HEART FAILURE: WHAT TO DO WHEN DRUGS “DON’T WORK”

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What to do when drugs don’t work in HF?

- A) Transplant
- B) Mechanical heart / LVAD
- C) Palliative care / +/- Hospice
- D) Why are we talking about HF & drugs. I wanted to hear Dr. Lombardi speak on his retrograde approach to CTO’s.
- E) A, B, C
Answer: What to do when drugs don’t work

- Answer = E
  - Transplant
  - Mechanical Heart / LVAD
  - Palliative Care / +/- Hospice

- I will talk about all of these therapies for Stage D Heart Failure.
- Fortunately, the vast majority of patients with HF due to systolic dysfunction are NOT Stage D

Disclosures

- None
- Patient photos/information used with pt’s permission
Introduction

- HF Terminology
- ACC/AHA Stages of HF & NYHA Class
- HF Epidemiology & Economics
- Reasons for Drugs Not Working...
- Management strategies for making the drugs work
- Approaches to Advanced or Stage D HF / “Tales from the trenches”. North Cascade Cardiology Heart Failure Prevention & Treatment Program Success Stories
- Cardiac Transplantation: A patient’s reflection
Heart Failure

“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.”

2009 ACC/AHA Heart Failure Guidelines

“impairs the ability of the ventricle to fill” = Diastolic HF

“impairs the ability of the ventricle to eject blood” = Systolic HF
Heart Failure Stages & NYHA Functional Class

What is Stage D HF: ACC/AHA Heart Failure Stages
2005 ACC/AHA Guidelines: New Classification of HF

<table>
<thead>
<tr>
<th>Stage</th>
<th>Clinical Characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>At risk for development of heart failure but no signs and symptoms of heart failure</td>
</tr>
<tr>
<td>B</td>
<td>Structural heart disease WITHOUT current or prior heart failure symptoms</td>
</tr>
<tr>
<td>C</td>
<td>Structural heart disease WITH current or prior symptoms of heart failure</td>
</tr>
<tr>
<td>D</td>
<td>Refractory heart failure requiring specialized interventions</td>
</tr>
</tbody>
</table>

New York Heart Association Functional Class

- I: No functional limitation
- II: Symptomatic with exercise
- III: Marked limitation, comfortable only at rest
- IV: Symptomatic at rest
Heart Failure Epidemiology & Economic Impact

- 5 million HF patients in US
- 4.5 million Stage C
- ~500,000 Stage D
- 2000 Transplants/year in US
- 1000 LVAD's/Year in US

Doing the Math..
Heart Failure Epidemic

500,000
CASES/YEAR
UNITED STATES

Who are these patients?
HF: A Condition of the Elderly

- 500,000 new cases of HF per year in US
- 1% or 5,000 pts age 65-69
- 4% or 20,000 pts age 70-79
- 50% or 250,000 pts > 80

Projected Elderly Population Age 65+

- 31.5 million in 1990
- 65.6 million in 2030

12.6% total US population in 1990

21.8% total US population in 2030

Demographic Trends

Elderly U.S. population will double with graying of “baby boomer” generation
Aging of population contributes to increasing incidence of HF

80% of pts hospitalized with HF > 65 yo

Primary reason for 12-15 million office visits/year

Primary reason for 6.5 million hospital days/year

HF most common Medicare DRG

Medicare pays hospital $6,000.00 per admission

Average stay ~ 5.8 days

More Medicare dollars spent for diagnosis and treatment of HF than for any other diagnosis

In 2010 estimated direct & indirect costs for HF 39 billion dollars

25% patients readmitted within 30 days – Medicare Will Not Pay!
Heart Failure: Natural History

