



Assigned readings (see course outline) Common Cardiovascular disorders in the elderly

Chapter: 7 (chest pain) Chapter 10: CV disorders

Revised and updated Dr. Delacroix 2023



Assessment of the Cardiovascular System (taped)

- Key symptoms
 - Dizziness
 - Syncope
 - Orthopnea
 - Angina
 - Edema
 - Claudication
- Differentiating normal from abnormal function in older adults

CVD RISK FACTORS

- Four major risk factors for CVD:
 - Hypertension
 - Diabetes mellitus
 - Dyslipidemia
 - **Smoking**
- Higher rates of CVD in older people: absolute number of cases per risk factor tends to increase with age
- Multiple risk factors act in concert with age-related CV changes to promote the development and progression of heart and vascular disorders

PRINCIPAL EFFECTS OF AGING ON THE CARDIOVASCULAR SYSTEM (1 of 2)

Age effect	Clinical implication
↑ Arterial stiffness	↑ Afterload and systolic BP
↓ Myocardial relaxation & compliance	↑ Risk of diastolic heart failure and atrial fibrillation
Impaired responsiveness to β-adrenergic stimulation	↓ Maximum cardiac output; impaired thermoregulation
↓ Sinus node function and conduction velocity in the atrioventricular node and infranodal conduction system	↑ Risk of sick sinus syndrome, left anterior fascicular block, and bundle branch block

PRINCIPAL EFFECTS OF AGING ON THE CARDIOVASCULAR SYSTEM (2 of 2)

Age effect	Clinical implication
Impaired endothelium- dependent vasodilation	↑ Demand ischemia and risk of coronary artery disease and peripheral arterial disease
↓ Baroreceptor responsiveness	↑ Risk of orthostatic hypotension
 ↓ Exercise response (↓ maximal heart rate, maximal cardiac output, VO₂ max, coronary blood flow, peripheral vasodilation) 	↓ Exercise capacity and ↑ cardiac complications (ischemia, heart failure, shock, arrhythmias, death) with illness

CLINICAL EFFECTS OF CV CHANGES

- In healthy older adults, age-related changes have modest clinically relevant effects on cardiac hemodynamics and performance at rest
 - Resting heart rate, ejection fraction, stroke volume, and cardiac output are well preserved even at very advanced age
- Ability to respond to increased demands associated with exercise or illness (either cardiac or noncardiac) declines progressively with advancing age
 - Peak aerobic capacity declines inexorably with age

Hypertension

- High prevalence of HTN only a third of elderly patients meets B/P target recommendations.
- symptoms
 - Often asymptomatic
 - If symptomatic: headache, CP, dyspnea or vision changes
- Description
 - Classified as essential or secondary

Hypertension

- Incidence:
- Affect 86 millions U.D. Adults 20 years or older
- Increase with age
- Half hypertensive patients are not well controlled
- 15% of hypertensive patients are not even aware they have HTN.
- Up to 95% are diagnosed with essential hypertension
- Higher in AA, with higher mortality rate
- Benefits of HTN control:
 - Reduction of 10 mmhg systolic and 5 mmhg diastolic at age 65 associated with
 - Reduction of 25% of MI, 40% stroke, 50% CHF, up to 20% mortality.
- Current HTN control remains extremely low despite obvious benefits.

Recommendation Summary

Population	Recommendation	Grade (What's This?)
Adults aged 18 years or older	The USPSTF recommends screening for high blood pressure in adults aged 18 years or older. The USPSTF recommends obtaining measurements outside of the clinical setting for diagnostic confirmation before starting treatment (see the Clinical Considerations section).	A

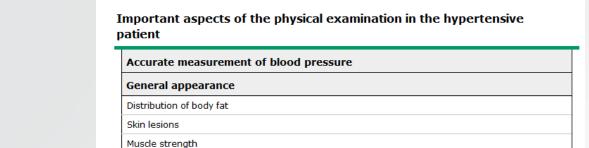
HTN: Risk factors





Important aspects of the history in the patient with hypertension

uration of hypertension	Presence of other risk factors
Last known normal blood pressure	Smoking
Course of the blood pressure	Diabetes
rior treatment of hypertension	Dyslipidemia
Drugs: types, doses, side effects	Physical inactivity
ntake of agents that may cause	Dietary history
nypertension	Sodium
Nonsteroidal antiinflammatory drugs	Processed foods
Estrogens	Alcohol
Adrenal steroids	Saturated fats
Cocaine	Psychosocial factors
Sympathomimetics	Family structure
Excessive sodium	Work status
amily history	Educational level
Hypertension	Sexual function
Premature cardiovascular disease or death	Features of sleep apnea
Familial diseases: pheochromocytoma,	Early morning headaches
renal disease, diabetes, gout	Daytime somnolence
Symptoms of secondary causes	Loud snoring
Muscle weakness	Erratic sleep
Spells of tachycardia, sweating, tremor	
Thinning of the skin	
Flank pain	
Symptoms of target-organ damage	
Headaches	
Transient weakness or blindness	
Loss of visual acuity	
Chest pain	
Dyspnea	
Claudication	



Alertness

Fundoscopy

Hemorrhage

Papilledema

Cotton wool spots

Neck

Thyroid

Heart

Size

Rhythm

Sounds

Lungs

Rhonchi

Rales

Abdomen

Renal masses

Arteriolar narrowing and arteriovenous nicking

Palpation and auscultation of carotids

Bruits over aorta or renal arteries

Neurologic assessment

UpToDate[®]

Visual disturbance Focal weakness Confusion

Femoral pulses

Extremities

Peripheral pulses

Edema

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Diagnosing

- All adults 18 years and older without known HTN should be screened for elevated BP (USPSTF et al., 2021).
- To diagnose HTN the provider should use an average of two or more readings obtained on two or more occasions Out of office BP measurements (ambulatory or home BP monitoring) are recommended to confirm the diagnosis of HTN before starting treatment

(USPSTF et al., 2021).

JNC8, ACC/AHA guidelines.... OMG! Confusion confusion

Updates in 2023 after years of resistance, primary care is slowly abandoning JNC8 guidelines and adopting the ACC/AHA 2017 guidelines

American Academy of Nurse practitioners 2017 ACC/AHA and JNC-8 hypertension guidelines

FYI: September 2018 - The American College of Cardiology (ACC) / American Heart Association (AHA) hypertension guideline published in November 2017 introduced new blood-pressure categories lowering the threshold for the diagnosis of hypertension. Items on each of the AANPCB certification examinations are reviewed each year by clinical experts for relevancy to current and best practice. The 2018 certification examinations use the 2017 ACC/AHA and JNC-8 guidelines to reference test items. While treatment targets may differ among various guidelines, it is important to keep evaluation of the individual's health as the central concern.

Diagnosis of HTN according to JNC8 Followed by AAFP and

- A diagnosis of HTN should be made under the following circumstances:
- Age <60 years: SBP ≥140 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits
- Age 60 years or older: SBP ≥150 mm Hg and/or DBP ≥90 mm Hg at
 ≥2 visits
- Age 60 years or older with CKD or diabetes: SBP ≥140 mm Hg and/or DBP ≥90 mm Hg at ≥2 visits
- Pre-hypertension
 - SBP ≥120 mm Hg and/or DBP ≥80 mm Hg at ≥2 visits

blood pressure classification acc/aha: 2017

Blood Pressure Categories



BLOOD PRESSURE CATEGORY	SYSTOLIC mm Hg (upper number)		DIASTOLIC mm Hg (lower number)
NORMAL	LESS THAN 120	and	LESS THAN 80
ELEVATED	120 – 129	and	LESS THAN 80
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 1	130 – 139	or	80 – 89
HIGH BLOOD PRESSURE (HYPERTENSION) STAGE 2	140 OR HIGHER	or	90 OR HIGHER
HYPERTENSIVE CRISIS (consult your doctor immediately)	HIGHER THAN 180	and/or	HIGHER THAN 120

Diagnostics: Hypertension

Initial Tests (lab, imaging)

- Hemoglobin or hematocrit or CBC
- Complete urinalysis (may reveal proteinuria, check kidney function)
- •CMP (Potassium, calcium, creatinine), and uric acid
- •TSH
- Lipid panel (total, HDL, LDL, triglyceride [TG])
- Fasting blood glucose or hemoglobin A1c
- Urinalysis (to evaluate albumin/creatinine ration)
- •EKG to evaluate possible presence of left ventricular hypertrophy (LVH) or rhythm abnormalities,

Follow-Up Tests & Special Considerations

consider echocardiogram stress test if initial diagnostic test are abnormal.

Special tests only if suggested by history, physical, or labs. In particular, consider possibility of sleep apnea.

Ambulatory (24-hour) BP monitoring if "white coat" HTN is suspected, episodic HTN, or autonomic dysfunction

Home BP monitoring is effective, especially when white coat HTN is a consideration; elevated home BPs correlate with adverse outcomes, possibly more so than office BPs, and normal readings are reassuring.

