

STATE OF THE ART MANAGEMENT OF HEART FAILURE

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HEART FAILURE

Definition:

- Heart failure is a complex clinical syndrome that can result from any structural or functional cardiac disorder that impairs the ability of the ventricle to fill with or eject blood.

HEART FAILURE

- HF is largely a clinical diagnosis and the syndrome is characterized by specific symptoms (dyspnea and fatigue) in the medical history and signs (edema, rales) on the physical examination.
- There is no single diagnostic test that “defines” HF.

HEART FAILURE

- Because not all patients have volume overload at the time of initial or subsequent evaluation, the term “heart failure” is preferred over the older term “congestive heart failure.”.

HEART FAILURE

- The clinical syndrome of HF may result from disorders of the pericardium, myocardium, endocardium, or great vessels.

HEART FAILURE

Clinical syndrome of HF may be caused by:

- Impaired LV myocardial function
- Valvular heart disease
- Infiltrative/Restrictive cardiomyopathies
- Hypertrophic cardiomyopathy
- Pericardial constriction or effusion
- Congenital heart disease
- Right ventricular dysfunction
- Other miscellaneous causes

HEART FAILURE

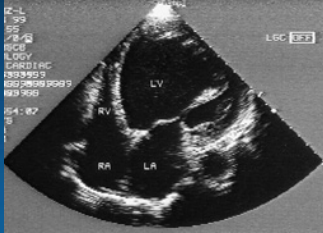
- It should be emphasized that HF is not equivalent to cardiomyopathy or to LV dysfunction; these latter terms describe possible structural or functional reasons for the development of HF.
- Even though majority of patients with HF have symptoms due to an impairment of LV myocardial function.

HEART FAILURE

- Impaired LV myocardial function could be LV systolic or diastolic function (HFNEF).
- In most patients, abnormalities of systolic and diastolic dysfunction coexist.
- HF patients with normal ejection fraction (HFNEF) may have a different natural history and may require different treatment strategies than patients with reduced EF.

HEART FAILURE

Left Ventricular Systolic Dysfunction



HEART FAILURE

At Risk for Heart Failure:

STAGE A High risk for developing HF

STAGE B Asymptomatic LV dysfunction

Heart Failure:

STAGE C Past or current symptoms of HF

STAGE D End-stage HF

HEART FAILURE

- Pharmacotherapy
- Device therapy
- Surgical therapy
- Adjunctive therapies
-Not so helpful therapies!

PHARMACOTHERAPY

- Left ventricular dysfunction begins with some injury to, or stress on, the myocardium and is generally a progressive process, even in the absence of a new identifiable insult to the heart.
- The principal manifestation of such progression is a change in the geometry and structure of the LV, such that the chamber dilates and/or hypertrophies and becomes more spherical—a process referred to as cardiac remodeling.

PHARMACOTHERAPY

- Although several factors can accelerate the process of LV remodeling, there is substantial evidence that the activation of endogenous neurohormonal systems plays an important role in cardiac remodeling and thereby in the progression of HF.
- Neurohormonal activation basically means elevated circulating or tissue levels of norepinephrine, angiotensin II, aldosterone, endothelin, vasopressin, and cytokines, which are seen in patients with HF.

PHARMACOTHERAPY

- These neurohormonal factors not only increase the hemodynamic stresses on the ventricle by causing sodium retention and peripheral vasoconstriction but may also exert direct toxic effects on cardiac cells and stimulate myocardial fibrosis, which can further alter the architecture and impair the performance of the failing heart.



PHARMACOTHERAPY

Goals of Pharmacotherapy

- Symptomatic relief: by affecting sodium retention/fluid loss, peripheral vasoconstriction and increasing cardiac contractility.
- Mortality benefit: still unclear of the target(s) for this benefit but interestingly all the medications imparting mortality benefit have a favorable effect on cardiac remodeling.

