Management of the RV in cardiogenic shock

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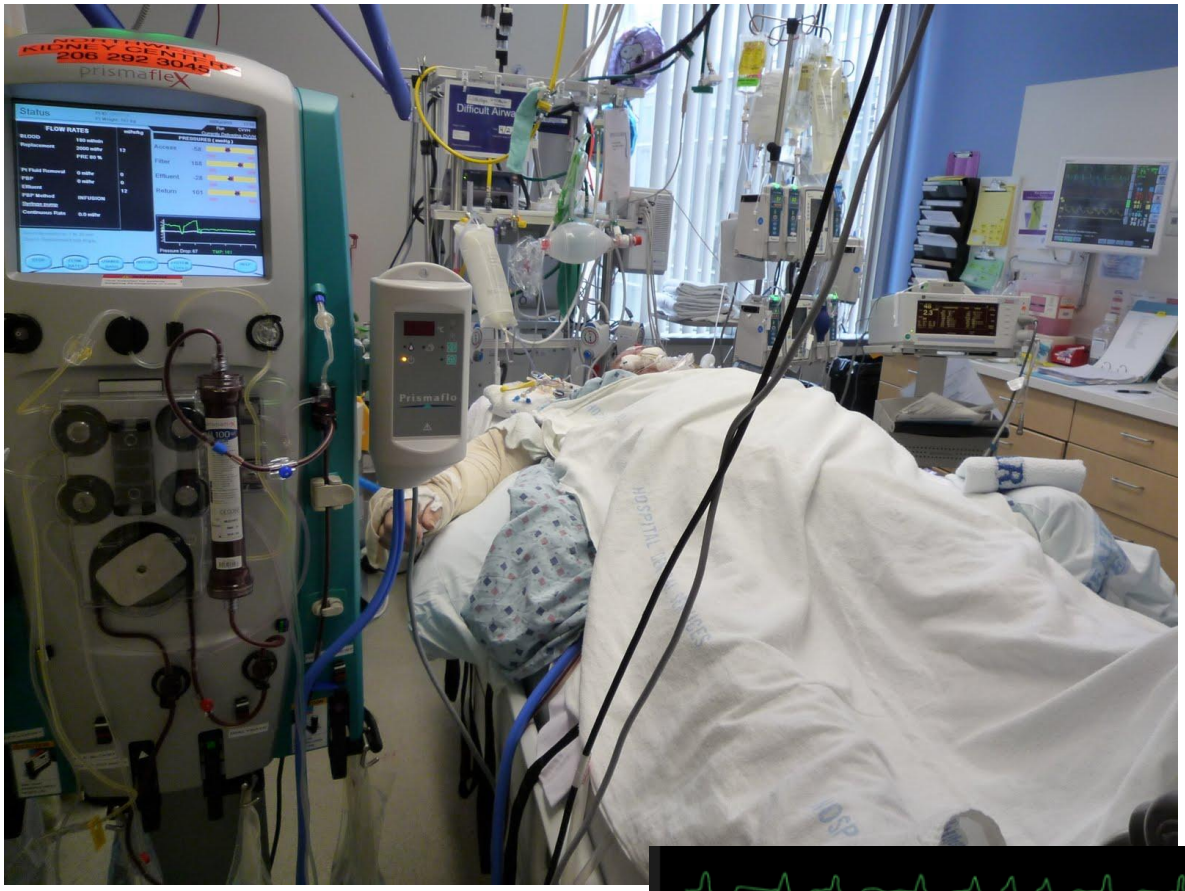
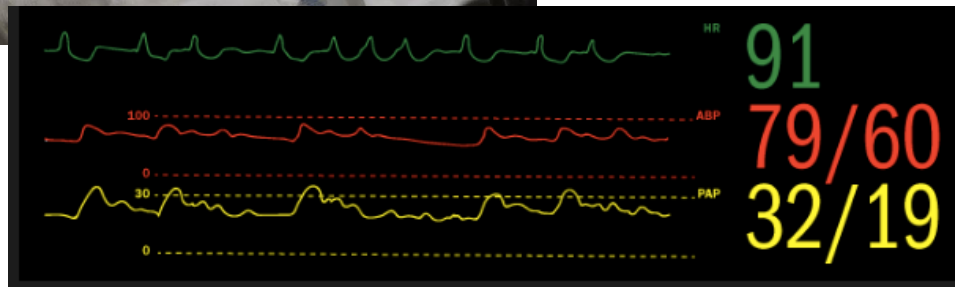
Disclosures

No disclosures/conflicts of interest

Cardiogenic shock





Society of Critical Care Medicine
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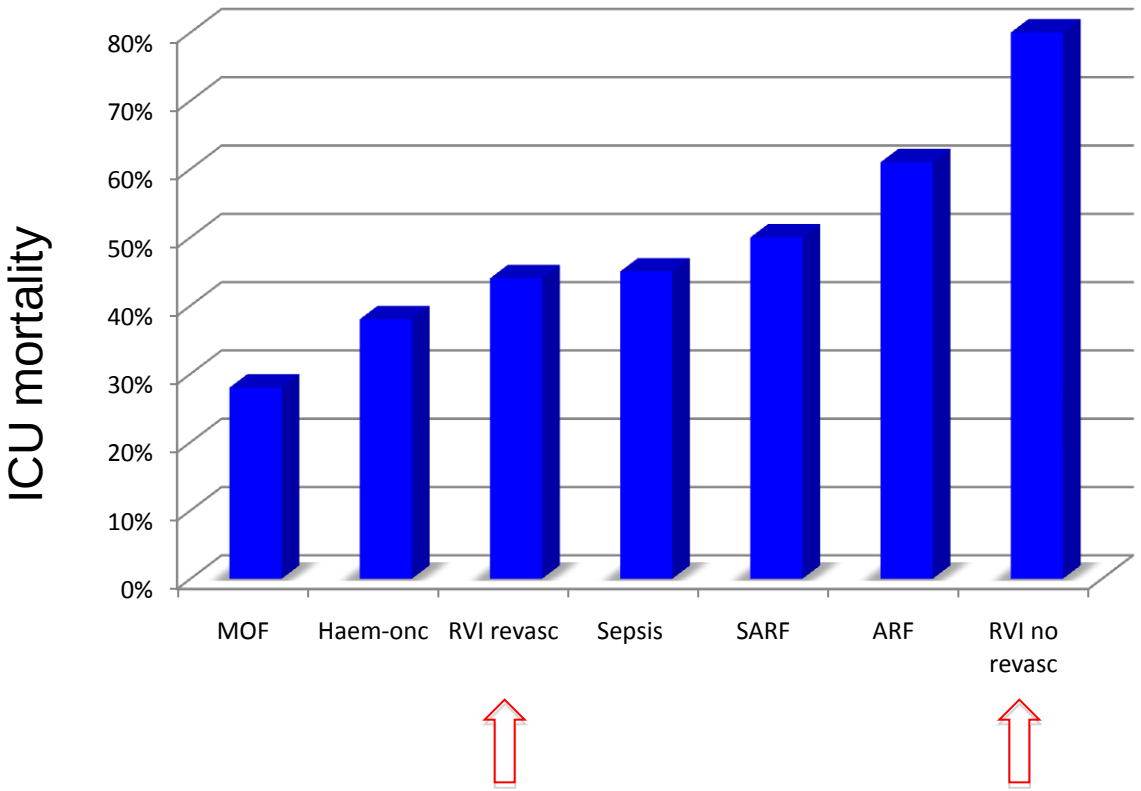

Lupi-Herrera et al., World J Cardiol, 2014
SCCM data, 2015. Parakh et al., Int Med J, 2015

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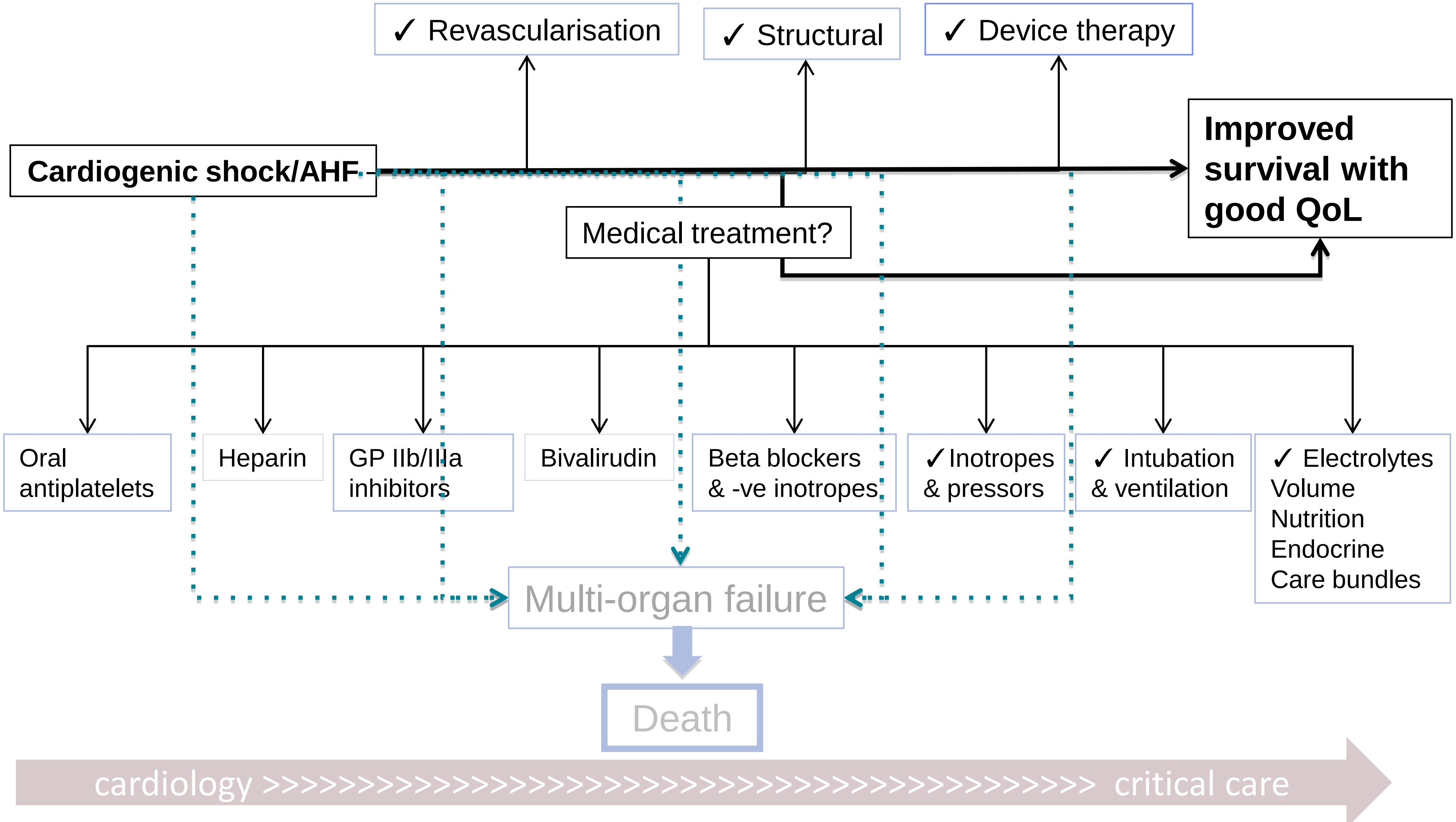
Cardiogenic shock management



Guidelines, 2016

European Journal of Heart Failure

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View issue TOC
Volume 18, Issue 3
March 2016
Pages 226–241

Statement

Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology

Veli-Pekka Harjola ✉, Alexandre Mebazaa, Jelena Čelutkienė, Dominique Bettex, Hector Bueno, Ovidiu Chioncel, Maria G. Crespo-Leiro, Volkmar Falk, Gerasimos Filippatos, Simon Gibbs, Adelino Leite-Moreira, Johan Lassus, Josep Masip, Christian Mueller, Wilfried Mullens, Robert Naeije, Anton Vonk Nordegraaf, John Parissis, Jillian P. Riley, Arsen Ristic, Giuseppe Rosano, Alain Rudiger, Frank Ruschitzka, Petar Seferovic, Benjamin Sztrymf, Antoine Vieillard-Baron, Mehmet Birhan Yilmaz, Stavros Konstantinides