Treatment: Shared decision-making

- Individual treatment goals should be jointly established with patients after discussion of the anticipated potential benefits and harm
- Educate on adverse effects of antihypertensive.
 - All antihypertensives associated with postural hypotension.
- Educate importance of compliance with medications.
 - Uncontrolled hypertension leads to end organ damage (e.g., stroke, heart failure, myocardial infarction, renal insufficiency, retinopathy)
- Stress the importance of blood pressure measurement techniques.
- Non-pharmacological: Recommend lifestyle improvements, including diet, exercise, and reducing or eliminating tobacco/alcohol. (see next slide)

HTN Management plan

- Behavioral modifications:
 - Diet modification: DASH diet with low sodium intake
 - Limit alcohol
 - Stop smoking
 - Routine exercise (30 minutes 5 times a week)
 - Engage in stress reduction activities
 - Discontinue unnecessary medications that can raise b/p

Effectiveness of lifestyle modifications for lowering BP

Modification	Recommendation	Approximate BP reduction
Weight loss	Maintain normal body weight (BMI 18.5-24.9)	5-20 mmHg per 20 lb weight loss
DASH diet	Diet rich in fruits, vegetables, and low-fat dairy products	8-14 mmHg
Physical activity	Aerobic exercise >30 min most days	4-9 mmHg
Low-salt diet	Reduce dietary sodium to max 2,400 mg/day (only if +HTN)	2-8 mmHg
Stress reduction	Practice a stress reduction modality such as meditation regularly	5 mmHg
Moderate alcohol consumption	Limit consumption to max 1 drink per day for women and 2 drinks per day for men	2-4 mmHg
Tobacco cessation	Incorporate cessation modality of choice	2-4 mmHg (1 week after cessation)

Pharmaceutical approach: JNC 8 or ACC/AHA First line: monotherapy

- Regardless of the guidelines avoid starting an older patient on two antihypertensive drug at the same time.
- Patient with no specific clinical reason/comorbidities
- 4 main class of drugs
 - 1. Thiazide diuretics (chlorthalidone preferred)
 - 2. Dihydropyridine Calcium channel Blockers (Example: Norvasc: Vasodilators, with minimal inotropic effect: No decrease heart rate)
 - 3. ACE inhibitors
 - 4. ARB if can't use ACE
- Monotherapy based upon age and race
 - FYI patient <50 yo: Ace inhibitor or ARB,
 - Patient > 60 yo: Thiazide or dihydropyridine CCBs.
 - Black patients any age: Thiazide or dihydropyridine CCBs

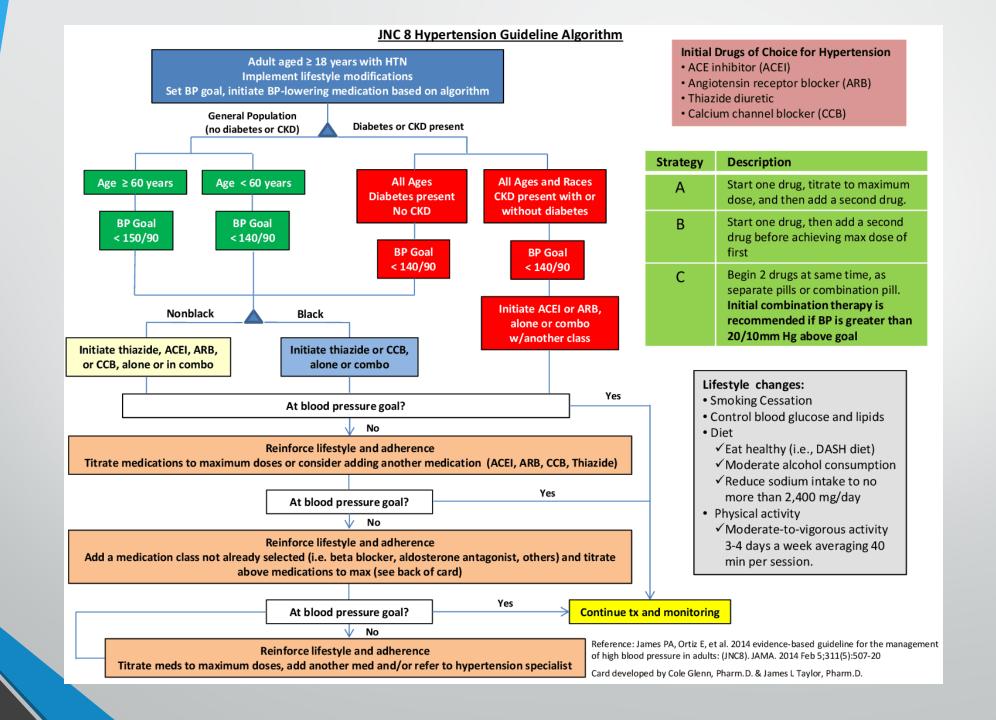
Sequential Drug therapy if does not respond to monotherapy

 Please do not follow strategy A. Too many issue and not beneficial in the long run, Usually followed for patient max on all drugs (resistant to all drugs.) So keep away.

For older patient: Follow strategy B.

- For instance: patient not at goal and compliant with Norvasc 10 mg daily, do not increase to 20 mg daily, instead you can add thiazide 12.5 mg daily.
- For patient less than 50 yo or if robust less than 60 yo you may follow strategy C.
- Avoid Strategy C for patient older than 60 yo.

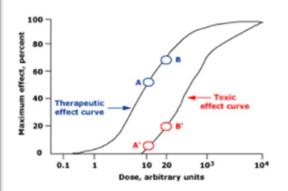
Strategy	Description
Α	Start one drug, titrate to maximum dose, and then add a second drug.
В	Start one drug, then add a second drug before achieving max dose of first
С	Begin 2 drugs at same time, as separate pills or combination pill. Initial combination therapy is recommended if BP is greater than 20/10mm Hg above goal



According to the ACC/AHA:

- The decision to initiate drug therapy should be individualized and involve shared decision-making between patient and provider. (CHART chart chart)
- Patient with a diagnosed of HTN without comorbidities
 - Patients with out-of-office daytime blood pressure ≥135 mmHg systolic or ≥85 mmHg diastolic
 - And/or an average office blood pressure ≥140 mmHg systolic or ≥90 mmHg diastolic
- Patient with a diagnosed of HTN with comorbidities
 - Patients with an out-of-office or in-office blood pressure (mean home or daytime ambulatory) ≥130 mmHg systolic or ≥80 mmHg diastolic and the following comorbidities
 - Established clinical cardiovascular disease (eg, chronic coronary syndrome [stable ischemic heart disease], heart failure, carotid disease, previous stroke, or peripheral arterial disease)
 - Type 2 diabetes mellitus
 - Chronic kidney disease
 - Age 65 years or older
 - An estimated 10-year risk of atherosclerotic cardiovascular disease of at least 10 percent

Dose relation between therapeutic effect and toxicity with antihypertensive drugs



The theoretical therapeutic and toxic effect curves of antihypertensive agents vary based upon the administered dose. The theoretical effects of a single drug given at two different doses (10 and 20 units) are shown. At a dose of 10 units, the antihypertensive agent has a minimal toxic effect (A') and a moderate therapeutic effect (A). Doubling the dose, however, is associated with substantial toxic effects (B') but little increase in therapeutic efficacy (B).

Titration for older patients

- Patient specific: Guidelines differ from one organization to the other
- Reasonable approach for older patients:
 - After monotherapy has been initiated, assess in 4 to 6 weeks
 - If inadequate response titrate up to mid range of the medication (ex: HCTZ 12.5 to 25 mg) then reassess in 4 to 6 weeks
 - Limit titration to one step with a given drug. Never max a drug (strategy B)
 - If one step titration fails, you can do the following:
 - Switch to a different more potent antihypertensive agent.
 - Divide current dose to twice daily (for instance instead of Norvasc 10 mg daily-> Norvasc 5 mg twice daily)
 - Not always the best: issue with compliance.
 - Add a second medications (lower end for instance for a older patient add Norvasc 5 mg daily to HTCZ 25 mg daily) and assess in 4 to 6 weeks.
 - If they can afford it and available, prescribe a combo med
 - Add a second med (dose specific to patient's B/p and tolerance: if less than b/p 20/10: lowest dose, higher dose if above and can tolerate it)

