ashp MIDYEAR 2017
Clinical Meeting & Exhibition

Getting to Dry:
Management of Acute Decompensated Heart Failure with Volume Overload

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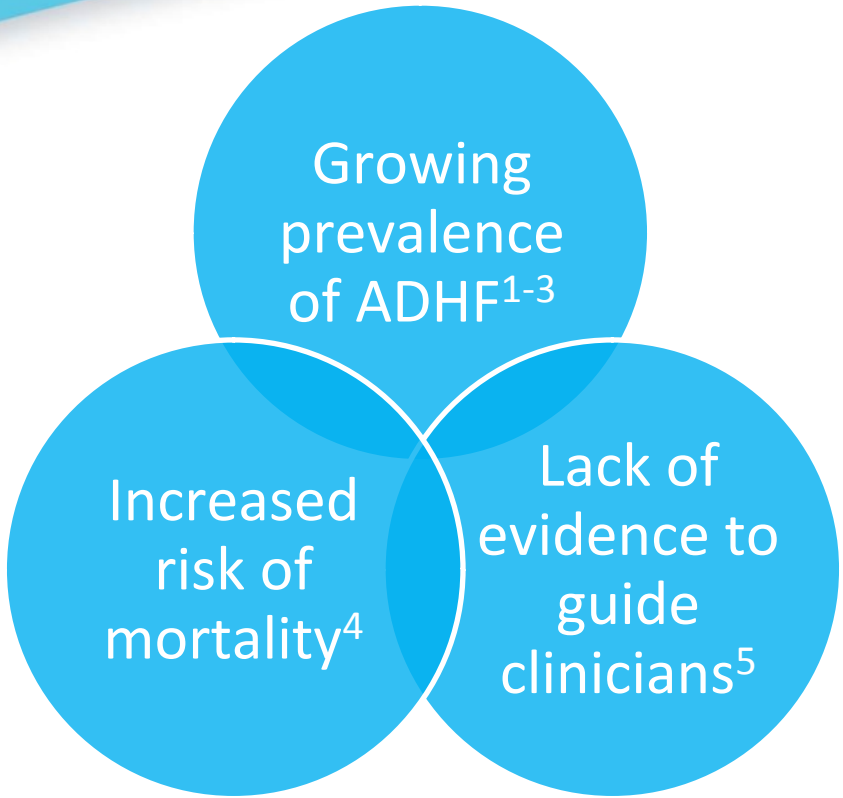
Disclosure

All planners, presenters, and reviewers of this session report no financial relationships relevant to this activity.

Learning Objectives

1. Given a patient presenting with acute decompensated heart failure and volume overload, design an individualized strategy for relieving congestive symptoms.
2. Given a patient failing to meet volume goals, determine potential etiologies of diuretic resistance and design a modified strategy for relieving congestive symptoms.
3. Given a patient preparing for discharge following an episode of acute decompensated heart failure, design a strategy for reducing the risk of hospital readmission.



ADHF acute decompensated heart failure

(1) *Circulation*. 2017 Mar 7;135(10):e146–603. (2) *Circ Heart Fail*. 2013 May;6(3):606–19. (3) *J Am Coll Cardiol*. 2014 Apr 1;63(12):1123–33. (4) *Am J Cardiol*. 2005 Sep 19;96(6A):32G–40G. (5) *J Am Coll Cardiol*. 2013 Oct 15;62(16):e147-239.

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DW is a 54 year-old white man with ischemic cardiomyopathy (EF 20%), hyperlipidemia, diabetes mellitus, and obstructive sleep apnea who presents with fatigue, shortness of breath, and abdominal discomfort of several weeks duration. He had a similar presentation 3 months ago. Today his breathing effort is labored and he has bilateral crackles over two-thirds the height of the lungs. Other pertinent findings include 2+ lower extremity edema and 10-kg weight gain. He is warm and well-perfused.

Current Medications:

- Aspirin 81 mg daily
- Atorvastatin 40 mg daily
- Lisinopril 10 mg daily
- Metoprolol succinate 100 mg daily
- Spironolactone 25 mg once daily
- Furosemide 40 mg twice daily
- Metformin 1000 mg twice daily
- Insulin glargine 25 units subq at night

Vitals: BP 118/78 mmHg, HR 71 bpm

134	98	28	182
4.5	26	1.4	

Hemoglobin A1c: 8.9%

NT-proBNP 12,800 pg/mL

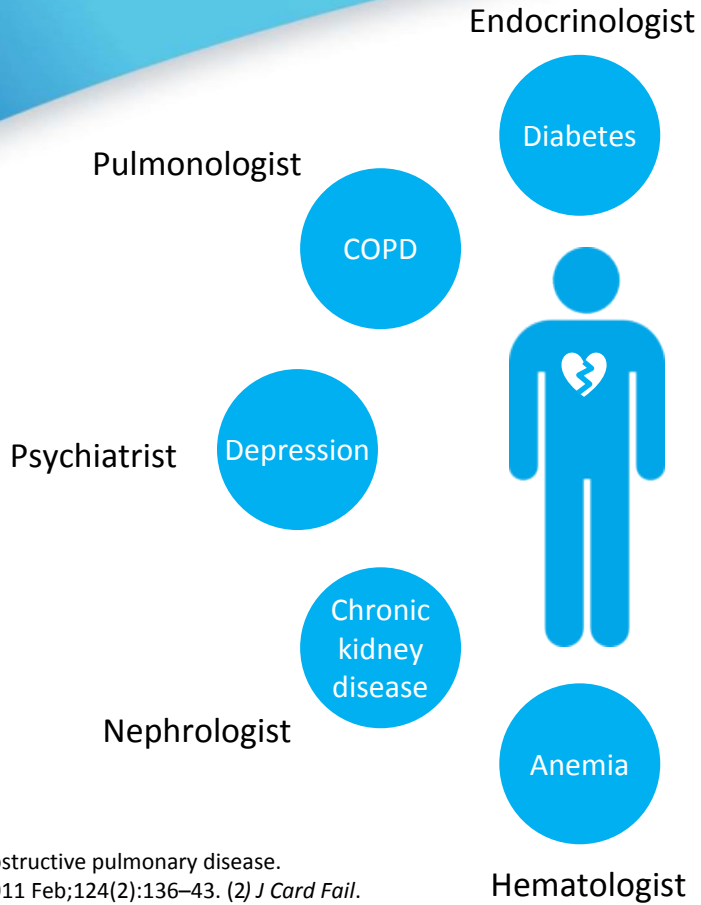
Chest x-ray: cardiomegaly, bilateral interstitial/alveolar edema; no effusions

Questions

1. What may have precipitated this heart failure exacerbation?
2. How should his congestive symptoms be managed? Provide recommendations regarding drug, dose, and frequency.
3. What should be done with his other guideline-directed medical therapy?

Questions

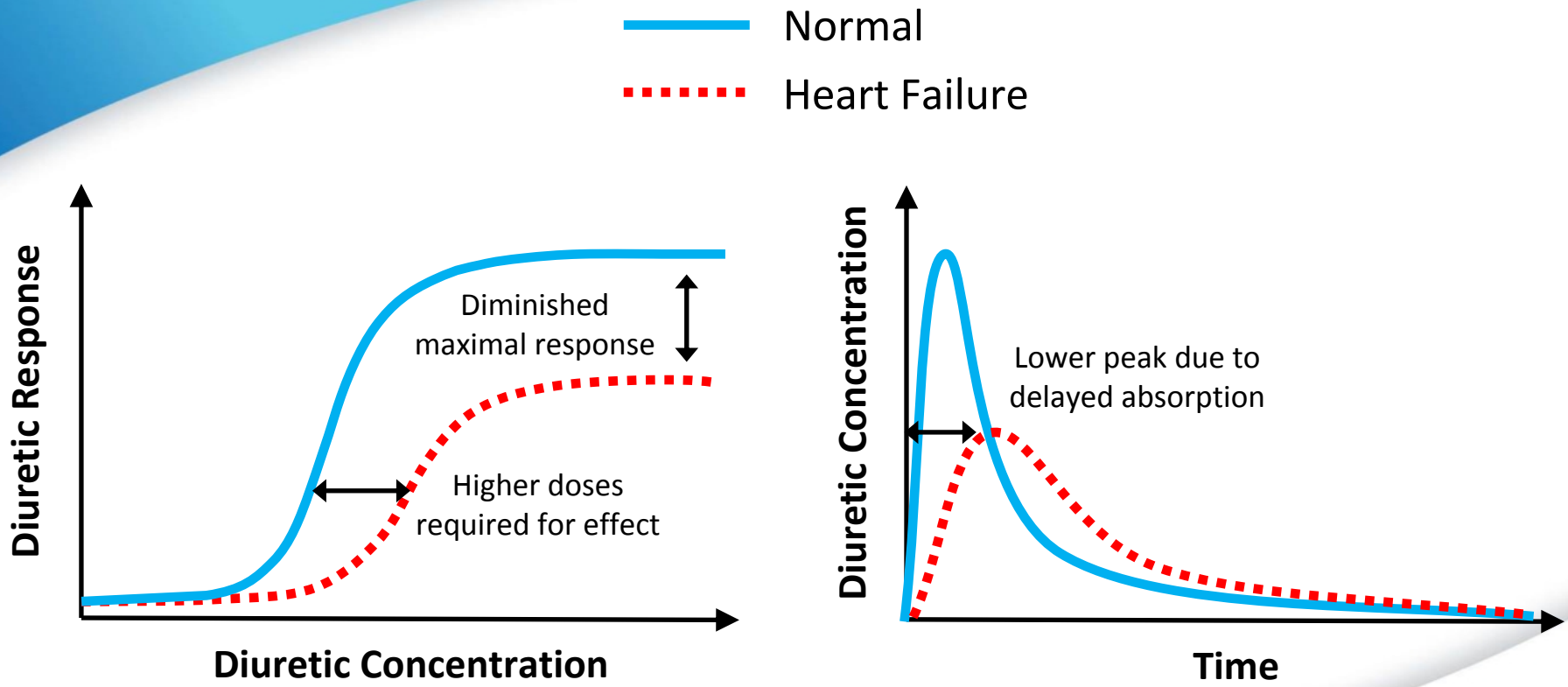
1. What may have precipitated this heart failure exacerbation?



Medication reconciliation to identify drug-related causes or contributors

- Most patients with heart failure have ≥ 5 comorbidities and take ≥ 6 chronic medications[1]
- Use of nonprescription medications may be as high as 93%[2]
- Nonadherence remains a major contributor to decompensation

COPD chronic obstructive pulmonary disease.
(1) *Am J Med*. 2011 Feb;124(2):136–43. (2) *J Card Fail*. 2009 Sep;15(7):600–6.

