
<td>1-2</td>
<td>10-30 min</td>
</tr>
</tbody>
</table>

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<tr>
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<tbody>
<tr>
<td>Labetalol</td>
<td>Vomiting, scalp tingling, bronchoconstriction, dizziness, nausea, heart block, orthostatic hypotension</td>
<td>Most HTN emergencies, ideal for preeclampsia</td>
</tr>
</tbody>
</table>
| Phentolamine| Tachycardia, flushing, HA                            | Pheochromocytoma and other catecholamine excess states | Adapted from Hypertension 2003;42:1206-52
Questions?