First published: 15 March 2016 Full publication history

Complex, management requires understanding of anatomy and mechanics,
Identification
Treat underlying causes
Support

Uncertainties remain

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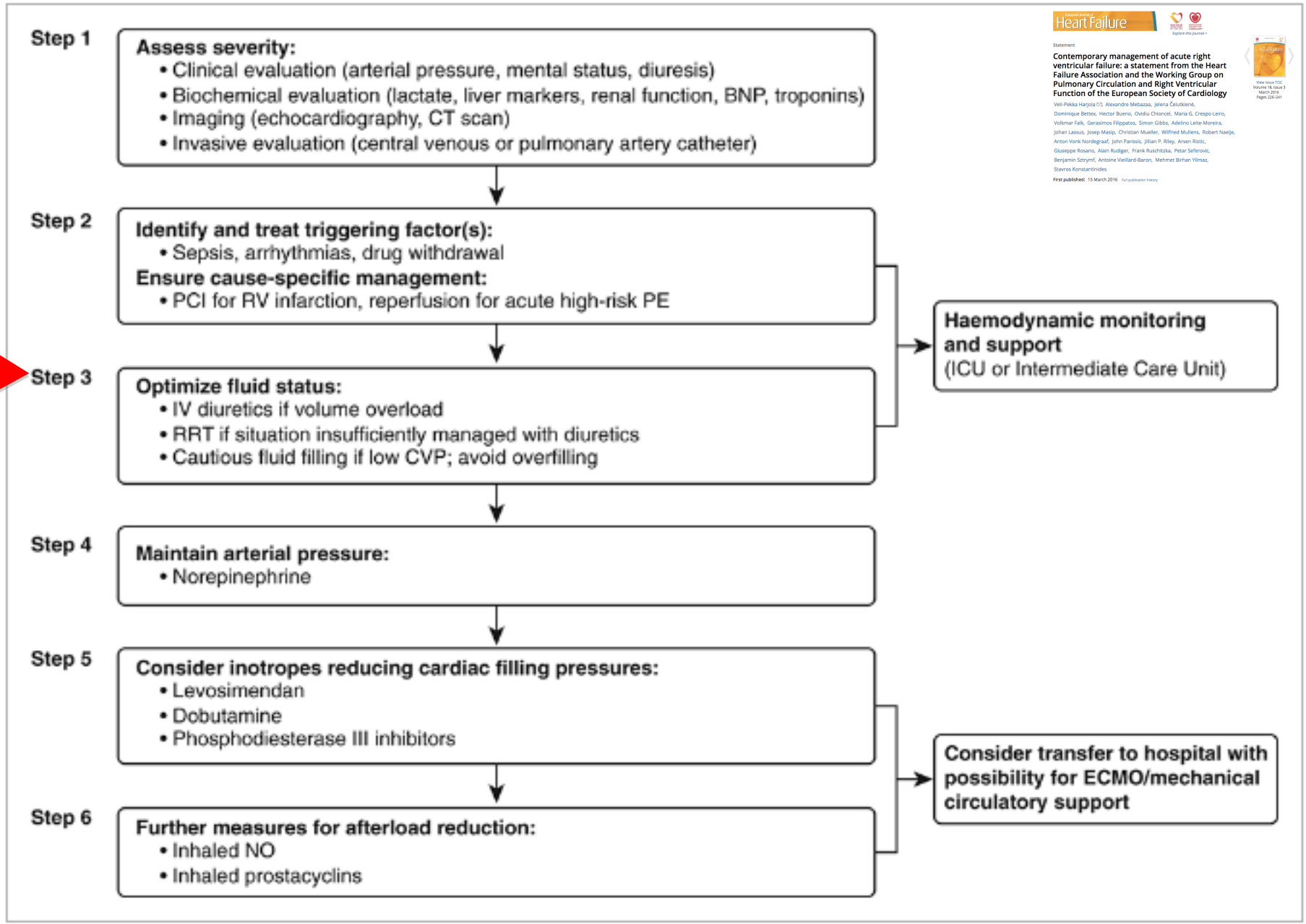

European Journal of Heart Failure

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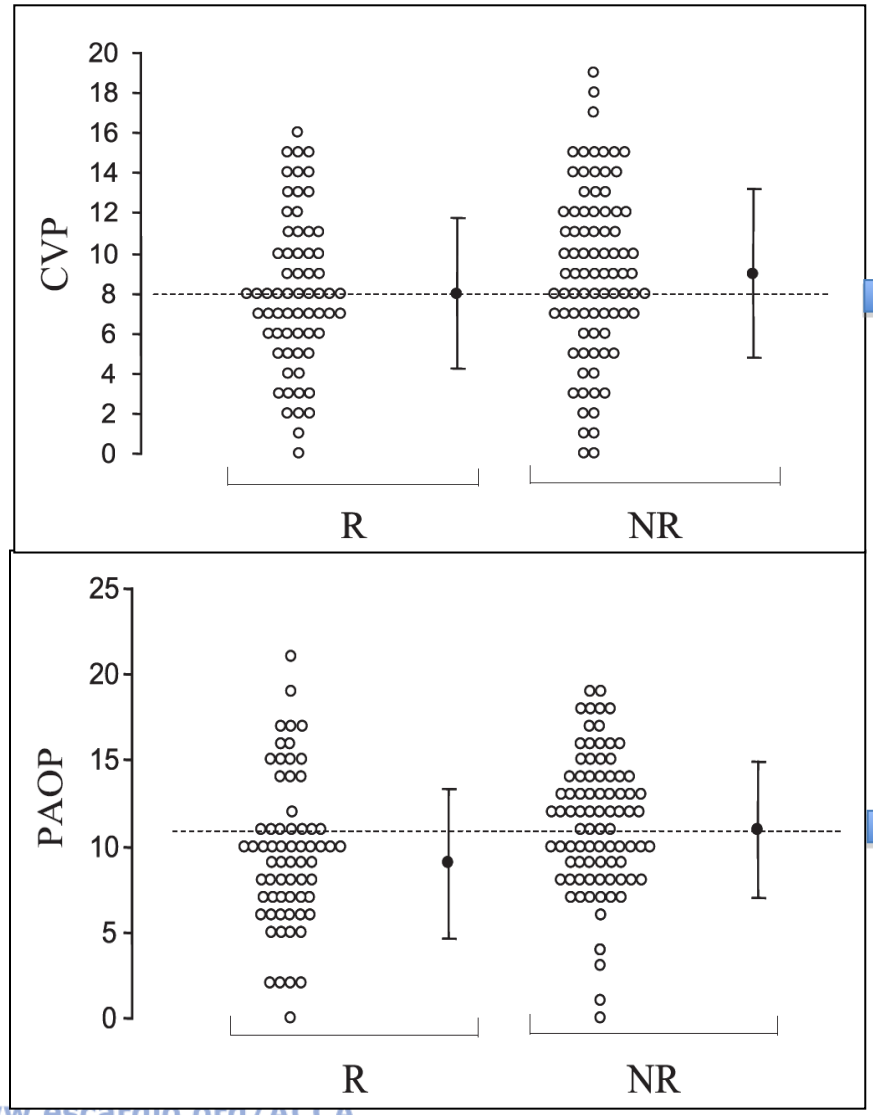
Volume?



CVP: +ve predictive value 47%

PCWP: +ve predictive value 54%

RV preload optimisation

- Initial studies:
  - Normal saline infusion, maintaining RAP <10mmHg
- Later clinical studies
  - Variable response reported
  - Aim target PCWP 18-24 mmHg

- **Berisha et al.,** 41 patients, electrocardiographic and haemodynamic criteria for RV infarction

  – maximal RV SWI with filling pressure 10-14mmHg

  - mean RAP >14mmHg associated with RV distension

  - haemodynamic response variable - optimal PCWP (corresponding to maximum LVSWI) 16mmHg

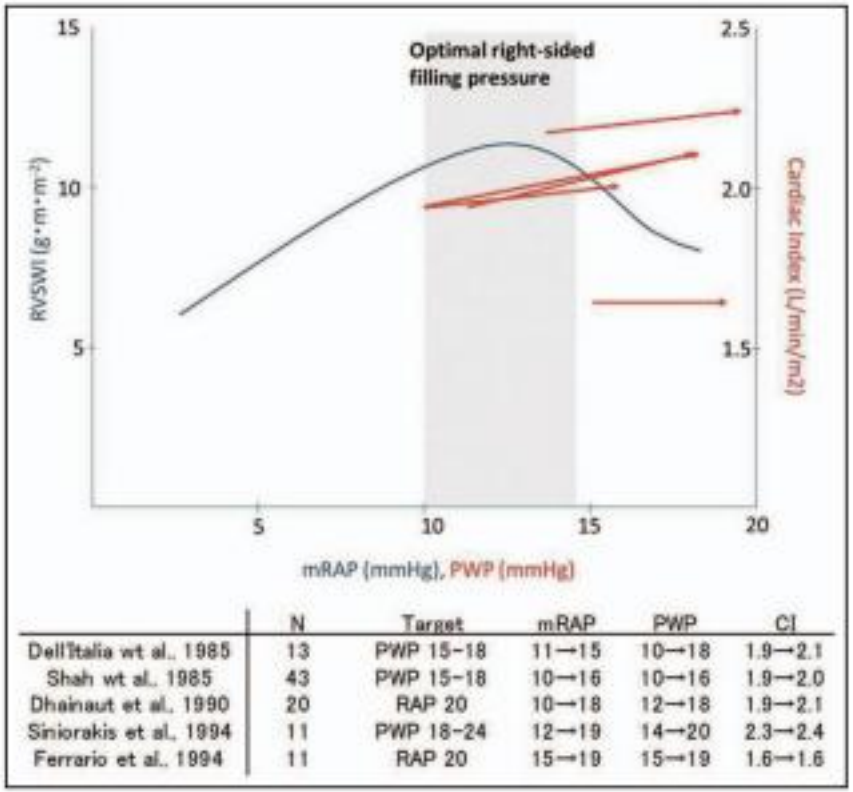
Inohara et al., EHJ Acute Cardiovascular Care 2013

RV preload optimisation

Smaller studies: Change in PCWP and CI
Wide variation in response
No linear association with higher mRAP target

Practically:
Aim transmural pressure 8-12mmHg

Measure CO and $ScvO_2$/systemic organ perfusion
(*not well-studied in acute RV failure*)



	N	Target	mRAP	PWP	CI
Dell'Italia wt al., 1985	13	PWP 15–18	11→15	10→18	1.9→2.1
Shah wt al., 1985	43	PWP 15–18	10→16	10→16	1.9→2.0
Dhainaut et al., 1990	20	RAP 20	10→18	12→18	1.9→2.1
Siniorakis et al., 1994	11	PWP 18–24	12→19	14→20	2.3→2.4
Ferrario et al., 1994	11	RAP 20	15→19	15→19	1.6→1.6

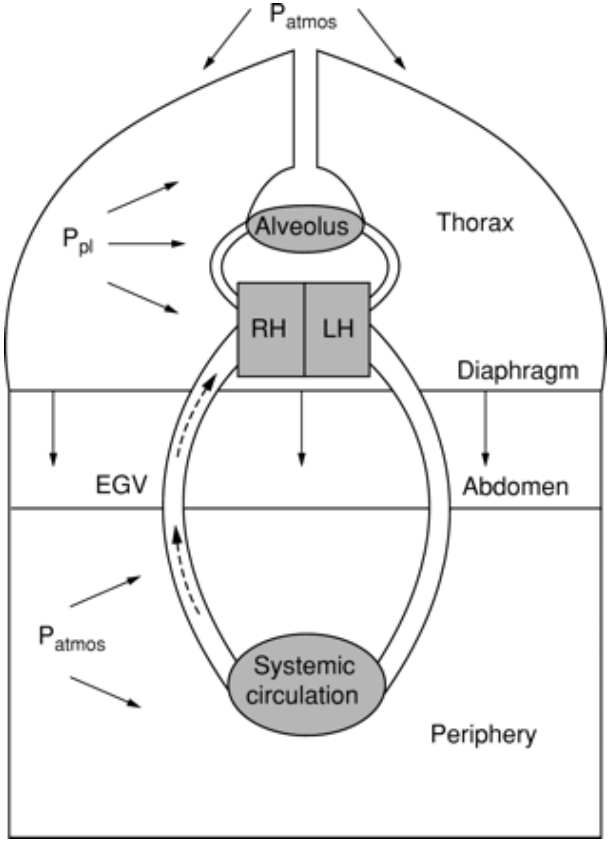
Inohara et al., EHJ Acute Cardiovascular Care 2013