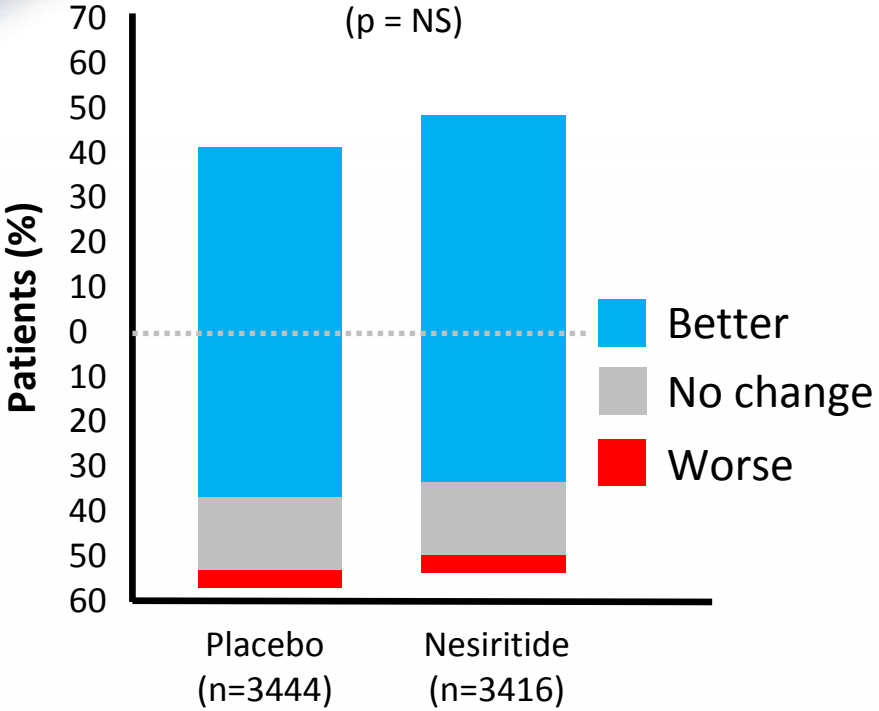


Left adapted from: *Cardiology*. 2001;96(3–4):132–43. Right adapted from: *Am J Med*. 1998 Jun 1;104(6):533–8.

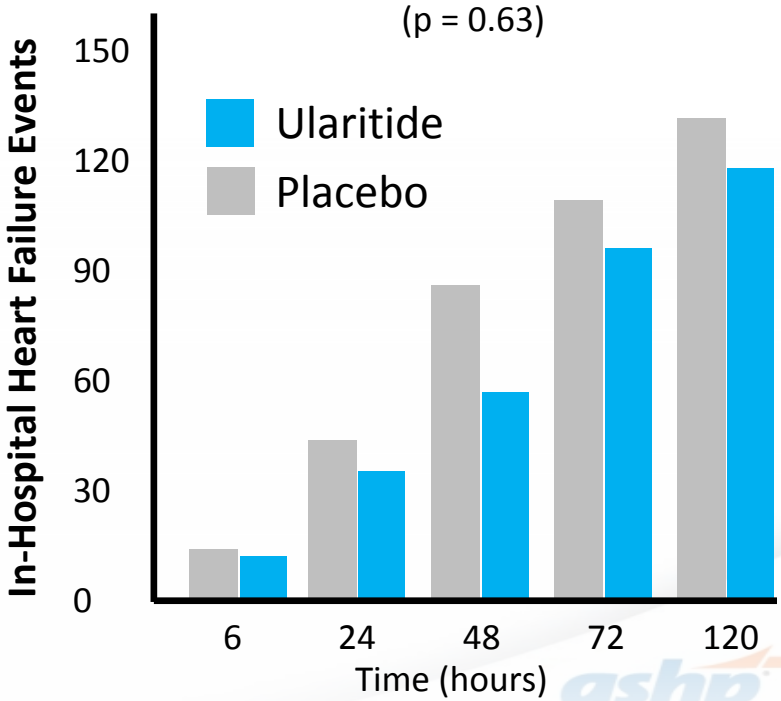
Questions

1. What may have precipitated this heart failure exacerbation?
2. How should his congestive symptoms be managed? Provide recommendations regarding drug, dose, and frequency.

ASCEND[1]
Dyspnea at 6 hours
(p = NS)

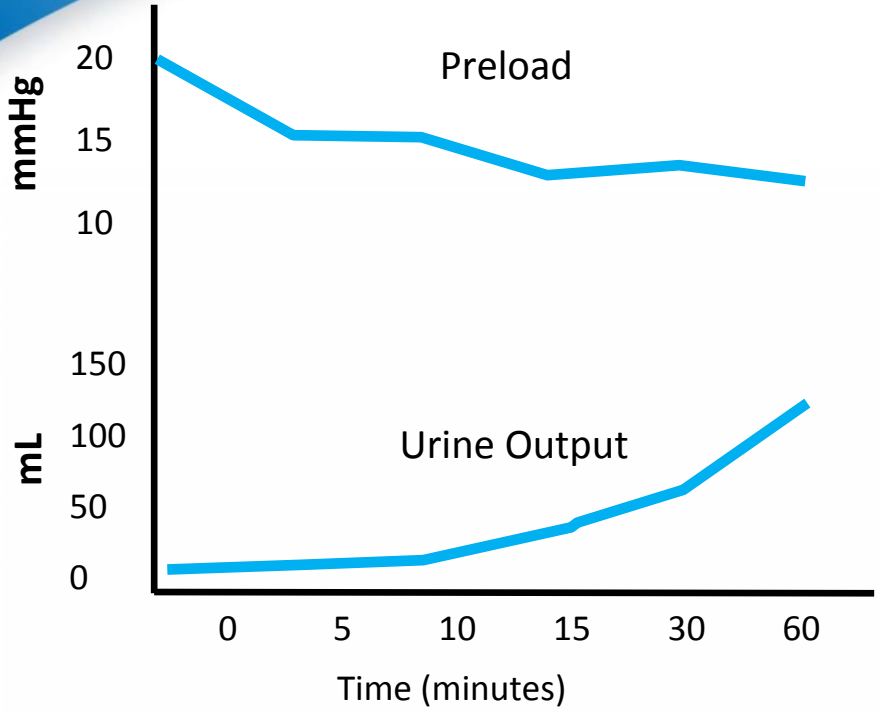


TRUE-AHF[2]
Persistent Heart Failure
(p = 0.63)

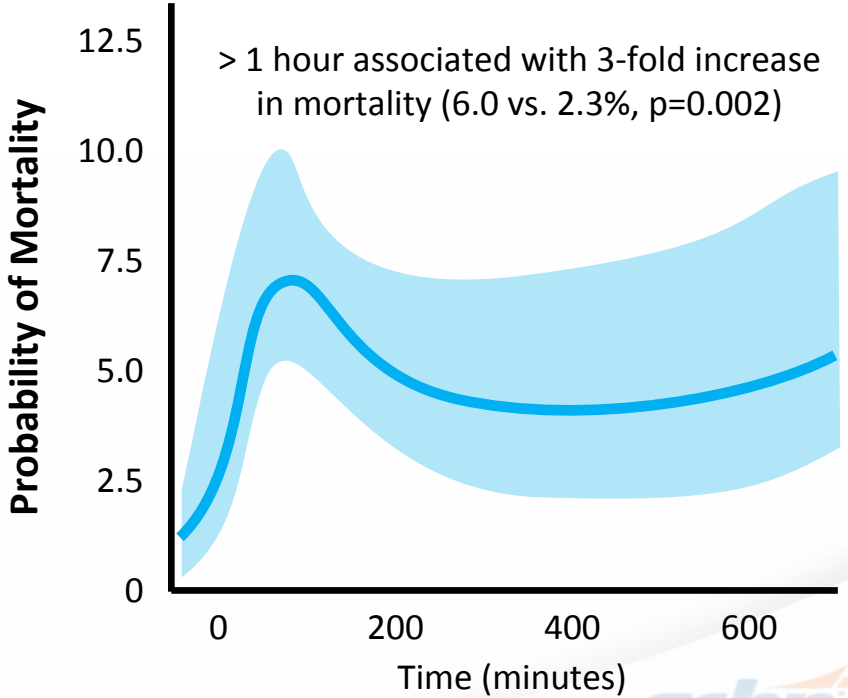


(1) *N Engl J Med*. 2011 Jul 7;365(1):32–43. (2) *N Engl J Med*. 2017 18;376(20):1956–64.

Effects of Furosemide Over Time[1]

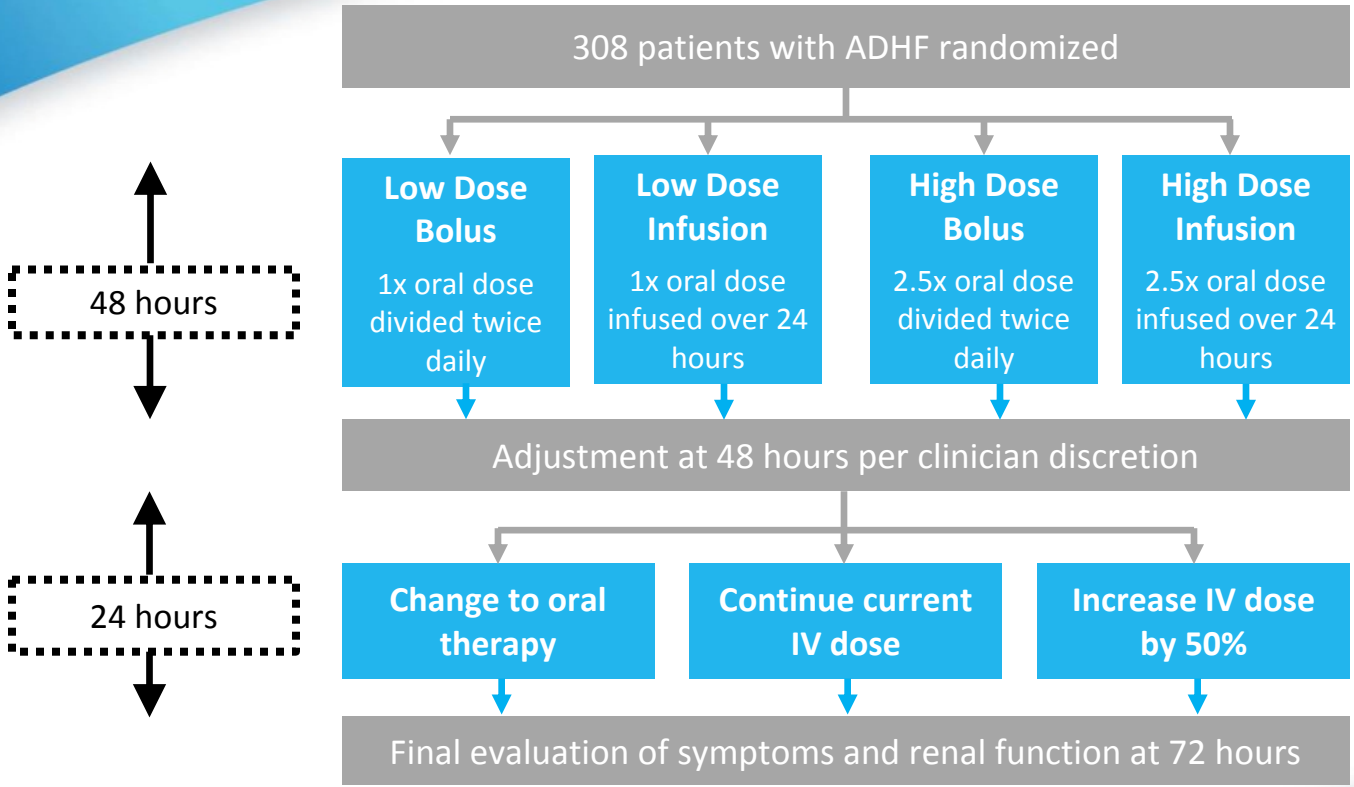


Door-to-Furosemide Time[2]



(1) *N Engl J Med*. 1973 May 24;288(21):1087–90. (2)*J Am Coll Cardiol*. 2017 Jun 27;69(25):3042–51.

