Heart Failure 101

the first in a collaborative Heart Failure webinar series by

GREAT PLAINS QUALITY INNOVATION
LAKE SUPERIOR QUALITY INNOVATION NETWORK

June 20, 2017
Welcome and Reminders

- Welcome!
- Thank you!
- Links to slides and recording will be available on our websites
  - [http://greatplainsqin.org](http://greatplainsqin.org) and [https://www.lsqin.org/](https://www.lsqin.org/)
- *6 to mute your line - *6 to unmute
- Utilize chat
Heart Failure 101

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Objectives

• Describe what heart failure is including its causes and progression.

• Understand the treatment of heart failure and the medications involved in both the chronic and acute setting

• Discuss heart failure prevention including prevention of recurrent exacerbations and readmissions
Heart Failure – Some Facts

• Heart Failure affects nearly 5 million Americans
• Heart Failure is the ONLY cardiovascular disorder on the rise
• An estimated 400,000 – 700,000 new cases are diagnosed each year
• Deaths from Heart Failure have more than doubled since 1979 averaging 250,000 annually
• Research funding is disproportionate to the numbers
  – 5 million Americans with Heart Failure = $28.7 million in research dollars
  – 390,000 Americans with Lung Cancer = $132 million in research dollars
Congestive Heart Failure

**Heart (or cardiac) failure** is the state in which the heart is unable to pump blood at a rate commensurate with the requirements of the tissues or can do so only from high pressures.

Type of Heart Failure Matters as it Determines Treatment

- **Systolic Heart Failure**: Less blood pumped out of ventricles, weakened heart muscle can’t squeeze as well.
- **Normal Heart**: No heart failure.
- **Diastolic Heart Failure**: Stiff heart muscle can’t relax normally.
Heart Failure with Reduced Ejection Fraction

- Weakening of the heart muscle for a variety of reasons
- Heart pumps ineffectively which can lead to fluid retention and symptoms of congestion
- Goal of therapy is to protect the heart from stress hormones, reverse underlying cause if possible and maintain fluid balance
- Increased risk of sudden cardiac death should EF stay below 35% despite optimal therapy.
Heart Failure with Preserved Ejection Fraction

- Preserved/normal strength of the heart
- Difficulty with filling time and relaxation of the heart muscle
- Poorly understood
- Goal of therapy is to control heart rate, control blood pressure and maintain fluid status
- Control other conditions which may contribute to the condition (blood pressure, sleep apnea, body weight)
Left Sided Congestive Heart Failure

- **Etiologies**
  - Ischemic heart disease
  - Alcohol/drug abuse
  - Valvular heart disease
  - Hypertension
  - Viral
  - Idiopathic
  - Tachyarrhythmias
  - Post/peri-partum
  - Genetic

- **Manifestations**
  - Congestion
    - Pulmonary edema
  - Fatigue
  - Orthopnea
  - PND
  - Arrhythmias
  - Weight gain
  - Cough
Right Sided Congestive Heart Failure

- **Etiologies**
  - Left sided heart failure
  - Chronic lung disease
  - Pulmonary hypertension
  - Congenital heart disease
  - Pulmonary embolism
  - Valvular heart disease

- **Manifestations**
  - Congestion
    - liver
    - GI tract
    - Limbs
  - Inability to pump to lungs/left side of heart
Case Study – A.D. 57 year old African American Male

- Admitted to hospital 7/24/2008 with dyspnea, lower extremity pain and edema
- CT of chest positive for pulmonary emboli, subacute bilateral pleural effusions, RLL infiltrate, moderately severe emphysema.
- Echocardiogram obtained: EF 10%, severe mitral regurgitation. LVEDD 70mm (normal 43-52), Right ventricle markedly dilated with markedly reduced function.
- Cardiac PET scan negative for ischemia.
- Developed acute renal insufficiency (Cr 1.8 GFR 43)
- Discharged 8/1/2008 on Coumadin, Aspirin and Coreg 3.125mg bid.
Comorbid Conditions

