Summary Table: Heart Failure & Related Conditions

Category	Key Details
Pharmacologic Treatments	* Beta blockers, ACE inhibitors, & ARBs target neurohormonal pathways * Neprilysin inhibitors ↑ natriuretic peptides (ANP, BNP) * Combination therapy with ARBs prevents angiotensin II buildup
Natriuretic Peptides Mechanism	* ANP & BNP released in response to increased atrial & ventricular pressure * Promote vasodilation & diuresis by increasing Na & water excretion * Neprilysin inhibition ↑ peptide levels but must be combined with ARBs
Cardiac Amyloidosis	* Presents with dyspnea, wt gain, edema * Restrictive cardiomyopathy due to extracellular amyloid protein deposits * Ventricular thickening leads to diastolic dysfunction * Diagnosed via **biopsy** (pink amorphous deposits) & echocardiogram
Renin-Angiotensin System in HF	* Reduced cardiac output lowers renal perfusion, triggering renin release * Angiotensin II causes vasoconstriction, increasing afterload * Aldosterone leads to Na retention, worsening fluid overload
Asymptomatic LV Dysfunction	* ↓ ejection fraction (<50%) without symptoms * Sympathetic activation & RAAS compensate initially * Leads to increased norepinephrine, vasoconstriction, & heart rate * Eventually causes maladaptive remodeling & HF progression
Pulmonary Congestion in HF	* Elevated LVEDP transmits to lungs * Causes pulmonary edema, alveolar hemorrhage * Hemosiderin-laden macrophages (heart failure cells) found in alveoli
Heart Failure Medications	* **Mortality-reducing drugs:** Beta blockers, ACE inhibitors, ARBs, aldosterone antagonists, neprilysin inhibitors, SGLT2 inhibitors * **Symptom management drugs:** Loop diuretics, thiazide diuretics, digoxin * **Contraindicated drugs:** Non-dihydropyridine calcium channel blockers
Acute Decompensated HF	* Sxs include fatigue, dyspnea, orthopnea, lower extremity edema * Hemodynamic effects: ↑ LV end-diastolic pressure, pulmonary congestion * Can be caused by anthracycline chemotherapy (doxorubicin)
High-Output Heart Failure	* Caused by arteriovenous fistula, decreasing systemic vascular resistance * Leads to increased stroke volume, venous return, & cardiac output

Category	Key Details
	* Ultimately results in LV overload & HF
Sodium Nitroprusside in HF	* Used for hypertensive emergency & acute heart failure * Causes balanced vasodilation (↓ preload & afterload) * Maintains stroke volume & cardiac output
Restrictive Cardiomyopathy vs Dilated Cardiomyopathy	* **Restrictive:** Normal LV size, reduced compliance, pulmonary congestion * **Dilated:** Enlarged LV, reduced contractility, systolic dysfunction
Secondary Mitral Regurgitation in HF	* LV dilation leads to mitral annulus widening, causing regurgitation * Diuretics & vasodilators ↓ preload & LV dilation
Neurohormonal Compensation in HF	* **Sympathetic activation:** ↑ heart rate & contractility * **RAAS activation:** Raises afterload & fluid retention * **Natriuretic peptides:** Promote *vasodilation* & *diuresis* but are overpowered by RAAS
Acute Decompensated HF on X-Ray	* Findings include cardiomegaly, Kerley B lines, pleural effusion, pulmonary edema * Different from ARDS, COPD, pulmonary HTN
Heart Failure & Pulmonary Congestion	* Pulmonary congestion leads to hemosiderin-laden macrophages * Identified with Prussian blue stain