1. Preload evaluation

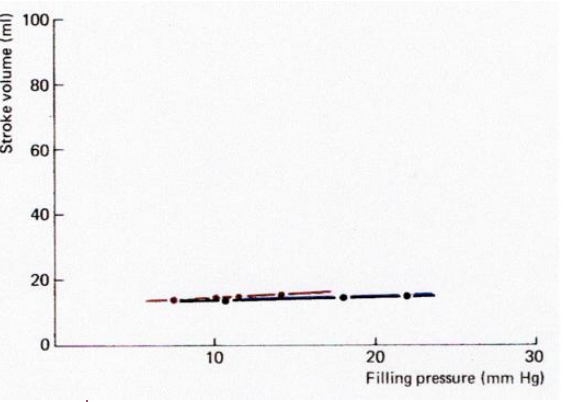
IPPV:
Increases ITP

Hypovolaemia:
Sepsis/SIRS
Vascular permeability
Insensible loss

P_{syst} reduced by analgesia & sedatives



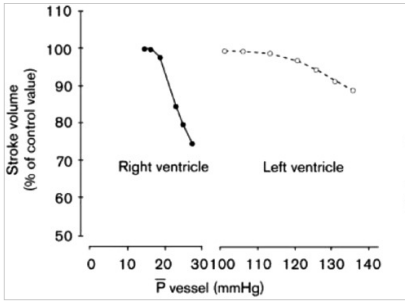
2. Pressure-volume



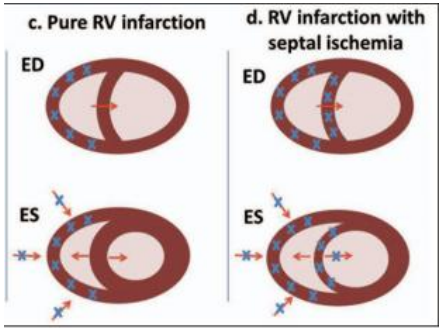
3. RV afterload evaluation

PVR normal: need increased RVEDP

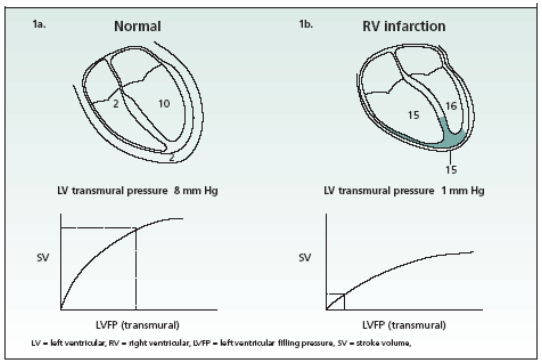
PVR elevated: increase in RVEDP will shift septum

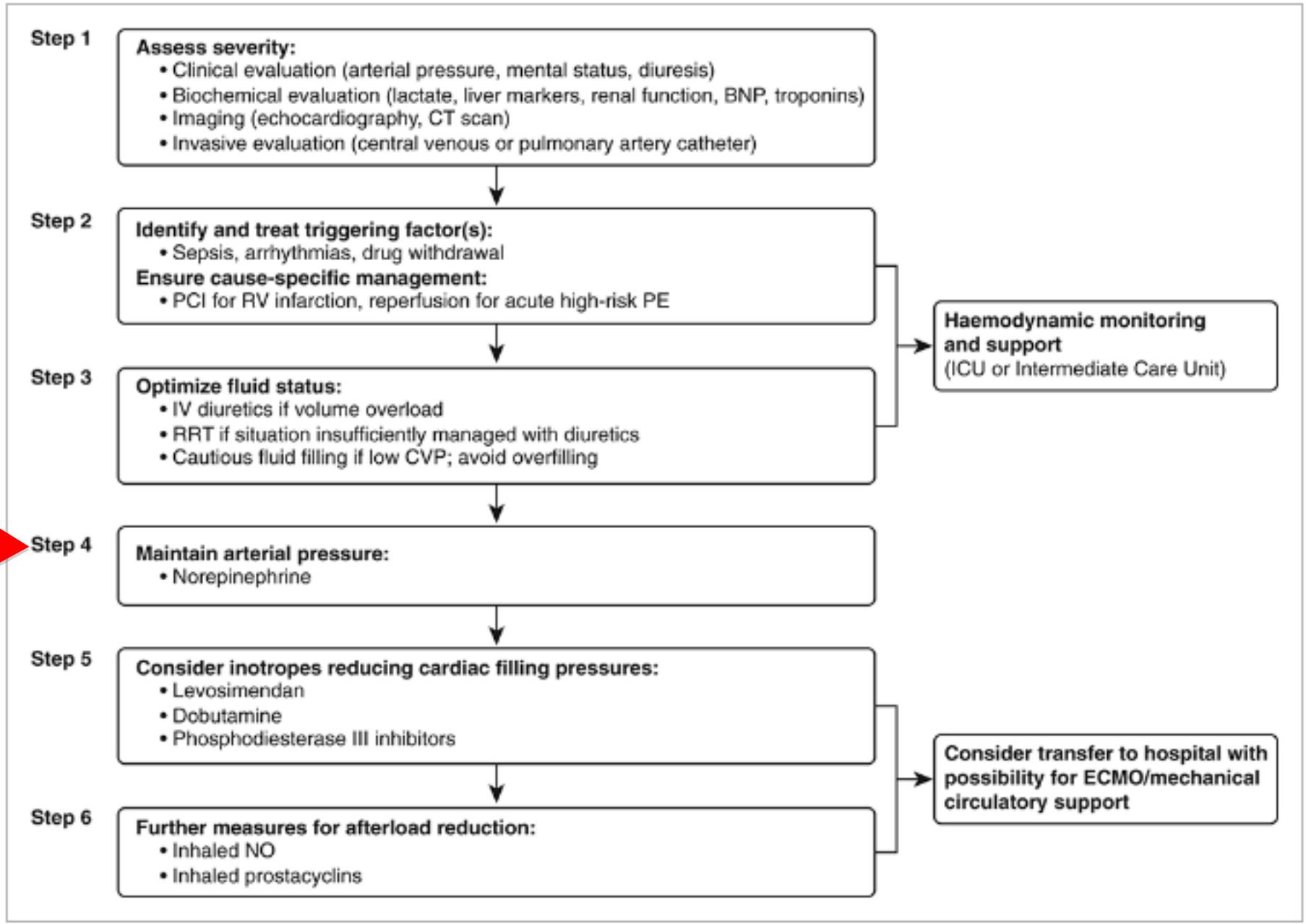


4. Septal involvement



5. The pericardium

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• Inhaled NO
• Inhaled prostacyclins
Consider transfer to hospital with possibility for ECMO/mechanical circulatory support

Vasoconstriction

Dopamine

- If >15mcg/kg/min is α-agonist
- Positive inotrope
- Elevation in PCWP

Noradrenaline

- Constrictor
- Antithrombotic
- Positive inotrope

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 MARCH 4, 2010 VOL. 362 NO. 9

Comparison of Dopamine and Norepinephrine in the Treatment of Shock

Daniel De Backer, M.D., Ph.D., Patrick Biston, M.D., Jacques Devriendt, M.D., Christian Madl, M.D., Didier Chochrad, M.D., Cesar Aldecoa, M.D., Alexandre Brasseur, M.D., Pierre Defrance, M.D.,

1678 patients with circulatory shock – 280 cardiogenic

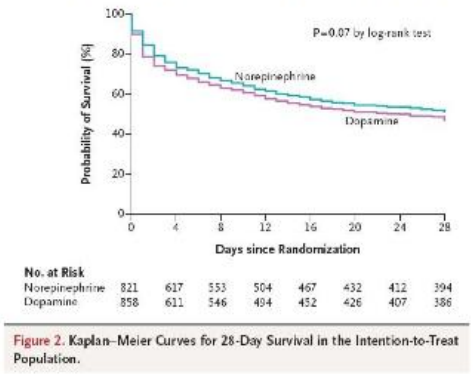

Figure 2. Kaplan–Meier Curves for 28-Day Survival in the Intention-to-Treat Population.

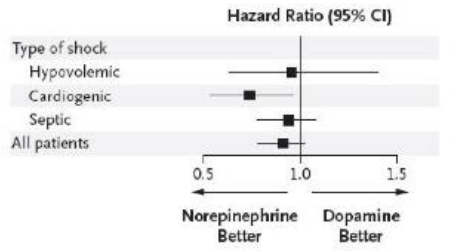

Figure 3. Forest Plot for Predefined Subgroup Analysis According to Type of Shock.

Systemic arterial pressure optimisation

Vasoactive drugs for management of acute right ventricular failure and their mechanism of action

	Receptor Binding					
Agent	**α_1**	**β_1**	**β_2**	**D**	**V1**	**Notes**
Norepinephrine	++	+				Improves PA/RV coupling in animals (73–75)
Phenylephrine	++					Increases PVR (71, 74, 77); may induce reflex bradycardia
Epinephrine	++	++	+			(79)
Vasopressin					+	Dose dependent pulmonary vasodilatation (0.01–0.03 U/min) and vasoconstriction (24, 82, 83)
Dopamine						Risk of arrhythmias
Low (<5 µg/kg/min)		+		++		
Medium (>10 µg/kg/min)	+	++		++		
High (>10 µg/kg/min)	++	++		++		
Dobutamine		++	+			β_2-mediated drop in SVR (31); risk of arrhythmias
Milrinone						Phosphodiesterase-3 inhibitor; inotropy and pulmonary vasodilatation; drop in LVEDP and SVR (72, 84, 89); risk of arrhythmias

Definition of abbreviations: D = dopaminergic receptor; LVEDP = left ventricular end-diastolic pressure; PA = pulmonary artery; PVR = pulmonary vascular resistance; RV = right ventricle; SVR = systemic vascular resistance; V1 = vasopressin receptor 1. + = low to moderate affinity, ++ = moderate to high affinity.