DOSE Study Design



ADHF acute decompensated heart failure

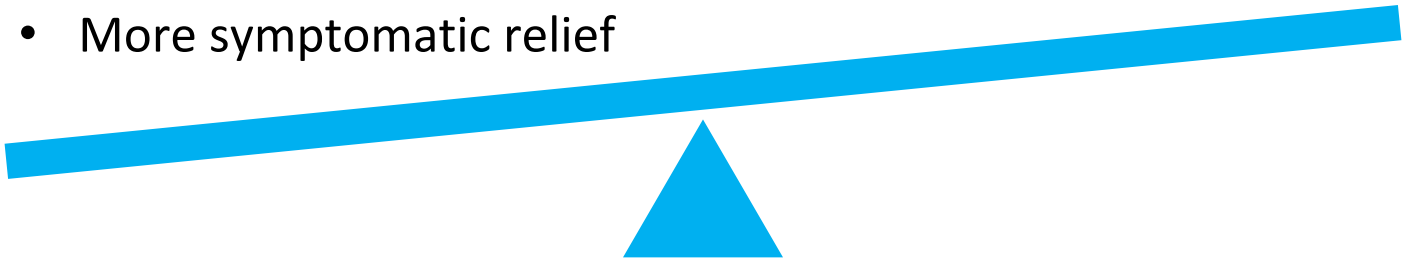
N Engl J Med. 2011 Mar 3;364(9):797–805.

High Dose

- Greater net fluid loss
- Greater weight loss
- More symptomatic relief

Low Dose

- Less transient worsening of renal function



- Low-dose less likely to be transitioned to oral diuretics and more likely to require a dose increase at 48 hours[1]
- *Transient* worsening of renal function in ADHF no worse than no change[2]

ADHF acute decompensated heart failure

(1) *N Engl J Med*. 2011 Mar 3;364(9):797–805. (2) *J Card Fail*. 2010 Jul;16(7):541–7.

Intravenous Bolus

Continuous Infusion

- Bolus arm 2x as likely to require a dose increase (21% vs. 11%, p=0.01) and receive thiazide-type diuretics (16% vs. 7%, p=0.02)[1]
- Prior trials have shown greater fluid and weight loss with continuous infusions[2]

(1) *N Engl J Med*. 2011 Mar 3;364(9):797–805. (2) *J Card Fail.* 2010 Mar;16(3):188–93.

Patients who may derive benefit from a continuous infusion?

- High bolus doses (toxicity risk)
- Delayed transcapillary refill rate (e.g., hypoalbuminemia)
- Hypotension with bolus administration
- Preload-dependent conditions (e.g., aortic stenosis, right ventricular failure)

Questions

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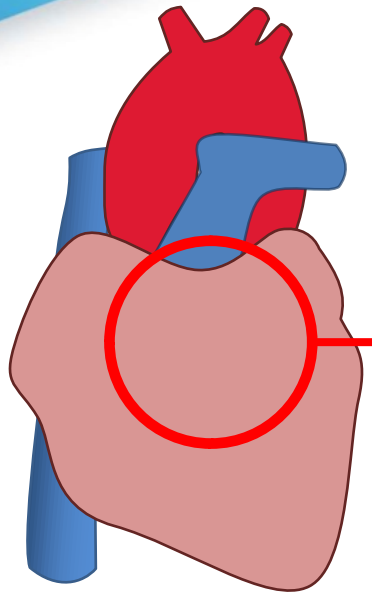
2. How should his congestive symptoms be managed? Provide recommendations regarding drug, dose, and frequency.
3. What should be done with his other guideline-directed medical therapy?



ACE inhibitor

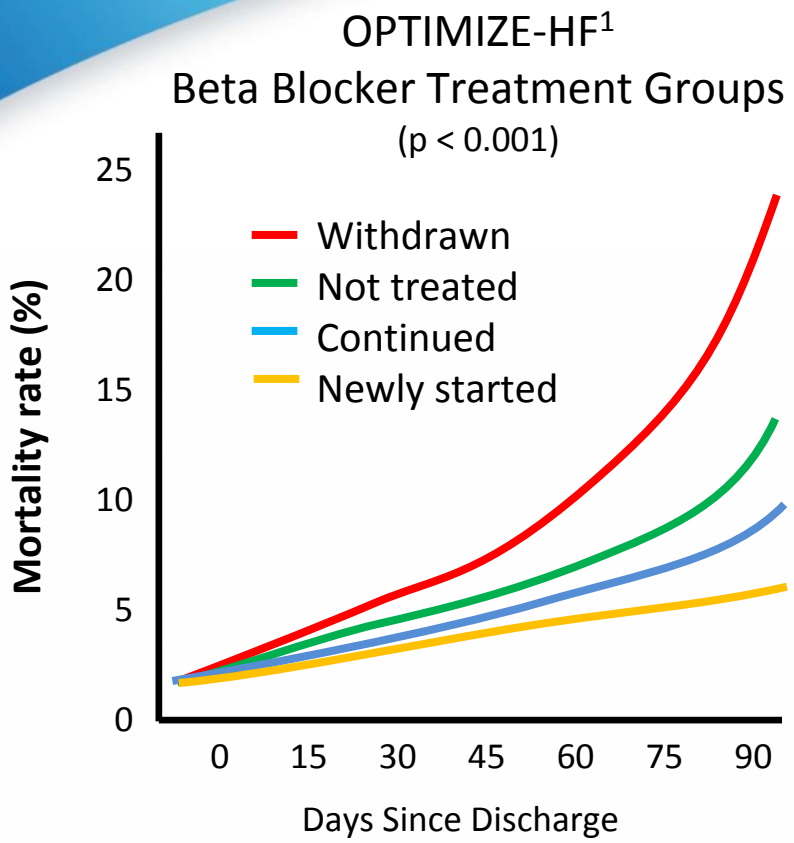
Renin-Angiotensin-Aldosterone System (RAAS)

- Increased vasoconstriction
- Increased volume retention
- Increased hypertrophy
- Increased fibrosis

Holding ACE inhibitor may increase length of stay (5.5 vs. 3.0 days, p=0.009)?

ACE angiotensin-converting enzyme

Cardiology. 2017;137(2):121–5.



- In OPTIMIZE-HF, beta blocker continuation was associated with lower risk of death (HR 0.60, 95% CI 0.37-0.99, p=0.044)[1]
- Confirmed in B-CONVINCED, which showed no worsening with continuation during hospitalization[2]

(1) *J Am Coll Cardiol* 2008; 52:190–9. (2) *Eur Heart J* 2009; 30: 2186-92.

ATHENA-HF

- Patients with ADHF receiving spironolactone 12.5-25 mg randomized to continuation vs. increasing dose to 100 mg
- No differences in congestive endpoints (NT-proBNP or dyspnea scores), urine output, or weight change

ADHF acute decompensated heart failure, NT-proBNP n-terminal pro-brain natriuretic peptide

JAMA Cardiol. 2017 Sep 1;2(9):950–8.

- What about metformin?

Questions

4. Would your recommendations for the management of congestion change if DW had HFpEF rather than HFrEF? Why or why not?

Ejection Fraction Breakdown in Recent ADHF Trials

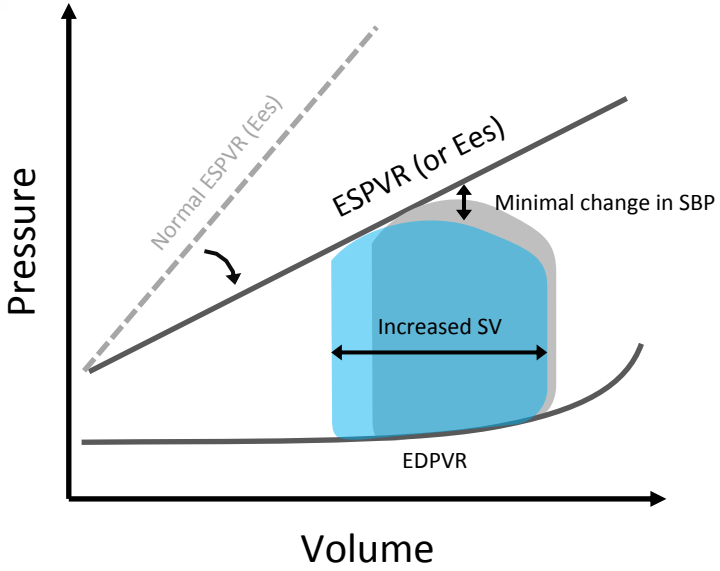
Trial	Year	Agent	Patients	Mean EF (%)	Patients with Preserved EF (%)
ASCEND-HF[1]	2011	Nesiritide	7147	NR	19.9%
DOSE[2]	2011	Loop diuretics	308	34.8%	27.0%
RELAX-AHF[3]	2012	Serelaxin	1161	38.7%	45.0%
ROSE-AHF[4]	2013	Dopamine/nesiritide	360	31.6%	24.4%
TACTICS-HF[5]	2017	Tolvaptan	257	33.0%	25.0%
TRUE-AHF[6]	2017	Ularitide	2157	NR	34.8%

EF ejection fraction, NR not reported

(1) *N Engl J Med*. 2011 Jul 7;365(1):32–43. (2) *N Engl J Med*. 2011 Mar 3;364(9):797–805. (3) *Lancet*. 2013 Jan 5;381(9860):29–39. (4) *JAMA* . 2013 Nov 18; (5) *J Am Coll Cardiol*. 2017 Mar 21;69(11):1399–406. (6) *N Engl J Med*. 2017 18;376(20):1956–64.