- Sleep apnea
- Microcytic anemia
- Deep vein thrombosis
- Severe peripheral vascular disease
- Tobacco dependence
- Alcohol dependence
- Impaired fasting glucose
First Clinic Visit - Subjective

- Dyspnea at 1/2 block
- Abdominal fullness
- Early satiety
- Peripheral edema
- Weight gain
- Denied orthopnea/PND
First Visit - Objective

• BP 108/75, HR 100, RR 22, O2 sat > 90%
• Ten pound weight gain since admission
• Lungs diminished throughout with poor air exchange
• S3, Grade 3/6 SEM at apex radiating to axilla
• Abdomen distended and firm/ascites
• +2-+3 bilateral peripheral edema to thighs
• JVD to crux of jaw at 90 degrees.
• Pro- BNP 9588 (N 0-125)
Where to Start? – Look for Reversible Causes

- Coronary artery disease had been ruled out
- Pulmonary Embolism
  - assure adequate anticoagulation
- Alcoholism
  - Emphasis on abstinence
  - Assess need for chemical dependency consult
- Sleep apnea
  - Emphasis on CPAP compliance
- Tachycardia
  - Work on rate control
    - Be cautious of compensatory tachycardia
- Anemia
  - Keep hemoglobin > 10
Evidence-Based Treatment Across the Continuum of Systolic LVD and HF

**Control Volume**
- Diuretics
  - Renal Replacement Therapy*

**ACEI**
- ARB
- ARNI

**Improve Clinical Outcomes**
- β-Blocker
- Aldosterone Antagonist

**Treat Residual Symptoms**
- CRT ±
  - an ICD*
  - HDZN/ISDN*
  - Ivabradine

**Digoxin**
Pharmacologic Therapy: Diuretics

• Diuretic therapy is recommended to restore and maintain normal volume status in patients with clinical evidence of fluid overload, generally manifested by:
  – Congestive symptoms
  – Signs of elevated filling pressures

  Strength of Evidence = A

• Loop diuretics rather than thiazide-type diuretics are typically necessary to restore normal volume status in patients with HF.

  Strength of Evidence = B
The Donkey Analogy:

Ventricular dysfunction impairs the patient’s ability to perform routine activities of daily living
Diuretic, ACE Inhibitors, ARB and ARNI therapy

Reduce the number of sacks on the wagon – lighten the load
# Loop Diuretics

<table>
<thead>
<tr>
<th>Agent</th>
<th>Initial Daily Dose</th>
<th>Max Total Daily Dose</th>
<th>Elimination: Renal – Met.</th>
<th>Duration of Action</th>
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Aldosterone Antagonist Therapy

• An aldosterone antagonist is recommended for patients on standard therapy, including diuretics, who have:
  – NYHA class II-IV HF with reduced LVEF (≤ 35%)
  – HF with preserved LVEF (≥ 45%) with elevated BNP levels or heart failure admission in last year

• Provided:
  – GFR >30 mL/min
  – Creatinine <2.5 mg/dL
  – Potassium <5.0 mEq/L

• Monitor renal function at 3 days, 1 week, monthly x3, then every 3 months
Aldosterone Antagonists in HF

RALES (Advanced HF)  EPHESUS (Post-MI)

Spironolactone  Placebo

RR = 0.70  P < 0.001

RR = 0.85  P < 0.008

## Potassium Sparing Diuretics

<table>
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</table>
Back to our Patient

- Started on Lasix 40mg daily
  - Titrated over the course of the week
    - Lasix 80mg in the am / 40mg in the afternoon
- Spironolactone 12.5mg daily
- Digoxin 0.125mg every other day.
  - Increased to daily as patient’s renal function remained stable.
- Encouraged adequate anticoagulation
Why Digoxin? Only in Select Patients!