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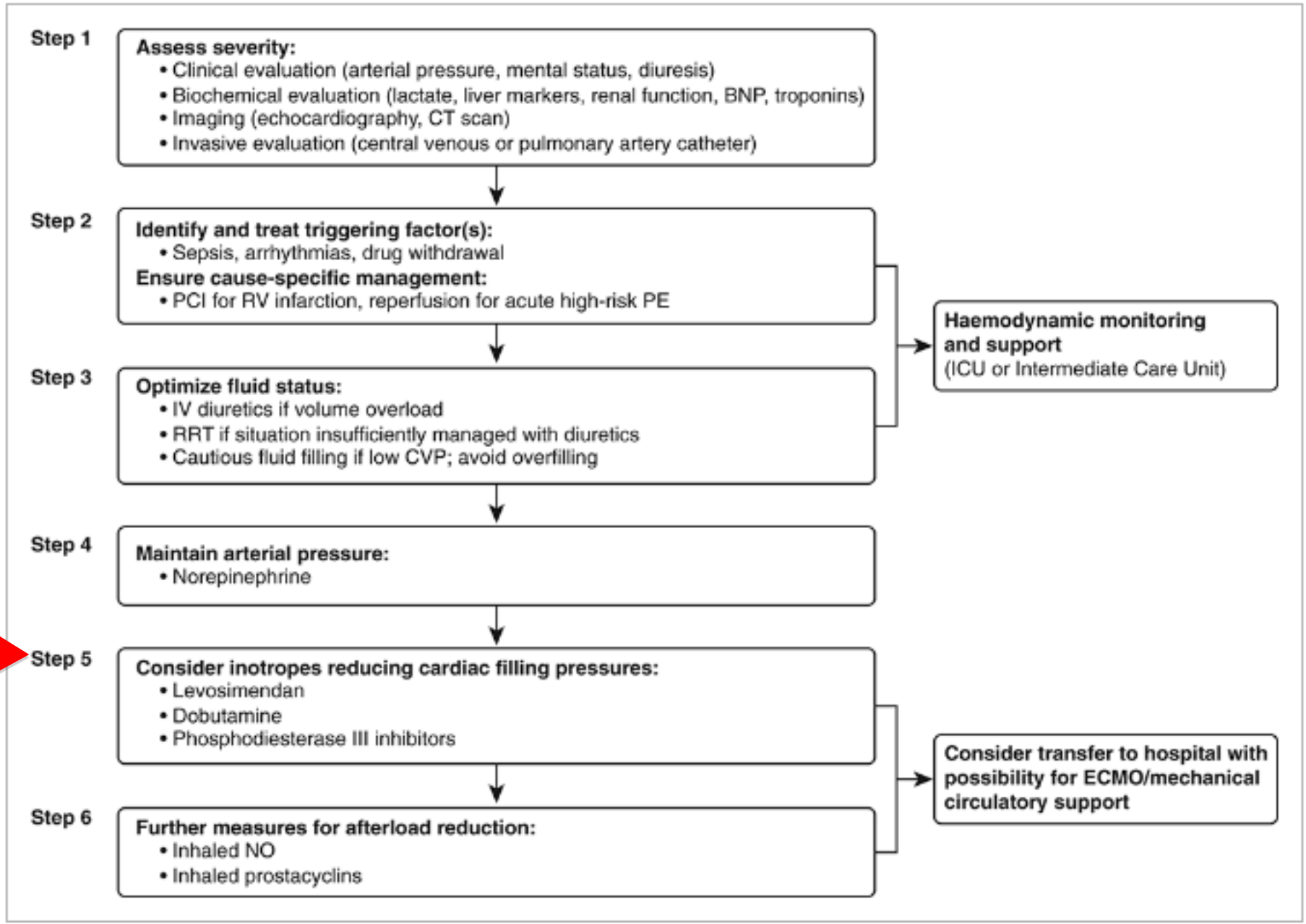
Harjola et al., Eur J Heart Failure 2016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Positive inotropic agents

- Diverse collection of pluripotent molecules
- Differing pharmacological properties
- Some shared activities – only one of which is positive inotropy
  - *Will increase dP/dt with variable effects on cardiac output/index*
  - Alteration in myocardial oxygen demand
  - Arrhythmia

Br J Pharmacol. 2012 Apr; 165(7): 2009–2011. PMCID: PMC3413839
doi: 10.1111/j.1476-5381.2011.01776.x

Inotropes and vasopressors: more than haemodynamics!

Hendrik Bracht,[1] Enrico Calzia,[1] Michael Georgieff,[1] Joel Singer,[2] Peter Radermacher,[1] and James A Russell[3]

- Alteration in bacterial metabolism and translocation
- Alteration in inflammatory markers and ROS
- Immune-modulatory effects
- Coagulation
- Differential effects on macrocirculation & microcirculation

OPEN ACCESS Freely available online

PLOS ONE

Conventional Hemodynamic Resuscitation May Fail to Optimize Tissue Perfusion: An Observational Study on the Effects of Dobutamine, Enoximone, and Norepinephrine in Patients with Acute Myocardial Infarction Complicated by Cardiogenic Shock

Corstiaan A. den Uil[1]*, Wim K. Lagrand[2], Martin van der Ent[3], Koen Nieman[1], Ard Struijs[1], Lucia S. D. Jewbali[1], Alina A. Constantinescu[1], Peter E. Spronk[4], Maarten L. Simoons[1]

	Dobutamine (n = 14)	Enoximone (n = 10)	Norepinephrine (n = 9)	P-value
ΔHR, bpm	+9 [0; +16]**	+4 [−11; +9]	+1 [−15; +4]	NS
ΔMAP, mmHg	+6 [−5; +21]	+8 [+1; +14]	+17 [+13; +32]**	NS
ΔCVP, mmHg	−1 [−3; +1]	−2 [−3; −1]*	+2 [−4; +4]	NS
ΔPCWP, mmHg	−2 [−4; −1]**	−2 [−3; −1]**	+5 [−1; +7]	NS
ΔMPAP, mmHg[a]	0 [−3; +3]	−1 [−9; 0]	+4 [−1; +7]	NS
ΔCI, L.min^{-1}.m^{-2}	+0.8 [+0.3; +1.4]**	+0.6 [−0.1; +1.5]	0.0 [−0.5; +0.1]	0.006
ΔSVR, dynes.sec.cm^{-5}	−201 [−623; +220]	−119 [−491; +175]	+390 [+237; +505]*	0.03
ΔSvO2, %	+6 [+2; +12]**	0 [−3; +4]	0 [−3; +6]	0.04
ΔLactate, mmol.L^{-1}	−0.4 [−2.5; −0.1]**	0.0 [−0.6; +0.2]	0.0 [−0.2; +0.5]	NS
ΔDelta-T, °C	−0.4 [−0.8; 0]	−1.1 [−1.9; +0.6]	0.0 [−2.2; +0.6]	NS
ΔPCD, mm.mm^{-2}	+0.6 [−0.9; +2.3]	+2.0 [+0.5; +3.4]*	−0.4 [−3.3; 0.0]	0.01

Abbreviations: HR, heart rate; MAP, mean arterial pressure; CVP, central venous pressure; PCWP, pulmonary capillary wedge pressure; MPAP, mean pulmonary artery pressure; CI, cardiac index; SVR, systemic vascular resistance; SvO_2, mixed-venous oxygen saturation; delta-T, central-peripheral temperature gradient; PCD, perfused capillary density.

Values represent median [interquartile range]. The p-value in the last column represents differences among groups. Asterisks indicate statistical significance versus baseline:

*, $p<0.05$;

**, $p<0.01$.

P-values>0.05 (NS, non-significant) are not shown.

[a]A pulmonary artery catheter was present in 27/33 (82%) of the measurements.

doi:10.1371/journal.pone.0103978.t003

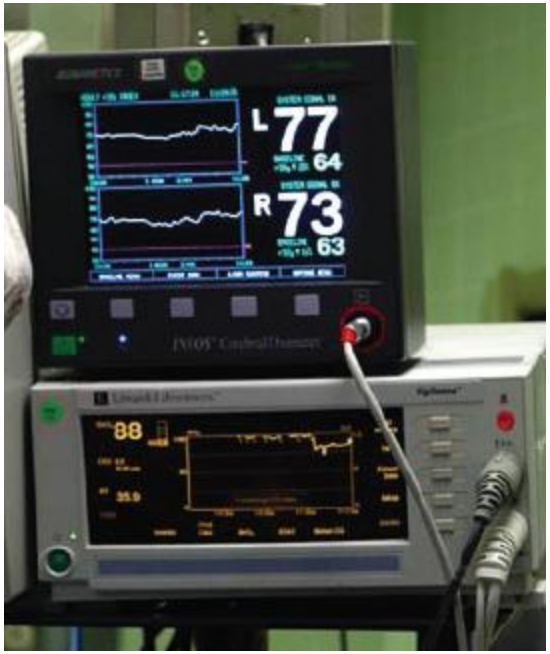
Cardiac output: global vs regional perfusion?

Regional resistance:

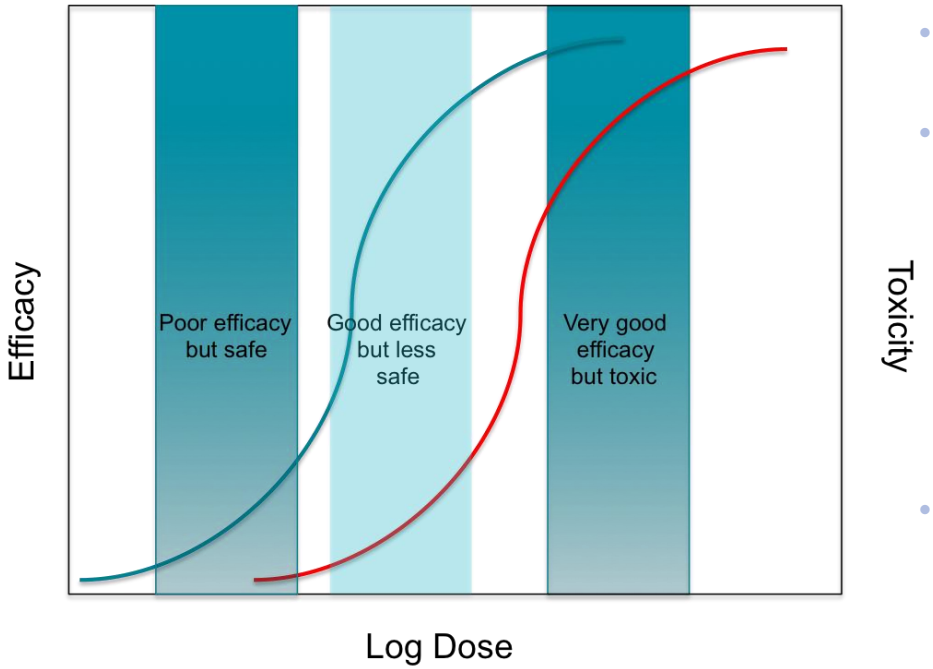
- neurohumoral factors related to inflammation and the sympathetic nervous system
- local factors related to autoregulation

Key (neglected) organs:

- GIT (gastric tonometry, splanchnic/hepatic saturations, indocyanine green)
- Brain



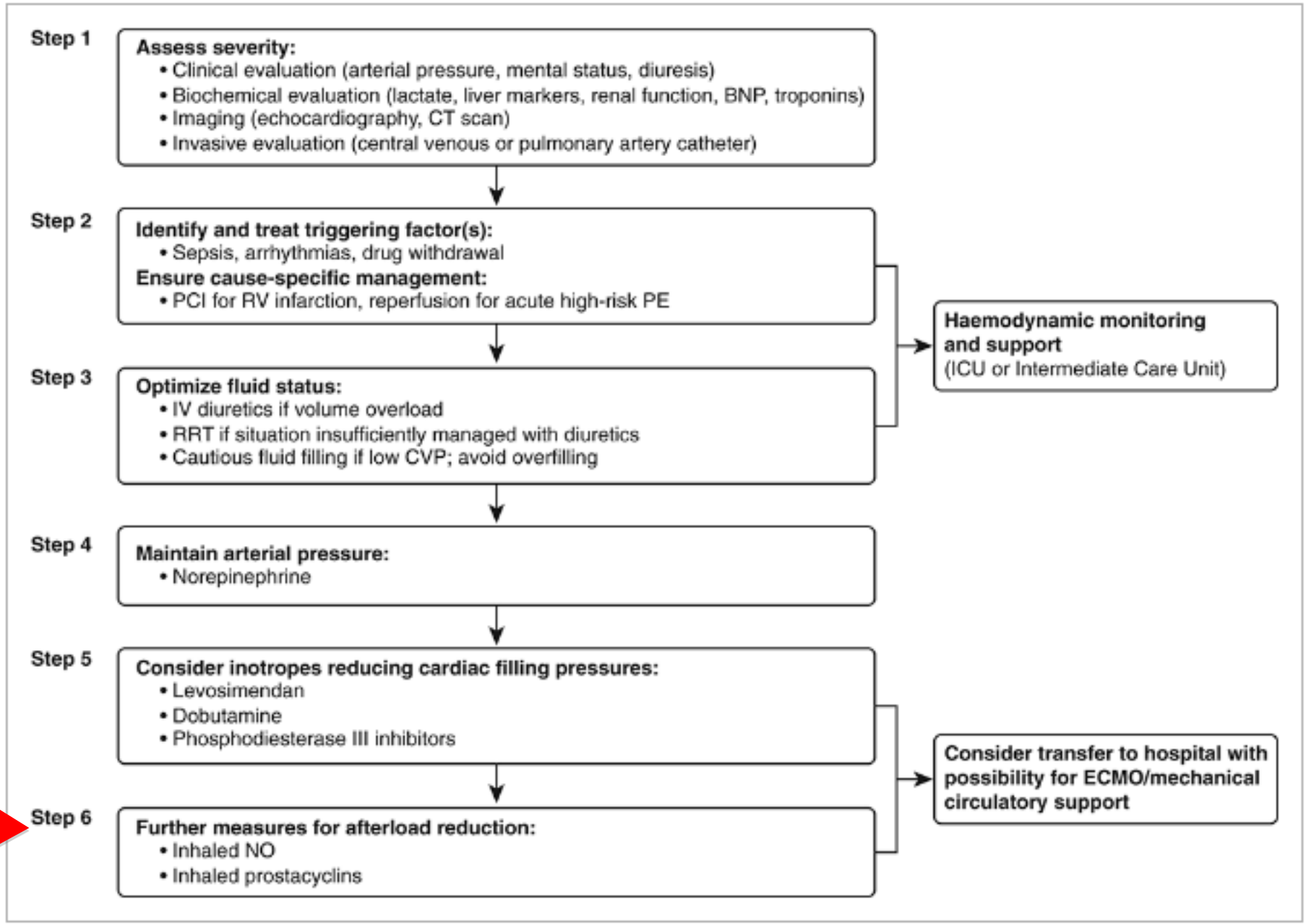
Each inotropic agent: efficacy vs toxicity