Considerations for individualizing antihypertensive therapy

Indication or contraindication	Antihypertensive drugs	
Compelling indications (major improvement in outcome independent of blood pressure)		
Heart failure with reduced ejection fraction	ACE inhibitor or ARB, beta blocker, diuretic, aldosterone antagonist*	
Postmyocardial infarction	ACE inhibitor or ARB, beta blocker, aldosterone antagonist	
Proteinuric chronic kidney disease	ACE inhibitor or ARB	
Angina pectoris	Beta blocker, calcium channel blocker	
Atrial fibrillation rate control	Beta blocker, nondihydropyridine calcium channel blocker	
Atrial flutter rate control	Beta blocker, nondihydropyridine calcium channel blocker	
Likely to have a favorable effec	t on symptoms in comorbid conditions	
Benign prostatic hyperplasia	Alpha blocker	
Essential tremor	Beta blocker (noncardioselective)	
Hyperthyroidism	Beta blocker	
Migraine	Beta blocker, calcium channel blocker	
Osteoporosis	Thiazide diuretic	
Raynaud phenomenon	Dihydropyridine calcium channel blocker	
Contraindications		
Angioedema	Do not use an ACE inhibitor	
Bronchospastic disease	Do not use a non-selective beta blocker	
Liver disease	Do not use methyldopa	
Pregnancy (or at risk for)	Do not use an ACE inhibitor, ARB, or renin inhibitor (eg, aliskiren)	
Second- or third-degree heart block	Do not use a beta blocker, nondihydropyridine calcium channel blocker unless a functioning ventricular pacemaker	
Drug classes that may have adverse effects on comorbid conditions		
Depression	Generally avoid beta blocker, central alpha-2 agonist	
Gout	Generally avoid loop or thiazide diuretic	
Hyperkalemia	Generally avoid aldosterone antagonist, ACE inhibitor, ARB, renin inhibitor	
Hyponatremia	Generally avoid thiazide diuretic	
Renovascular disease	Generally avoid ACE inhibitor, ARB, or renin inhibitor	

ACE: angiotensin-converting enzyme; ARB: angiotensin receptor blocker.

Adapted from: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. JAMA 2003; 289:2560.

^{*} A benefit from an aldosterone antagonist has been demonstrated in patients with NYHA class III-IV heart failure or decreased left ventricular ejection fraction after a myocardial infarction.

Contraindications

- Diuretics may worsen gout.
- Avoid thiazide and loop diuretic in patient with sulfa allergy Cross sensitivity
- \blacksquare θ -Blockers (relative) in reactive airway disease, heart block, diabetes, and peripheral vascular disease; probably should be avoided in patients with metabolic syndrome or insulin-requiring diabetes.
- θ -Blockers : Wean slowly after chronic use.
- Diltiazem or verapamil: Do not use with systolic dysfunction or heart block.
- Amlodipine may cause peripheral edema and GERD.
- CCB: don't prescribe to patients with heart block without a pacemaker.

Key Clinical Points

- 1. The prevalence of hypertension increases steadily with age.
- 2.Older people develop systolic hypertension due to the age-related increase in arterial stiffness. Systolic blood pressure and pulse pressure, both closely associated with arterial stiffness, confer the greatest significance as cardiovascular and cerebrovascular risk factors.
- 3.Age-related changes in systems that regulate blood pressure result in greater blood pressure variability. Therefore, careful attention is needed to accurately measure and diagnose hypertension, as well as monitoring for adverse drug events—especially postural hypotension—throughout treatment.
- 4. Older hypertensive individuals commonly have physiologic characteristics that respond effectively to lifestyle modifications.

Hypertension.

- Follow-up
 - Frequency based on level of hypertension:
 - Prehypertensive: 3 to 6 months
 - If systolic and/or diastolic B/P less than 20 mmHg from target four to 6 weeks
 - If systolic and/or diastolic B/P more than 20 mmHg from target within two to 4 weeks.
 - Best practice: one month to bring B/P to target. Usually not possible for the elderly as you have to go slow and start low.
 - Repeat electrolytes, BUN/creatinine about 3 to 6 weeks after initiating thiazide diuretics, ARB, or ACEi, to evaluate for drug-induced complications
 - When patient is stable: OK to follow up q 6 to 12 months.

Hypertension

Prevention/prophylaxis

- Lifestyle modification
- Routine BP monitoring: Home self b/p.
- Monitor quality of life (address dizziness, fatigue, sexual function, fall)

Referral

- Unusual signs and symptoms
- Suspected secondary cause

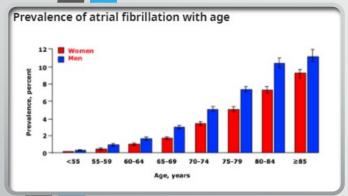
Education

- Routine BP monitoring
- Proper BP measurement technique
- Encourage compliance

Atrial fibrillation (Afib)

 Afib: paroxysmal or continuous supraventricular tachyarrhythmia characterized by rapid, uncoordinated atrial electrical activity and an irregularly irregular ventricular response. In most patients

Tachyarrhythmias – Atrial fibrillation- updated 2022



- The most common sustained arrhythmia seen in clinical practice
- Incidence and prevalence increase exponentially with age, such that the prevalence in octogenarians is approximately 10%
- Patients over age 80 account for approximately 35 percent of patients with AF and the prevalence of AF is about 10 percent
- rate control has often been preferred in the elderly patients for the following reasons:
 - They are more sensitive to the proarrhythmic effects of drugs
 - AF is often permanent
 - AF is often asymptomatic

Types of Atrial fibrillation

- 1. Paroxysmal: Episodes last < 7 days vast majority less than 24 hours. Respond to cardioversion
- 2. Persistent: Last > 7 days, respond to cardioversion but may occur again
- 3. Permanent: Present > 1 year, usually failed cardioversion

Valvular Afib vs Nonvalvular Afib

Valvular Afib: affects people who have moderate to severe valve diseases only or an artificial valve.

NonValvular AFIB: atrial fibrillation that isn't caused by a problem with a heart valve

Risk and contributing factors:

- Most common Risk factors:
 - Age
 - HTN,
 - Obesity
- Other risk factors
 - Male sex
 - European ancestry (Caucasians)
 - Diabetes

- Most are idiopathic.
- Coronary artery disease
- Heart failure
- Cardiomyopathy (ischemic or nonischemic)
- Valvular diseases (e.g., mitral regurgitation, mitral stenosis)
- Hyperthyroidism
- Cardiac procedures or surgeries
- Alcohol abuse
- Sleep apnea
- COPD
- Serious infections (e.g., pneumonia)

AF: CLINICAL FEATURES

- Symptoms related to AF are highly variable
 - Often asymptomatic specially if ventricular response is 60 to 100 beat per minute
 - Most common: palpitations, shortness of breath, impaired exercise tolerance, dizziness, presyncope or syncope, fatigue, anxiety, chest pressure.
- Physical exam:
 - Irregularly irregular heart rate and pulse with pulse deficit

Diagnostics (Class)

- History and physical:
- In patients with ongoing AF, the standard 12-lead electrocardiogram is diagnostic
 - Absence p wave, irregular QRS interval, and usually tachycardia
- Ambulatory rhythm monitoring (Holter monitoring) is helpful in diagnosing paroxysmal AFib or AFlut and monitoring for recurrence
- Initial Lab studies:
 - CBC, CMP, thyroid function, renal functions, Toxicology screen (if you suspect drug use), D-dimer, BNP (r/o HF),
 - PT/INR if you contemplate anticoagulation.
 - troponin if suspect unstable angina or MI. (if you feel you need this, transfer to ER)
 - If patient is on digoxin, order a level.



Follow-up tests

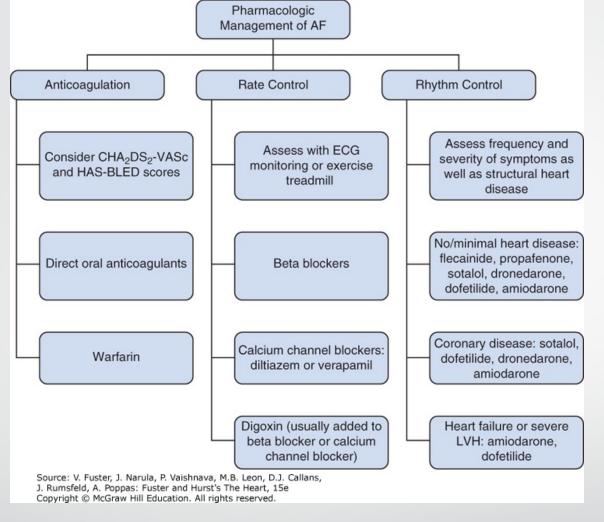
- Exercise stress test
- Holter monitor
- Chest x-ray (CXR) for cardiopulmonary disease
- Echocardiogram
- TEE (Transesophageal echocardiogram) to detect left atrial appendage thrombus if cardioversion is planned
- Sleep study may be useful if sleep apnea is suspected.