PHARMACOTHERAPY

- Vasodilators
- Beta blockers
- Aldosterone receptor blockers
- Statins
- Aspirin
- Diuretics
- Digoxin
- Calcium Channel blockers
- Coumadin

PHARMACOTHERAPY

- Vasodilators
- Beta blockers
- Aldosterone receptor blockers
- Statins
- Aspirin (ischemic cardiomyopathy)
- Diuretics
- Digoxin
- Calcium Channel blockers
- Coumadin

PHARMACOTHERAPY

Vasodilators:

- ACE-I
- ARB
- Isosorbide dinitrate / hydralazine (Bidil)

Key points:

- HF patient should be on at least one of these medications or combination of medications.
- Uptitration to goal dosages is necessary to attain maximal mortality benefit.

PHARMACOTHERAPY

Drug	Initial Daily Dose(s)	Maximum Dose(s)
ACE Inhibitors		
Captopril	6.25 mg 3 times	50 mg 3 times
Enalapril	2.5 mg twice	10 to 20 mg twice
Fosinopril	5 to 10 mg once	40 mg once
Lisinopril	2.5 to 5 mg once	20 to 40 mg once
Perindopril	2 mg once	8 to 16 mg twice
Quinapril	5 mg twice	20 mg twice
Ramipril	1.25 to 2.5 mg once	10 mg once
Trandolapril	1 mg once	4 mg once
Angiotensin Receptor Blockers		
Candesartan	4 to 8 mg once	32 mg once
Losartan	25 to 50 mg once	50 to 100 mg once
Valsartan	20 to 40 mg twice	160 mg twice

PHARMACOTHERAPY

Beta Blockers

- Carvedilol (Coreg)
- Metoprolol succinate (Toprol XL)
- Bisoprolol

Key Points

- Should not be started or uptitrated when patient is volume overloaded.
- Gradual uptitration is the key and every two to four weeks is an acceptable interval.
- Goal dosages give maximal mortality benefit.

PHARMACOTHERAPY

Beta-Blockers		
Bisoprolol	1.25 mg once	10 mg once
Carvedilol	3.125 mg twice	25 mg twice
		50 mg twice for patients over 85 kg
Metoprolol succinate extended release (metoprolol CR/XL)	12.5 to 25 mg once	200 mg once

PHARMACOTHERAPY

Aldosterone receptor blockers

- Spironolactone
- Eplerenone (only post MI LV dysfunction)

Key points

- At this point indicated for NYHA class III or IV.
- Contraindicated in renal failure and hyperkalemia
(Creatinine should be less than or equal to 2.5 mg/dL in men or less than or equal to 2.0 mg/dL in women and potassium should be less than 5.0 mEq/L)

PHARMACOTHERAPY

Drug	Initial Daily Dose(s)	Maximum Dose(s)
Aldosterone Antagonists		
Spironolactone	12.5 to 25 mg once	25 mg once or twice
Eplerenone	25 mg once	50 mg once

PHARMACOTHERAPY

- Statins
 - Indicated both in ischemic or non-ischemic cardiomyopathy regardless of the cholesterol level.
- Aspirin
 - Indicated in ischemic cardiomyopathy.

PHARMACOTHERAPY

- Diuretics
- Preferably loop diuretics are used for congestive symptoms.
 - Addition of a thiazide diuretic sometimes necessary to promote excess diuresis, for sequential nephron blockade.

PHARMACOTHERAPY

Drug	Initial Daily Dose(s)	Maximum Total Daily Dose	Duration of Action
Loop diuretics			
Bumetanide	0.5 to 1.0 mg once or twice	10 mg	4 to 6 hours
Furosemide	20 to 40 mg once or twice	600 mg	6 to 8 hours
Torsemide	10 to 20 mg once	200 mg	12 to 16 hours
Thiazide diuretics			
Chlorthalidone	250 to 500 mg once or twice	1000 mg	6 to 12 hours
Chlorthalidone	12.5 to 25 mg once	100 mg	24 to 72 hours
Hydrochlorothiazide	25 mg once or twice	200 mg	6 to 12 hours
Indapamide	2.5 once	5 mg	36 hours
Metolazone	2.5 mg once	20 mg	12 to 24 hours
Sequential nephron blockade			
Metolazone	2.5 to 10 mg once plus loop diuretic		
Hydrochlorothiazide	25 to 100 mg once or twice plus loop diuretic		
Chlorthalidone (IV)	500 to 1000 mg once plus loop diuretic		

