ACUTELY DECOMPENSATED HEART FAILURE AN OVERVIEW OF THE TRANSITION

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DISCLOSURES

- None

OBJECTIVES

- Explore clinical presentation of acute decompensation.
- Explore hemodynamic subsets of acutely decompensated heart failure (ADHF).
- Explore approaches to the management of ADHF.
- Determine guideline based management aimed at each stages explored.
- Explore characteristics that determine transition to advanced heart failure.

PARADIGM SHIFTS IN HEART FAILURE

Period	Clinical Endpoint	Pathophysiology	Therapy
Pre-1970	Edema	Na+ retention	Diuretics
1970-1985	Symptoms	Hemodynamics	Inotropes Vasodilators Diuretics
1985-1995	Survival	Neurohormonal activation	ACE inhibitors/ARBs β-Blockers Spironolactone NO donors
Since 1995	Cellular Mechanics	Apoptosis	Antioxidants* Cytokine antagonists* NO-regulators*
Since 2001	Symptoms and Survival	Replacement	Assist devices or TAH

* Ongoing

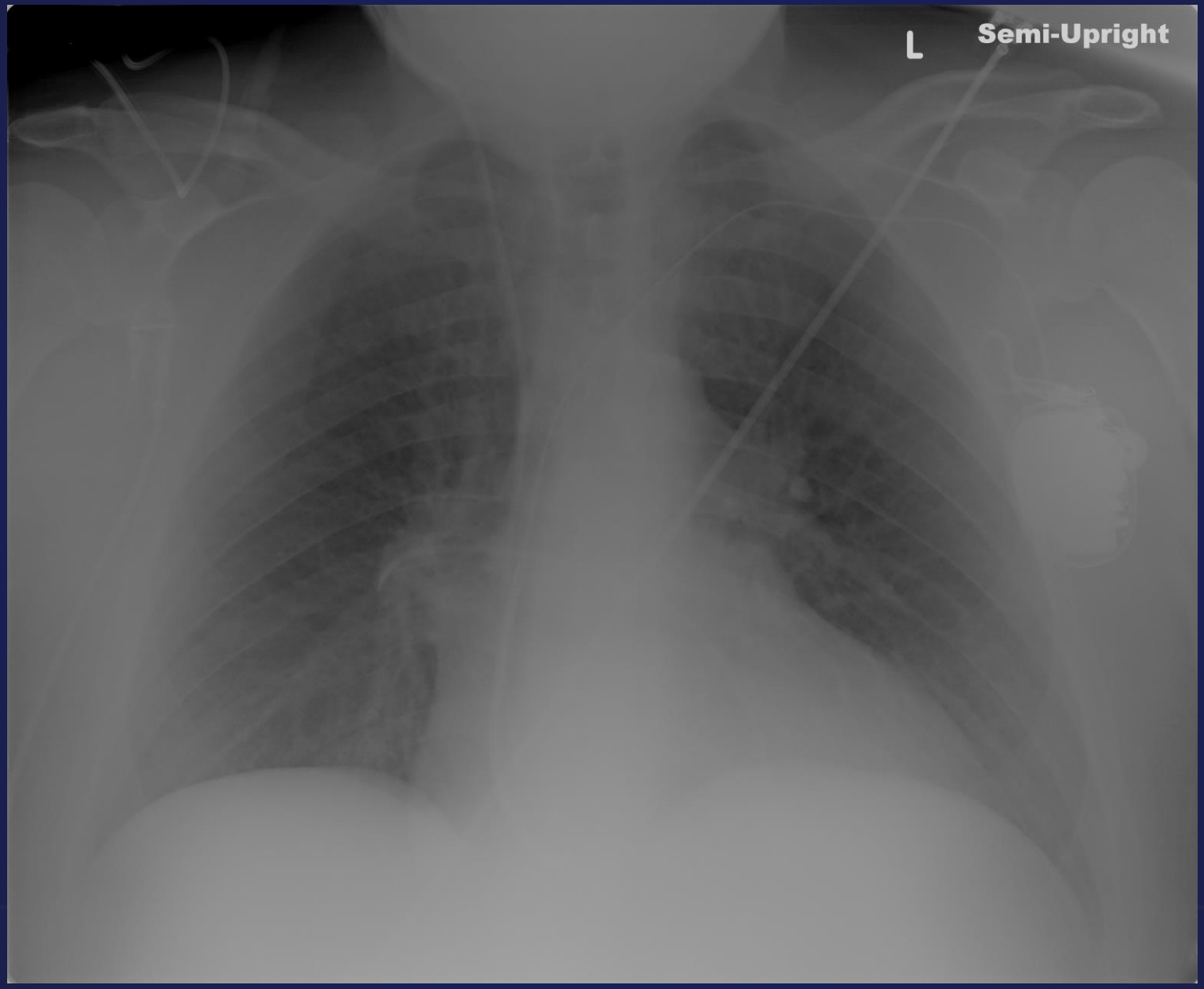
PATIENT #1: IS HE DECOMPENSATED?

- 44M with acute decompensated HF
- NIDCM (EF 19%), ICD 2006
- NYHA Class III at baseline
  - VO2 max 19.6 mL/kg/min
- 3 hospitalizations in 3 months

- Off medications x 2 weeks
- Discharged from outside hospital (2 days)
  - Minimal relief from IV diuresis
  - Cr 1.7, BNP 1143
- Exertional chest pain, SOB at rest, dry heaves, presyncope, palpitations
- 50 pounds above dry weight

- BP: 130/85, HR: 90
- Comfortable at rest only
- S1S2, + RV heave, 3/6 SEM, no S3, S4
- Lungs clear
- Nonpitting edema to thighs bilaterally

- Hgb 13
- Cr 1.3
- BUN 28
- LFTs normal
- BNP 1160
- Troponins negativeLVEF 25%, hypokinesis
- Mild MR, annular dilatation
- Mildly dilated IVC, normal inspiratory collapse
- CVP not that high but marked increased in filling pressures


L
Semi-Upright

PATIENT #2
IS HE DECOMPENSATED?