Reasons drugs “don’t work”
When drugs don’t work: Stage C vs Stage D HF

PERFECT STORM

Patient

Physician/Provider

Healthcare system

BIOLOGIC FAILURE

B-Blocker Receptor Down Regulation

B-Blocker Receptor Upregulation & Reverse Remodeling

Pathologic Remodeling

STAGE C
Structural heart disease WITH current or prior symptoms of heart failure

STAGE D
Refractory heart failure requiring specialized interventions

Noncompliant Patient
Transplant

Noncompliant Physician / Provider
Mechanical Hearts / LVAD

Health care delivery system failure
Palliative Care / +/- Hospice

Regenerative Medicine

Noncompliant Patient Transplant

Noncompliant Physician / Provider Mechanical Hearts / LVAD

Health care delivery system failure Palliative Care / +/- Hospice

Regenerative Medicine
The Noncompliant Patient

Who are these Noncompliant Patients?
HF Patient “failures”

- Dietary & medical noncompliance – usually due to failure to educate
- Failure to seek early care with escalating symptoms
- Failure of patient social support system – failure to educate family, care givers, Cook

Causes of HF Readmissions

- 24% dietary non-compliance
- 24% medical non-compliance
- 19% non-evidenced based care
- 7% other
- 15% fail to seek care
Conclusion:

- Drugs don’t work when:
  - Poorly educated patient & family & cook
    - Role for a Heart Failure Nurse Educator
  - Patient who cannot be educated due to psychiatric illness or dementia?
    - Role for a Heart Failure RN Case Manager
  - Deconditioned patient
    - Exercise Physiologist / Cardiac rehab
  - Complex medical program
    - Greater role for a dedicated HF Pharmacist

Physician Compliance with the “Guidelines”
Non-Compliant HF Physicians

- Improvement International Survey
  - 15 countries / 1363 physicians / year 2000
  - 11,063 patients with stage C HF
  - Outpatient HF Meds.................................
    - 60% ACE-Inhibitors
    - 34% Beta-Blockers
    - 20% ACE-Inhibitor & Beta-Blocker
    - 12% Aldosterone Receptor Antagonist

Clinicians Failures in the care of HF Patients

- Failure to treat with Evidenced Based Medications
- Failure to stop medications that exacerbate HF
- Failure to titrate meds to target doses
- Failure to address comorbidities
- Failure to consider device therapies
- Failure to provide dietary counseling
Evidenced Based Medical Interventions for HF Patients (LV systolic dysfunction)

Reduce Mortality

Diagnose & Treat Comorbidities

Control Volume

Promote Self Management & Enhance Compliance

Treat Residual symptoms

Interventions for LV systolic dysfunction

Meds are expensive. Nobody takes them! They don’t work! The patients are just too old for all these meds!
HF Is Expensive and Deadly: Outcomes During and After a HF Admission

- Mean length of stay = 6.2 days
- In house mortality = 4.1%
- Hospital Readmissions =
  - 20% @ 30 days
  - 50% @ 6 Months
- Longer Term Mortality
  - 30 Day mortality – 11.6%
  - 1 Year mortality – 33%

Fonarow GC et al JACC 2007; 297: 61-67

The Case for Optimism – Myocardial Recovery & Reverse Remodeling

- Spontaneous Recovery
  - Peripartum Cardiomyopathy
  - Acute Myocarditis

- Recovery after removal of cardiac insult
  - ETOH
  - Sepsis
  - Tachycardia-Induced
The Case for Optimism - Myocardial Recovery & Reverse Remodeling

Recovery following medical intervention
- Beta Blocker
- ACE-I / ARB
- Aldosterone Receptor Antagonist
- Hydralazine / Nitrates
- Volume Management

Recovery following surgical intervention
- Revascularization (CABG or PCI)
- CRT (Bi-V Pacing)
- Valve repair/replacement
- VAD

Evidenced Based Treatment of HF

- **Reduce Mortality**:
  - ACE-I / ARB, Beta Blockers, Aldosterone Receptor Antagonists
  - Hydralazine / Nitrates

- **Control Volume**:
  - Salt Restriction, Diuretics

- **Treat Residual Symptoms**:
  - Digoxin

- **Enhance Compliance**:
  - Education, Disease Management, Performance Improvement Programs