Control Volume
- Diuretics
  - Renal Replacement Therapy*
- ACEI
- ARB
- ARNI

Improve Clinical Outcomes
- β-Blocker
- Aldosterone Antagonist
- CRT ±
- an ICD*
- HDZN/ISDN*
- Ivabradine

Treat Residual Symptoms
- Digoxin
2013 ACCF/AHA Guideline for the Management of Heart Failure: Digoxin

- Clinicians may consider adding digoxin in patients with persistent symptoms of HFrEF during GDMT. Digoxin may also be added to the initial regimen in patients with severe symptoms who have not yet responded symptomatically during GDMT.

- Initiate and maintain at a dose of 0.125 to 0.25 mg daily.

- Low doses (0.125 mg daily or every other day) should be used initially if the patient is >70 years of age, has impaired renal function, or has a low lean body mass.
Digoxin

What does it do?

• Enhances inotropy of cardiac muscles
• Reduces activation of SNS and RAAS
• Controlled trials have shown long term therapy:
  – Reduces symptoms
  – Increases exercise tolerance
  – Improves hemodynamics
  – Decreases risk of heart failure progression
  – Reduces hospitalization rates for decompensation
  – Does not improve survival
A.D – Ten Days Later

- Weight is down 19 pounds
- Dyspnea at ¾ block
- Swelling improved but continues:
  - +2 on right / +1 on left
- 2 pillow orthopnea
- Cr 1.1, BUN 20, Chloride 98, K+ 3.8
- BP 123/89
- HR 115
- INR 1.7
Evidence-Based Treatment Across the Continuum of Systolic LVD and HF

Control Volume

Diuretics
Renal Replacement Therapy*

Improve Clinical Outcomes

ACEI
ARB
ARNI

β-Blocker
Aldosterone Antagonist

CRT ±
an ICD*
HDZN/ISDN*
Ivabradine

Treat Residual Symptoms

Digoxin

*Essentia Health Here with you
2013 ACCF/AHA Guideline for the Management of Heart Failure: Beta Blockers

• Use of 1 of the 3 beta blockers proven to reduce mortality (eg, bisoprolol, carvedilol, and sustained-release metoprolol succinate) is recommended for all patients with current or prior symptoms of HFrEF, unless contraindicated, to reduce morbidity and mortality. (Level of Evidence: A)

• Beta blockers shown to be effective in clinical trials are recommended for symptomatic and asymptomatic patients with an LVEF ≤ 40%. (Level of Evidence: A)

• Cardioprotective effects due to the blockade of excessive SNS stimulation

• In the short term, beta blocker decreases myocardial contractility, it’s effects on EF improvement are generally seen after 1-3 months of treatment
Beta Blockers – Limit the Donkey’s speed – saving energy
# Effect of Beta Blockade on Outcomes in Patients With HF and Post-MI LV Dysfunction

<table>
<thead>
<tr>
<th>Study</th>
<th>Drug</th>
<th>HF Severity</th>
<th>Target Dose (mg)</th>
<th>Outcome</th>
</tr>
</thead>
</table>

Work to get to target doses!

General considerations

If symptoms worsen or other side effects appear

If up-titration continues to be difficult
2013 ACCF/AHA Guideline for the Management of Heart Failure: ACE/ARB

• ACE inhibitors can reduce the risk of death and reduce hospitalization in HFrEF. The benefits of ACE inhibition were seen in patients with mild, moderate, or severe symptoms of HF and in patients with or without CAD. ACE inhibitors should be prescribed to all patients with HFrEF.

• Clinicians should prescribe an ACE inhibitor with caution if the patient has very low systemic blood pressures (systolic blood pressure <80 mm Hg), markedly increased serum levels of creatinine (>3 mg/dL), bilateral renal artery stenosis, or elevated levels of serum potassium (>5.0 mEq/L).

• Clinicians should attempt to use doses that have been shown to reduce the risk of cardiovascular events in clinical trials.