MANAGEMENT OF AF

- Decision making:
 - Heart rate versus rhythm control.
 - Anticoagulate or not.
 - Behavioral modifications:
 - Avoid stimulant (caffeine, nicotine, decongestant) and ETOH above recommended guidelines
 - Encourage physical activities and weight reduction

MANAGEMENT OF AF

- Identify and treat the underlying cause(s), such as:
 - Treat the hyperthyroidism.
 - Treat the hypertension.
 - Manage electrolyte unbalance
- Principal strategies for relieving symptoms: control of heart rate and/or maintenance of normal sinus rhythm
 - > 80 to 100 beats/min at rest
 - > 90 to 110 beats/minutes minute with moderate exercise



Pharmacologic management of AF. The three pillars of management of AF are anticoagulation, rate control, and rhythm control. The decision on anticoagulation is based on the CHA₂DS₂-VASc and HAS-BLED scores. Direct oral anticoagulants are preferred over warfarin except in specific situations, such as the presence of a mechanical heart valve. Rate control is best accomplished with β-blockers or calcium channel blockers, with digoxin added in selected cases when rate control is not adequate. Rate control should be assessed with 24-hour ambulatory monitoring, and in some cases, exercise treadmill testing. The choice of an antiarrhythmic drug is based on the presence or absence of underlying heart disease. Flecainide and propafenone may only be used in patients without structural heart disease. Amiodarone is a second-line agent in patients with no or minimal heart disease, whereas it is a first-line agent in patients with HF and severe LV hypertrophy.



MANAGEMENT OF AF: STROKE RISK STRATIFICATION

Clinical risk factors for stroke, transient ischemic attack, and systemic embolism in the CHA₂DS₂-VASc score

(A) The risk factor-based approach expressed as a point based scoring system, with the acronym CHA₂DS₂-VASc

(NOTE: maximum score is 9 since age may contribute 0, 1, or 2 points)

HA ₂ DS ₂ -VASc risk factor	Points	
Congestive heart failure Signs/symptoms of heart failure or objective evidence of reduced left-ventricular ejection fraction	+1	
Hypertension Resting blood pressure >140/90 mmHg on at least two occasions or current antihypertensive treatment	+1	
Age 75 years or older	+2	
Diabetes mellitus Fasting glucose >125 mg/dL (7 mmol/L) or treatment with oral hypoglycaemic agent and/or insulin	+1	
Previous stroke, transient ischaemic attack, or thromboembolism	+2	
Vascular disease Previous myocardial infarction, peripheral artery disease, or aortic plaque	+1	
Age 65-74 years	+1	
Sex category (female)	+1	

Clinical characteristics comprising the HAS-BLED bleeding risk score

Letter	Clinical characteristic*	Points
Н	Hypertension (ie, uncontrolled blood pressure)	1
Α	Abnormal renal and liver function (1 point each)	1 or 2
S	Stroke	1
В	Bleeding tendency or predisposition	1
L	Labile INRs (for patients taking warfarin)	1
Е	Elderly (age greater than 65 years)	1
D	Drugs (concomitant aspirin or NSAIDs) or excess alcohol use (1 point each)	1 or 2
		Maximum 9 points

HAS-BLED score (total points)	Bleeds per 100 patient-years¶
0	1.13
1	1.02
2	1.88
3	3.74
4	8.70
5 to 9	Insufficient data

The HAS-BLED bleeding risk score has only been validated in patients with atrial fibrillation receiving warfarin. Refer to UpToDate topics on anticoagulation in patients with atrial fibrillation and on specific anticoagulants for further information and other bleeding risk scores and their performance relative to clinical judgment.

INR: international normalized ratio; NSAIDs: nonsteroidal antiinflammatory drugs.

- * Hypertension is defined as systolic blood pressure >160 mmHg. Abnormal renal function is defined as the presence of chronic dialysis, renal transplantation, or serum creatinine ≥200 micromol/L. Abnormal liver function is defined as chronic hepatic disease (eg, cirrhosis) or biochemical evidence of significant hepatic derangement (eg, bilirubin more than 2 times the upper limit of normal, plus 1 or more of aspartate transaminase, alanine transaminase, and/or alkaline phosphatase more than 3 times the upper limit of normal). Bleeding predisposition includes chronic bleeding disorder or previous bleeding requiring hospitalization or transfusion. Labile INRs for a patient on warfarin include unstable INRs, excessively high INRs, or <60% time in therapeutic range.
- ¶ Based on initial validation cohort from Pisters R. A novel-user-friendly score (HAS-BLED) to assess 1-year risk of major bleeding in patients with atrial fibrillation: the Euro Heart Survey. Chest 2010; 138:1093. Actual rates of bleeding in contemporary cohorts may vary from these estimates.

Original figure modified for this publication. Lip GY. Implications of the CHA2DS2-VASc and HAS-BLED Scores for thromboprophylaxis in atrial fibrillation. Am J Med 2011; 124:111. Table used with the permission of Elsevier Inc. All rights reserved.

a score of 0 indicates low risk 1-2 indicates moderate risk and ≥3 indicates high risk.

Please note: Decision to anticoagulate patient or not should not be based solely on the has-bled score

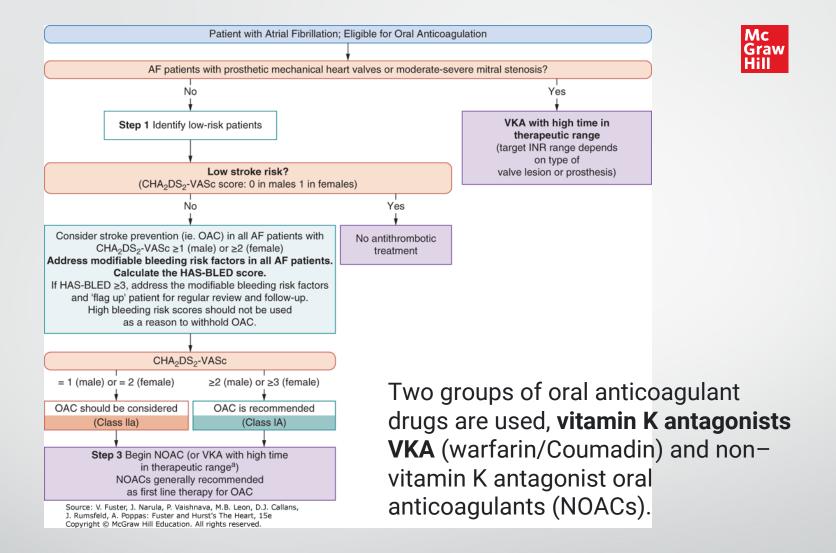
Your goal is to deal with modifiable risk factors and decrease the has-bled score

- 1- Control HTN
- 2- D/c NSAIDS or aspirin
- 3- ETOH management
- 4- If on warfarin. Tighter control with more frequent monitoring and diet management or switch them to a NOAC

5- preserve kidney and liver function: Pharm and non pharm approach. Only two nonmodifiable risk factors are age, bleeding predisposition (ITP) or stroke.

Recommendations: Management with anticoagulants
Benefits outweigh the risk of bleeding in following group regardless of the
has-bled score.

For a CHA_2DS_2 -VASc score ≥ 2 in males or ≥ 3 in females For a CHA_2DS_2 -VASc score of 1 in males and 2 in females based on age 65 to 74 years,. Age 65 to 74 years is a stronger risk factor than the other factors conferring one CHA_2DS_2 -VASc score point



Decision pathway for anticoagulation use in patients with AF. Key: AF, atrial fibrillation; CHA2DS2-VASc, Congestive heart failure, Hypertension, Age >_75 years, Diabetes mellitus, Stroke, Vascular disease, Age 65–74 years, Sex category (female); HAS-BLED, Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly (>65 years), Drugs/alcohol concomitantly; INR, international normalized ratio; NOAC, non—vitamin K antagonist oral anticoagulant; OAC, oral anticoagulant; SAMe-TT2R2, Sex (female), Age (<60 years), Medical history, Treatment (interacting drug(s)), Tobacco use, Race (non-Caucasian) (score); TTR, time in therapeutic range; VKA, vitamin K antagonist. If a VKA is being considered, calculate SAMe-TT2R2 score: if score 0–2, may consider VKA treatment (eg, warfarin) or NOAC; if score >2, should arrange regular review/frequent INR checks/counselling for VKA users to help good anticoagulation control, or reconsider the use of NOAC instead; TTR ideally >70

MANAGEMENT OF AF: STROKE PROPHYLAXIS

DOAC (direct-acting oral anticoagulant)/NOAC (novel oral anticoagulant) agents are preferred for most patients with nonvalvular atrial fibrillation who should receive anticoagulant therapy Dabigatran 150 mg q12h when creatinine clearance (CrCl) ≥ 50 mL/min (75 mg q12h when CrCl = 15–30 mL/min). No INR monitoring needed

- Rivaroxaban (Xarelto) 20 mg/d when CrCl ≥50 mL/min (15 mg/d when CrCl = 15-50 mL/min)
- Apixamab (Eliquis) 5 mg twice daily (2.5 mg twice daily when 2 or more of the following are present: age ≥80 years, weight ≤60 kg, or creatinine ≥1.5 mg/dL)
- Edoxaban (savaysa) 60 mg po daily
- Warfarin
 - Patients with mechanical valves should be treated with warfarin to maintain an INR of 2.0 to 3.0 or 2.5 to 3.5 dependent on the type and location of the prosthesis.
 - titrated to maintain an INR of 2-3
 - Initial dose Coumadin 5 mg for adults, but start at 2.5 mg elderly patient older than 70 yo
 - Check INR q 2-3 days until therapeutic for two consecutive checks, then recheck in a week if therapeutic check once monthly.