- **Each inotropic agent**
- **Each organ system**
  - Cardiac
  - Renal
  - Hepatic
  - Cerebral
  - GIT
  - Microcirculation
- **Each pathological situation:**
  - Sepsis
  - AMI+CS
  - DCM+CS
  - Haemorrhagic shock
- **In context of different ICU interventions**

Which inotrope?

- No real evidence to support one over another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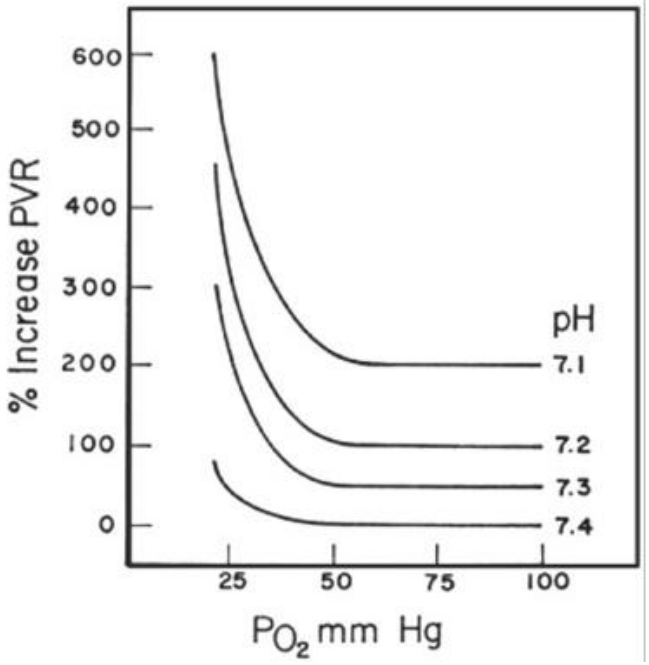
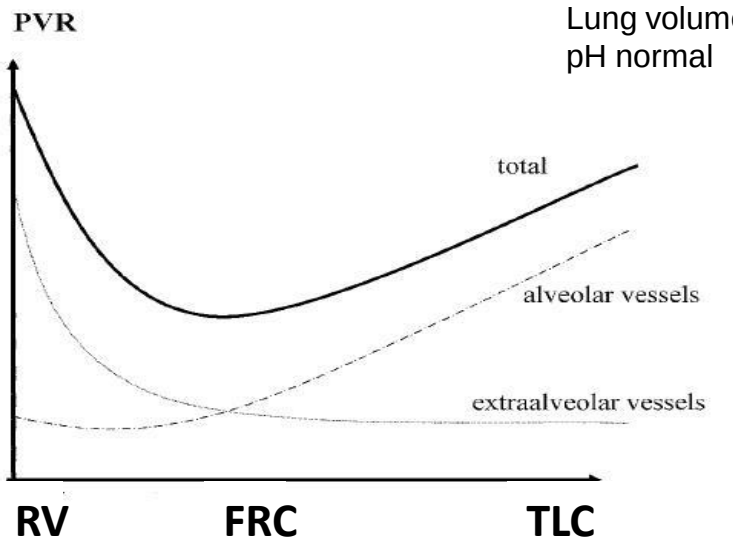
Afterload reduction

- Critical illness frequently associated with increased PVR
- HPV – alveolar, pulmonary arterial/bronchial arterial hypoxaemia, worsened with acidemia
- **Focus on:**
  - Reducing pulmonary vascular tone
  - Judicious use of pulmonary vasodilators
  - Awareness of the effects of positive pressure ventilation





Aim: normoxia, normocarbia
Lung volumes near FRC
pH normal

Ventetuolo & Klinger, Ann Am Thorac Soc 2014

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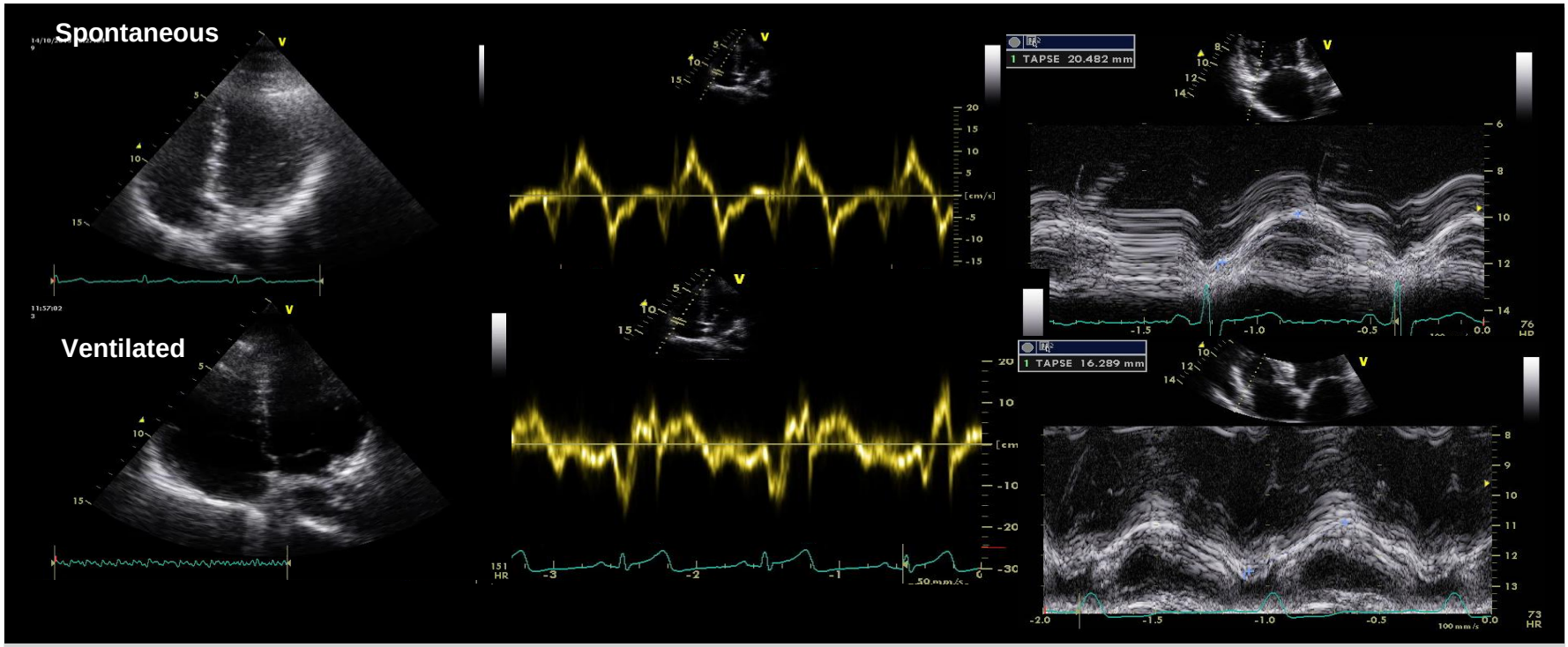
Potentially injurious effects of ventilation



Tavazzi G, ESICM 2014

Circulation

American Heart Association.

Articles

Characterization of Right Ventricular Diastolic Performance After Complete Repair of Tetralogy of Fallot

Restrictive Physiology Predicts Slow Postoperative Recovery

Presented in part at the 65th Scientific Sessions of the American Heart Association, New Orleans, La, November 1992.

Seamus Cullen; Darryl Shore; Andrew Redington

From the Royal Brompton Hospital and The National Heart and Lung Institute, London, UK.

Correspondence to Dr Andrew Redington, Royal Brompton Hospital and The National Heart and Lung Institute, Sydney Street, London 5W3 6NP, UK.

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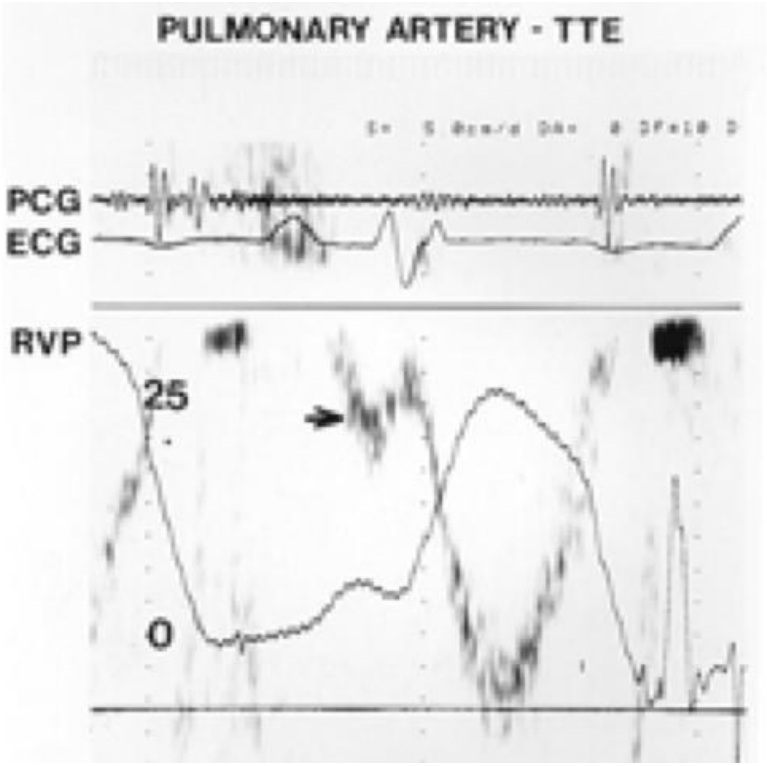
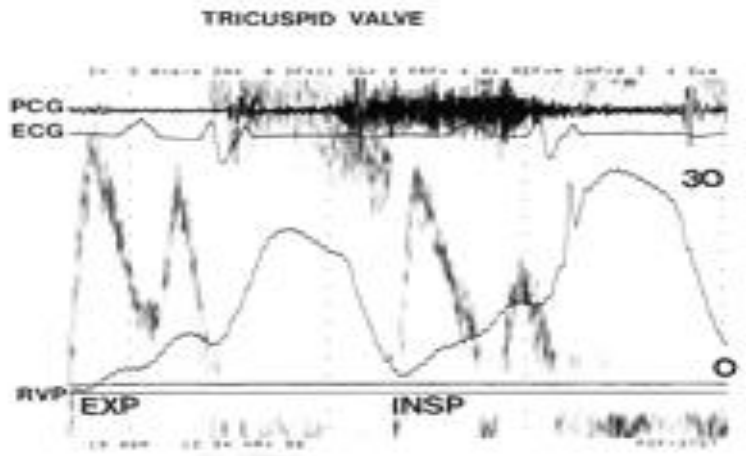
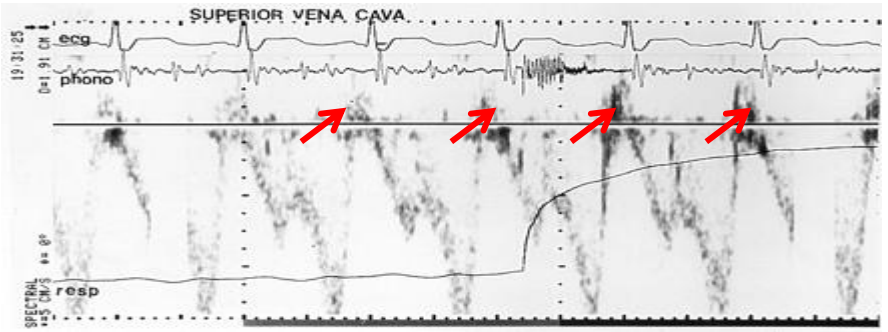