- 54 year old man referred with 4 years of HF from DCM. NYHA III-IV, 3 ADHF admissions in 9 months
- Echo EF 15%, EDD 7.4, Mod MR, Mod TR, Mod RV dysfunction
- Cr 1.6, BUN 47, Na 136
- 6MWD = 220 m

- Comfortable

BP 95/75, HR 94, JVD 12, clear lungs, +S3, +S4 , P2, Palpable liver, Cool ext. 2+ edema

- Meds:

Carvedilol, Lisinopril, Aldactone, Digoxin, Lasix

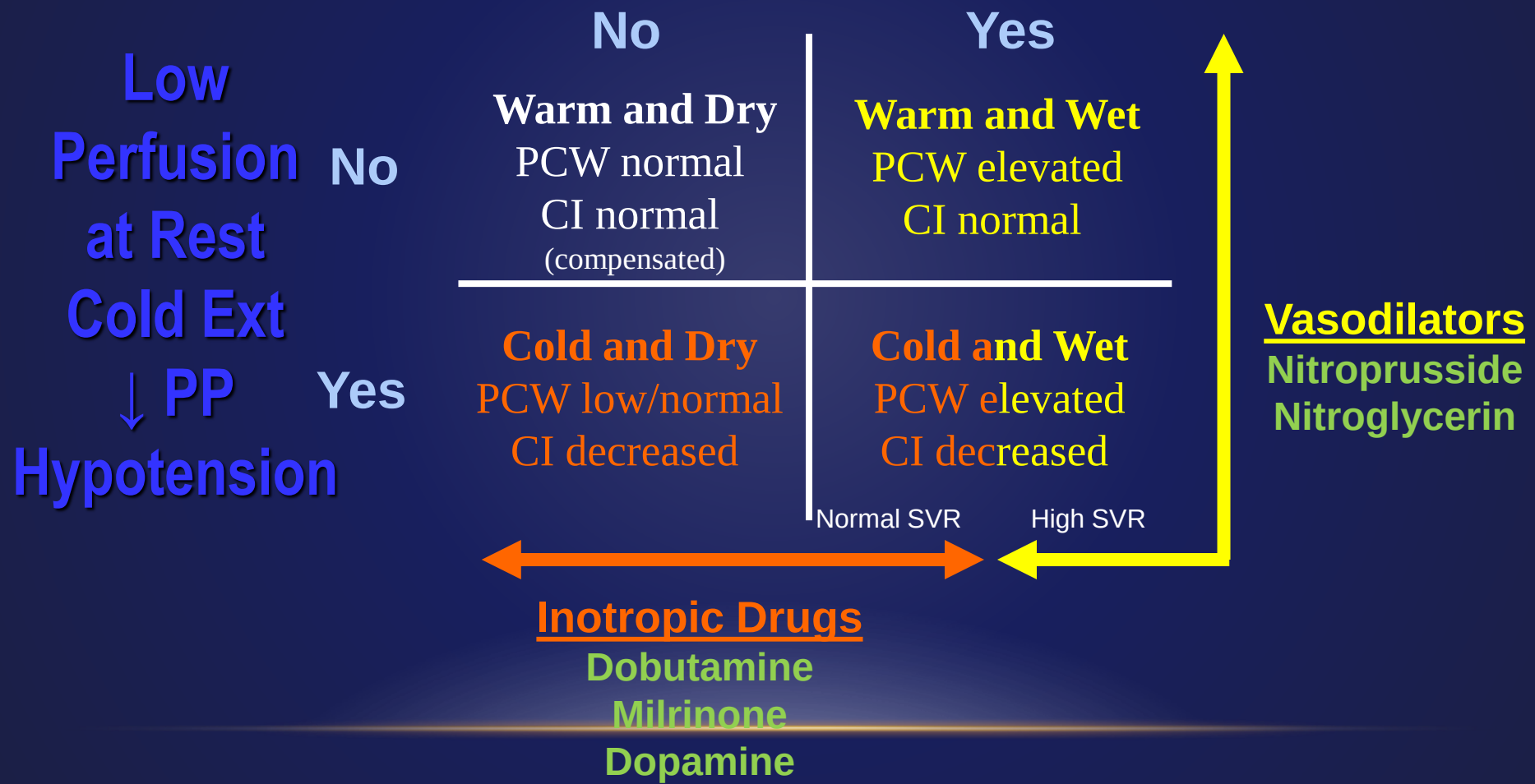
FORRESTER CLASSIFICATION

Class	Description	CI	PCWP	Mort (%)
I	No congestion/peripheral hypo-perfusion	2.7	12	2
II	Isolated congestion	2.3	23	10
III	Isolated peripheral hypo-perfusion	1.9	12	22
IV	Both congestion and peripheral hypo-perfusion	1.7	27	55

Forrester JS. Am J Cardiol 1977;39;137

Before starting treatment
Determine Hemodynamic Subset

Congestion at Rest (Orthopnea, JVD, ascites, edema, S3)