Rhythm control versus rate control in atrial fibrillation

- Both strategies can fail both in the short and in the long terms.
- Most patient with new onset AFIB spontaneously convert within 72 hours after start of rate control medication.
- If they do not spontaneously convert. At least one attempt at cardioversion should be attempted.
 - Cardioversion is most often performed electrically but may also be achieved using antiarrhythmic drug therapy in some instances, <u>by experienced clinicians.</u> (not in <u>primary care</u>)
 - If duration of AFib/AFlut is ≥48 hours or unknown, anticoagulated for ≥3 weeks before cardioversion to reduce the risk of stroke.
 - Cardioversion attempted only after TEE is performed, continue anticoagulation 4
 weeks post cardioversion.
 - Hemodynamically unstable: Hospitalization: Rhythm control is favored. Chemically or via cardioversion. See above.

Pharmacology intervention Rate control in stable patients: Go Slow start low

- Optimal target for ventricular rate has not been firmly established, but there is evidence that aggressive control of the ventricular rate (<80 bpm) offers no benefit beyond more modest rate control (i.e., resting heart rate <110 bpm). So a range between range 80 to 100 is acceptable at rest
- 1- Beta blockers (e.g., metoprolol tartrate [Lopressor], atenolol [Tenormin])
 - Example: metoprolol tartrate at 12.5–100 mg PO BID.
 - Use in those with CAD and/or reduced systolic function.
- 2- Nondihydropyridine calcium channel blockers (e.g., verapamil [Calan, Verelan], diltiazem [Cardizem])
 - Example: diltiazem (immediate release) 60–120 mg PO TID (max: 360 mg/day).
 - Avoid use in those with reduced systolic function.
 - Avoid combo therapy with a beta blocker increases bradycardia, syncope, and hemodynamic instability.
- 3- Digoxin (least effective) for sedentary older adult only (80-year-old and above)
 - Example: digoxin 125 mcg PO daily.
 - Can be taken with a beta blocker or calcium channel blocker.
- 4- Amiodorone: (considered mostly a rhythm control meds, but it does slow down ventricular rate)
 - Due to short- and long-term effects: AHA/ACC strongly recommends to only as a second line therapy if other meds fail

A-FIB: Rhythms control

Restoration of sinus rhythm using electrical or pharmacologic cardioversion – Patient must be hemodynamically stable

- Electrical: Cardioversion
 - without anticoagulant therapy if Afib started less than or equal to 48 hours
 - If duration of AFib is >48 hours or unknown, anticoagulate for ≥3 weeks before cardioversion to reduce the risk of stroke.
 - Usually cardiologist will perform a TEE to r/o atrial thrombus
 - Continue anticoagulation for at least 4 weeks post successful cardioversion

A-FIB: Rhythm control strategies.

- Involve a cardiologist:
- Preferred for patients:
 - at high risk for a CV event
 - Fail rate control
 - AF diagnosed within one year who have not spontaneously converted
 - hemodynamically instable patients
 - Younger patient less than 60 yo or who are extremely active and need optimal cardiac performance.
- Restoration of sinus rhythms can be achieved with cardioversion: electrically or with antiarrhythmic drug therapy, ablation can also be performed as a last resort.
- Antiarrhythmic therapy for long-term maintenance of sinus rhythm:
 - Options for pharmacologic cardioversion include Flecainide, dofetilide, propafenone, ibutilide and amiodorone if LVH, HF, or CAD. **Amiodarone is effective and is commonly prescribed**.(and a horrible drug)
- There is currently no standard/optimal rhythm control drug therapy in older adults with symptomatic AF

ISSUES FOR REFERRAL

- Management of AFib refractory to standard medical therapy
 - unable to achieve adequate rate control with medication
 - development of significant bradycardia and hemodynamic instability with rate control
 - the use of more aggressive treatments.
 - These may include pacemaker implantation
 - to allow for more intensive pharmacologic blocking of the AV node
 - or an ablation procedure. AFlut in particular is often very amenable to ablation;
 - thus, consideration should be given to early expert referral in appropriate patients.
 Antiarrhythmic drug therapy can often be very effective but should be prescribed by experienced practitioners.

Ongoing care

- FOLLOW-UP RECOMMENDATIONS
 - Patient Monitoring
 - Adequate anticoagulation levels with warfarin should be determined weekly during initiation and monthly when stable. If NOACs are employed, hepatic and renal functions should be reevaluated annually
 - DIET
 - Patients on warfarin should attempt to consume a stable amount of vitamin K.
 - PATIENT EDUCATION
 - For overweight and high BMI patients, weight loss combined with risk factor modification have demonstrated beneficial effects on controlling AFib

Heart Failure- Updated guidelines 2022

- Signal symptoms
 - Dyspnea on exertion, orthopnea, fatigue, leg edema, paroxysmal nocturnal dyspnea, progressive activity intolerance,
- Description
 - Pump failure: Clinical syndrome caused by the heart's inability to eject enough blood to maintain tissue perfusion
- Different types:
 - Right vs left Heart failure
 - Heart failure with reduced ejection fraction (HFrEF) AKA systolic heart failure
 - Heart failure with preserved ejection fraction (HFpEF). AKA diastolic heart failure

PROGNOSIS

- Median survival rates of 2–3 years
 - > 25%—30% of patients die within 1 year after initial diagnosis
 - > 50% survive 1-5 years
 - > 20%-25% survive >5 years
 - More than 250 thousands Americans die from HF each year.
- patients with HFPEF (Diastolic heart failure) have somewhat better survival rates than patients with systolic HF (HFREF= EF less than 40%)

EPIDEMIOLOGY OF HEART FAILURE (HF)

- Incidence and prevalence increase with age
 - 6 millions Americans and >850,000 new cases annually
 - Leading cause of hospitalization and rehospitalization in older adults
 - Median age of patients hospitalized with HF is 75 years, and approximately two thirds of deaths attributable to HF are in patients age 75 years or older
- HF is a major cause of chronic disability and impaired quality of life in older adults

Risk factors

- older age
- Higher in AA with poorer outcomes
- physical inactivity
- cigarette smoking
- lipid abnormalities
- overweight/obesity
- Hypertension: (lead to structural and functional abnormalities-> increase mortality. Controlling B/p is primordial especially in HFpEF.
- diabetes mellitus
- metabolic syndrome
- Insulin resistance
- Outcomes and progression of disease
- Worst among patients with diabetes and/or insulin resistance and/or lipid abnormalities.
- High B/p and elevated BMI are associated with better outcomes in HF patients.

Heart Failure

Signs and Symptoms of Heart Failure

"A N-E-W L-E-A-F"

A: Acute Agitation/Anxiety

N: Nighttime shortness of breath or ↑ nighttime urination

E: Edema in lower extremities

W: Weight gain (2–4 lb/week)

L: Lightheadedness

E: Extreme shortness of breath lying down

A: Abdominal symptoms (nausea, pain, decreased appetite, distention)

F: Fatigue

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Cardinal S/S of HF
Dyspnea
fatigue
decrease exercise tolerance
fluid retention
S/s of pulmonary and/or splanchnic congestion
and/or peripheral edema.

Clinical approach: In primary care

- probability of HF if patient presents with symptoms or signs of HF:
 - based on the patient's prior clinical history, presenting symptoms, physical examination, and resting electrocardiogram.
 - If these elements are normal, HF is unlikely, and other diagnoses should be considered.
 - If at least one of these elements is abnormal, plasma NPs should be measured, if abnormal order an echocardiography.
 - American College of Cardiology/American Heart Association (ACC/AHA) guidelines recommend that when patients present with dyspnea, measurement of BNP or NT-proBNP can be useful to support a diagnosis and severity or to exclude HF.

Recognizing worsening HF

Clinical clues that a patient may have worsening or advanced HF include the folllowing:

Persistent New York Heart Association (NYHA) III-IV symptoms

≥2 emergency department (ED) visits or hospitalizations for acute HF in 12 months

High risk biomarker profile (hyponatremia, very elevated NPs or troponin)

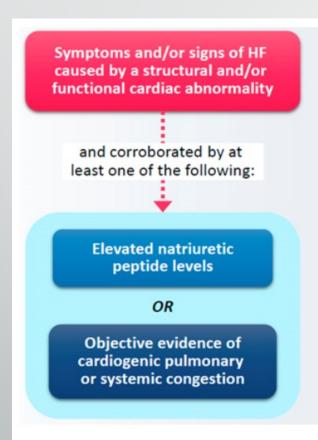
Inability to up titrate guideline-directed medical therapies because of hypotension (SBP ≤90 mmHg), dizziness, excessive fatigue, nausea, etc.