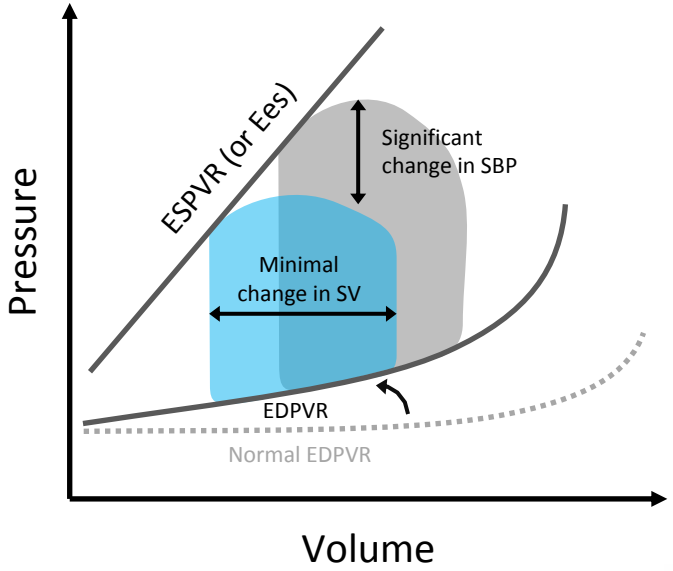
A

Changes in Pressure-Volume Relationships with Reduced EF



B

Changes in Pressure-Volume Relationships with Preserved EF



EF ejection fraction, EDPVR end-diastolic pressure-volume relationship, ESPVR end-systolic pressure-volume relationship, Ees end-systolic elastance, SV stroke volume

Adapted from data provided in *J Am Coll Cardiol*. 2012 Jan 31;59(5):442–51.

ROPA-DOP Preliminary Results

Patients (n=90) with ADHF and HFpEF randomized to bolus vs. continuous infusion

Outcome at 72 hours	Intermittent Bolus	Continuous Infusion	p (adjusted)
Increased serum creatinine	4.6%	16.0%	0.03
Worsening renal function	11.6%	36.2%	0.02
Volume output	10.3 L	10.7 L	0.98

Presented at 2017 Heart Failure Society of America Meeting.

Stiles, S. Avoid Low-Dose Dopamine, Prefer Bolus Furosemide in Preserved-EF Acute HF: ROPA-DOP. Medscape Cardiology. Available at: http://www.medscape.com/viewarticle/886049#vp_2. Accessed 2017 Oct 1.

DW experiences some minor improvement in dyspnea but his urine output is not robust and he fails to meet goal diuresis for two consecutive days (goal 2-3 L negative per day, but less than 2 L negative total for past 48 hours). He reports worsening abdominal discomfort and nausea/vomiting over the past 24 hours which is only partially relieved by antiemetics.

New medications:

- Furosemide 120 mg IV BID
- Insulin aspart sliding scale ACHS

Vitals: BP 112/72 mmHg, HR 74 bpm

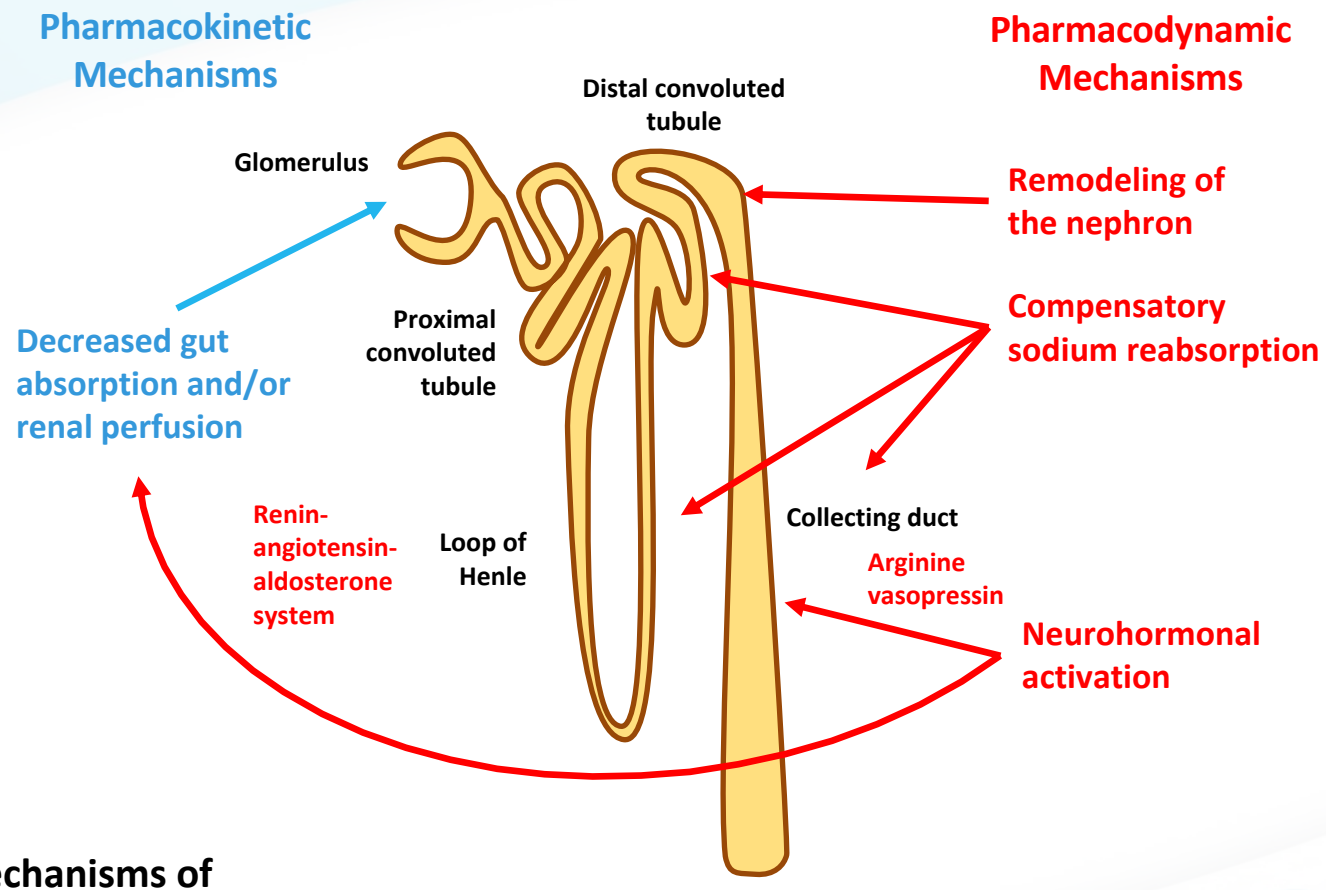
130	94	24	118
3.8	28	1.4	

Questions

5. What mechanisms might explain diuretic resistance in this patient?
6. What should be done to augment diuresis at this time? Provide recommendations regarding drug, dose, and frequency for at least two pharmacologic options.

Questions

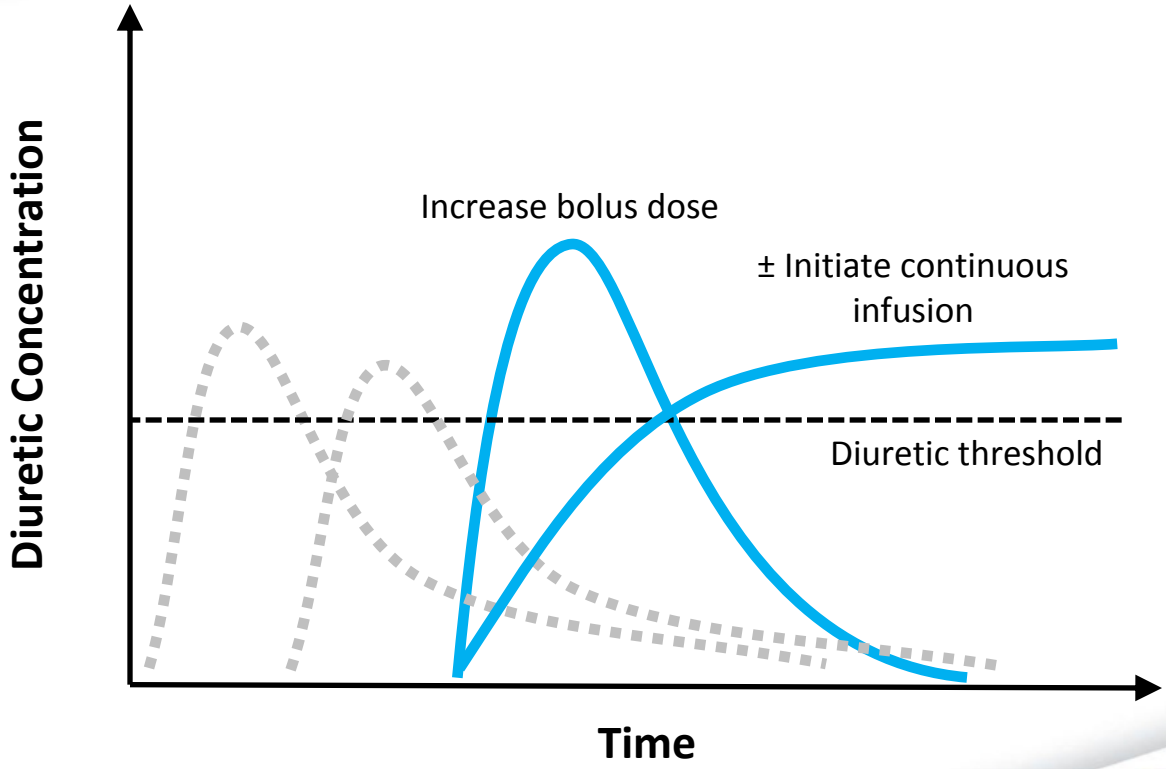
5. What mechanisms might explain diuretic resistance in this patient?



Common Mechanisms of Diuretic Resistance

Questions

5. What mechanisms might explain diuretic resistance in this patient?
6. What should be done to augment diuresis at this time? Provide recommendations regarding drug, dose, and frequency for at least two pharmacologic options.