Effects of IPPV in RV restrictive physiology

- **Inspiration increases E/A ratio**
- **Abolishes PA diastolic wave**
- **Relative contribution of "restrictive" antegrade a wave to forward flow:**
  - Inspiration: 7 ±8%
  - Expiration: 22 ±10%
- **43% patients with SARF**
- **Inducible by IPPV**





Cullen, Circulation. 1995 Mar 15;91(6):178

Pulmonary vasodilators

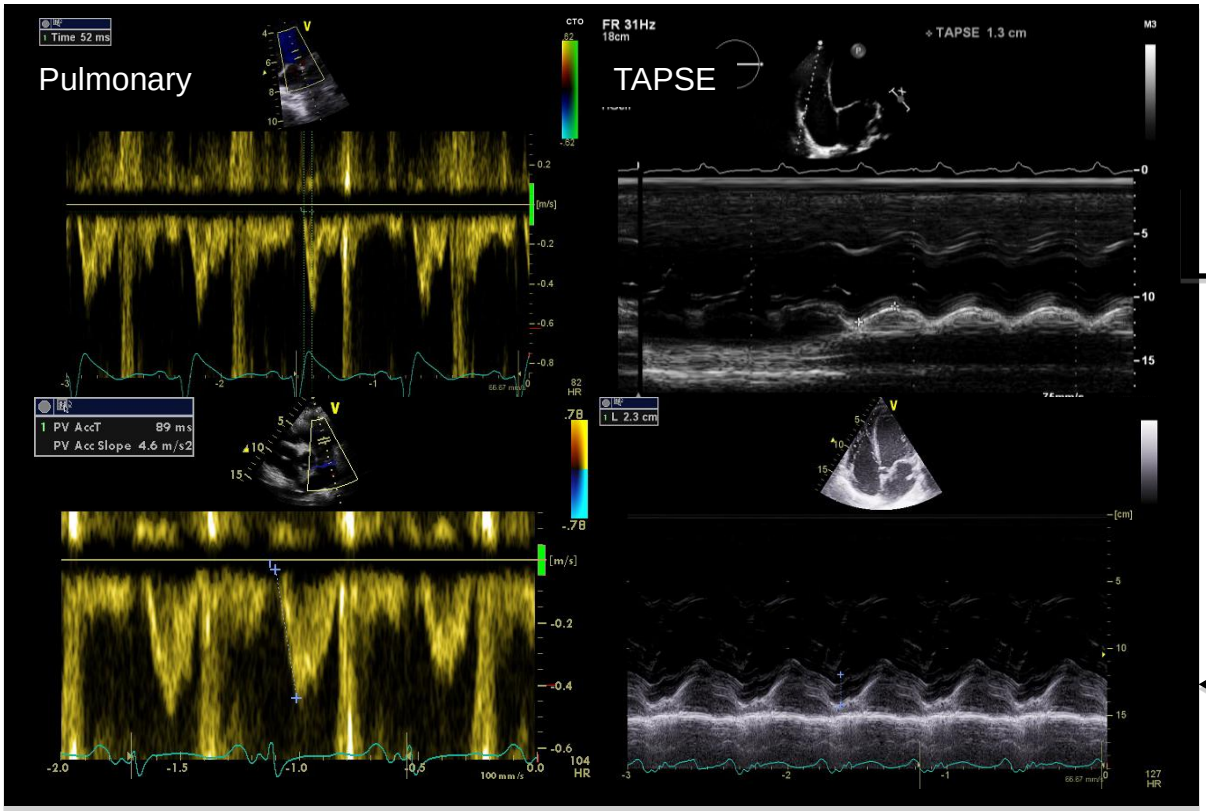
- None approved for treatment of RV failure in critically ill
- All have systemic & pulmonary effects
- Systemic administration may alter V/Q mismatch, and worsen hypoxaemia

Currently available pulmonary vasodilator medications

Name	Drug Class	Action	Route of Administration	Terminal Half-Life
Ambrisentan	Endothelin receptor antagonist	Blocks endothelin receptor A	Oral	15 h
Bosentan	Endothelin receptor antagonist	Blocks endothelin receptor A and B	Oral	5.4 h
Macitentan	Endothelin receptor antagonist	Blocks endothelin receptor A	Oral	14–18 h
Sildenafil	Phosphodiesterase type-5 inhibitor	Slows metabolism of intracellular cGMP	Oral or intravenous	4 h orally
Tadalafil	Phosphodiesterase type-5 inhibitor	Slows metabolism of intracellular cGMP	Oral	17.5 h
Epoprostenol	Prostacyclin	Increases intracellular cAMP	Intravenous or inhaled*	<6 min
Treprostinil	Prostacyclin derivative	Increases intracellular cAMP	Intravenous, subcutaneous, inhaled, or oral	4 h
Iloprost	Prostacyclin derivative	Increases intracellular cAMP	Inhaled	20–30 min
Nitric oxide	Soluble guanylate cyclase stimulator	Increases intracellular cGMP	Inhaled	Seconds
Riociguat	Soluble guanylate cyclase stimulator	Increases intracellular cGMP	Oral	7–12 h

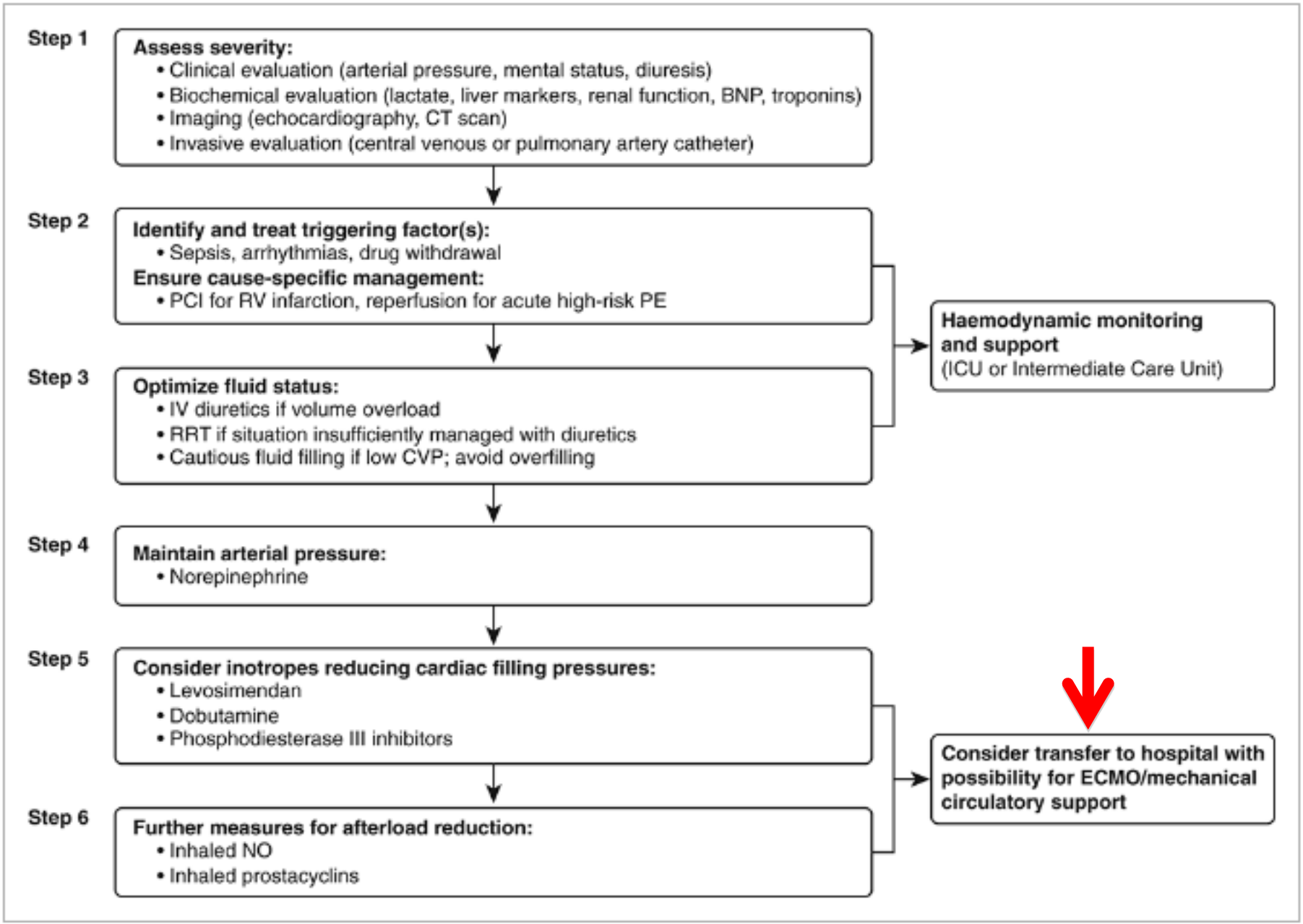
Harjola et al., Eur J Heart Failure 2016

Right heart afterload

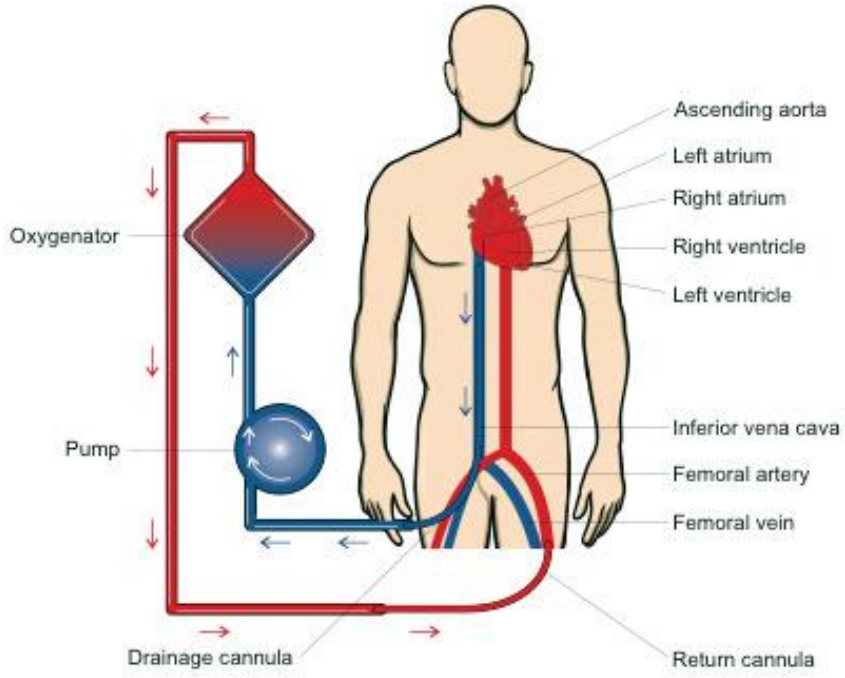


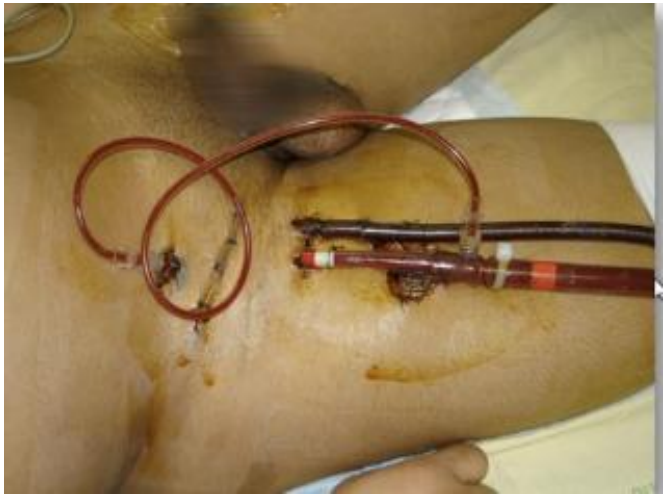
Maximal pulmonary vasodilatation

- iNO
+ Levosimendan
+ Nebulised prostacyclin
+ Low dose vasopressin
+ Nebulised milrinone