Onset of arrhythmias (atrial fibrillation, ventricular tachycardia with implantable cardioverter-defibrillator [ICD] shocks)

Escalating doses of diuretics (e.g., >160 mg/d furosemide) or persistent edema despite escalating diuretic doses

Need for intravenous inotropes

Heart Failure: Revised guidelines 2022



Elevated NP levels	Ambulatory	Hospitalized/ Decompensated
BNP, pg/mL	≥ 35	≥ 100
NT-pro-BNP, pg/mL	≥ 125	≥ 300

Figure 1. Universal definition of HF³

Biomarkers in HF

Early diagnosis of HF is challenging

Nonspecific clinical presentation of HF

Use of biomarker: BNP, NT pro-BNP: essential in patient's evaluation and diagnostic process.

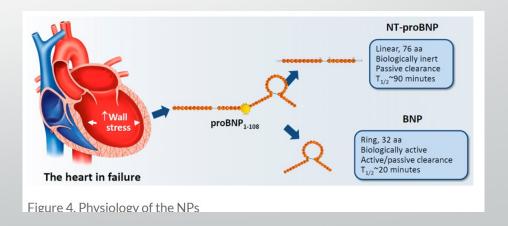
Note: patients with HFpEF (more than 50%),

AA and high BMI may have low BNP baseline

cardiac troponin is not specific for establishing a diagnosis of HF.

 Elevated high-sensitivity cardiac troponin (hscTn) is associated with worse clinical outcomes.

Elevated NP levels	Ambulatory	Hospitalized/ Decompensated
BNP, pg/mL	≥ 35	≥ 100
NT-pro-BNP, pg/mL	≥ 125	≥ 300



American College of Cardiology and American Heart Association revised guidelines for the diagnosis of Health failure

TABLE 79-7 III DIAGNOSTIC EVALUATION OF PATIENTS WITH HEART FAILURE

Class I (indicated in most patients)

- · Complete blood count
- · Blood chemistries: electrolytes, creatinine, blood urea nitrogen, glucose, magnesium, calcium, liver function tests, and lipid profile
- · Thyroid-stimulating hormone (TSH)
- . B-type natriuretic peptide (BNP) or N-terminal pro-BNP level
- Urinalysis
- · Chest radiograph and electrocardiogram (ECG)
- · Echocardiogram: two-dimensional with Doppler
- · Cardiac catheterization and coronary angiography in patients with angina or significant ischemia unless the patient is not eligible for revascularization

Class II (acceptable in selected patients; see text)

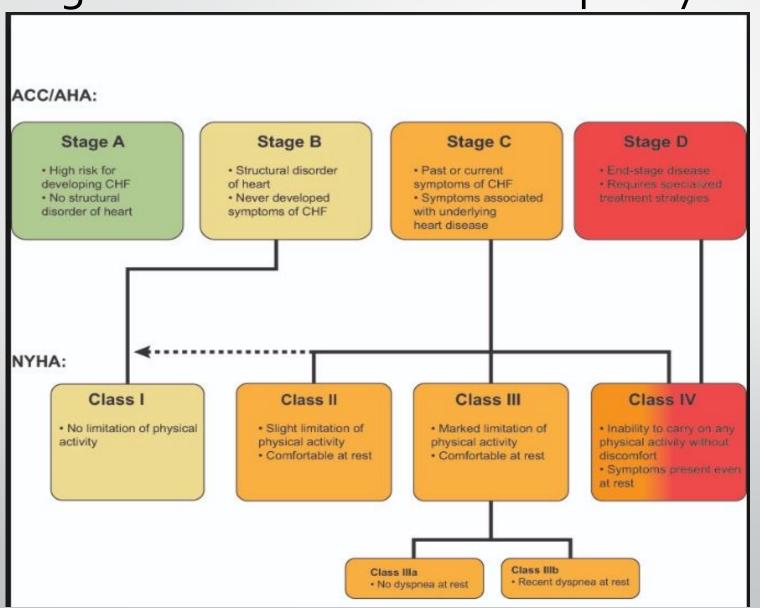
- · Serum iron and ferritin
- · If suspected, assessment for rheumatologic disease, human immunodeficiency virus, amyloidosis, or pheochromocytoma
- · Screening for sleep-disordered breathing
- · Stress test to evaluate for ischemia in patients with unexplained heart failure who are potential candidates for revascularization
- · Coronary angiography if ischemia may be contributing to heart failure in patients who are potential candidates for revascularization
- · Endomyocardial biopsy when a specific diagnosis is suspected that would influence therapy

Class III (not routinely indicated)

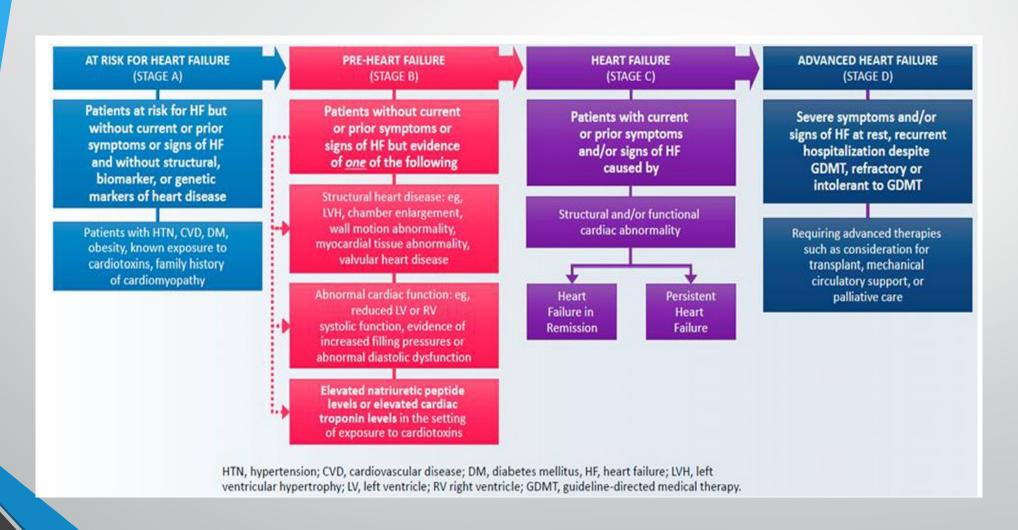
- · Routine repeat measurement of left ventricular function in stable patients
- Endomyocardial biopsy as a routine procedure in the evaluation of patients with heart failure

Yancy CW, Jessup M, Bozkurt B, et al. 2013 ACCF/AHA guideline for the management of heart failure. J Am Coll Cardiol. 2013;62:e147-e239.

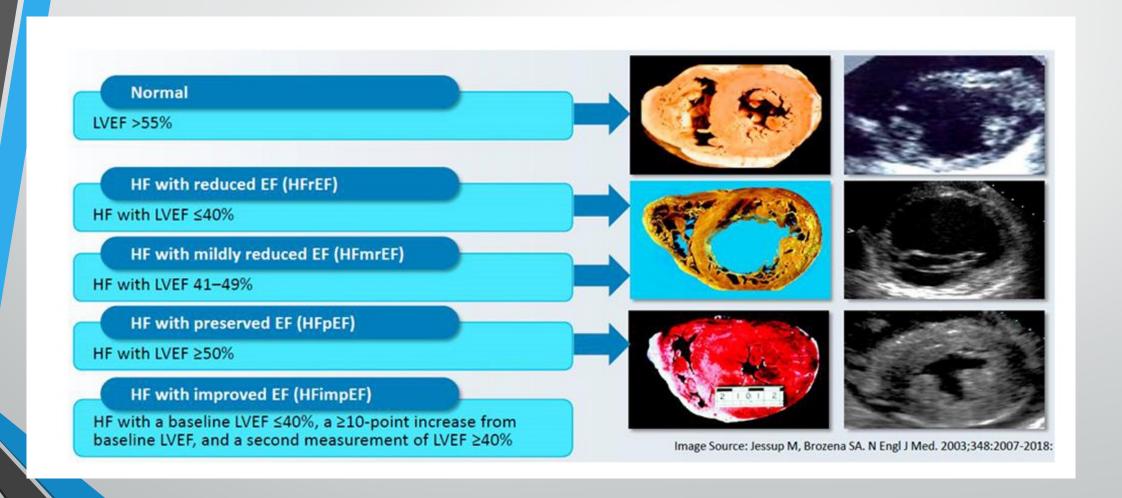
Classification of heart failure: ACC/AHA objective stages vs NYHA functional capacity class