- **Diagnose & Treat Comorbidities**
  - Vascular (CAD, PAD, AAA)
  - DM
  - COPD
  - Renal Failure
What to do when .......... doesn’t work

& ARBS

ACE Inhibitors:

Why bother:

- 20-25% relative risk reduction in HF mortality

HFSA 2010 Practice Guideline
ACE Inhibitors

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Daily Dose</th>
<th>Target Dose</th>
<th>Mean Dose in Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Captopril</td>
<td>Capoten</td>
<td>6.25 mg tid</td>
<td>50 mg tid</td>
<td>122.7 mg/day</td>
</tr>
<tr>
<td>Enalapril</td>
<td>Vasotec</td>
<td>2.5 mg bid</td>
<td>10 mg bid</td>
<td>16.6 mg/day</td>
</tr>
<tr>
<td>Fosinopril</td>
<td>Monopril</td>
<td>5-10 mg qd</td>
<td>80 mg qd</td>
<td>N/A</td>
</tr>
<tr>
<td>Lisinopril</td>
<td>Zestril, Prinivil</td>
<td>2.5-5 mg qd</td>
<td>20 mg qd</td>
<td>4.5 mg/day, 33.2 mg/day*</td>
</tr>
<tr>
<td>Quinapril</td>
<td>Accupril</td>
<td>5 mg bid</td>
<td>80 mg qd</td>
<td>N/A</td>
</tr>
<tr>
<td>Ramipril</td>
<td>Altace</td>
<td>1.25-2.5 mg qd</td>
<td>10 mg qd</td>
<td>N/A</td>
</tr>
<tr>
<td>Trandolapril</td>
<td>Mavik</td>
<td>1 mg qd</td>
<td>4 mg qd</td>
<td>N/A</td>
</tr>
</tbody>
</table>

*No mortality difference between high and low dose groups, but 12% lower risk of death or hospitalization in high dose group vs. low dose group.
HFSA 2010 Practice Guideline

**ARBs**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Daily Dose</th>
<th>Target Dose</th>
<th>Mean Dose in Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Candesartan</td>
<td>Atacand</td>
<td>4-8 mg qd</td>
<td>32 mg qd</td>
<td>24 mg/day</td>
</tr>
<tr>
<td>Losartan</td>
<td>Cozaar</td>
<td>12.5-25 mg qd</td>
<td>150 mg qd</td>
<td>129 mg/day</td>
</tr>
<tr>
<td>Valsartan</td>
<td>Diovan</td>
<td>40 mg bid</td>
<td>160 mg bid</td>
<td>254 mg/day</td>
</tr>
</tbody>
</table>

Problem                          | Solution                                           |
Cough :                         | ~Change to an Angiotensin Receptor Blocker         |
  5-10% in white pts of European descent  |                                                   |
  50% in Chinese pts            |                                                   |
  ***Cough could also be due to decompensated HF*** |                                                   |
Hypotension or Acute Renal Failure | Occurs in pts most dependent on renin angiotensin system (i.e. Class IV hyponatremic pt) 15-30% pts with Class III & IV HF have >0.3 mg/dl in sCr. Consider bilateral renal artery stenosis Consider NSAID use
### ACE-I “Failures”

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angioedema</td>
<td>All ACE-I absolutely contraindicated</td>
</tr>
<tr>
<td>~Occurs in 1% pts</td>
<td></td>
</tr>
<tr>
<td>~More common in</td>
<td>ARBs – relatively contraindicated</td>
</tr>
<tr>
<td>african americans</td>
<td></td>
</tr>
</tbody>
</table>

### What to do when ........doesn’t work

- Beta Blockers
  - Beta-Blockers:
  - Why Bother:
    - 34-40% relative risk reduction in HF mortality
**Beta Blocker “Failures”**