• ARBs are recommended in patients with HFrEF with current or prior symptoms who are ACE inhibitor intolerant, unless contraindicated, to reduce morbidity and mortality.108,345,415,450 (Level of Evidence: A)
ACE Inhibitors in Heart Failure: From Asymptomatic LVD to Severe HF

<table>
<thead>
<tr>
<th>SOLVD Prevention (Asymptomatic LVD)</th>
<th>CONSENSUS (Severe Heart Failure)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20% death or HF hosp.</td>
<td>40% mortality at 6 mos.</td>
</tr>
<tr>
<td>29% death or new HF</td>
<td>31% mortality at 1 year</td>
</tr>
<tr>
<td>SOLVD Treatment (Chronic Heart Failure)</td>
<td>27% mortality at end of study</td>
</tr>
<tr>
<td>16% mortality</td>
<td>▪ No difference in incidence of sudden cardiac death</td>
</tr>
</tbody>
</table>

New Kids On the Block

Sacubitril/Valsartan (Entresto)
Ivabradine (Corlanor)
LCZ696: Angiotensin Receptor Neprilysin Inhibition

LCZ696

Angiotensin receptor blocker + Inhibition of neprilysin

Essentia Health
Here with you
Neprilysin Inhibition Potentiates Actions of Endogenous Vasoactive Peptides That Counter Maladaptive Mechanisms in Heart Failure

**Endogenous vasoactive peptides**
(natriuretic peptides, adrenomedullin, bradykinin, substance P, calcitonin gene-related peptide)

Neprilysin

Inactive metabolites

Neprilysin inhibition

- Neurohormonal activation
- Vascular tone
- Cardiac fibrosis, hypertrophy
- Sodium retention
Angiotensin–Neprilysin Inhibition versus Enalapril in Heart Failure

John J.V. McMurray, M.D., Milton Packer, M.D., Akshay S. Desai, M.D., M.P.H., Jianjian Gong, Ph.D., Martin P. Lefkowitz, M.D., Adel R. Rizkala, Pharm.D., Jean L. Rouleau, M.D., Victor C. Shi, M.D., Scott D. Solomon, M.D., Karl Swedberg, M.D., Ph.D., and Michael R. Zile, M.D., for the PARADIGM-HF Investigators and Committees*

(all comparisons are versus enalapril 20 mg daily, not versus placebo)
PARADIGM-HF: Cardiovascular Death or Heart Failure Hospitalization (Primary Endpoint)

Kaplan-Meier Estimate of Cumulative Rates (%)

Days After Randomization

Patients at Risk

<table>
<thead>
<tr>
<th></th>
<th>LCZ696</th>
<th>Enalapril</th>
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</thead>
<tbody>
<tr>
<td>LCZ696</td>
<td>4187</td>
<td>4212</td>
</tr>
<tr>
<td>Enalapril</td>
<td>3922</td>
<td>3883</td>
</tr>
<tr>
<td>3663</td>
<td>3579</td>
<td></td>
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<tr>
<td>3018</td>
<td>2922</td>
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<tr>
<td>2257</td>
<td>2123</td>
<td></td>
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<tr>
<td>1544</td>
<td>1488</td>
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<tr>
<td>896</td>
<td>853</td>
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<tr>
<td>249</td>
<td>236</td>
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</table>

Enalapril (n=4212)

LCZ696 (n=4187)

HR = 0.80 (0.73-0.87)

P = 0.0000002

Number needed to treat = 21
PARADIGM-HF: Cardiovascular Death

- **Enalapril** (n=4212): HR = 0.80 (0.71-0.89), P = 0.00004, Number need to treat = 32

- **LCZ696** (n=4187)

<table>
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<tr>
<th>Days After Randomization</th>
<th>Patients at Risk</th>
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<tr>
<td></td>
<td>LCZ696</td>
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<tr>
<td>0</td>
<td>4187</td>
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<tr>
<td>180</td>
<td>4056</td>
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<td>360</td>
<td>3891</td>
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<td>540</td>
<td>3282</td>
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<td>720</td>
<td>2478</td>
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<td>900</td>
<td>1716</td>
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<tr>
<td>1080</td>
<td>1005</td>
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<tr>
<td>1260</td>
<td>693</td>
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</table>
In heart failure with reduced ejection fraction, when compared with recommended doses of enalapril:

LCZ696 was more effective than enalapril in . . .