PHARMACOTHERAPY

Digoxin

- Provides symptomatic benefit and has been shown to reduce hospitalizations.
- Start therapy at 0.125 mg PO daily for normal renal function and no uptitration necessary. Dosage should be reduced or medication not used in renal failure.
- Maximal benefit in HF patients with atrial arrhythmias and in this case dose can be up to 0.25mg daily with normal renal function.
- If HF patient on digoxin it should not be withdrawn until EF normalizes or toxicity seen.
- Compared to men not as beneficial in women.

PHARMACOTHERAPY

Calcium Channel blockers

- Dihydropyridine calcium antagonists, preferably Norvasc, are safe to use in HF patients for added BP control but provide neutral effect on mortality.

Coumadin

- Routine use of coumadin in low EF is not recommended but is obviously indicated with atrial arrhythmias or in some case of ventricular aneurysms or LV thrombus.

DEVICE THERAPY

Implantable Cardioverter-Defibrillator (ICD)
Cardiac resynchronization therapy (CRT)

ICD

- Sudden cardiac death claims about 335,000 lives each year in the United States, according to the American Heart Association.
- The most common heart-rhythm disorders (arrhythmias) that lead to cardiac arrest are:
 - Ventricular tachycardia
 - Ventricular fibrillation

ICD

- An implantable cardioverter-defibrillator (ICD) continuously monitors your heartbeat and delivers precisely calibrated electrical shocks to restore a normal heart rhythm.

WHO BENEFITS FROM AN ICD

Group Addressed in Recommendation	2005 ACC/AHA HF
LVD d/t MI, LVEF 30% or less, NYHA II, III	<i>Class I, LOE: B</i>
LVD d/t MI, LVEF 30% to 35%, NYHA II, III	<i>Class IIa, LOE: B</i>
LVD d/t MI, LVEF 30% to 40%, NSVT, positive EP study	N/A
LVD d/t MI, LVEF 30% or less, NYHA I	<i>Class IIa, LOE: B</i>
LVD d/t MI, LVEF 21% to 35% or less, NYHA I	N/A
NICM, LVEF 30% or less, NYHA II, III	<i>Class I, LOE: B</i>
NICM, LVEF 30% to 35%, NYHA II, III	<i>Class IIa, LOE: B</i>
NICM, LVEF 30% or less, NYHA I	<i>Class IIb, LOE: C</i>
NICM, LVEF 31% to 35% or less, NYHA I	N/A

WHO BENEFITS FROM AN ICD

Bottom line.....

- Non-ischemic cardiomyopathy with LVEF \leq 35%.
- Ischemic cardiomyopathy with LVEF \leq 35%.
- Ischemic cardiomyopathy with LVEF between 35-40% with either NSVT or a positive EP study.

ICD

Waiting periods to put the device:

- For NICM, ranges from 3 - 9 months of optimal therapy without improvement in LVEF.
- For ICM, at least 40 days after MI or revascularization with optimal medical therapy and no improvement in LVEF.

CRT

- Studies have shown that the presence of ventricular dyssynchrony (uncoordinated beating of the left and right ventricles) in HF patients promotes LV enlargement (**cardiac remodeling**), leading to further functional decline, and puts patients at risk of decompensated HF, potentially life-threatening arrhythmias, and death.

CRT

- Cardiac resynchronization therapy (CRT) utilizes a triple-lead biventricular pacing device to correct ventricular dyssynchrony in patients with left ventricular dilatation and systolic dysfunction.



CRT

- How to determine if a HF patient has ventricular dyssynchrony?
- RCT's have used prolongation of QRS duration as a measure of dyssynchrony, basically suggesting there is delay in activation of one or the other ventricles.

CRT

- The clinical evidence to date demonstrates that CRT can:
- Restore LV synchrony
 - Increase functional capacity
 - Improve quality of life
 - Reduce HF hospitalizations
 - Potentially reduce all-cause mortality in selected patients.