Diuretic Concentration
Increase bolus dose
± Initiate continuous infusion
Diuretic threshold
Time

Disadvantages to continuous infusion diuretics

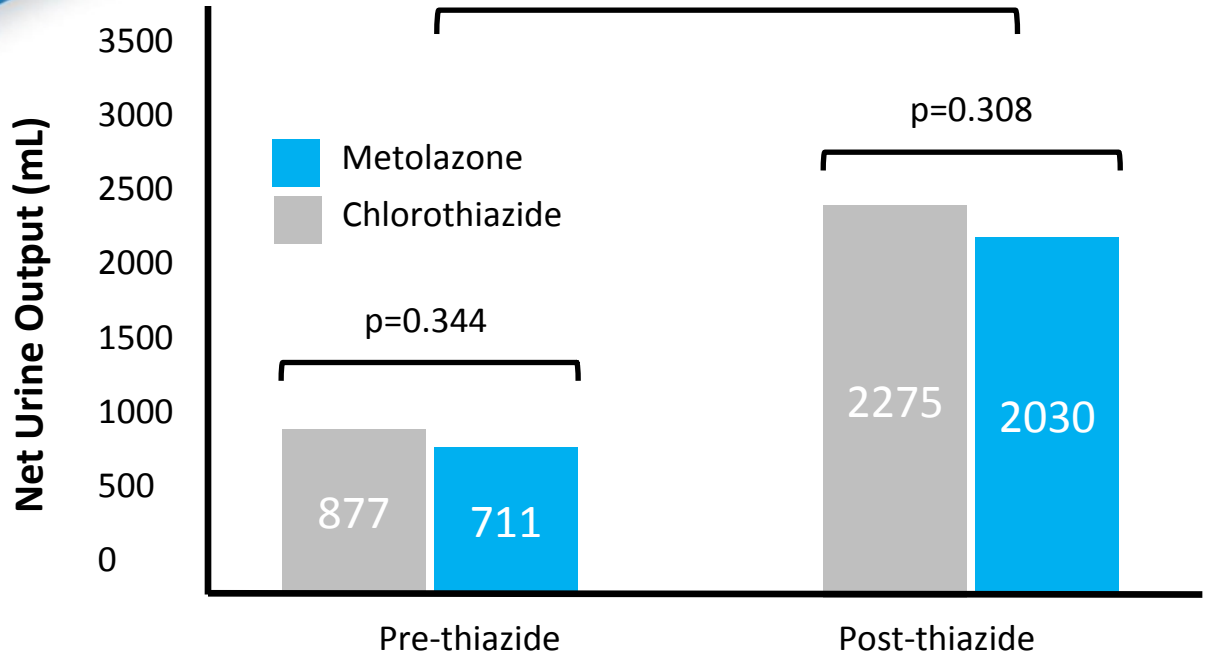
- May encourage a “set it and forget it” mentality
- Overnight urination (i.e., fall risk, decreased sleep quality)
- Unknown safety of high-dose infusions
- Drug mismanagement (omitting boluses, “titrate” orders)

Thiazide-Type Diuretics

Agent	Metolazone	Chlorothiazide	Hydrochlorothiazide
Oral bioavailability	40-65%	N/A	65-75%
Usual dose (maximum/day)	2.5–5 mg once daily (20 mg)	500–1000 mg once to twice daily (2000 mg)	25–50 mg once to twice daily (100 mg)
Onset (peak)	2–3 h (6-8 h)	2 h (3–6 h)	2 h (3–6 h)
Duration of action	12–24 h	6–12 h	6–12 h

Metolazone vs. Chlorothiazide[1]

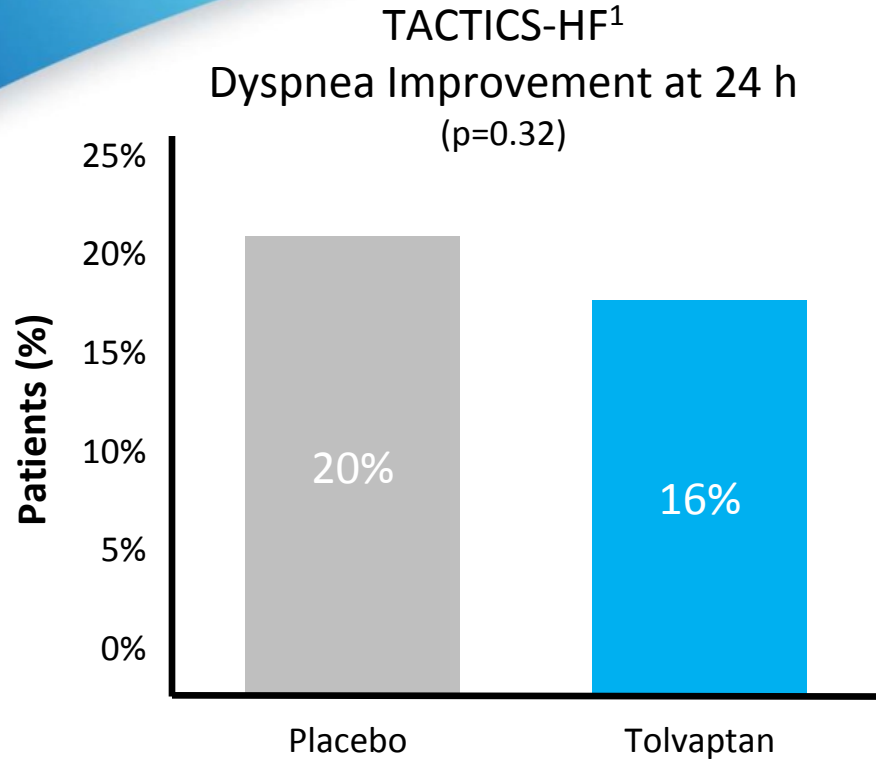
(p=0.026 for noninferiority)



From the available studies in ADHF[1-3]:
HCTZ < CTZ = MTZ

ADHF acute decompensated heart failure, CTZ chlorothiazide, HCTZ hydrochlorothiazide, MTZ metolazone

(1) *Pharmacotherapy*. 2016 Aug;36(8):852–60. (2) *Pharmacotherapy*. 2014 Aug;34(8):882–7. (3) *Cardiovasc Ther*. 2015 Apr;33(2):42–9.



- Improvements in weight and fluid loss but not symptoms[1]
- Confirmed the results of EVEREST[2]
- 48 hours of therapy: $1200
- Have less expensive options for hyponatremia (furosemide plus hypertonic saline)[3-5]

(1) *J Am Coll Cardiol*. 2017 Mar 21;69(11):1399–406. (2) *JAMA*. 2007 Mar 28;297(12):1332–43. (3) *Eur J Heart Fail*. 2000 Sep;2(3):305–13. (4) *Am Heart J*. 2003 Mar;145(3):459–66. (5) *Int J Cardiol*. 2013 Jul 15;167(1):34–40.

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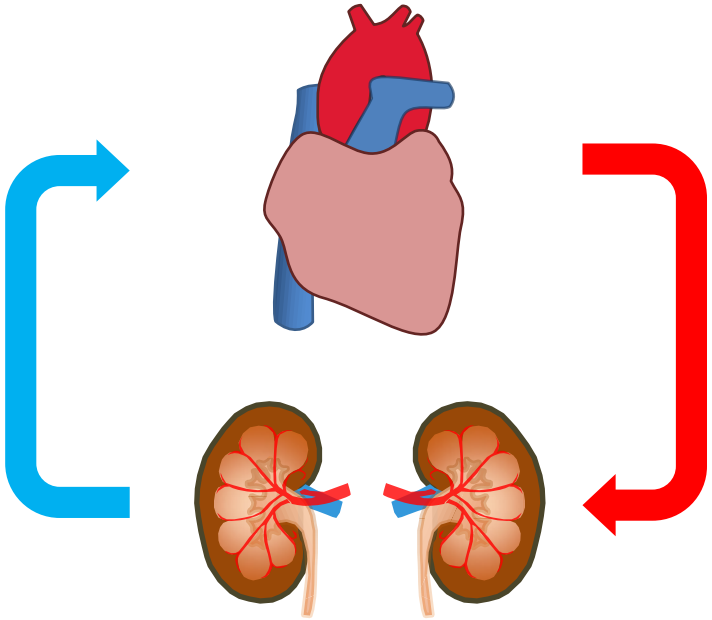
Venous Vasodilation

- Mobilizes fluid in periphery
- Decongests kidneys

Nitroglycerin*
Nitroprusside
Nesiritide



Arterial Vasodilation

- Improved renal blood flow due to reduced impedance

Nitroprusside
Nesiritide

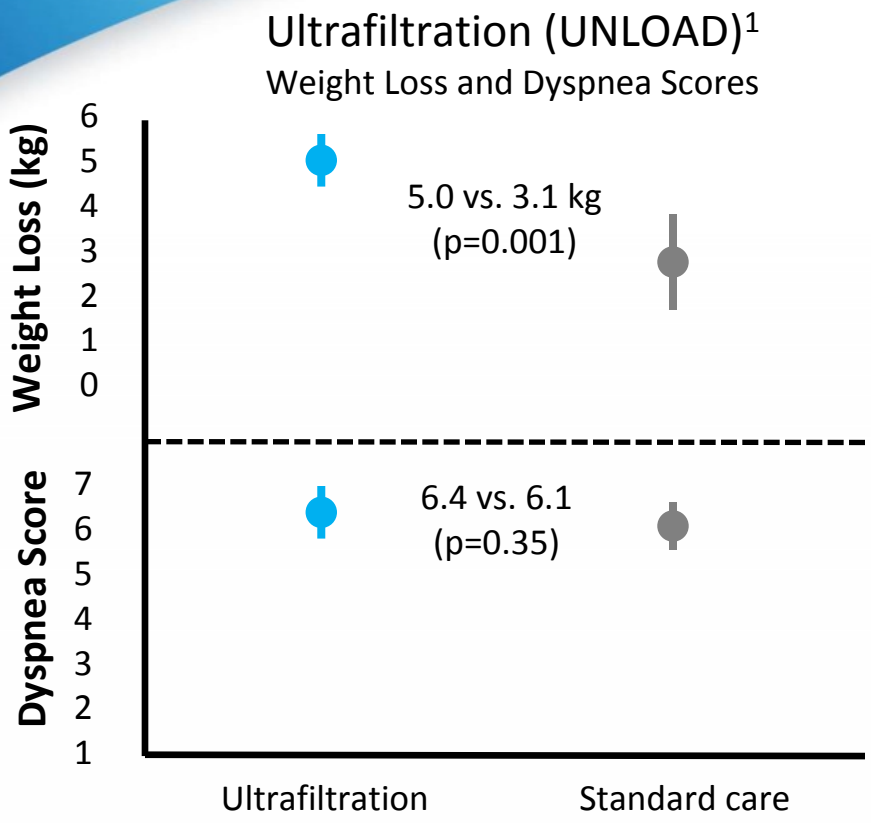
- Nitroglycerin and nesiritide improve hemodynamics and some congestive symptoms but follow-up trials of nesiritide have been equivocal[1-2]

*At high-doses (> 100 mcg/min), nitroglycerin exerts venous and arterial dilating effects.