- Reducing the risk of CV death and HF hospitalization
- Reducing the risk of CV death by incremental 20%
- Reducing the risk of HF hospitalization by incremental 21%
- Reducing all-cause mortality by incremental 16%
- Incrementally improving symptoms and physical limitations

LCZ696 was better tolerated than enalapril . . .

- Less likely to cause cough, hyperkalemia or renal impairment
- Less likely to be discontinued due to an adverse event
- More hypotension, but no increase in discontinuations
- Not more likely to cause serious angioedema
†Hydral-Nitrates green box: The combination of ISDN/HYD with ARNI has not been robustly tested. BP response should be carefully monitored.
‡See 2013 HF guideline.
§Participation in investigational studies is also appropriate for stage C, NYHA class II and III HF.
ACEI indicates angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor-blocker; ARNI, angiotensin receptor-neprilysin inhibitor; BP, blood pressure; bpm, beats per minute; CI/i, contraindication; COR, Class of Recommendation; CrCl, creatinine clearance; CRT-D, cardiac resynchronization therapy–device; Dx, diagnosis; GDMT, guideline-directed management and therapy; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; ICD, implantable cardioverter-defibrillator; ISDN/HYD, isosorbide dinitrate hydral-nitrates; K+, potassium; LBBB, left bundle-branch block; LVAD, left ventricular assist device; LVEF, left ventricular ejection fraction; MI, myocardial infarction; NSR, normal sinus rhythm; and NYHA, New York Heart Association.

Yancy et al, 2017 ACC/AHA/HFSA Heart Failure Focused Update
Systolic Heart failure treatment with the If inhibitor ivabradine Trial

SHIFT Trial

Ivabradine and outcomes in chronic heart failure (SHIFT): a randomised placebo-controlled study

Karl Swedberg, Michel Komajda, Michael Böhm, Jeffrey S Borer, Ian Ford, Ariane Dubost-Brama, Guy Lerebours, Luigi Tavazzi, on behalf of the SHIFT Investigators*

http://www.lancet.com published online August 29, 2010 DOI:10.1016/S0140-6736(10)61198-1
Background of SHIFT Trial

• Elevated heart rate is associated with poor outcome in a number of cardiovascular conditions including heart failure

• Heart rate remains elevated in many heart failure patients despite treatment by beta-blockers

• Ivabradine is a novel heart rate-lowering agent acting by inhibiting the $I_f$ current in the sino-atrial node

• It was hypothesized that the addition of ivabradine to recommended therapy would be beneficial in heart failure patients with elevated heart rate
Hospitalizations for Heart Failure

Ivabradine n=514 (9.4%PY)  Placebo n=672 (12.7%PY)

HR = 0.74, P < 0.001

-26%
Time to first event of primary composite endpoint (Cardiovascular death or Heart Failure Hospitalization)

Placebo + SOC: 937 events \( [n = 3,264] \)

Corlanor + SOC: 793 events \( [n = 3,241] \)

\[ HR \{95\% CI \} = 0.82 \{0.75 - 0.90\} \]

18% ARR

\( P < 0.001 \)
Treatment of HFrEF Stage C and D

Yancy et al, 2017 ACC/AHA/HFSA Heart Failure Focused Update
Contraindications of Corlanor

- Acute decompensated HF
- Blood pressure < 90/50
- Sick sinus syndrome, AV block without the protection of a PM
- Resting heart rate < 60
- Severe hepatic impairment
- PM set to HR > 70
- Concomitant use of strong P450 3A4 (CYP3A4) inhibitors
How about our patient??

In the Clinic......

• Initiated Lisinopril 2.5mg daily

• Coreg increased to 6.25 mg twice a day

• Enrolled in our Coumadin Clinic

• Digoxin increased to daily

• Appointment in 1 week with lab prior

One week later......