I. Must use a “heart failure” approved Beta Blocker.
- Carvedilol
- Bisoprolol
- Metoprolol Succinate

II. Patient must be euvolemic when initiating or uptitrating beta blockers in a patient with LV dysfunction

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**HFSA 2010 Practice Guideline Beta Blockers**

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Daily Dose</th>
<th>Target Dose</th>
<th>Mean Dose in Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bisoprolol</td>
<td>Zebeta</td>
<td>1.25 mg qd</td>
<td>10 mg qd</td>
<td>8.6 mg/day</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>Coreg</td>
<td>3.125 mg bid</td>
<td>25 mg bid</td>
<td>37 mg/day</td>
</tr>
<tr>
<td>Carvedilol</td>
<td>Coreg CR</td>
<td>10 mg qd</td>
<td>80 mg qd</td>
<td></td>
</tr>
<tr>
<td>Metoprolol Succinate CR/XL</td>
<td>Toprol XL</td>
<td>12.5-25 mg qd</td>
<td>200 mg qd</td>
<td>159 mg/day</td>
</tr>
</tbody>
</table>

**HFSA 2010 Practice Guideline**

**Beta Blockers**

**Recommendation 7.9**

Beta blocker therapy is recommended in the great majority of patients with HF and reduced LVEF, even if there is concomitant diabetes, chronic obstructive lung disease or peripheral vascular disease.

- Beta blocker therapy should be used with caution in patients with diabetes with recurrent hypoglycemia, with asthma or with resting limb ischemia.
- Considerable caution should be used if beta blockers are initiated in patients with marked bradycardia (<55 beats/min) or marked hypotension (SBP < 80 mmHg).
- Beta blockers are not recommended in patients with asthma with active bronchospasm. *Strength of Evidence = C*

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**HFSA 2010 Practice Guideline**

**Beta Blockers—Summary of Recommendations**

| General | Initiate at low doses  
|         | Up-titrate gradually, generally no sooner than at 2 week intervals  
|         | Use target doses shown to be effective in clinical trials  
|         | Aim to achieve target dose in 8-12 weeks  
|         | Maintain at maximum tolerated dose |

| Considerations if symptoms worsen or other side effects appear | Adjust dose of diuretic or other concomitant vasoactive medication  
|                                                              | Continue titration to target dose after symptoms return to baseline |

| Considerations if up-titration continues to be difficult | Prolong titration interval  
|                                                         | Reduce target dose  
|                                                         | Consider referral to a HF specialist |

| If an acute exacerbation of chronic HF occurs | Maintain therapy if possible  
|                                              | Reduce dosage if necessary  
|                                              | Avoid abrupt discontinuation  
|                                              | If discontinued or reduced, reinstate gradually before discharge |

Last word on Beta Blockers!

What to do when .... Doesn’t work.

- Aldosterone Receptor Antagonists
  - Why bother:
    - EPHESUSial
      - Mortality reduced from 13.6% to 11.8% at one year
      - no survival benefit if sCr > 1.1
    - 2 year mortality reduced from 46% to 35%
    - 35% reduction in HF hospitalization
HFSA 2010 Practice Guideline
Aldosterone Antagonists

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Daily Dose</th>
<th>Target Dose</th>
<th>Mean Dose in Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spironolactone</td>
<td>Aldactone</td>
<td>12.5-25 mg qd</td>
<td>25 mg qd</td>
<td>26 mg/day</td>
</tr>
<tr>
<td>Eplerenone</td>
<td>Inspra</td>
<td>25 mg qd</td>
<td>50 mg qd</td>
<td>42.6 mg/day</td>
</tr>
</tbody>
</table>

Recommendation 7.14

Administration of an aldosterone antagonist is recommended for patients already receiving standard therapy, including diuretics, who have:

- NYHA class IV HF (or class III, previously class IV) from reduced LVEF (≤ 35)

Strength of Evidence = A
HFSA 2010 Practice Guideline
Aldosterone Antagonists

Recommendation 7.15

Administration of an aldosterone antagonist should be considered in patients following an acute MI, with clinical HF signs and symptoms or history of diabetes mellitus and an LVEF <40%.

