-SE (side effects): hypokalemia, dehydration, hyperglycemia, hyperuricemia

• Loop diuretics (furosemide) not routinely used for HTN
  - produce much greater diuresis than thiazides
  - reduce BP and promoting vasodilation
  - mainly used for greater diuresis needed or a patient with low GFR
  - side effects similar to thiazide diuretics**

• Potassium sparing diuretics (spironolactone)
  - diuresis small
  - can balance potassium loss found in other two types of diuretics
  - most important side effect: hyperkalemia
  - should not be used with potassium supplements, ACE inhibitors, angiotensin II receptor blockers, aldosterone antagonists (b/c they all promote hyperkalemia)
    • Can reduce BP when used alone or enhance effects of other hypotensive drugs

  Beta-1 Adrenergic Blockers

• E.G. Propranalol
• Suppress SNS effect on heart & blood vessels
  – Block cardiac beta receptors to decrease HR & contractility
  – Suppress tachycardia
  – Block beta 1 receptors on kidney cells to reduce renin release
  – Reduce peripheral vascular resistance
• SE’s
  – Bradycardia, decreased atrioventricular conduction, reduced contractility, bronchoconstriction, can mask s/s hypoglycemia

• How they reduce BP is uncertain
• 4 uses of beta blockers:
  -blocks cardiac beta receptors decreasing heart rate and contractility → cardiac output declines
  - suppress reflex tachycardia caused by vasodilators
  - blockade of beta receptors of the kidney reduces the release of kidneys
  - long term use reduces peripheral vascular resistance ( mechanism unknown)

• Patients should not use it if they have asthma
• Glycogensis- converts glycogen into glucose
  - glucose levels rise
• If beta blockers are administered during glycogensis, the glucose levels fall too low

• Beta blockers also block beta 1 receptors in the heart
• Beta blockers decrease tremors, perspiration
• Tremors are NOT a good indicator of hypoglycemia
• Perspiration NOT a good indicator of hypoglycemia