(1) *JAMA*. 2002 Mar 27;287(12):1531–40. (2) *N Engl J Med*. 2011 Jul 7;365(1):32–43.

Intravenous Vasodilators

Agent	Nesiritide	Nitroglycerin	Nitroprusside
Dilation	Arterial, venous	Venous (mostly)	Arterial, venous
Onset	15 minutes	Immediate	Immediate
Half-life	20 minutes	4 minutes	2 minutes
Dosing	0.01–0.03 mcg/kg/min (± 2 mcg/kg bolus)	5–200 mcg/kg/min	0.3–3 mcg/kg/min
Disadvantages	• Longer half-life • Cost	• High doses required for arterial vasodilation • Tachyphylaxis with extended duration	• Toxic metabolites in severe renal or hepatic impairment • Cost



- Excluded patients on inotropes or vasodilators and those who required initiation were deemed treatment failures
- Average removal rate 250 mL/h to target 80% of excess body weight
- Ultrafiltration was also associated with fewer rehospitalizations (18 vs. 32%, p=0.037)

J Am Coll Cardiol. 2007 Feb 13;49(6):675–83.

Despite the changes you made to DW's therapy, his urine output does not improve and he is less than 1 L negative overnight. His latest vital signs include a blood pressure of 104/62 mmHg and heart rate of 82 bpm. Morning labs are significant for a serum creatinine of 1.9 (up from 1.4 mg/dL at admission). The team decides to place a pulmonary artery catheter which reveals the following information:

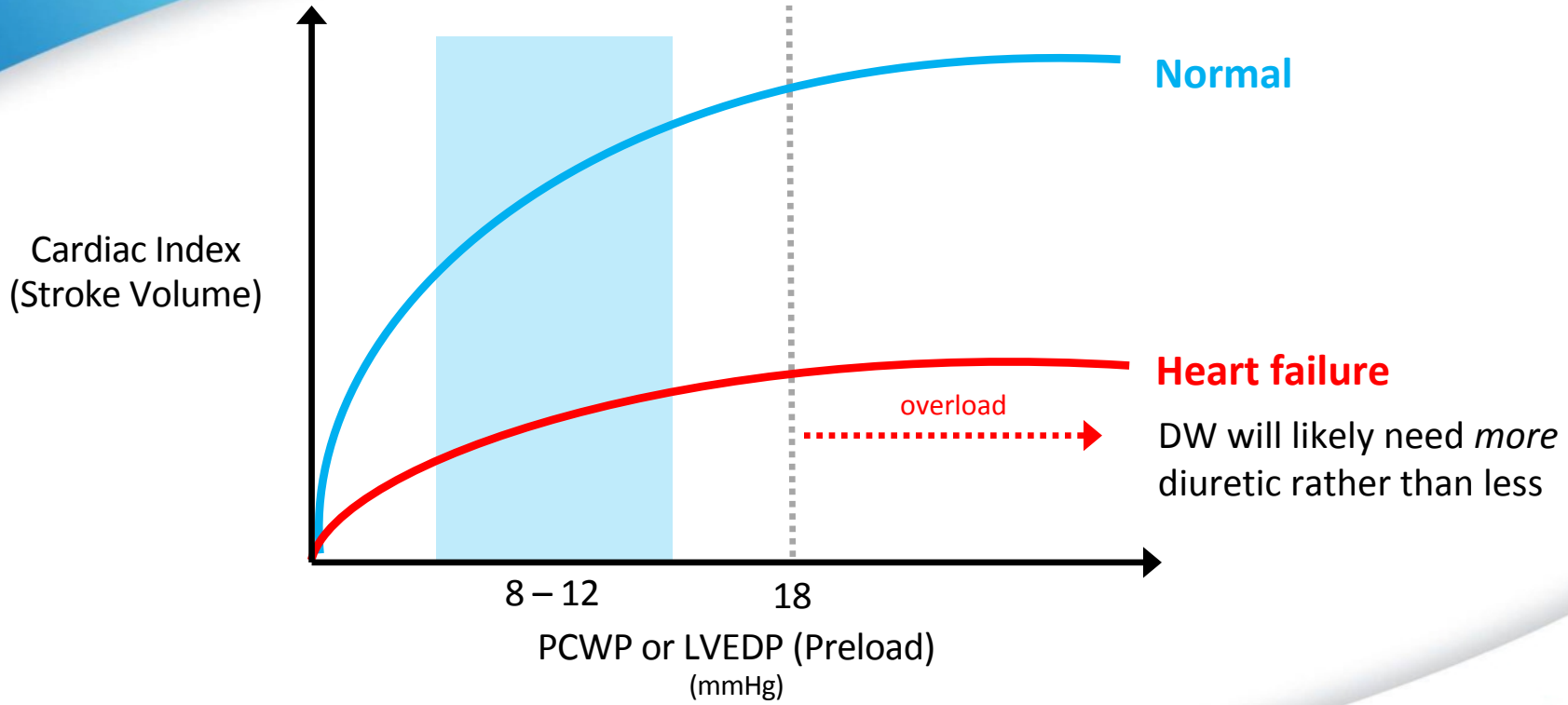
Right atrium: 22 mmHg
Right ventricle: 42/20 mmHg
Pulmonary artery: 40/22 (28) mmHg
Wedge pressure: 26 mmHg

Cardiac output: 4.2 L/min
Cardiac index: 2.0 L/min/m^2
Systemic vascular resistance: 910 dynes·sec/cm^5

The patient is subsequently placed on dobutamine at 3 mcg/kg/min.

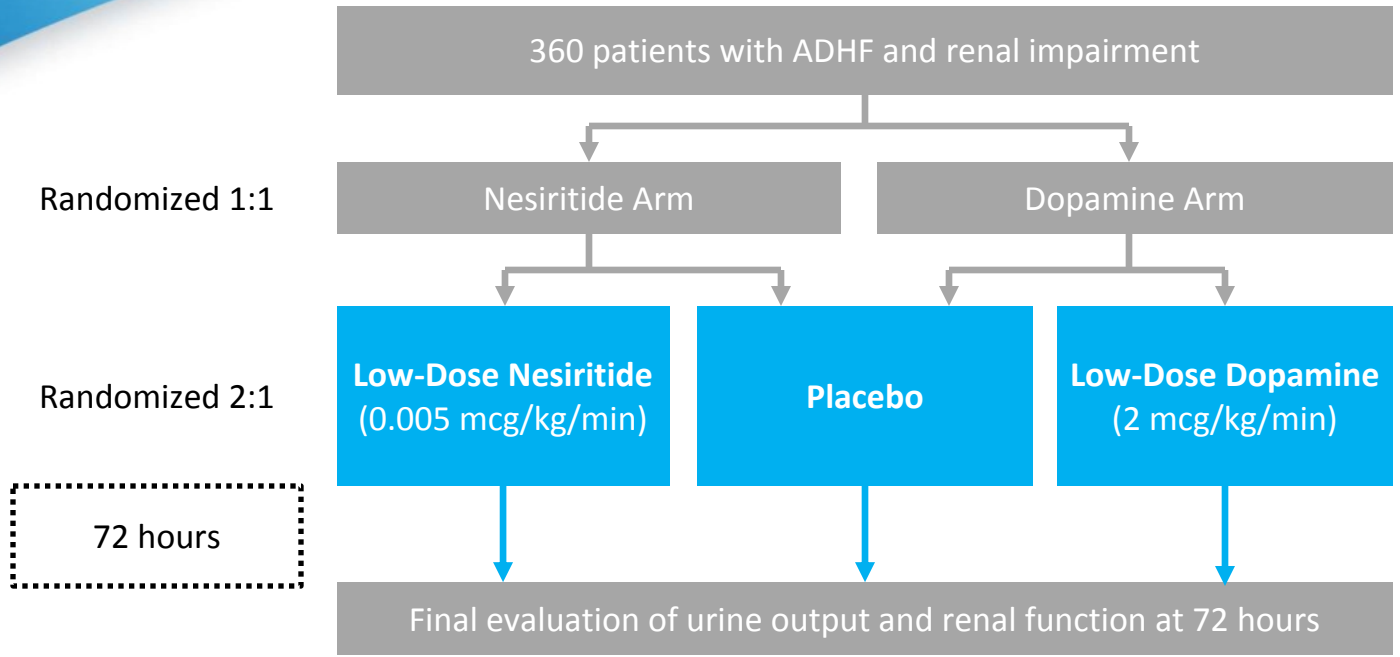
Questions

7. Would your recommendations for volume management change at this time? Why or why not?



LVEDP left ventricular end-diastolic pressure
PCWP pulmonary capillary wedge pressure

ROSE-AHF Study Design



ADHF acute decompensated heart failure

JAMA. 2013 Dec 18;310(23):2533-43.

ROSE Study Results

Dopamine Arm

Outcome	Placebo	Dopamine	P
Cumulative urine output	8296	8524	0.59
Change in cystatin C	0.11	0.12	0.72
Patient-reported symptoms (AUC)	4704	4553	0.43
Drug discontinued due to tachycardia	0.9%	7.2%	< 0.001

Nesiritide Arm

Outcome	Placebo	Nesiritide	P
Cumulative urine output	8296	8574	0.49
Change in cystatin C	0.11	0.07	0.36
Patient-reported symptoms (AUC)	4704	4498	0.62
Drug discontinued due to hypotension	10.4%	18.8%	0.07

JAMA. 2013 Dec 18;310(23):2533-43.