- Patients should be on standard therapy, including an ACE inhibitor (or ARB) and a beta blocker.

Strength of Evidence = A

HFSA 2010 Practice Guideline
Aldosterone Antagonists

Recommendation 7.16

Aldosterone antagonists are not recommended when:

- creatinine is > 2.5 mg/dL (or creatinine clearance is < 30 ml/min)
- serum potassium is > 5.0 mmol/L
- in conjunction with other potassium-sparing diuretics.

Strength of Evidence = A
HFSA 2010 Practice Guideline
Aldosterone Antagonists

Recommendation 7.17

It **is recommended** that serum potassium concentration be monitored frequently following initiation or change in an aldosterone antagonist.

Monitoring should reflect protocols followed in clinical trials.

*Strength of Evidence = A*

---

Aldosterone Receptor Antagonist
“Failure”

Indications:

1) Spironolactone:
   - RALES Trial
     - Class III & IV Aldactone 12.5 mg q2d
     - 2 year mortality reduced from 46% to 35%
     - 35% reduction in HF hospitalization
   - Note in RALES trial: ....
     - Pts with sCr > 2.5 excluded
     - Few pts with sCr > 1.5 enrolled

2) Eplerenone:
   - Ephesus Trial
     - LVEF ≤ 40%
     - Clinical evidence of HF or DM within 14 days post MI
     - Mortality reduced from 13.6% to 11.8% at one year

<table>
<thead>
<tr>
<th>Problem</th>
<th>Solution</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperkalemia</td>
<td>Stop Spironolactone or Eplerenone</td>
</tr>
<tr>
<td>Gynecomastia</td>
<td>Stop Spironolactone</td>
</tr>
<tr>
<td></td>
<td>Start Eplerenone</td>
</tr>
</tbody>
</table>
Hydralazine & Nitrates

HFSA 2010 Practice Guideline
Hydralazine and Oral Nitrates

Recommendation 7.19

A combination of hydralazine and isosorbide dinitrate is recommended as part of standard therapy in addition to beta-blockers and ACE-inhibitors for African Americans with HF and reduced LVEF:

- NYHA III or IV HF  
  Strength of Evidence = A
- NYHA II HF  
  Strength of Evidence = B
### HFSA 2010 Practice Guideline
#### Other Vasodilators

<table>
<thead>
<tr>
<th>Generic Name</th>
<th>Trade Name</th>
<th>Initial Daily Dose</th>
<th>Target Dose</th>
<th>Mean Dose in Clinical Trials</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fixed dose hydralazine/isosorbide dinitrate</td>
<td>BiDil</td>
<td>37.5 mg hydralazine/20 mg isosorbide dinitrate tid</td>
<td>75 mg hydralazine/40 mg isosorbide dinitrate tid</td>
<td>142.5 mg hydralazine/76 mg isosorbide dinitrate/day</td>
</tr>
<tr>
<td>Hydralazine</td>
<td>Apresoline</td>
<td>37.5 mg qid</td>
<td>75 mg qid</td>
<td>270 mg/day</td>
</tr>
<tr>
<td>Isosorbide dinitrate</td>
<td>Isordil</td>
<td>20 mg qid</td>
<td>75 mg qid</td>
<td>270 mg/day</td>
</tr>
</tbody>
</table>

### Volume Management

![Volume Management](image)
## 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>
</tr>
</thead>
<tbody>
<tr>
<td>Furosemide</td>
<td>20-40mg qd or bid</td>
<td>600 mg</td>
<td>65%R/35%M</td>
<td>4-6 hrs</td>
</tr>
<tr>
<td>Bumetanide</td>
<td>0.5-1.0 mg qd or bid</td>
<td>10 mg</td>
<td>62%R/38%M</td>
<td>6-8 hrs</td>
</tr>
<tr>
<td>Torsemide</td>
<td>10-20 mg qd</td>
<td>200 mg</td>
<td>20%R/80%M</td>
<td>12-16 hrs</td>
</tr>
<tr>
<td>Ethacrynic acid</td>
<td>25-50 mg qd or bid</td>
<td>200 mg</td>
<td>67%R/33%M</td>
<td>6 hrs</td>
</tr>
</tbody>
</table>