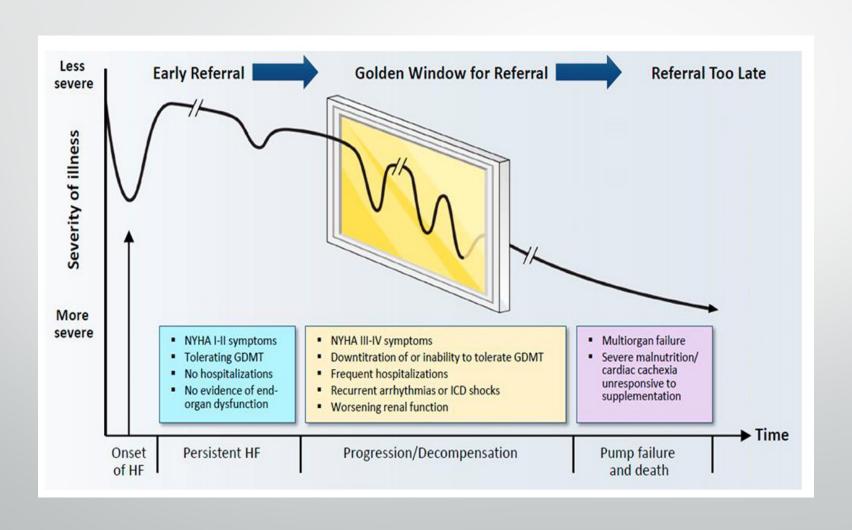
New classification of Heart failure stages



Classification according to left ventricular ejection fraction



Golden window for referral for consideration of advanced HF therapies



HF management approach: Goals

- Improving survival and reducing morbidity
- Improving functional capacity and quality of life (QoL).
- Controlling modifiable Risk factors for HF:
- Physical conditioning by exercise training can improve exercise tolerance, health-related QoL, and HF hospitalization rates in patients with HF.
- A shared decision-making approach is recommended

TABLE 79-8 NONPHARMACOLOGIC ASPECTS OF HEART FAILURE MANAGEMENT Patient education · Symptoms and signs of heart failure · Detailed discussion of all medications · Emphasize importance of adherence · Specific information about when to contact nurse or physician for worsening symptoms Daily weight chart · Specific directions on when to contact nurse or physician for changes in weight · Self-management of diuretic dosage based on daily weights in selected patients · Involve family/significant other when feasible Dietary consultation · Individualized and consistent with needs/lifestyle • Avoidance of excess sodium intake (> 2.3 g/d) • Avoidance of excess fluid intake (> 2 L/d) · Weight loss, if appropriate · Low fat, low cholesterol, if appropriate · Adequate caloric intake · Emphasize adherence while allowing flexibility Medication review · Heart failure therapy in accordance with guidelines · Eliminate unnecessary medications · Simplify regimen whenever possible · Consolidate dosing schedule Social services · Assess social support structure · Evaluate emotional and financial needs · Intervene proactively when feasible Intensive follow-up · Telephone and/or telemedicine contacts · Home health visits as needed · Outpatient clinic

Palliative care consultation in patients with advanced symptoms or frequent hospitalizations

Contact information

- · Names and phone numbers of nurse and physician
- · 24-hour availability

Management of HFrEF

- targeting the RAAS is a cornerstone of the medical management of HFrEF.
 - inhibition of the RAAS with
 - angiotensin receptor-neprilysin inhibitor (ARNI)
 - Or angiotensin converting enzyme (ACE) inhibitors,
 - Or angiotensin receptor blockers (ARBs),
 - ARNI is now the preferred RAAS inhibitor for HFrEF. (reimbursed by insurance)
- mineralocorticoid receptor antagonists (MRA)
- \bullet in conjunction with evidence-based β -blockers (carvedilol, bisoprolol, metoprolol succinate).
- SGLT2 inhibitor
- Mechanisms of drug actions:
 - <u>ACE, ARB and ARNI</u>: inhibit conversion of angiotensin I to angiotensin II, which prevents vasoconstriction and induces relaxation of the vasculature-> decreasing cardiac workload.
 - MRAs slow HF progression, prevent/reverse cardiac remodeling, and prevent the development of arrhythmias by blocking aldosterone,
 - **B-blockers** prevents the ventricular remodeling
 - SGLT2 inhibitor: (see following slide)

HFrEF/Systolic Heart failure Reduced EF less or equal 40-50 %.

Chronic treatment

- TARGETING THE RASS first: IT is the cornerstone of the management of HF
- ARNI is preferred approach above ace or arb (use ot be extremely expensive now it is covered by medicare)
 - Initial: Sacubitril 49 mg/valsartan 51 mg twice daily. Double the dose as tolerated after approximately 2 weeks to the target maintenance dose
 - ACE inhibitors (ACEIs) or ARB if can tolerate ACEIs. But never both together or in addition with an ARNI
 - Ace inhibitor for instance:
 - Enalapril: Initial: 2.5 mg twice daily; as tolerated, may increase dose (eg, double) every ≥2 weeks to a target dose of 10 to 20 mg twice dailyFirst-line therapy in individuals with systolic dysfunction. They prevent left ventricular remodeling. The significance of ACEIs and ARBs in those with diastolic heart failure is uncertain.
 - Use an ARB, such as valsartan (Diovan) 20–40 mg PO BID (max: 320 mg/day), if unable to tolerate an ACEI due to side effect, such as cough.
 - Monitor for cough (excluding ARBs), renal impairment, angioedema (rare with ARBs), and hyperpotassemia.

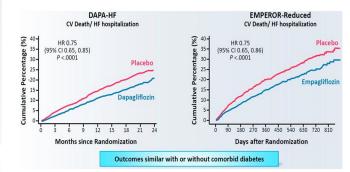
Beta blockers

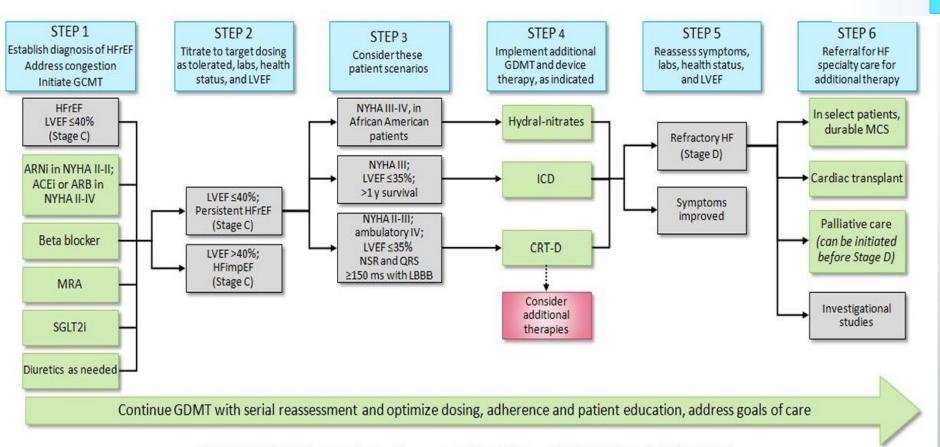
- Example: Start carvedilol at 3.125 mg BID (max: 50 mg/day).
- Monitor closely for bradycardia, hypotension, and fatigue
- SGLT2 I: two approved for HF: Dapagliflozin (Farxiga) 10 mg once daily or Empagliflozin (Jardiance) Oral: 10 mg once daily Diuretics
- mineralocorticoid receptor antagonist (e.g., spironolactone [Aldactone]).
 - Can be added at any time.
 - Example: Start spironolactone at 12.5–50 mg PO QD.
 - Monitor renal function and electrolytes closely.

ACE inhibitors, ARBs, and ARNIs

- with HFrEF, treatment with sacubitril-valsartan was superior to enalapril in reducing the risk of death and hospitalization due to HF.
- ACE is superior to ARB.
- In patients with chronic symptomatic HFrEF NYHA class II or III who tolerate an ACE inhibitor or ARB, replacement with an ARNI is recommended to further reduce morbidity and mortality.
- An ARNI should not be administered concomitantly or within 36 hours of the last dose of an ACE inhibitor, and it should also not be administered to patients with a history of angioedema.

SGLT2 inhibitors





Dapagliflozin is

approved for adults with NYHA class II-IV HFrEF, with or without type 2 diabetes mellitus (T2DM), to decrease the risk of hospitalization for HF and cardiovascular death. Empagliflozin was recently approved to reduce the risk of cardiovascular death and HF hospitalization in adults with HF

2022 ACC/AHA/HFSA Guideline for the Management of Heart Failure - DOI: 10.1016/j.cardfail.2022.02.010

Heidenreich PA, et al. J Card Fail 2022

Primary care: Step 1 and 2 only. Referral might be needed at step one, depending on the provider but definitely at step 4 GDMT: Guideline-Directed Medical Therapy

STEP 1: Management of HFrEF

- medications may be started simultaneously at initial (low) doses recommended for HFrEF even for elderly.
- Or these medications may be started sequentially for frailed patient, with sequence guided by clinical or other factors, without need to achieve target dosing before initiating next medication.
 - Might be preferred for the elderly. Start with ARNI low dose, then add Beta blocker ASAP, MRA, then SGLT2 inhibitors. Titrate up as tolerated and depending on the clinical presentations and comorbidities.