CARRESS-HF

Patients with ADHF and renal impairment randomized

Ultrafiltration 200 mL/h for 96 hours

or

Home Dose	Furosemide Starting Dose
≤ 80 mg	40 mg IVB, then 5 mg/h
81-160 mg	80 mg IVB, then 10 mg/h + MTZ 5 mg
161-240 mg	80 mg IVB, then 20 mg/h + MTZ 5 mg BID
> 240 mg	80 mg IVB, then 30 mg/h + MTZ 5 mg BID

If patient fails to meet urine output goals:

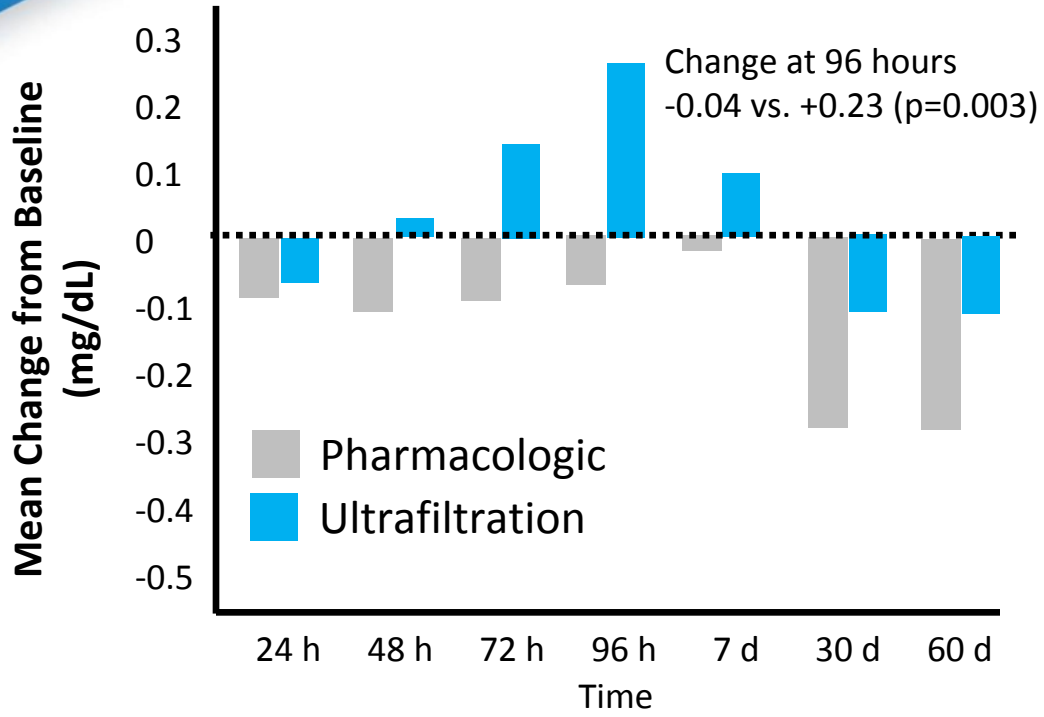
1. At 24 hours, advance diuretics
2. At 48 hours, Step 1 and consider vasodilators/inotropes
3. At 72-96 hours, Step 1-2 and consider hemodynamic guided-therapy ± MCS

ADHF acute decompensated heart failure, IVB intravenous bolus, MCS mechanical circulatory support, MTZ metolazone

JAMA. 2013 Dec 18;310(23):2533-43.

CARRESS-HF[1]

Changes in Serum Creatinine



- More adverse effects also seen with ultrafiltration (72% vs. 57%, p=0.03)
- Disrupted renal counter-regulatory response?
- Masked low output?

(1) *JAMA.* 2013 Dec 18;310(23):2533-43.

After a week of inotropic support and aggressive diuresis, DW's symptoms have significantly improved. He has been successfully weaned from dobutamine and is approaching his baseline weight. The team plans to send him home in the next several days and is preparing a discharge plan. Numerous changes have been made to his medication regimen during the hospitalization.

Current Medications:

- Aspirin 81 mg daily
- Atorvastatin 40 mg daily
- Isosorbide dinitrate 20 mg TID
- Hydralazine 50 mg TID
- Spironolactone 25 mg once daily
- Furosemide 80 mg IV once daily
- Insulin glargine 40 units subq at night
- Insulin aspart sliding scale ACHS

Vitals: BP 114/80 mmHg, HR 70 bpm

136	96	26	124
4.3	24	1.3	

Questions

8. What changes to this patient's medication regimen should be considered as he approaches discharge?
9. What non-pharmacologic strategies might also reduce his risk of readmission?

Questions

8. What changes to this patient's medication regimen should be considered as he approaches discharge?

PARADIGM-HF

Should the patient be changed to sacubitril/valsartan?

Inclusions	Exclusions
• NYHA Class II-IV symptoms • Ejection fraction ≤ 35% • NT-proBNP ≥ 600 pg/mL or ≥ 400 if hospitalized in the last 12 months • Enalapril equivalent ≥ 10 mg/day	• Symptomatic hypotension • Blood pressure < 100/95 mmHg • GFR < 30 mL/min • Serum potassium ≥ 5.4 • Unacceptable side effects

- Compared to enalapril, sacubitril/valsartan reduced the composite of death or first hospitalization for heart failure (21.8 vs. 26.5%, p<0.001)

NYHA New York Heart Association, GFR glomerular filtration rate, NT-proBNP n-terminal pro-brain natriuretic peptide

N Engl J Med. 2014 Sep 11;371(11):993–1004.

Diuretic Observation Prior to Discharge

Patients (n=123) observed on discharge diuretic < 24 or ≥ 24 hours prior to discharge

Outcome	< 24 hour (n=61)	≥ 24 hour (n=62)	p (adjusted)
30-day heart failure readmission	11 (18%)	2 (3.2%)	<0.001
60-day heart failure readmission	18 (29.5%)	6 (9.7%)	<0.001
90-day heart failure readmission	23 (37.7%)	12 (19.4%)	<0.001
Any heart failure readmission	34 (55.7%)	23 (37.1%)	<0.001

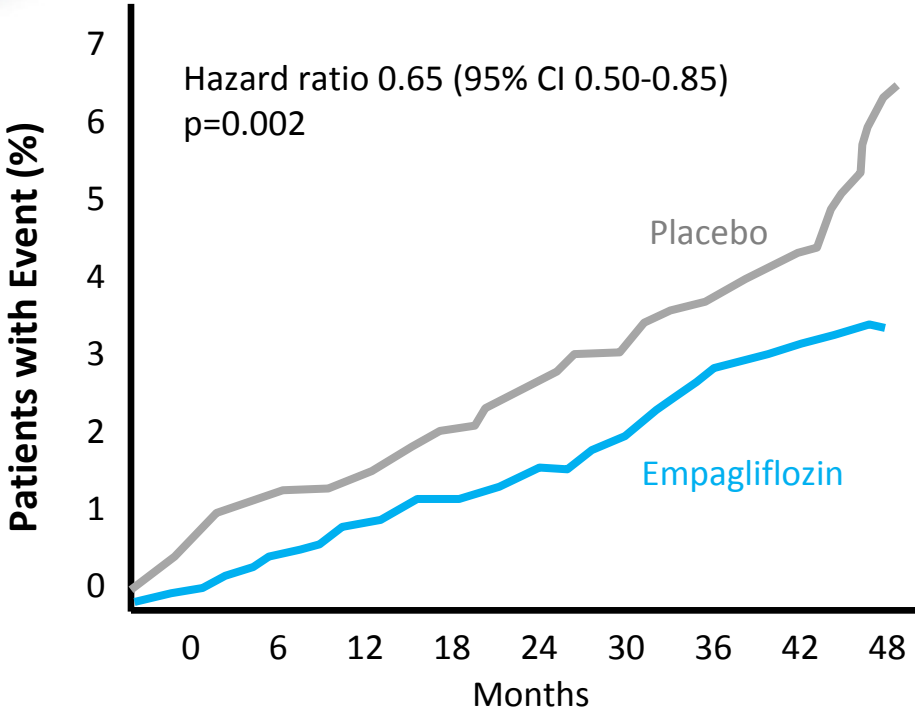
J Card Fail. 2017 Jul 5. pii: S1071-9164(17)30201-4

Other Medication Adjustments

- Beta blocker?
- Ivabradine or digoxin?
- Non-heart failure medications?

EMPA-REG OUTCOME[1]

Hospitalization for Heart Failure



- Empagliflozin also associated with reduction in cardiovascular death (3.7% vs. 5.9%, p<0.001)
- Patients may require reduction in diuretic dose

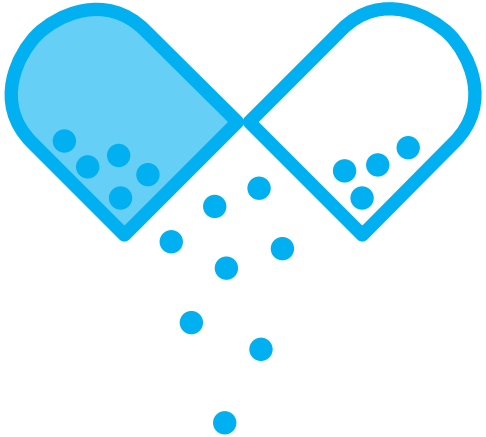
N Engl J Med 2015;373:2117-28.

Questions

8. What changes to this patient's medication regimen should be considered as he approaches discharge?
9. What non-pharmacologic strategies might also reduce his risk of readmission?

- Pharmacist-provided patient education associated with > 40% reduction in readmissions across several trials[1,2]
- Largest trial (PILL-CVD) did not impact readmissions but compared individualized to standardized education[3]
- A single session at discharge unlikely to reduce readmissions significantly

(1) *Farm Hosp Organo Of Expresion Cient*. 2006 Dec;30(6):328–42. (2) *Am J Health-Syst Pharm*. 1999 Jul 1;56(13):1339–42. (3) *Ann Intern Med*. 2012 Jul 3;157(1):1–10.



- Medication adherence remains a major contributor to readmissions
- Pharmacists improve adherence rates, which have been associated with reductions in readmission of 19-43%[1-3]
- Benefits greatest with longitudinal programs vs. single intervention

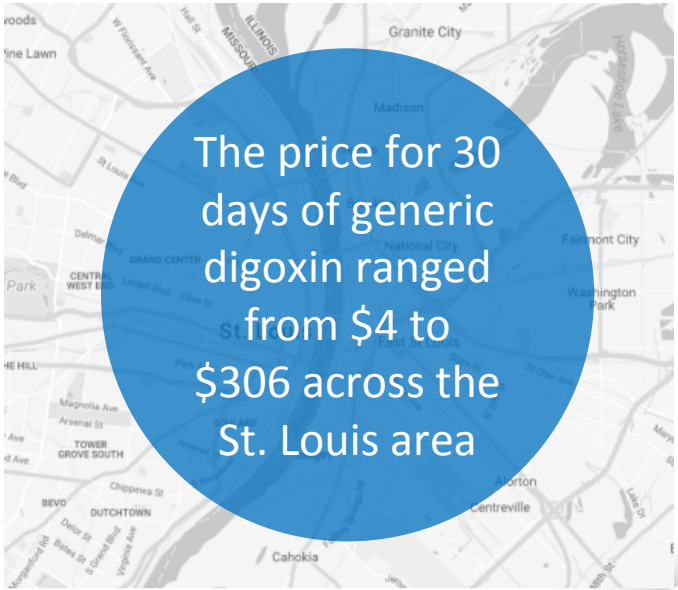
(1) *Arch Intern Med*. 1998 May 25;158(10):1067–72. (2) *Prog Cardiovasc Dis*. 2017 Aug 18. pii: S0033-0620(17)30113-5. (3) *Ann Intern Med*. 2007 May 15;146(10):714–25.