## Thiazide Diuretics

<table>
<thead>
<tr>
<th>Agent</th>
<th>Initial Daily Dose</th>
<th>Max Total Daily Dose</th>
<th>Elimination</th>
<th>Duration of Action</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chlorothiazide</td>
<td>250-500 mg qd or bid</td>
<td>1000 mg</td>
<td>Renal</td>
<td>6-12 hrs</td>
</tr>
<tr>
<td>Chlorthalidone</td>
<td>12.5-25 mg qd</td>
<td>100 mg</td>
<td>65% Renal, 10% into Bile, 25% Unknown</td>
<td>24-72 hrs</td>
</tr>
<tr>
<td>Hydrochlorothiazide</td>
<td>25 mg qd or bid</td>
<td>200 mg</td>
<td>Renal</td>
<td>6-12 hrs</td>
</tr>
<tr>
<td>Metolazine</td>
<td>2.5 mg qd</td>
<td>20 mg</td>
<td>80% Renal, 10% into Bile, 10% Unknown</td>
<td>12-24 hrs</td>
</tr>
<tr>
<td>Idapamide</td>
<td>2.5 mg qd</td>
<td>5 mg</td>
<td>Metabolic</td>
<td>36 hrs</td>
</tr>
</tbody>
</table>
## Diuretic Resistance

<table>
<thead>
<tr>
<th>Patient Noncompliance</th>
<th>HF Progression</th>
</tr>
</thead>
<tbody>
<tr>
<td>High Salt Diet</td>
<td>Bowel edema</td>
</tr>
<tr>
<td>NSAID’s</td>
<td>Intestinal hypoperfusion</td>
</tr>
<tr>
<td>COX-II Inhibitors</td>
<td>Renal hypoperfusion</td>
</tr>
</tbody>
</table>

## Treatment of Diuretic Resistance

- Oral diuretics not absorbed in an edematous gut
  - Change to Torsemide
  - IV diuretics
- Use sequential nephron blockade
- Positive Inotropes
A role for Digoxin?

- Initiate at .125mg daily
- DIG trial = increased mortality as plasma concentrations > 1.0 ng/ml
- Toxicity may occur at levels < 2.0 if hypo- K & Mg, or Hypothyroid
- No mortality benefit
- Improves functional capacity in pts with class III & IV HF, especially in pts with afib

Digoxin: I started my patient on Dig and they became toxic.

- Initiate at .125mg daily
- DIG trial = increased mortality as plasma concentrations > 1.0 ng/ml
- Toxicity may occur at levels < 2.0 if hypo- K & Mg, or Hypothyroid
- No mortality benefit
- Improves functional capacity in pts with class III & IV HF, especially in pts with afib
When drugs don’t work because the system don’t work!

- Failure to...
  - A) Provide adequate discharge planning
    - Is outpatient HF provider in the loop?
    - Is family/care giver/NH staff in the loop?
    - Role for a HF Rehab Unit
  - B) Provide outpatient follow up & monitoring
    - Can the outpatient provider f/u within 1 week
    - Accessible/unified medical record
    - Telehealth & device based remote monitoring
  - C) Failure to address patient & care giver needs
    - Role for Palliative Care

The CORE MEASURES DO NOT EQUAL A HF TREATMENT STRATEGY

- A Comprehensive HF Disease Management Program:
  - “Getting Beyond the Core Measures”
  - Only 1 core measure reduces mortality
Benefits of HF Disease Management Program