• Patient able to walk 4 blocks

• No PND / orthopnea

• No peripheral edema

• HR 96

• Pro-BNP 3566

• Coreg increased to 12.5mg twice a day

• Potassium increased to 5.0 – started on low potassium diet. Recheck K+ in 1 week
Two Weeks after That

- Patient with no complaints
- Remains euvoletic on exam
- HR decreased to 76 BP stable
- K+ increased to 5.5 on recheck and spironolactone discontinued
- Increased Coreg to 25mg bid
- Unable to further titrate lisinopril secondary to continued hyperkalemia
Echo after 3 months of optimal medical management.

At Diagnosis

Following Therapy
Mitral Regurgitation

At Diagnosis

After Therapy
How about inpatient management? Same approach!

- Look for reversible causes and treat
- Assess patient and determine appropriate treatment.
- Decongest/Diurese
- Start guideline based therapy
- Prepare for discharge and anticipate barriers to optimal management.
Box 1: Proposed causes of acute decompensated heart failure due to left ventricular dysfunction

**Primary cardiac**
- Progressive cardiomyopathy with remodelling
- Acute cardiomyopathy (myocarditis, postpartum cardiomyopathy)
- Myocardial ischemia
- Arrhythmia (tachy- or bradyarrhythmia)
- Valvular dysfunction (stenosis or regurgitation)
- Pericardial syndrome (tamponade, constriction)

**Pressure overload**
- Hypertensive urgency or emergency

**Volume overload**
- Sodium or volume load
- Decreased compliance with diuretics
- Renal dysfunction
- Hepatic dysfunction

**High output**
- Shunt (intra- or extracardiac)
- Anemia
- Septicemia
- Thyroid disease

**Other**
- Inflammation or infection
- Major surgery
- Lack of compliance with heart failure medications
- New medications (excess β-blockade)
- Substance abuse (alcohol, stimulants)
“Warm or Cold?.........................Wet or Dry?”

- **Sepsis / vasodilatory shock**
  - Volume
  - Vasopressors

- **Normal**
  - “Dry and warm”

- **Pulmonary edema**
  - “Wet and warm”
  - Loop diuretics
  - Nitrates
  - Bilevel or continuous positive airway pressure
  - Nesiritide
  - Ultrafiltration?

- **Hypovolemic shock**
  - Volume

- **Cardiogenic shock**
  - Inotropes?
  - Intra-aortic balloon pump or ventricular assist device?
  - “Dry and cold”
  - “Wet and cold”

- **Volume depletion**
  - Low jugular venous pressure, skin tenting, orthostasis

- **Volume overload**
  - Peripheral edema, ascites, elevated jugular venous pressure, crackles

- **Cardiac index,**
  - L/min/m²
  - 2.2

- **High output**
  - Warm extremities, shunting with coexisting low tissue perfusion

- **Low output**
  - Cool extremities, fatigue, decreased urine output / elevated serum urea

18 **Pulmonary capillary wedge pressure, mm Hg**
Physical Exam in Acute Decompensated CHF

Box 2: Findings in patients with suspected acute decompensated heart failure

- Prior history of heart failure or myocardial injury
- Dyspnea on exertion, orthopnea or paroxysmal nocturnal dyspnea
- Fatigue
- Increasing edema, weight or abdominal girth

Physical examination

- Elevated jugular venous pressure
- Peripheral edema or ascites
- Rales, hypoxia or tachypnea
- Tachycardia, arrhythmia
- Diffuse point of maximal intensity
- Ventricular filling gallop (S3)
- Atrial gallop (S4)
- Cool extremities above the hands and feet
- Poor urine output
Inpatient Diuresis

- Typically IV loop diuretics at doses 1-1/2 – 2 times patients oral dosing
- If inadequate diuresis consider increasing dose, addition of oral diuretic in different class (metolazone, spironolactone), change to continuous infusion versus bolus, or ultrafiltration
- Careful monitoring of renal function, electrolytes, onset of hypotension, symptoms, weight and strict I&O recommended
- 2gm sodium diet and 2L fluid restriction recommended in most patients (strength of evidence=C)
- In the absence of symptomatic hypotension IV nitro, nitroprusside or nesieritide may be considered in addition to diuretic therapy (SOE=B)
- IV vasodilators may be considered in patients with ADHF who have persistent severe HF despite aggressive treatment with diuretics and standard oral therapy.