WHO BENEFITS FROM CRT

- Non-ischemic or ischemic cardiomyopathy with LVEF \leq 35%.
- QRS duration of more than or equal to 120 msec.
- Symptomatic HF mainly class III or IV and may include class II also.
- Waiting periods are the same as for ICD's.

PITFALLS WITH CRT

- Although CRT has been shown to benefit the vast majority, between 20% and 30% of patients who receive the devices do not respond well to therapy.
- Potential problems could be how we are measuring dyssynchrony, using it too late or waiting for symptoms to develop.
- Secondly once the device is inserted we need to "tune it up" and keep following it.

CRT

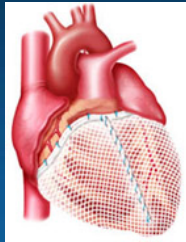
- Recent studies have started using echocardiographic measures of looking at dyssynchrony to screen for eligibility of CRT, which appears more promising.
- More importantly optimizing the A-V and V-V delays under echo guidance.
- Lastly looking at early signs of reverse remodelling to measure effect of CRT and revisiting optimization on follow ups.

SURGICAL THERAPY

Coronary revascularization
Mitral valve repair
Ventricular remodelling surgery
Acorn device
Bariatric surgery
Left ventricular assist device (LVAD)
Biventricular assist device (BiVAD)

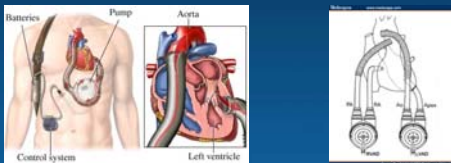
SURGICAL THERAPY

Acorn device (CorCap)



SURGICAL THERAPY

Left ventricular assist device (LVAD)
Biventricular assist device (BiVAD)



ADJUNCTIVE THERAPIES

- Heart failure clinic referral
- Enhanced External Counterpulsation (EECP)
- Non-invasive hemodynamic monitoring
- Sleep apnea treatment
- Psychosocial assessment
- Exercise prescription
- Continuous aortic flow augmentation (Cancion device)
- Inspiratory muscle training

HEART FAILURE CLINIC

There are three elements that are crucial to the success of these programs.

- First, there must be ready access to physicians trained in HF.
- Second, specially trained HF nurses.
- Finally, efforts should be made to educate patients and their caregivers about HF, precipitating factors, and need for compliance with medication and dietary advice

HEART FAILURE CLINIC

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Heart Failure

Multidisciplinary Strategies for the Management of Heart Failure Patients at High Risk for Admission

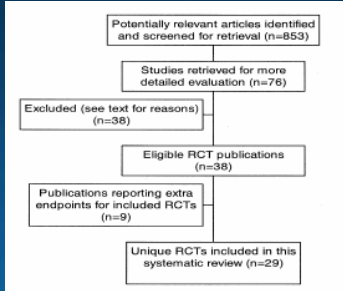
A Systematic Review of Randomized Trials

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HEART FAILURE CLINIC



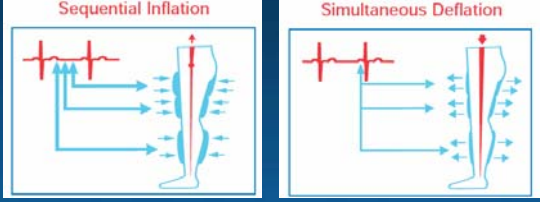
HEART FAILURE CLINIC

- Multidisciplinary strategies for the management of patients with HF reduce mortality and all-cause hospitalizations.
- According to the ACC guidelines, referral to a specialized heart failure clinic is a Class I indication, especially for patients with Stage D HF.



ECCP

- Compressive cuffs (similar to large blood pressure cuffs) are securely wrapped around the patient's calves, thighs and buttocks.
- These cuffs inflate in a distal to proximal sequence in early diastole, and deflate simultaneously in late diastole just prior to the onset of systole.
- Inflation and deflation are specifically timed to the patient's ECG to optimize therapeutic benefit.