- Increased use of evidenced based medications
- Improved symptom status and functional capacity
- Improved patient reported QOL
- Decreased hospitalizations
- Decreased total medical costs
- Increased survival suggested in some studies

Comprehensive HF Disease Management Program

- Optimize HF medications
- Educate patient / family / caregivers / Cook
- Volume Management - Heart Failure Diary - daily weights, BP and HR, fluid restriction PRN, and weight adjusted diuretics, Integration of device related remote monitoring systems
- Ongoing support of ETOH & nicotine abstinence
- Support for structured exercise program - Cardiac Rehab
- Vigilant follow up – via frequent office visits, phone follow-up, remote monitoring via device
HF Disease Management Program: The UCLA Experience

- Fonarow GC et al JACC 1997; 30;725-732
- 85% reduction in readmissions at 6 months compared to usual care.
- Multiple randomized controlled trials validate the UCLA experience and the utility of HF Management Programs

Who should manage inpatients with HF: Comparative Percentage of HF patients receiving a Beta blocker at discharge. Ansari, Circulation 2003

<table>
<thead>
<tr>
<th>Percentage of HF Patients Treated with Beta-Blockers</th>
<th>Usual Care</th>
<th>Provider Notification</th>
<th>ARNP</th>
<th>Impact-HF Carvedilol Pre-discharge Initiation</th>
</tr>
</thead>
<tbody>
<tr>
<td>27</td>
<td>16</td>
<td>67</td>
<td>91</td>
<td></td>
</tr>
</tbody>
</table>
APPROACHES TO ADVANCED OR STAGE D HF &
“TALES FROM THE TRENCHES“. NORTH CASCADE CARDIOLOGY HEART FAILURE PREVENTION & TREATMENT PROGRAM SUCCESS STORIES

HF Interventionist Tool Box

- PCI
- Valvuloplasty / Percutaneous Valves
- Endovascular Procedures
Surgical Team

PEACEHEALTH ST JOSEPH MEDICAL CENTER

INLAND NORTHWEST THORACIC ORGAN TRANSPLANT AND MECHANICAL HEART PROGRAM

If you can't explain it simply, you don't understand it well enough.
Albert Einstein
THANKSGIVING DINNER 2006: THE MEAL THAT NEVER WAS FOR DR LEONE. A TALE OF CABG & A RESCUE LVAD AS A BRIDGE TO TRANSPLANT

Subtotally Occluded RCA

Occluded LMCA
Rescue LVAD as a Bridge to Transplantation

70 SHOCKS FROM ELLENSBERG TO SEDRO WOOLEY: A TALE OF STENTS, ENDOGRAFTS, AND A DESTINATION LVAD.
CRITICAL CAROTID STENOSIS

POST CAROTID STENT

70 Shocks from Ellensberg to Sedro Wooley:
A tale of stents, endografts, and a Destination LVAD.

CTA – 3D RECONSTRUCTION

CT
70 Shocks from Ellensburg to Sedro Woolley: 
A tale of stents, endografts, and a Destination LVAD.

PRE-EVAR

POST-EVAR

HEARTMATE 2 VS XVE

1 YEAR ANNIVERSARY
MIGO’S LONG ROAD BACK HOME! THE TALE OF CABG, STENTS, ICD’S, SILDENAFIL AND A NEW HEART.
BILATERAL RENAL ARTERY STENOSIS

TEACHING POINT

- ACE-I intolerance
- Incidental finding of a prior non-contrast CT of small kidneys
Migo's long road back home! The tale of CABG, stents, ICD's, Sildenafil and a new heart.