R. Bourge, UAB Cardiology (adapted from L. Stevenson)
Stevenson LW. *Eur J Heart Failure* 1999;1:251-257

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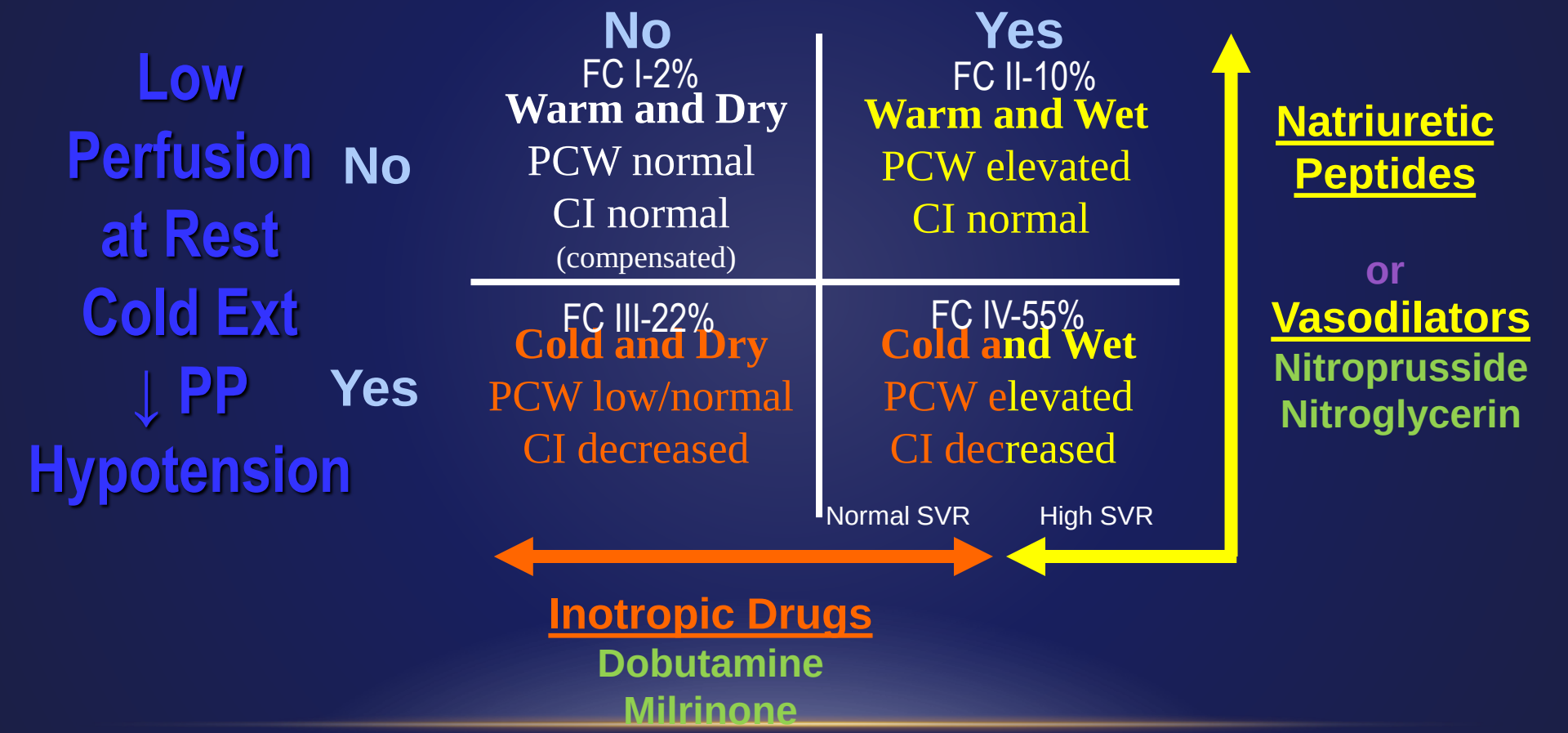

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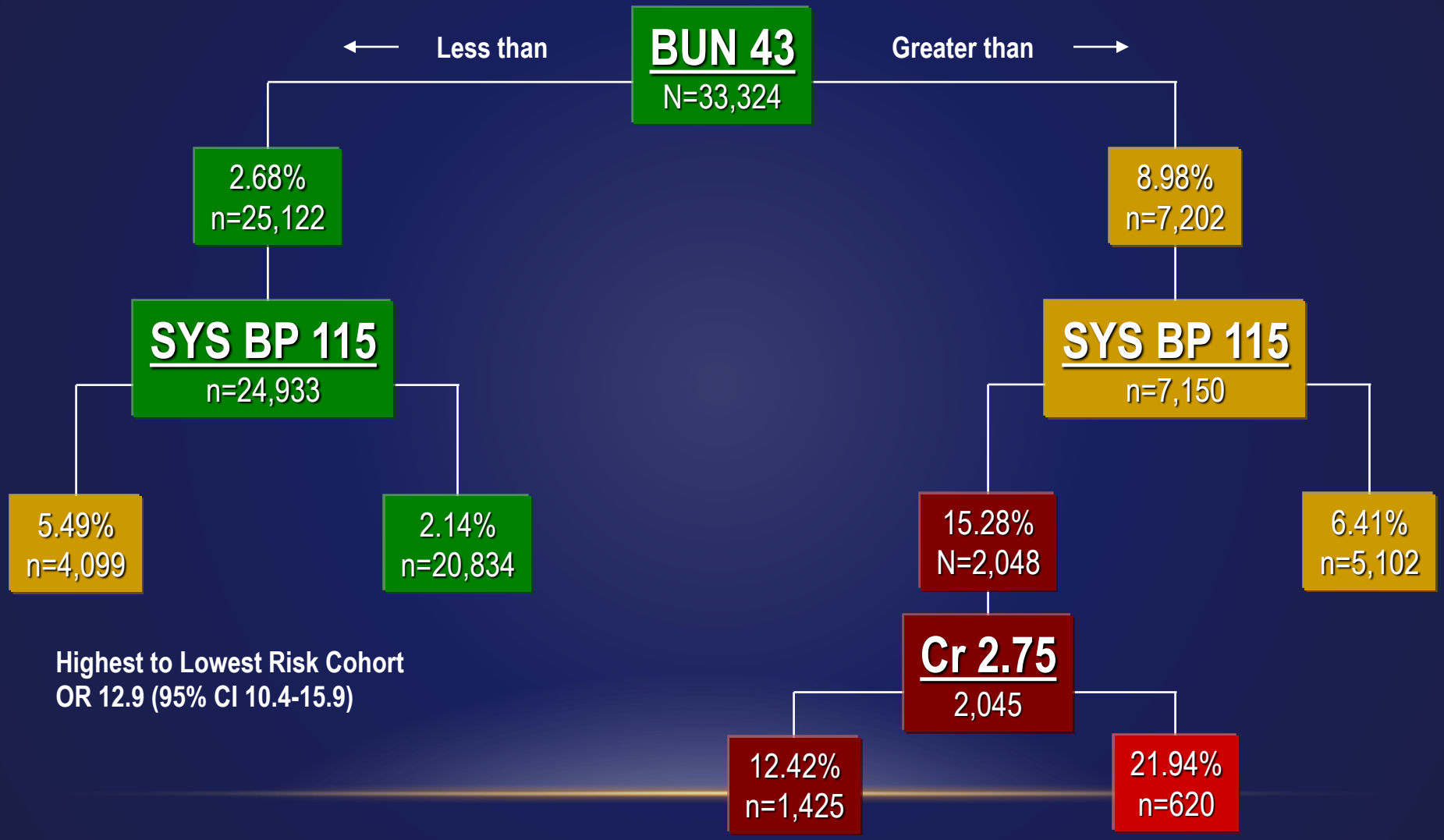
ADHERE® CART: PREDICTORS OF IN-HOSPITAL MORTALITY



Highest to Lowest Risk Cohort
OR 12.9 (95% CI 10.4-15.9)

Reference: Fonarow GC, et al. Risk stratification for in-hospital mortality in heart failure using classification and regression tree (CART) methodology. *JAMA*. 2005;293:572-580.

PATIENT #1:
WHAT HEMODYNAMIC QUADRANT IS HE IN?