Step 1
Assess severity:
• Clinical evaluation (arterial pressure, mental status, diuresis)
• Biochemical evaluation (lactate, liver markers, renal function, BNP, troponins)
• Imaging (echocardiography, CT scan)
• Invasive evaluation (central venous or pulmonary artery catheter)
Step 2
Identify and treat triggering factor(s):
• Sepsis, arrhythmias, drug withdrawal
Ensure cause-specific management:
• PCI for RV infarction, reperfusion for acute high-risk PE
Step 3
Optimize fluid status:
• IV diuretics if volume overload
• RRT if situation insufficiently managed with diuretics
• Cautious fluid filling if low CVP; avoid overfilling
Haemodynamic monitoring and support
(ICU or Intermediate Care Unit)
Step 4
Maintain arterial pressure:
• Norepinephrine
Step 5
Consider inotropes reducing cardiac filling pressures:
• Levosimendan
• Dobutamine
• Phosphodiesterase III inhibitors
Step 6
Further measures for afterload reduction:
• Inhaled NO
• Inhaled prostacyclins
Consider transfer to hospital with possibility for ECMO/mechanical circulatory support

Peripheral VA-ECMO





23Fr venous, 19-21Fr arterial
(Legmo: 10-12Fr)

Cardiac (or cardiopulmonary) support
Percutaneous, rapid access
Awake or ventilated
Up to 8L/min – high, stable flow, 2-4 weeks
Better kit – transportation and monitoring
Cheaper than Tandem Heart and Impella
Expanding indications

Guidelines?

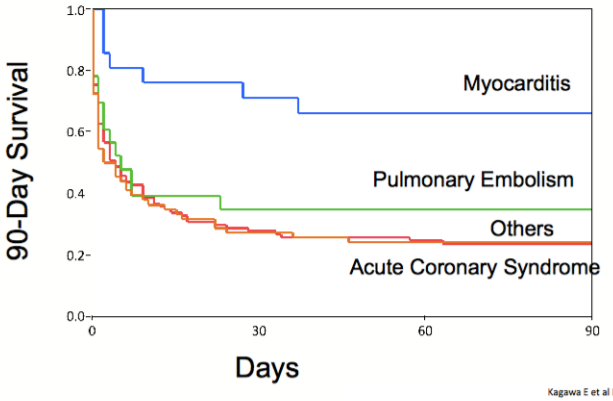
European Heart Journal (2014) **35**, 2541–2619
doi:10.1093/eurheartj/ehu278

ESC/EACTS GUIDELINES

2014 ESC/EACTS Guidelines on myocardial revascularization

The Task Force on Myocardial Revascularization of the European Society of Cardiology (ESC) and the European Association for Cardio-Thoracic Surgery (EACTS)

IABP insertion should be considered in patients with haemodynamic instability/cardiogenic shock due to mechanical complications.	IIa	C
Patients with mechanical complication after acute myocardial infarction require immediate discussion by the Heart Team.	I	C
Short-term mechanical circulatory support in ACS patients with cardiogenic shock may be considered.	IIb	C



Kagawa E et al ESC 2015

??RV failure

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J Heart Lung Transplant. 2015 Dec;34(12):1549-60. doi: 10.1016/j.healun.2015.08.018. Epub 2015 Sep 8.

Benefits of a novel percutaneous ventricular assist device for right heart failure: The prospective RECOVER RIGHT study of the Impella RP device.

Anderson MB[1], Goldstein J[2], Milano C[3], Morris LD[4], Kormos RL[5], Bhama J[5], Kapur NK[6], Bansal A[7], Garcia J[8], Baker JN[8], Silvestry S[9], Holman WL[10], Douglas PS[11], O'Neill W[12].

METHODS: Thirty patients with RVF refractory to medical treatment received the Impella RP device at 15 United States institutions. The study population included 2 cohorts: 18 patients with RVF after left ventricular assist device (LVAD) implantation (Cohort A) and 12 patients with RVF after cardiotomy or myocardial infarction (Cohort B). The primary end point was survival to 30 days or hospital discharge (whichever was longer). Major secondary end points included indices of safety and efficacy.

Transfemoral insertion

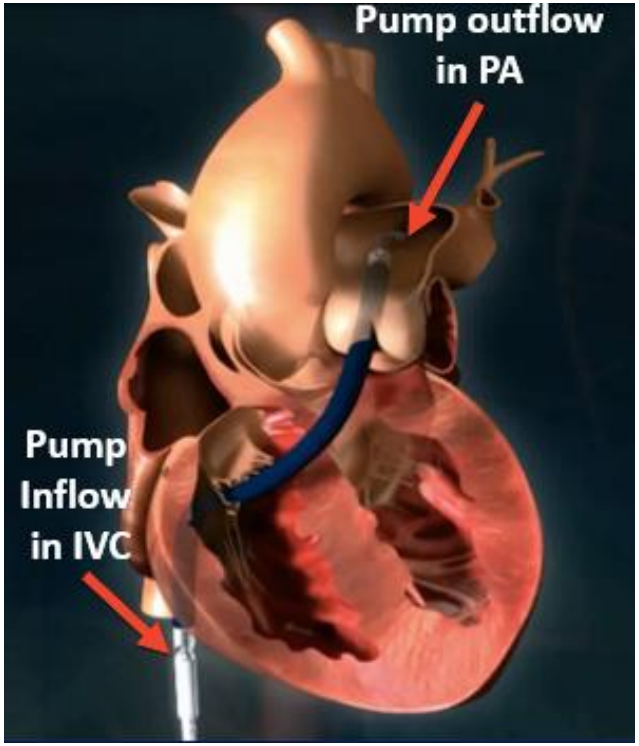
3D shaped cannula

22Fr motor housing
Pump on 1Fr catheter

4L/min @33,00rpm

ACT160-180

COHORT B: 58.3% survival (cohort predicted survival 40%)



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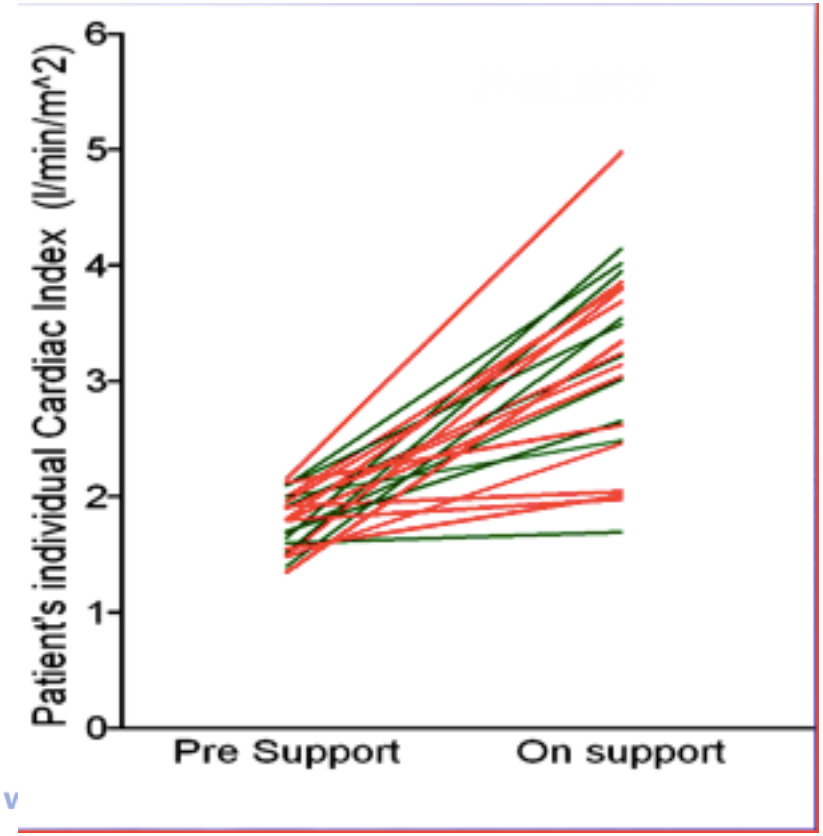