EECP

Sequential Inflation


Simultaneous Deflation

The diagram illustrates two methods of External Counterpulsation (EECP). On the left, 'Sequential Inflation' shows a series of cuffs on the lower leg inflating one after another, creating a retrograde pressure wave that travels up the leg. On the right, 'Simultaneous Deflation' shows all cuffs inflating together and then deflating rapidly at the same time, which decreases systemic vascular resistance and afterload.



EECP


- The sequential cuff inflation creates a retrograde pressure wave that augments diastolic pressure, increasing coronary perfusion pressure and venous return to the right heart.
- Rapid, simultaneous cuff deflation decreases systemic vascular resistance, afterload, and cardiac workload.
- Patients typically undergo 35 one hour sessions to complete treatment.



EECP

Although the specific mechanisms responsible for the beneficial clinical effects of EECP therapy remain unclear, recent studies have demonstrated:

- Enhanced peripheral endothelial function.
- Improved stress myocardial perfusion both at baseline and at maximal exercise levels
- Reduced plasma levels of BNP.
- Improved regional myocardial oxygen metabolism.



EECP

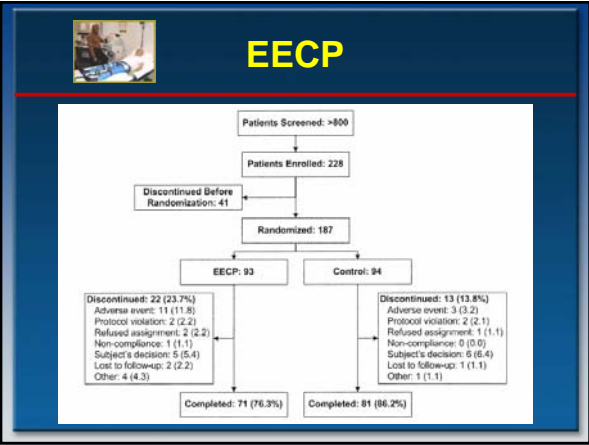
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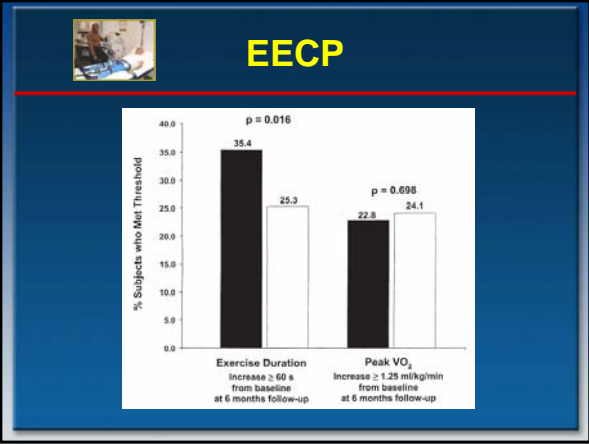
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Enhanced External Counterpulsation

Enhanced External Counterpulsation Improves Exercise Tolerance in Patients With Chronic Heart Failure

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Not so Helpful therapies

Outpatient IV natreacor infusion
Intermittent inotropic infusion
Continuous inotropic infusion
Vasopressin receptor antagonists
Hawthorn extract
Immunosuppressive therapy
Antiviral therapy
Growth hormone
Pentoxifylline

Ineffective therapies

Endothelin receptor antagonist
Vasopeptidase inhibitors
Flosequinan
Moxonidine
Coenzyme Q10
Anti-TNF alpha

CONCLUSION

- HF is a clinical diagnosis.
- Syndrome of HF can be caused by a variety of structural heart and vascular diseases.
- HFNEF not completely understood and requires additional investigation.
- LV systolic dysfunction is a progressive disorder linked to neurohormonal activation and cardiac remodelling.
- Cardiac remodelling is an important target for therapy and to assess response to therapy.

CONCLUSION

- Goals of management for HF remain symptomatic relief and mortality benefit for all types of therapy.
- Early HF clinic referral has an impact not only on morbidity but reduces mortality.
- EECF is a novel therapy and has possible benefits symptomatically.
- Non-invasive hemodynamic monitoring in clinic setting can add to clinical assessment and help guide therapy.

Questions?