- 44M with acute decompensated HF
- NIDCM (EF 19%), ICD 2006
- NYHA Class III at baseline
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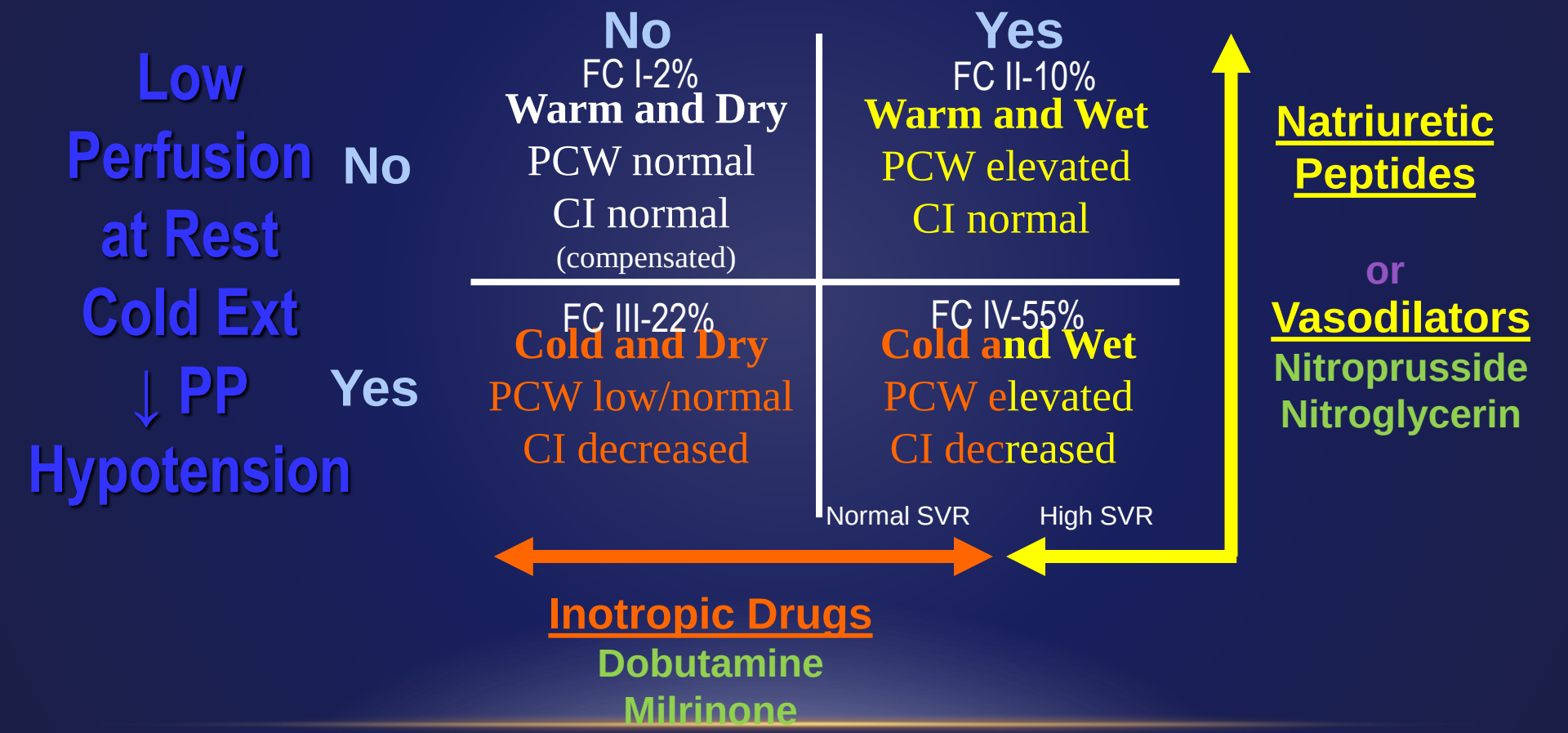

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PATIENT #1: HEMODYNAMICS

- BP: 134/92 mmHg (mean 104), HR: 85
- RAP: 24 mmHg
- RVP: 64/20 mmHg
- PAP: 63/43 mmHg (mean 51)
- PAW: 40 mmHg

- PA sat: 35%
- SVR: 2782 dynes-sec-cm5
- PVR: 4.9 Wood units

- FCO 2.8 L/min, FCI 1.2 L/min/m2
- TDCO: 2.3 L/min, TDCI 1L/min/m2

PATIENT #1: HOSPITAL COURSE

- Initiated on IV Vasodilator and IV diuresis
  - SBP 105/68 (79)
  - PA 42/13 (24)
  - CO 6.6, CI 2.9
  - CVP 4
- Neurohumoral agents added as tolerated
- NYHA Class I within 2 days

LIMITATIONS OF POSITIVE INOTROPES FOR ACUTE CHF

- Increased mortality
  - Milrinone[1,2]
  - Enoximone[3]
  - Imazodan[4]
  - Vesnarinone[5]
  - Dobutamine[6,7]
  - Xamoterol[8]
  - Ibopamine[9]
- Increased risk of hospitalization[1]

- Aggravation and induction of arrhythmias (need telemetry)
  - Milrinone[10,11]
  - Dobutamine[12]
  - Dopamine[13]
- Tachycardia[14]
- Tachyphylaxis (dobutamine)[15]
- Neurohormonal activation and/or lack of suppression[16]
- Physiologic effects antagonized by β-blockade (dobutamine, dopamine)

[1]Packer M, et al. *New Engl J Med* 1991; 325: 1468-75.
[2]DiBianco R, et al. *New Engl J Med* 1989; 320: 677-83.
[3]Uretsky BF, et al. *Circulation* 1990; 82: 774-80.
[4]Goldberg AD, et al. *Circulation* 1990; 82: Suppl III: III-673.
[5]Cohn JN, et al. *New Engl J Med* 1998; 339: 1810-16.
[6]Dies F, et al. *Circulation* 1986;74: Suppl II: II-38.
[7]O' Connor CM, et al. *Am Heart J* 1999; 138: 78-86.
[8]The Xamoterol in Severe Heart Failure Group. *Lancet* 1990; 336: 1-6.
[9]Hampton JR, et al. *Lancet* 1997; 349: 971-7.
[10]Kleiman NS, et al. *J Am Coll Cardiol* 2000; 36; 310-25
[11]Thackray S, et al. *Eur J Heart Fail* 2000; 2: 209-212
[12]Burger AJ, et al. *Am J Cardiol* 2001 Jul 1;88(1):35-9
[13]Chiolero, et al, *Cardiovasc Surgeon* 1991; 39: 81-84
[14] Colucci WS. *J Card Fail* 2001;7(1):92-100.
[15] B. Hoffman and R. Lefkowitz. Chapter 10, The Pharmacologic Basis of Therapeutics, Goodman and Gilman, Eds, 9th. Edition (CD-ROM) 1996.
[16]Aronson D, et al. J Card Fail 2001; 7 (No. 3 Suppl 2): 28.