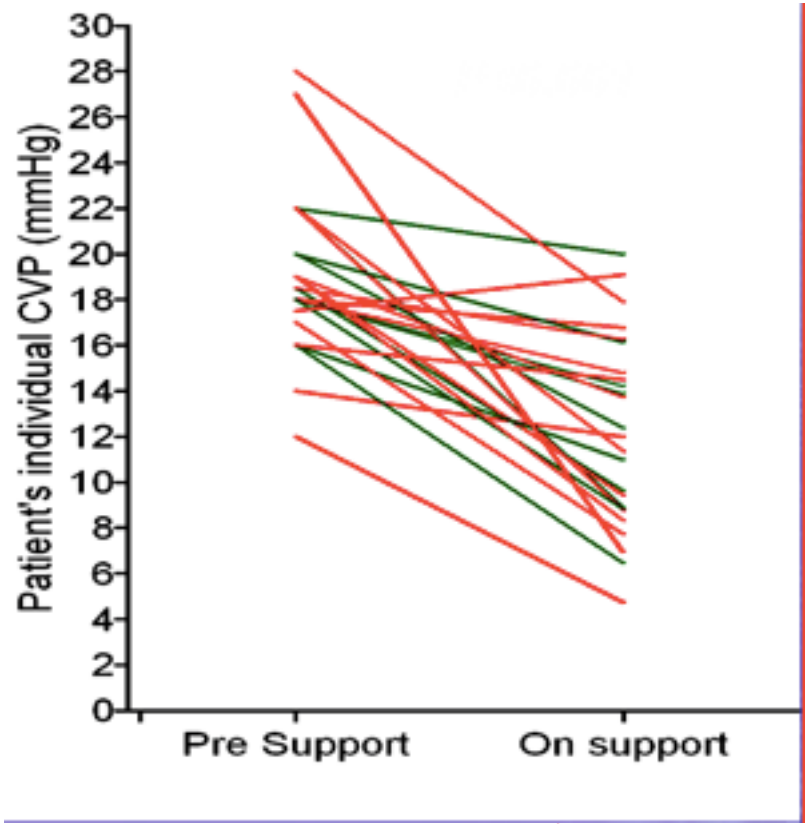

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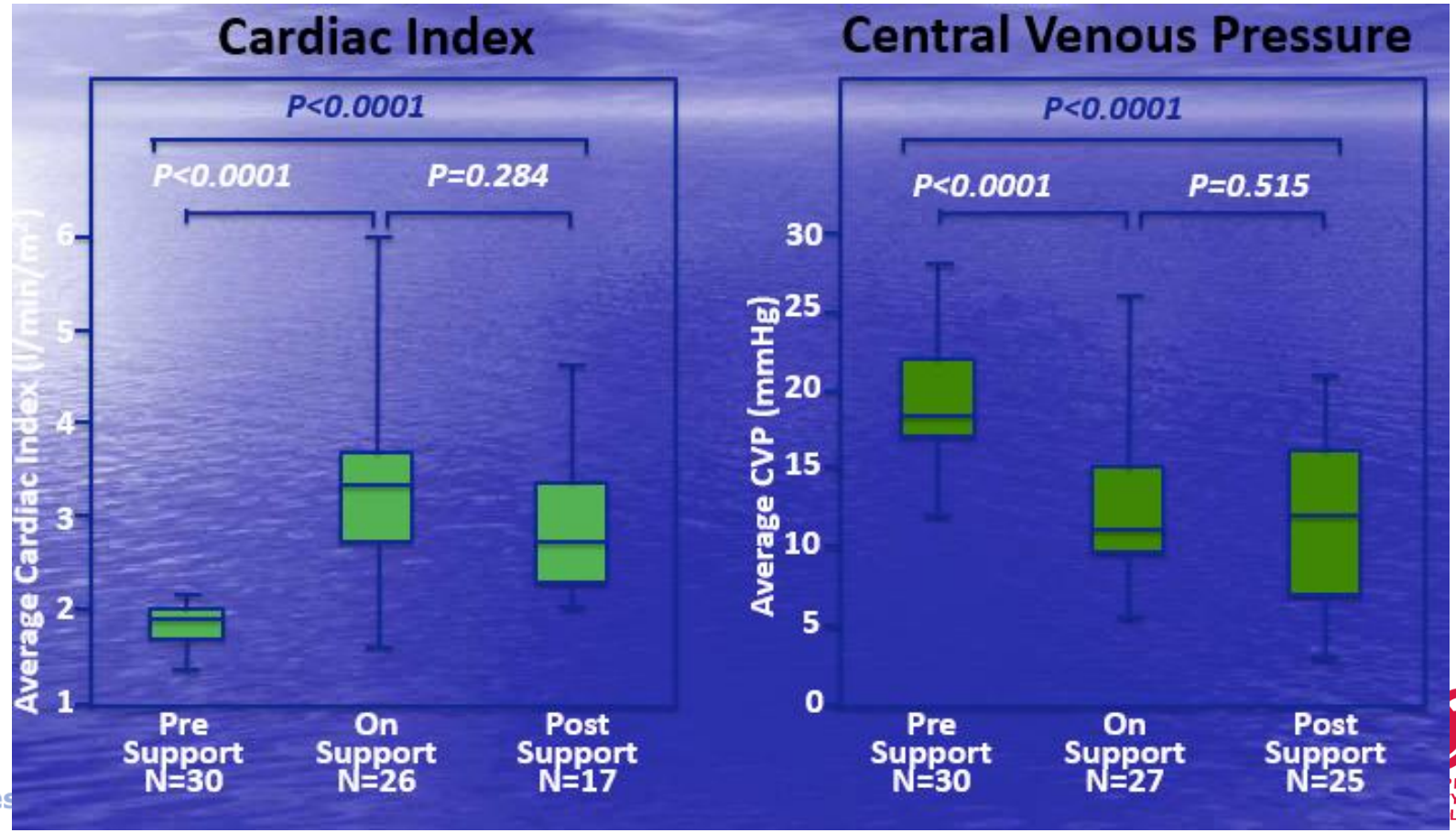

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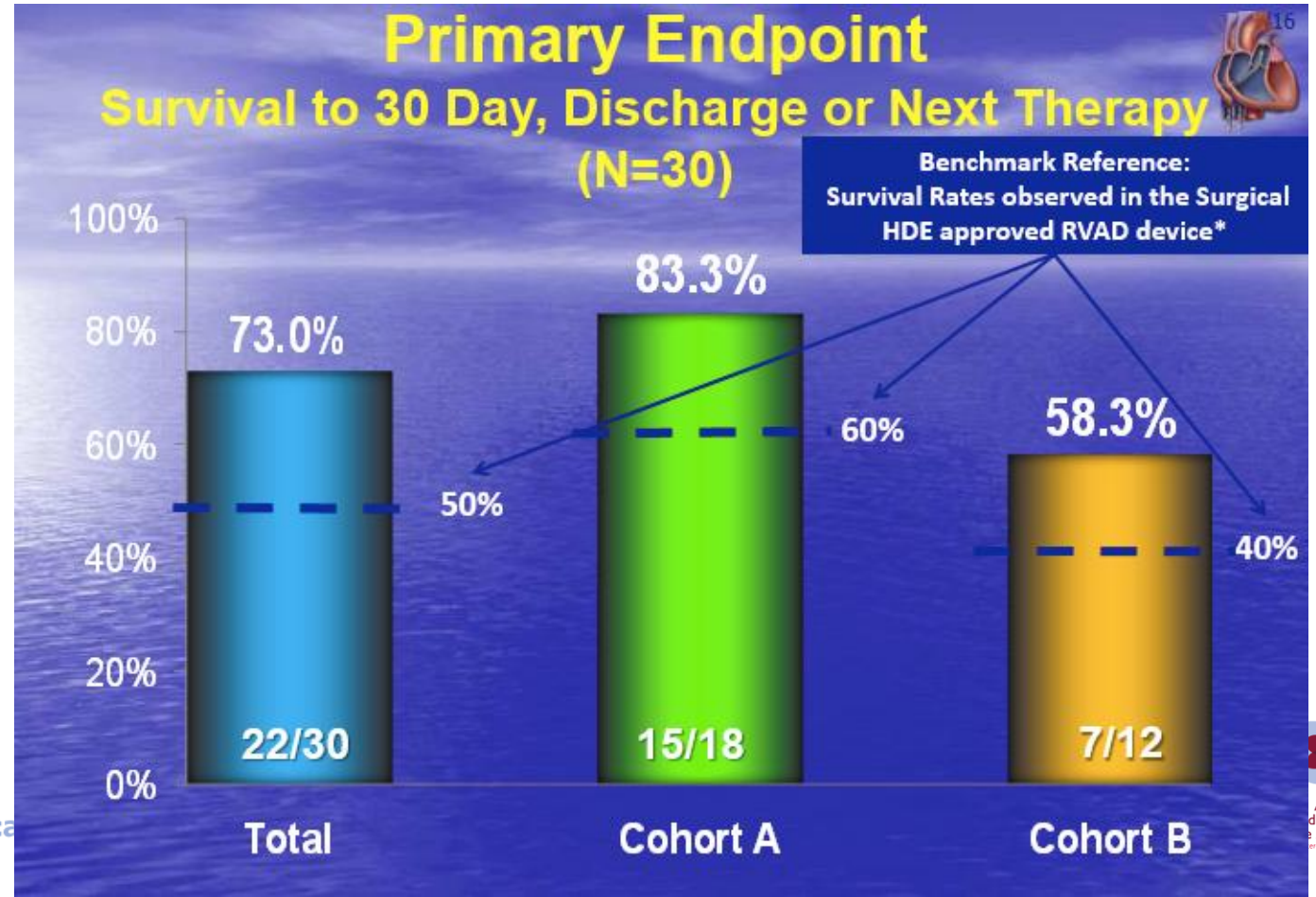

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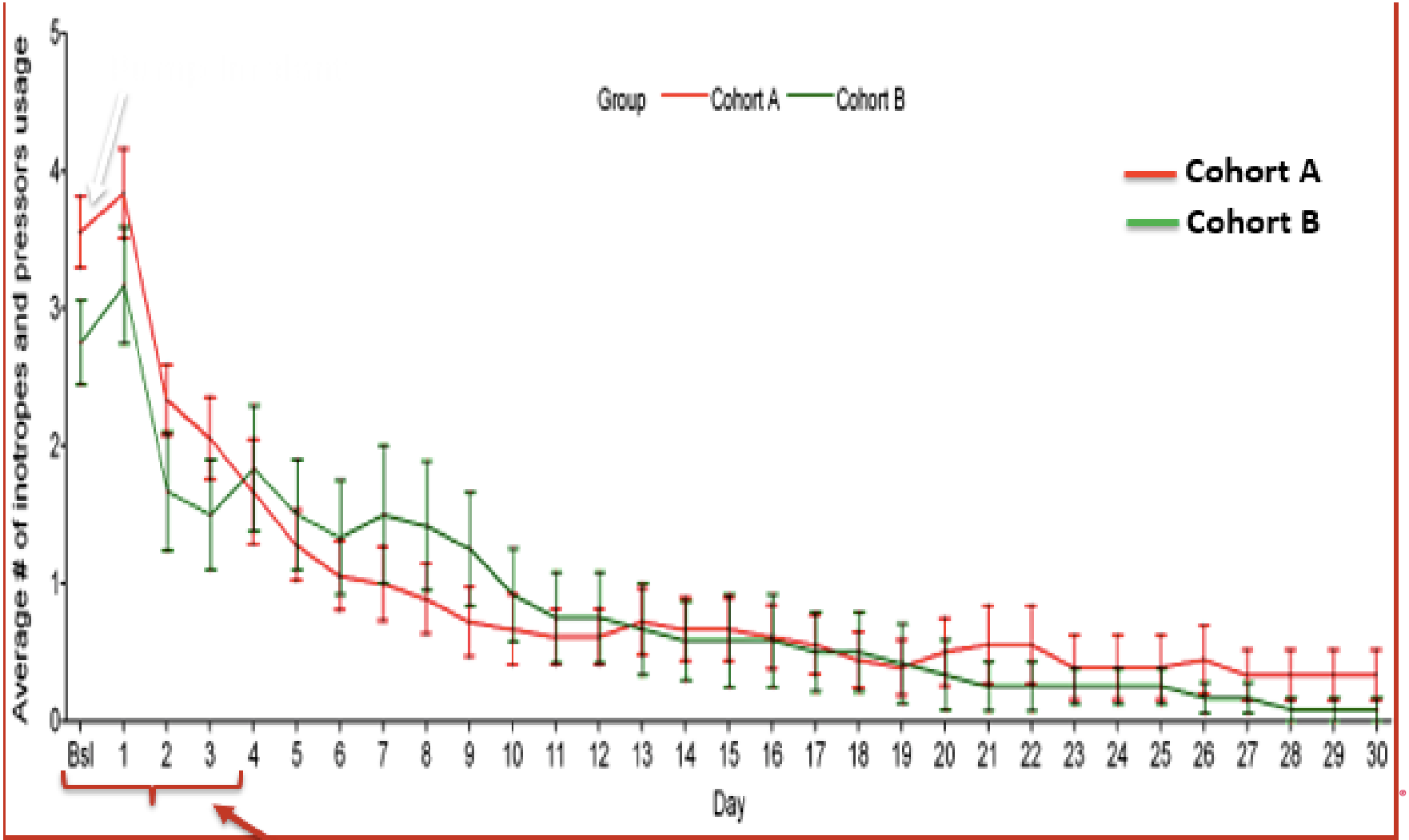

Statement from ESC

European Journal of Heart Failure

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Statement

Contemporary management of acute right ventricular failure: a statement from the Heart Failure Association and the Working Group on Pulmonary Circulation and Right Ventricular Function of the European Society of Cardiology

Veli-Pekka Harjola ✉, Alexandre Mebazaa, Jelena Čelutkienė, Dominique Bettex, Hector Bueno, Ovidiu Chioncel, Maria G. Crespo-Leiro, Volkmar Falk, Gerasimos Filippatos, Simon Gibbs, Adelino Leite-Moreira, Johan Lassus, Josep Masip, Christian Mueller, Wilfried Mullens, Robert Naeije, Anton Vonk Nordegraaf, John Parissis, Jillian P. Riley, Arsen Ristic, Giuseppe Rosano, Alain Rudiger, Frank Ruschitzka, Petar Seferovic, Benjamin Sztrymf, Antoine Vieillard-Baron, Mehmet Birhan Yilmaz, Stavros Konstantinides

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Conclusions

Acute RV failure is a complex clinical scenario and its appropriate management requires an understanding of RV anatomy and mechanics, rapid identification and treatment of underlying causes, and knowledge of supportive treatment measures. Many uncertainties remain, and there is a need for randomized trials to investigate the efficacy and safety of pharmacological and mechanical interventions for the treatment of acute RV failure.

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Many interventions seem physiologically/intuitively sensible – but that doesn't mean they are right

Sir Iain Chalmers, co-founder Cochrane collaboration, BBC Radio 4, 2013