Example Adherence-Improvement Strategies

- Simplifying complex regimens (e.g., less frequently dosed medications, reducing unnecessary polypharmacy)
- Individualized education (e.g., adjusting diuretic based on weight)
- Improving medication-taking behavior (e.g., pillboxes, alerts, integrating medications into daily routines)
- Referral to pharmacist-managed bridge clinic[1]
- Improving access by identifying lower cost alternatives

(1) *Ann Pharmacother*. 2017 Jul;51(7):555–62.

- Financial limitations are a major barrier
- Even within the same geographic area, 75-fold variability in cost observed
- Made more challenging by the fragmented health payment system
- Efforts to improve access requires a committed outpatient/community pharmacy team



JAMA Intern Med. 2017 Jan 1;177(1):126–8.

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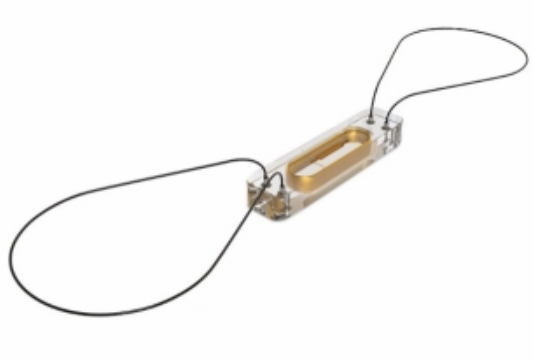
Pulmonary Artery Pressure Monitoring



Sensor

Hospital System

Patient System

Images used with permission from St. Jude Medical.

Patient Management in CHAMPION

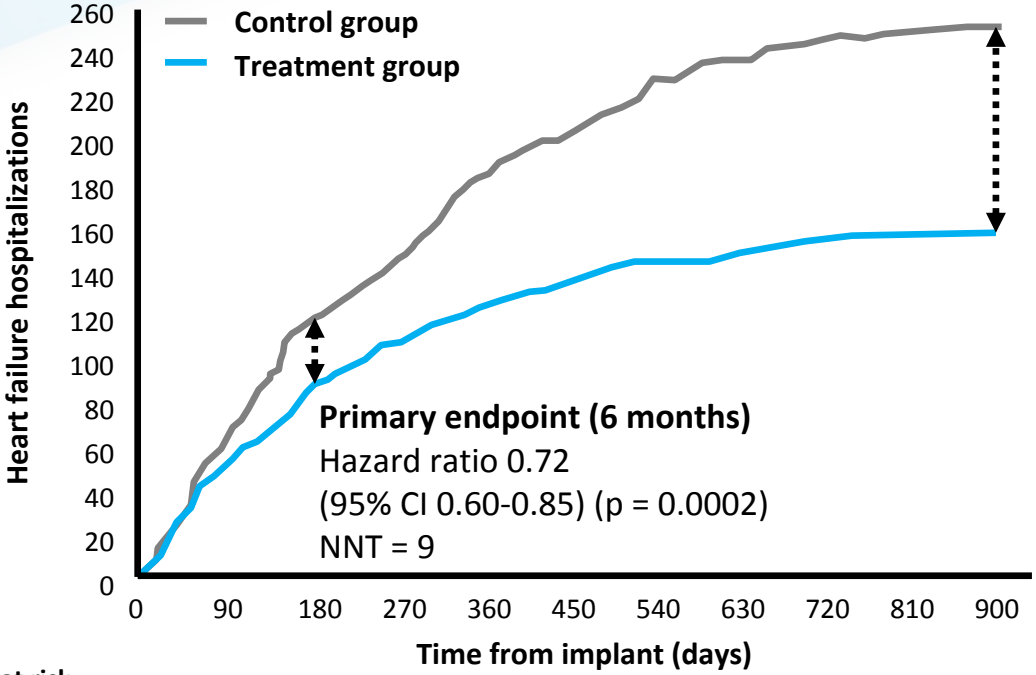
Adults with NYHA Class III heart failure and hospitalization within prior 12 months (n=550)

	Hypovolemic	"Optivolemic"	Hypervolemic
Definition	Poor perfusion without congestive symptoms PA pressures below goal	No congestive symptoms PA systolic 15-35 mmHg PA diastolic 8-12 mmHg PA mean 10-25 mmHg	Congestive symptoms PA pressures above goal
Treatment	Decrease intensity of diuretics or vasodilators Liberalize fluid/salt intake Hold ACEi/ARB if renal function worsened Consider admission	Continue diuretic regimen Optimize GDMT	Increase intensity of diuretics or vasodilators Re-educate on fluid/salt intake
Follow-Up	≥ 2-3 days/week	Weekly (2-3 days/week if GDMT adjusted)	≥ 2-3 days/week

ACEi angiotensin-converting enzyme inhibitor, ARB angiotensin receptor blocker, GDMT guideline-directed medical therapy, PA pulmonary artery

J Card Fail. 2011 Jan;17(1):3–10.

CHAMPION Results



Number at risk

Control group	280	267	252	215	179	137	105	67	25	10	0
Treatment group	270	262	244	210	169	131	108	82	29	5	1

Supplementary endpoint (mean 15 months of follow-up)
Hazard ratio 0.63
(95% CI 0.52-0.77) (p < 0.0001)
NNT = 4

Also improved with W-IHM (p < 0.05):

- Mean PA pressures
- Days alive
- Quality of life

PA pulmonary artery, W-IHM wireless implantable hemodynamic monitor
Lancet. 2011 Feb 19;377(9766):658–66.

DW has a pulmonary artery (PA) pressure monitor placed during his admission and you are assigned to follow his PA pressures. Approximately 6 weeks after discharge, he calls the clinic with complaints of “feeling funny” and wants to know if it is related to his new medication (i.e., sacubitril/valsartan). You ask him to transmit readings from his PA pressure monitor, which reveals:

PA systolic 12 mmHg, PA diastolic 6 mmHg, and PA mean 8 mmHg

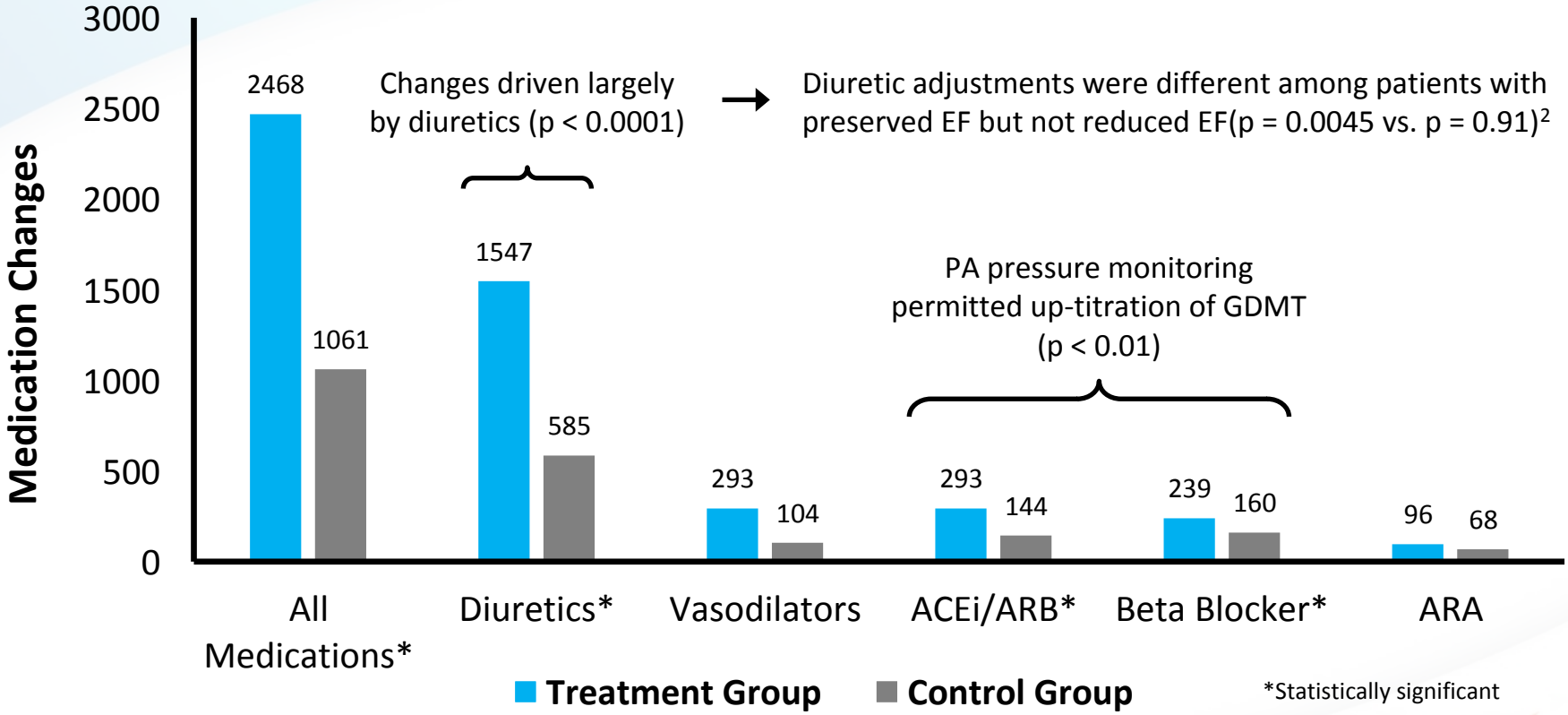
Heart failure medications:

- Sacubitril/valsartan 49/51 mg BID
- Metoprolol succinate 25 mg once daily
- Spironolactone 25 mg once daily
- Torsemide 60 mg once daily

Questions

10. What changes would you like to make to his heart failure regimen?

Frequency of Medication Changes in CHAMPION[1]



ACEi angiotensin-converting enzyme inhibitor, ARA aldosterone receptor antagonist, ARB angiotensin receptor blocker, EF ejection fraction, GDMT guideline directed medical therapy, PA pulmonary artery. *JACC Heart Fail*. 2016 May;4(5):333–44. *Circ Heart Fail*. 2014 Nov;7(6):935–44.

ashp MIDYEAR 2017
Clinical Meeting & Exhibition

Getting to Dry:
Management of Acute Decompensated Heart Failure with Volume Overload

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