Recommendations for Inotropic Support, MCS, and Cardiac Transplantation.

Recommendations	COR	LOE	References
Inotropic support			
Cardiogenic shock pending definitive therapy or resolution	I	C	N/A
BTT or MCS in stage D refractory to GDMT	IIa	B	647, 648
Short-term support for threatened end-organ dysfunction in hospitalized patients with stage D and severe HF/EF	IIb	B	592, 649, 650
Long-term support with continuous infusion palliative therapy in select stage D HF	IIb	B	651–653
Routine intravenous use, either continuous or intermittent, is potentially harmful in stage D HF	III: Harm	B	416, 654–659
Short-term intravenous use in hospitalized patients without evidence of shock or threatened end-organ performance is potentially harmful	III: Harm	B	592, 649, 650
MCS			
MCS is beneficial in carefully selected* patients with stage D HF in whom definitive management (eg, cardiac transplantation) is anticipated or planned	IIa	B	660–667
Nondurable MCS is reasonable as a "bridge to recovery" or "bridge to decision" for carefully selected* patients with HF and acute profound disease	IIa	B	668–671
Durable MCS is reasonable to prolong survival for carefully selected* patients with stage D HF/EF	IIa	B	672–675
Cardiac transplantation			
Evaluation for cardiac transplantation is indicated for carefully selected patients with stage D HF despite GDMT, device, and surgical management	I	C	680

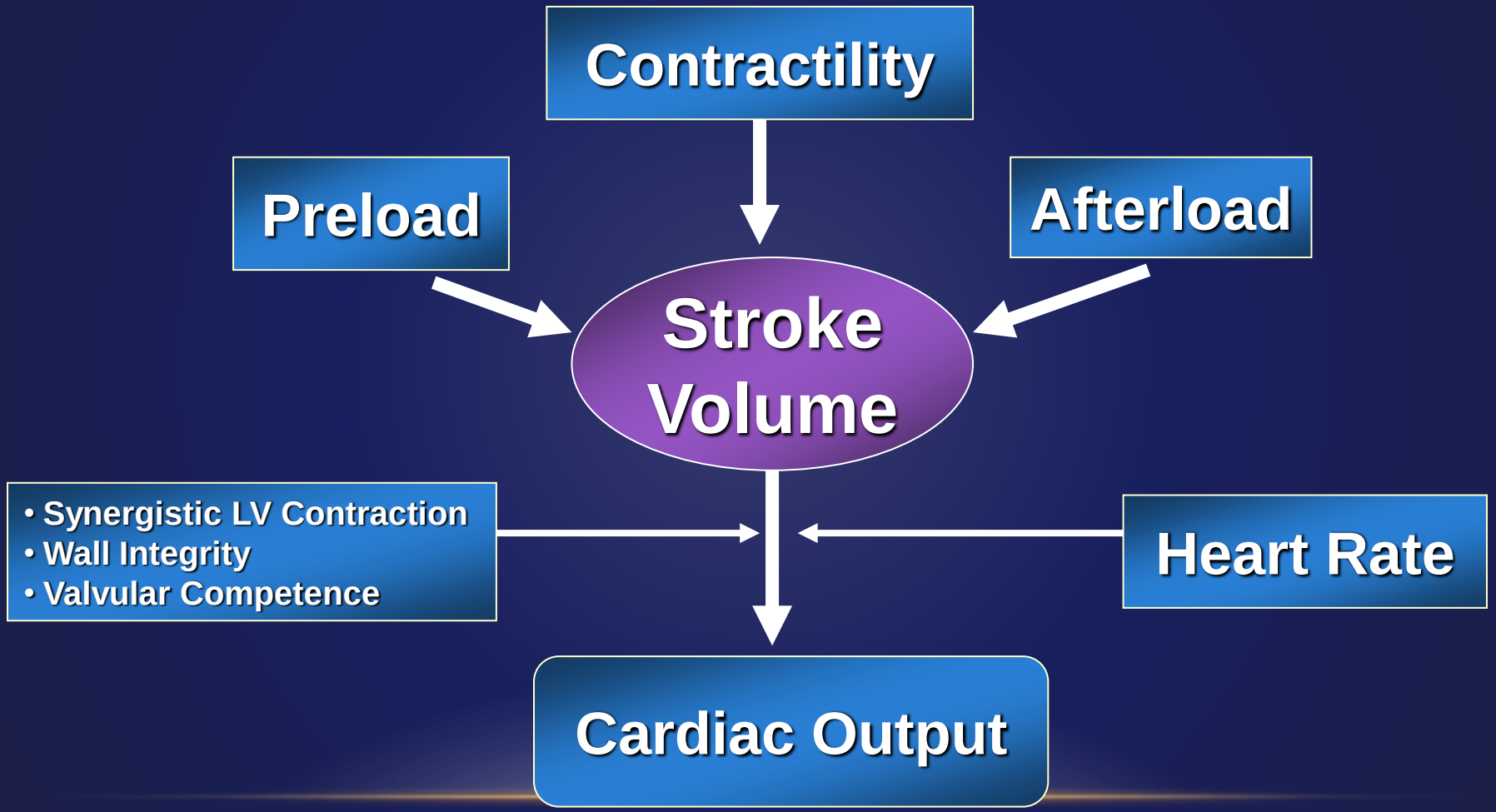
*Although optimal patient selection for MCS remains an active area of investigation, general indications for referral for MCS therapy include patients with LVEF <25% and NYHA class III–IV functional status despite GDMT, including, when indicated, CRT, with either high predicted 1- to 2-year mortality (eg, as suggested by markedly reduced peak oxygen consumption and clinical prognostic scores) or dependence on continuous parenteral inotropic support. Patient selection requires a multidisciplinary team of experienced advanced HF and transplantation cardiologists, cardiothoracic surgeons, nurses and ideally, social workers and palliative care clinicians.