Medication doses should be increased to target as tolerated.

Other new treatment for management of HFrEF

Vericiguat

- The 2022 AHA/ACC/HSFA guideline recommends that in selected high-risk patients with HFrEF and recent worsening of HF already on GDMT, an oral sGC stimulator (vericiguat) may be considered to reduce HF hospitalization and cardiovascular death
- This is beyond step 2 management: Cardiologist should be starting this medication

Management for HFmEF

HF with mildly reduced EF (HFmrEF)

HF with LVEF 41-49%

Recommendations

In patients with HFmrEF, **SGLT2i** can be beneficial in decreasing HF hospitalizations and cardiovascular mortality

Among patients with current or previous symptomatic HFmrEF, use of evidence-based beta blockers for HFrEF, ARNi, ACEi, or ARB, and MRAs may be considered, to reduce the risk of HF hospitalization and cardiovascular mortality, particularly among patients with LVEF on the lower end of this spectrum

HFpEF/Diastolic Heart failure Preserved EF more than 50%.

- In diastolic HF: SGLT2, ACEIs or ARBs (ARBs > ACE1s) ARNI and MRA(spironolactone) can be used to potentially reduce hospitalizations and mortality
- Best approach: Continue to be the management of associated symptoms (HTN...)
- Treatment of HFpEF is largely governed by management of associated conditions and symptoms: hypertension, lung disease, coronary artery disease, atrial fibrillation (AF), obesity, anemia, diabetes mellitus, kidney disease, and sleep disordered breathing
- Goal: Improved ventricular relaxation, decrease heart rate, maintain sinus rhythm and treat HTN to avoid LV remodeling and exacerbation.

Management of HFpEF

HF with preserved EF (HFpEF)

HF with LVEF ≥50%

Recommendations

In patients with HFpEF, **SGLT2i** can be beneficial in decreasing HF hospitalizations and cardiovascular mortality

In selected patients with HFpEF, **MRAs** may be considered to decrease hospitalizations, <u>particularly among patients with LVEF on the lower end</u> of this spectrum

In selected patients with HFpEF, **ARNi** may be considered to decrease hospitalizations, particularly among patients with LVEF on the lower end of this spectrum

Summary

- medical therapies for the treatment of HFrEF and HFpEF is rapidly evolving
- The four pillars ARNI + beta-blocker + MRA + SGLT2 inhibitor are considered the new "standard of care" for HFrEF
- ARNIs and SGLT2 inhibitors have benefits in HF across the spectrum of EF, including in those with HFmrEF and select patients with HFpEF
- Morbidity, mortality, and hospitalizations can be reduced by attention to a multidisciplinary, comprehensive disease management program.

Coronary Artery disease (Taped)

- Prevalence:
 - 6% of American older than 75 years of age and account for 60% CAD-
 - Leading cause of death in elderly
- Age, gender, ethnicity factors
 - Markedly increased prevalence with age
 - Greater incidence in men

Coronary Artery Disease

- Description
 - Imbalance between the supply and demand for blood flow to the myocardium
- Etiology
 - Most often cause by atherosclerosis
 - Imbalance between myocardial demand and coronary perfusion

CAD Manifestation

- Early: asymptomatic
- Stable angina: Predictable discomfort related to exertion (physical or emotional)
- Acute Coronary syndrome
 - Unstable angina: Unpredictable symptomatology, often at night. EKG changes but no enzymes abnormality (Troponin, CPK)
 - NSTEMI
 - STEMI
 - Both NSTEMI and STEMI with EKG changes and enzymes elevations.

CAD

• Epidemiology:

- CAD is the leading cause of death for adults both in the United States and worldwide.
- The cost of CAD in the United States was \$555 billion in 2016 and is expected to rise to \$1.1 trillion by 2035.
- ~80% of CAD is preventable with a healthy lifestyle.

Incidence

In the United States, the lifetime risk of a 40-year-old developing CAD is 49% for men and 32% for women.

Prevalence

 In the United States, 28.4 million people carry a diagnosis of CAD, whereas 7.12 million have angina pectoris.

CAD signal symptoms

- Signal symptoms
 - Substernal Chest pain, tightness, or discomfort frequently radiate to jaw, back or arms last 2 minutes to 15 with stable or unstable angina or constant with STEMI NSTEMI and sometimes unstable angina.
 - Atypical symptoms in the elderly:
 - Dyspnea and exertional fatigue common signal symptoms in the elderly
 - Often with stable angina, Patient may present with non cardiac symptoms and nonspecific symptoms
 - Dyspnea, diaphoresis, fatigue, belching, nausea, dizziness, or indigestion that usually occurs with exertion or stress.

Risk factors

Modifiable Risk factors:

- Hypertension:
- Hyperlipidemia:
- Smoking
- Diet: high in fat, and low in grains and fruits and vegetables
- Diabetes:
- Obesity
- Sedentary life style
- ETOH above recommendations
- Stress

Non Modifiable risk factors

- Genetic
 - First degree relative with CAD
 - Thrombogenic disorders
- Age
 - 45 or above for males
 - 55 or above for females
- Male gender
- Socioeconomic factors
- Type A personality
- Racial minorities :
 - AA (sociecomonic, access to care and genetically higher sensitivity to salt)
 - Hispanic paradox: Higher rate of DM, HTN, obesity but less Heart disease than white.
- Stress

Symptomatic CAD: Stable Angina (class)

- Diagnosis: initial
- Complete blood count, lipid profile, HgbA1c, lipid panel, for risk stratification
- CMP to rule out electrolyte abnormalities and assess renal function
- ECG
- Chest Xray to r/o other causes of pain.
- F/U tests:
- Stress testing is most helpful for patients at intermediate risk of CAD.
- Echocardiogram (FYI: A normal Echo does not r/o CAD)
- CT coronary angiography or cardiac MRI can be considered as a supplement/alternative to stress testing in patients with continued symptoms despite negative stress testing
- Cardiac catheterization with coronary angiography is the gold standard for confirmation and delineation of coronary disease and direction of interventional therapy or surgery.

Prevention and treatment of CAD

- General measures
 - Hypertension: <130/80 mm Hg if tolerated
 - Smoking cessation
 - Diet: Low fat, high in grains, fruits, vegetables and omega 3 fatty acids
 - Hyperlipidemia: Follow ATP IV guidelines
 - Diabetes: A1C less than 7 (though avoid hypoglycemic episodes)
 - Weight management
 - ETOH intake one or less drink equivalent daily

Pharmaceutical treatment of symptomatic CAD

Antianginal therapy:

- Beta blockers: Metoprolol, carvedilol.
 - Decrease O2 demand, and improve symptoms of angina
 - Coreg, Metoprolol start low and titrate slowly.
- CCBs: Dihydropyridine CCBs (nifedipine, Amlodipine) preferred.
 - Arterial vasodilation, decrease O2 demand, and improve coronary blood flow
 - Nondihydropyridine CCBs (diltiazem, verapamil) have negative inotropic effects (decrease HR-> decrease Cardiac output) Do not use specially subtype if concomitant Systolic dysfunction

Nitrates:

- Dilate veins and arteries including Coronary circulation.
- o.4 mg sublingual every 5 minutes for 2 t o3 doses during acute angina symptoms

Long acting Nitrate

- For patient with frequent angina symptoms to prevent angina
- Isosorbide mononitrate start at 30 mg ER daily.
- May cause headache and hypotension but usually improved with continued usage

Pharmaceutical treatment of CAD

Post MI with CAD: Ace or ARB is absolutely indicated and recommended in combo with BB to prevent cardiac remodeling and improve survival rate.

lipid-lowering agents:

- Per guidelines: High-intensity statin therapy is indicated for all patients with CAD regardless of lipid levels.
 - LDL goal: 100 mg/dl the more aggressive goal is <70 mg/dl.
- Statin therapy should also be encouraged for those with high CAD risk (lifetime risk ≥7.5–10%).
- Atorvastatin (20 to 80 mg/day) and rosuvastatin (10 to 40 mg/day) are high-intensity statins. Again think twice about a max dose on elderly patient
- Ezetimibe may be added to statin therapy if LDL is not at goal

Pharmaceutical treatment of CAD

- Antiplatelets: decrease risk of thrombosis
- All patient if not contraindicated should get a daily dose of
- Aspirin (75 to 162 mg/day)
- Clopidogrel (75 mg/day) may be used in patients with contraindications to aspirin.
- Dual antiplatelet therapy with aspirin + clopidogrel, prasugrel, or ticagrelor is indicated after MI or percutaneous coronary intervention (PCI) (use prasugrel only after PCI. Do not use in patient with CVA history).

Ongoing care

- Lifestyle modifications should be aggressively stressed at every visit.
- Compliance with treatment plan. Diet/exercise/Medications/Follow-ups
- Frequent follow-up after initial event: every 3 months in first year and then 1 to 2 times per year