- Journal of Cardiac Failure Vol 16 No 6 June 2010
The “Dilators”

Venodilators
Nitroglycerin, Morphine

• “Preload reduction with venodilators is thought to be helpful in acute decompensated heart failure by reducing congestion and minimizing cardiac oxygen demand”

Arterial Vasodilators
Nitrates, Nitroprusside, Dobutamine

• “Afterload reduction is also thought to be helpful in some patients with acute decompensated heart failure by decreasing myocardial oxygen demand and improving forward flow”

• CMAJ 2007 Mar 13; 176(6) 797-805
Inotrope Therapy

- May be considered in patients with advanced HF with LV dilatation, reduced RF and diminished peripheral perfusion or end organ dysfunction (low output).

- Similar patients as above who are responding poorly to IV diuretics with worsening renal function.

- Are not recommended unless left heart filling pressure are known to be elevated or CI is severely impaired based on direct measurement or clear clinical signs.

- If symptomatic hypotension or worsening tachyarrhythmias develop dose should be reduced or discontinuation of agent should occur.
Ultrafiltration: Aqua/natriuresis

Bilevel or continuous positive airway pressure: Preload reduction

Nitrates, nitroprusside, dobutamine: Arterial vasodilation

Dobutamine, dopamine, milrinone: Increased inotropy

Nitrates, morphine: Venodilation

Furosemide: Natriuresis

Essentia Health
Here with you
<table>
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<th>Medication</th>
<th>Mechanism</th>
<th>Setting</th>
<th>Dosing</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>Natriuresis (preload reduction)</td>
<td>Volume overload with elevated left and right ventricular filling pressures</td>
<td>Bolus intravenous infusion (dose is often about twice the patient’s usual dose at home); adjust dose based on urine output; add thiazide (metolazone 2.5-5 mg orally daily or chlorothiazide 250-500 mg intravenously once or twice daily), or switch furosemide to a continuous infusion (5-30 mg/h), or both in severe cases with diuretic resistance</td>
<td>Foundation of treatment for acute decompensated heart failure in patients with symptoms of congestion (“wet and warm”)</td>
</tr>
<tr>
<td>Ultrafiltration</td>
<td>Venovenous filter to remove free water</td>
<td>Alternative to loop diuretics for treatment of volume overload</td>
<td>Ultrafiltration/hemofiltration system; fluid removal rates as dictated by clinical assessment, adequate blood pressure and system capabilities</td>
<td></td>
</tr>
<tr>
<td>Nitroglycerin</td>
<td>Venodilation (preload reduction), coronary vasodilator (anti-ischemic)</td>
<td>Volume overload with adequate blood pressure, cardiac ischemia</td>
<td>1-2 sprays of sublingual nitroglycerin (0.3-0.8 mg) every 3-5 min at first. Consider transition to continuous intravenous infusion (v. topical paste); 10-20 μg/min intravenously at first; increase by 5-20 μg/min every 3-5 min as blood pressure allows</td>
<td>Probably underused in patients presenting with acute decompensated heart failure and adequate blood pressure</td>
</tr>
<tr>
<td>Positive pressure ventilation</td>
<td>Positive intrathoracic pressure (preload reduction)</td>
<td>Volume overload with (or without) dyspnea or hypoxia</td>
<td>Continuous positive airway pressure (with or without bilevel positive airway pressure) at pressure of 9-20 cm H₂O</td>
<td>Consider short-term use (hours) in patients with acute decompensated heart failure in acute respiratory distress</td>
</tr>
<tr>
<td>Morphine</td>
<td>Venodilator (preload reduction)</td>
<td>Volume overload with adequate blood pressure after nitroglycerin treatment</td>
<td>Bolus 2-4 mg intravenously</td>
<td>No evidence of efficacy; second-line treatment</td>
</tr>