BTT indicates bridge to transplant; COR, Class of Recommendation; CRT, cardiac resynchronization therapy; GDMT, guideline-directed medical therapy; HF, heart failure; HF/EF, heart failure with reduced ejection fraction; LOE, Level of Evidence; LVEF, left ventricular ejection fraction; MCS, mechanical circulatory support; N/A, not applicable; and NYHA, New York Heart Association.

Yancy C W et al. Circulation. 2013;128:e240-e327

American Heart Association.

DETERMINANTS OF VENTRICULAR FUNCTION



Goals for Treatment of Acutely Decompensated Heart Failure

Hemodynamic

SBP ≥ 80 mm Hg

PCWP < 15 mm Hg

RAP < 8 mm Hg

SVR < 1200 dyne-s-cm^{-5}

Clinical

SBP ≥ 80 mm Hg

No orthopnea

No peripheral edema

No hepatomegaly/ascites

JVP < 8 cm

Warm extremities

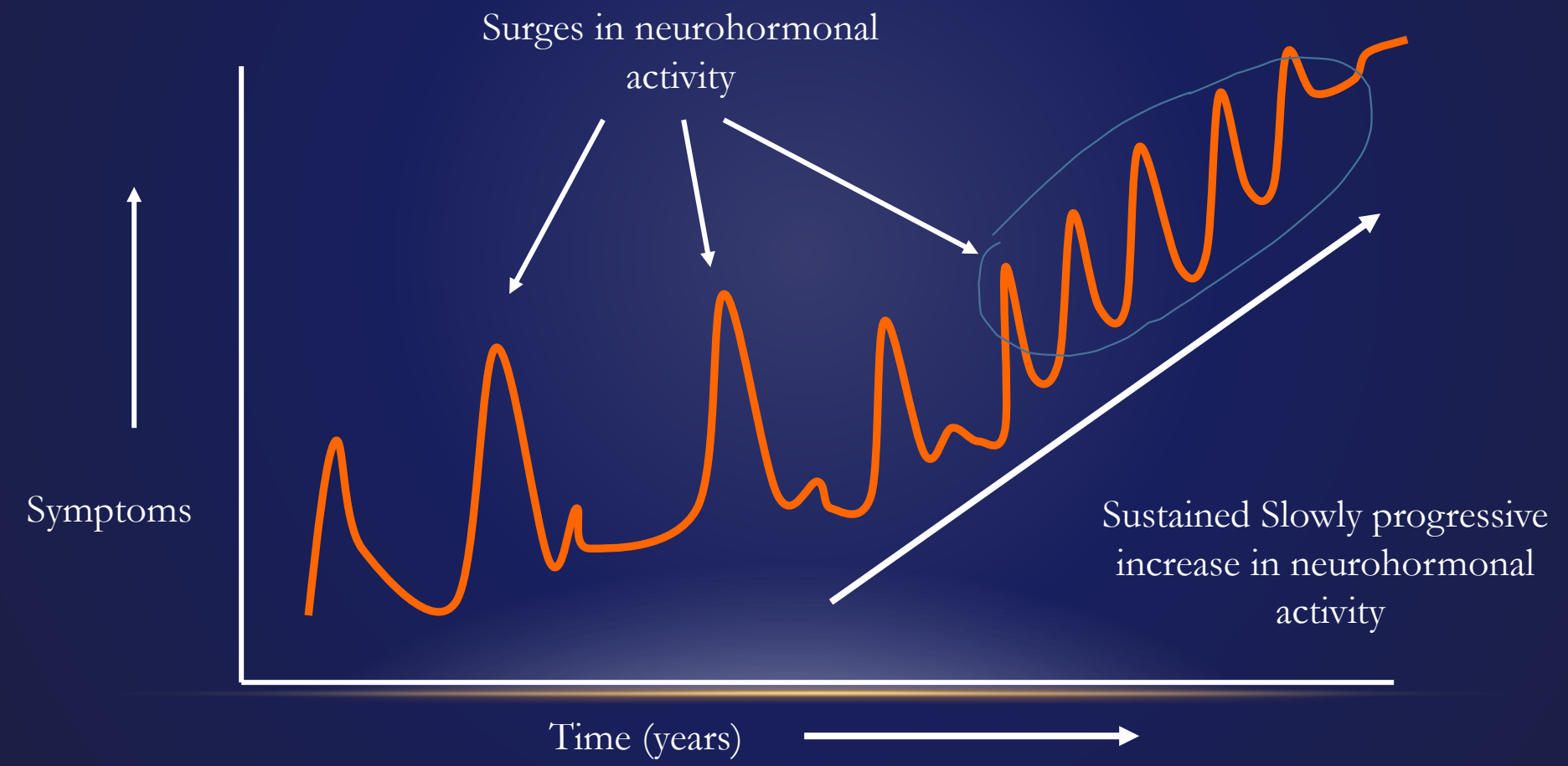
Pathophysiologic

Achieving neurohormonal attenuation and balance

Functional Class Improvement

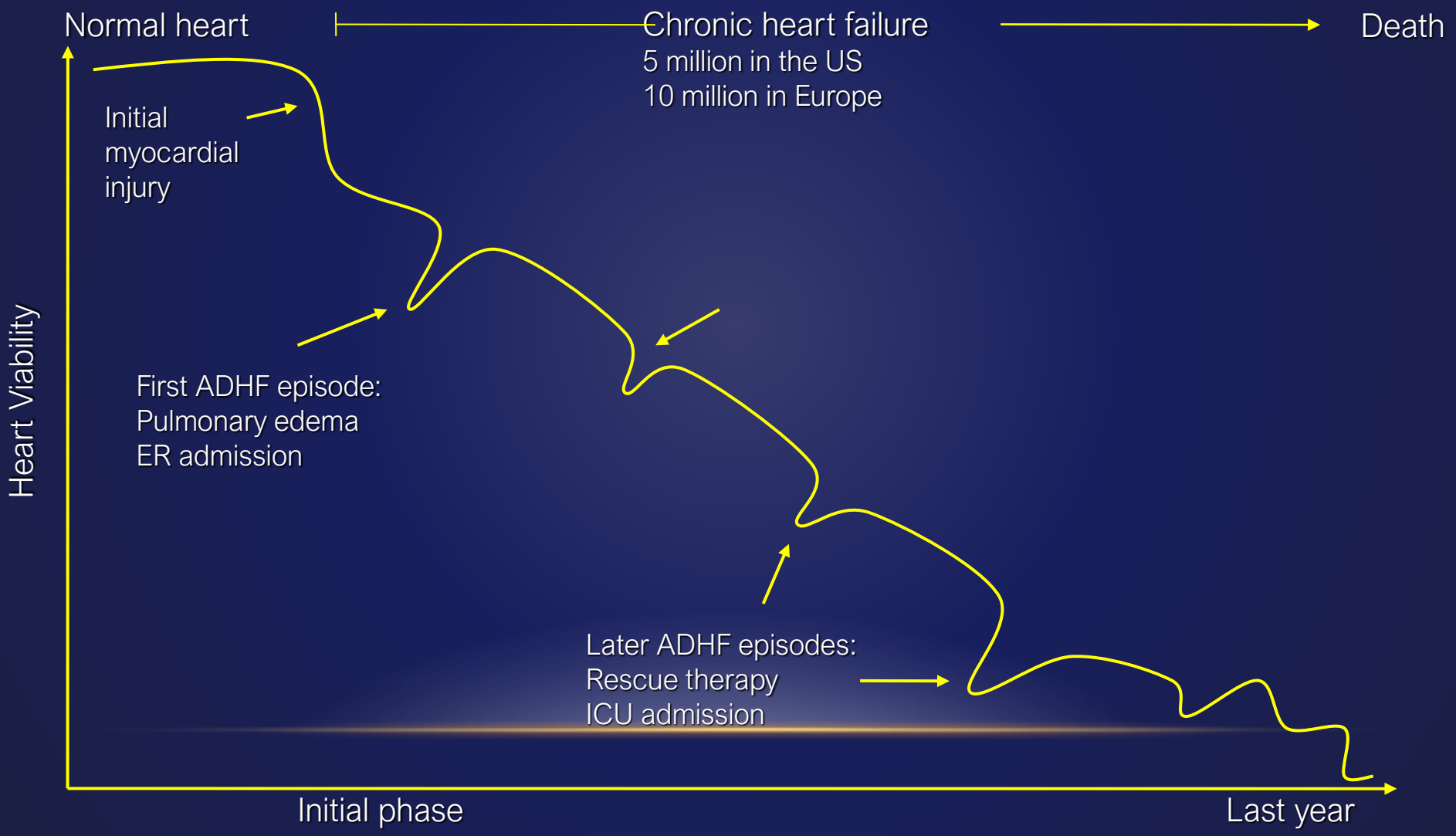
JVP = jugular venous pressure.

PROGRESSION OF CHRONIC HEART FAILURE RELATED TO DECOMPENSATION (SYMPTOMS)



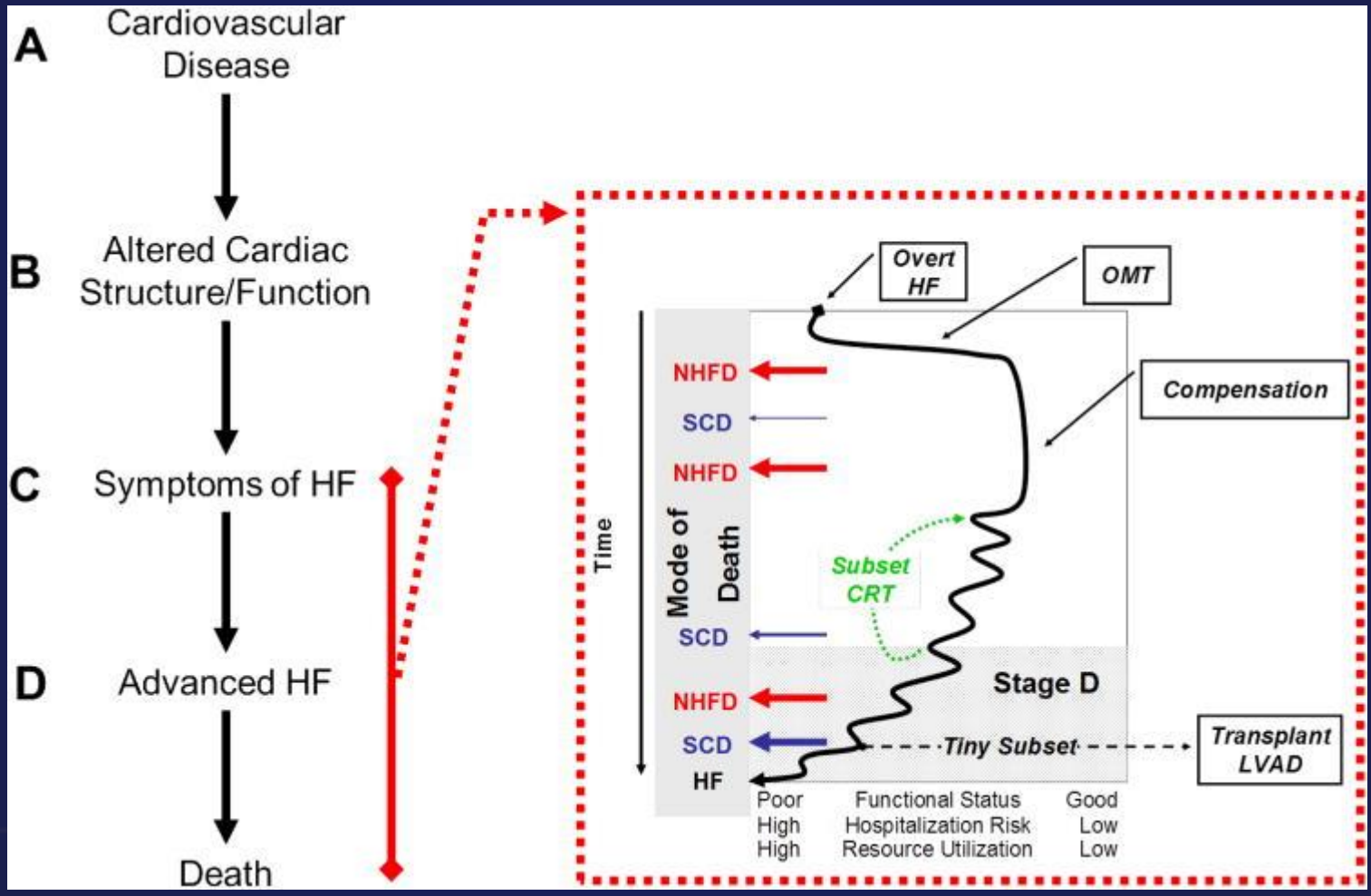
Courtesy; Milton Packer, M.D.