PRE-TXP LCA

POST-TXP LCA

WHAT GOOD IS FIXING MY HEART IF I CAN’T WALK.
“PERCUTANEOUS CABG AND ABF GRAFT”
OCCLUDED RIGHT EXTERNAL ILIAC

OCCLUDED LEFT EXTERNAL ILIAC

What good is fixing my heart if I can’t walk. “Percutaneous CABG and ABF graft”

WIRE ACROSS TOTAL AORTIC OCCLUSION

KISSING AORTO-ILIAC STENTS
What good is fixing my heart if I can’t walk. “Percutaneous CABG and ABF graft”

SEVERE MID LAD STENOSIS

STENT MID LAD

Percutaneous CABG

CRITICAL MID RCA STENOSIS

POST-PCI
5 years s/p HeartMate II

1 year Anniversary Post-Txp
A year has gone by since my heart transplant surgery.

In the first three months the medical follow-up was somewhat grueling: in the first month, two biopsies a week. The transplant staff kept a very close eye on my progress. In the second month, the biopsies were down to once a week, and I began my physical rehab several times a week. Then, in month three, it was biopsy every two weeks.

The reason for the schedule was to keep ahead of any rejection issues that usually surface in the early stages post operatively. As time passes, the chances of rejection lessen quite a bit.

While a hospital resident, I was ambulatory, and I met a few people who had had a transplant, were waiting for transplant, and some who were waiting for transplant and had temporary VADs (ventricular assist devices) implanted. While the center had a great survival rate record for their patients, all of that was statistical, and, individually meant nothing to me. First, I had to make it to the point of actually having a transplant, then, bit by bit, survive the whole process. I saw clearly that this all a pretty dicey affair: no one was guaranteeing anything. And not everyone survives. Some people are still waiting for a donor, some have had troubles in recuperating, some have died waiting, and yet others have died of complications post transplant. But many have made it, and have survived for considerable periods of time: ten, twenty years.

And I was to learn what it takes to survive.
First, there is the medicating and the monitoring: no deviations. And immune system suppression is forever: the donor heart remains a foreign part, no matter how friendly we get. And, at first, when my new heart seemed to pound when I lay down to rest, I had to make friends with it, so that we could learn to live together.

Then, there is the doctor community: every symptom must be investigated and followed, as the suppressed immune system is so vulnerable to most foreign invasions. One doctor told me, when the idea of transplanting the heart was first broached, that managing that was just exchanging my heart failure management for another. And he was right. But in reality, it was not just the exchange, because in the process, I got back my ability to do things, physically, that were simply undoable as a heart failure patient.
As I progressed, I also began to think in terms of tomorrow, in other words, I began to allow myself to make plans again. Baby steps at first, but even now, those plans are pretty short term ones. But that’s how I function: got to have some kind of goals...

I went back to work as planned on the 1st of February. I see a lot of doctors and pretty frequently. But that is a good thing. I see them often (or they see me often, as the case may be) so that they can keep close tabs on my progress. And to tell the truth, I have been doing much better that I ever expected.

So, a year post transplant, with the exception of some drug induced side effects (when the moon is full…), my life has been given back to me, albeit on a much shorter leash.

But that is a small price to pay for hiking to altitude, playing racquetball, and in general, doing what I have always enjoyed. Seeing my youngest daughter’s son growing, my son’s daughters growing up: that is its own reward.
And so, my dear friends, I apologize for taking so much of your time with my travails, but it seemed like a good time for putting some kind of closure to this great dramatic moment of my life, of Vince’s life, and to let you know that I am well and so happy to be still among you all.

1) Heart failure is managed & never cured
2) A heart transplant / mechanical heart is a not a cure but a more manageable disease
3) Never give up on medical therapy!
4) Heart failure must be managed by a team not an individual
5) A comprehensive heart failure program must also provide state of the art Palliative care
6) Ischemic cardiomyopathy is one manifestation of pan-atherosclerotic vascular disease. Search for athero-occlusive disease in other critical vascular beds

7) Young patients with advanced HF frequently present with low output HF rather than overt congestion

8) Targeted / thoughtful interventions! Don’t swing for the fences!

9) Cardiac rehab is highly underutilized

10) Never forget the usual nonvascular comorbidities:
   - DM/Renal Disease/COPD/Anemia/Thyroid/OSA