<tr>
<td>Nesiritide</td>
<td>Venodilator (preload reduction)</td>
<td>Volume overload with adequate blood pressure</td>
<td>Bolus 2 μg/kg; then infusion 0.01 μg/kg per min, adjusting dose up to 0.03 μg/kg per min</td>
<td>Not currently available in Canada</td>
</tr>
<tr>
<td>Nitroprusside</td>
<td>Arterial vasodilator (afterload reduction)</td>
<td>Acute heart failure with severe hypertension, or mitral valve regurgitation with adequate blood pressure</td>
<td>Continuous intravenous infusion of 0.3 μg/kg per min at first; titrate rapidly to desired blood pressure; maximum dose 10 μg/kg per min</td>
<td>Use nitroglycerin instead in most patients with acute decompensated heart failure; tight sensitive; toxic levels of thiocyanate may accumulate</td>
</tr>
<tr>
<td>Vasodilating inotropes (dobutamine, milrinone)</td>
<td>Inotrope, chronotrope, systemic vasodilator, pulmonary vasodilator</td>
<td>Acute heart failure unresponsive to above therapies, worsening renal function</td>
<td>Dobutamine: 2-20 μg/kg per min intravenously Milrinone: 0.125-0.75 μg/kg per min intravenously (may load 50 μg/kg intravenously over 10 min, but not necessary); renal adjustment necessary</td>
<td>For short-term use in patients with significantly impaired cardiac output; may increase arrhythmia and risk of death; milrinone has longer half-life than the β-agonists</td>
</tr>
<tr>
<td>Vasopressor inotropes (dopamine, norepinephrine)*</td>
<td>Inotrope, chronotrope, vasoconstrictor</td>
<td>Shock with inadequate blood pressure (possibly low-dose dopamine in cardiological syndrome)</td>
<td>Dopamine: 1-50 μg/kg per min intravenously Norepinephrine: 0.01-0.4 μg/kg per min intravenously</td>
<td>Used in critically ill patients with hypotension; typically avoided in pure heart failure with high systemic vascular resistance, but such resistance may be low in acute decompensated heart failure owing to activation of systemic inflammatory response or total circulatory collapse</td>
</tr>
</tbody>
</table>

*Vasopressin and phenylephrine would not typically be used in acute decompensated heart failure.
Discharge Instructions for All CHF Patients

- Exacerbating factors addressed
- Near optimal volume status observed
- Transition from IV to oral diuretics successfully completed.
- Patient and family education completed including CLEAR discharge instructions (medication reconciliation!!)
- LVEF documented
- Smoking cessation counseling documented
- Near optimal pharmacologic therapy achieved including ACE and beta blocker (for reduced LVEF) or intolerance documented.
- Follow up clinic visit scheduled within 1 week
Additional recommendations for Advanced HF or Recurrent HF Admissions

• Oral medication regimen stable for 24 hours
• No IV vasodilator or inotropic agent for 24 hours
• Plans for post discharge management (scale present in the home, visiting nurse or telephone follow up no longer than 3 days after discharge)
• Referral to disease management

• Journal of Cardiac Failure Vol 16 No 6 June 2010
Need to Shift to a Focus on Prevention

Stages in the development of HF - Recommended therapy by stage

2013 ACCF/AHA Guideline for the Management of Heart Failure
Thank you, I’ll be happy to take questions.
Questions and Discussion

- Questions for our speakers
  - Via phone
    *6 to unmute → *6 to mute again
  - Via chat
Heart Failure Webinar Series

- June 20 – Heart Failure 101
- July 27 – Improve Heart Failure Self Care and Reduce Readmissions Using Data to Understand the Problem
- August 23 – Standardizing Heart Failure Education and Engaging Your Patient in the Standard
- September (TBD) – Support CHF Patients in the Home Setting

Recordings will be available at http://greatplainsqin.org and https://www.lsqin.org/