NATURAL HISTORY OF HF



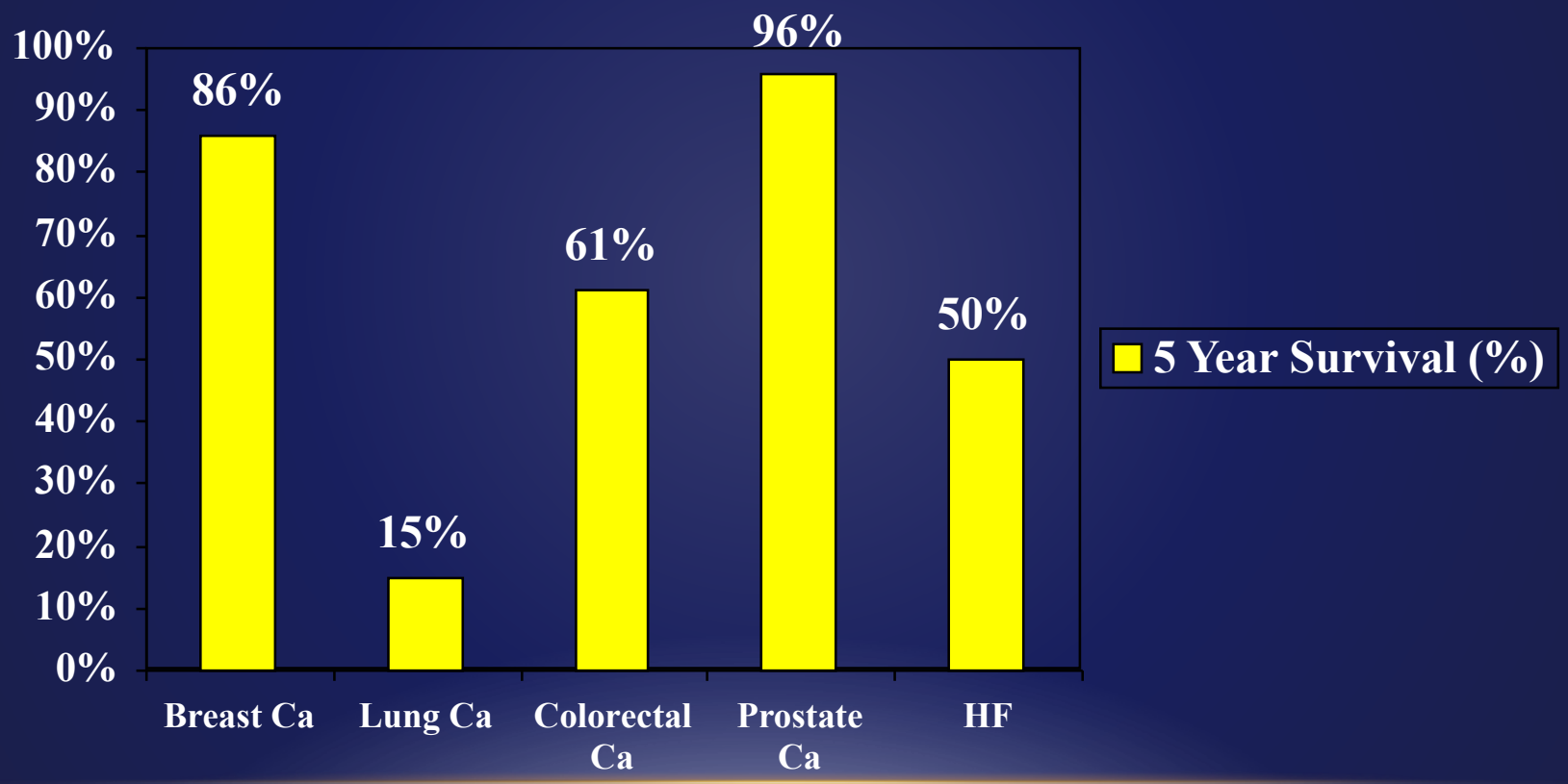
Gheorghiade M. Am J Cardiol. 2005;96(suppl 6A):1-4G.

EVERY DECOMPENSATION TRANSITIONS PATIENT TO ADVANCED HEART FAILURE



Congest Heart Fail. 2011 Jul-Aug;17(4):160-8. doi: 10.1111/j.1751-7133.2011.00246.x. Epub 2011 Jul 21.

FIVE-YEAR SURVIVAL CANCER VERSUS HF



2002 Heart and Stroke statistical update and 2002 Cancer Facts and Figures, American Cancer Society

ESC CRITERIA FOR ADVANCED HEART FAILURE

- ***NYHA Class III-IV Symptoms***
- ***Episodes of volume overload and/or peripheral hypoperfusion***
- ***Objective evidence of severe cardiac dysfunction***
(EF<30%, Doppler Pseudonormal or Restrictive filling pattern, PCWP>16mmHg or RAP >12 mmHg)
- ***Severely impaired functional capacity***
(Inability to exercise, 6MWD<300m, Peak VO2<12-14 ml/kg/min)
- ***HF Hospitalizations***
(≥1 in past 6 months)
- ***Above occurring despite attempts to optimize diuretics, RAAS antagonists, BB, CRT or in the setting of intolerance to OMT***

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DOES HE HAVE ADVANCED HEART FAILURE?

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DOES HE HAVE ADVANCED HEART FAILURE?

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- Meds:

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CLOSING REMARK
ACUTE DECOMPENSATION OF HEART FAILURE IS AFTERLOAD MISMATCH

- Primary pathophysiology: elevation of LV filling pressure and fluid redistribution to the lungs[1] as a result of afterload mismatch (excess vasoconstriction) rather than decrease in contractility[2]
- The interaction between a rise in SVR and myocardial systolic and diastolic reserve is the major mechanism for elevated filling pressures and decompensation[1]
- Reduction of LV filling pressure (via balanced vasodilation +/- diuresis) results in rapid relief in symptoms and is associated with reduced risk of rehospitalization and improved survival[3]

1. Fonarow GC. *Rev Cardiovasc Med.* 2002;3(suppl 4):S19–S29.
2. Shah M et al. *Rev Cardiovasc Med.* 2001;2(suppl 2):S2–S6.
3. Aghababian RV. *Rev Cardiovasc Med.* 2002;3(suppl 4):S3–S9.

THANK YOU!