Management
Post operative
Low Cardiac Output Syndrome

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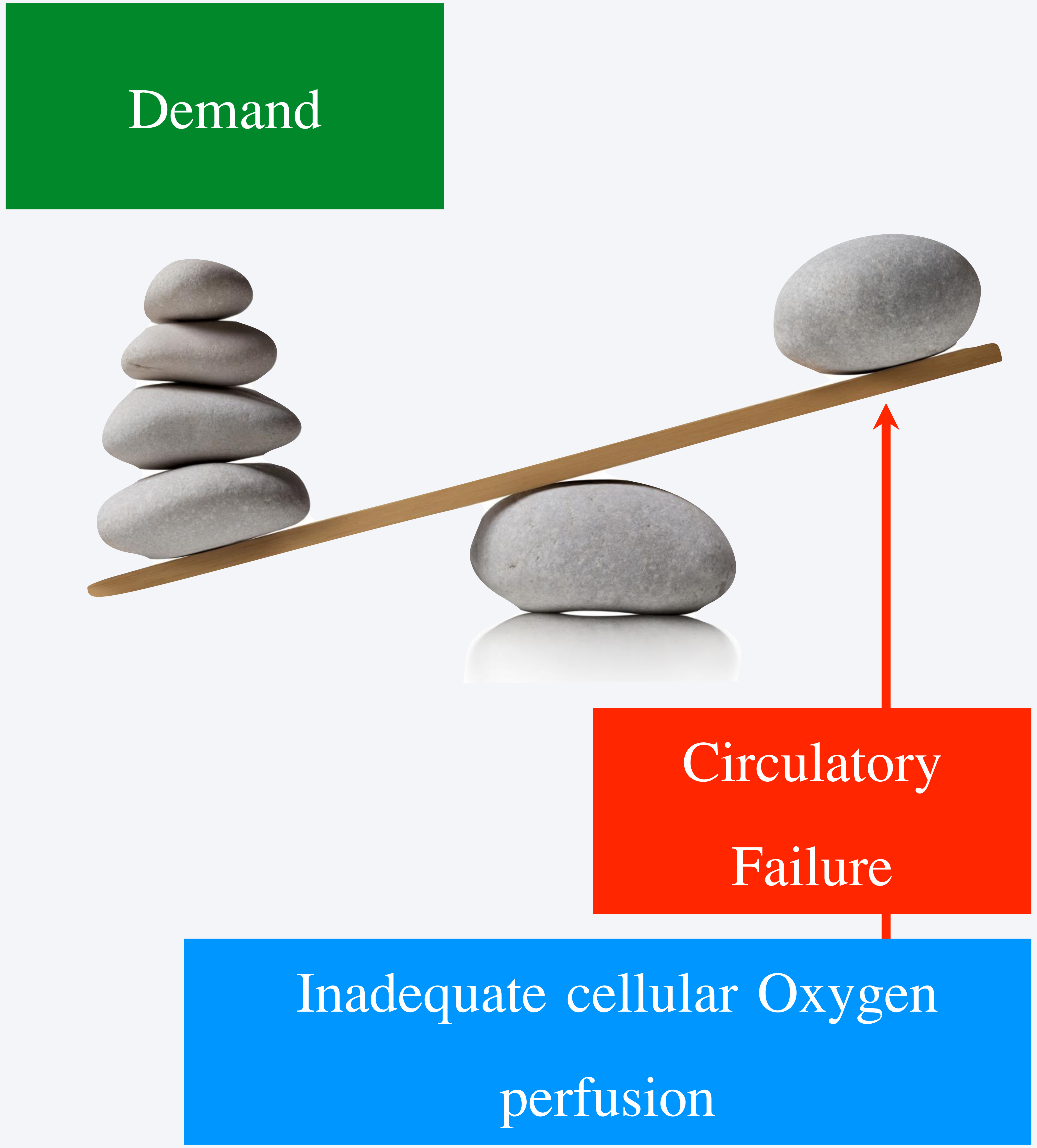
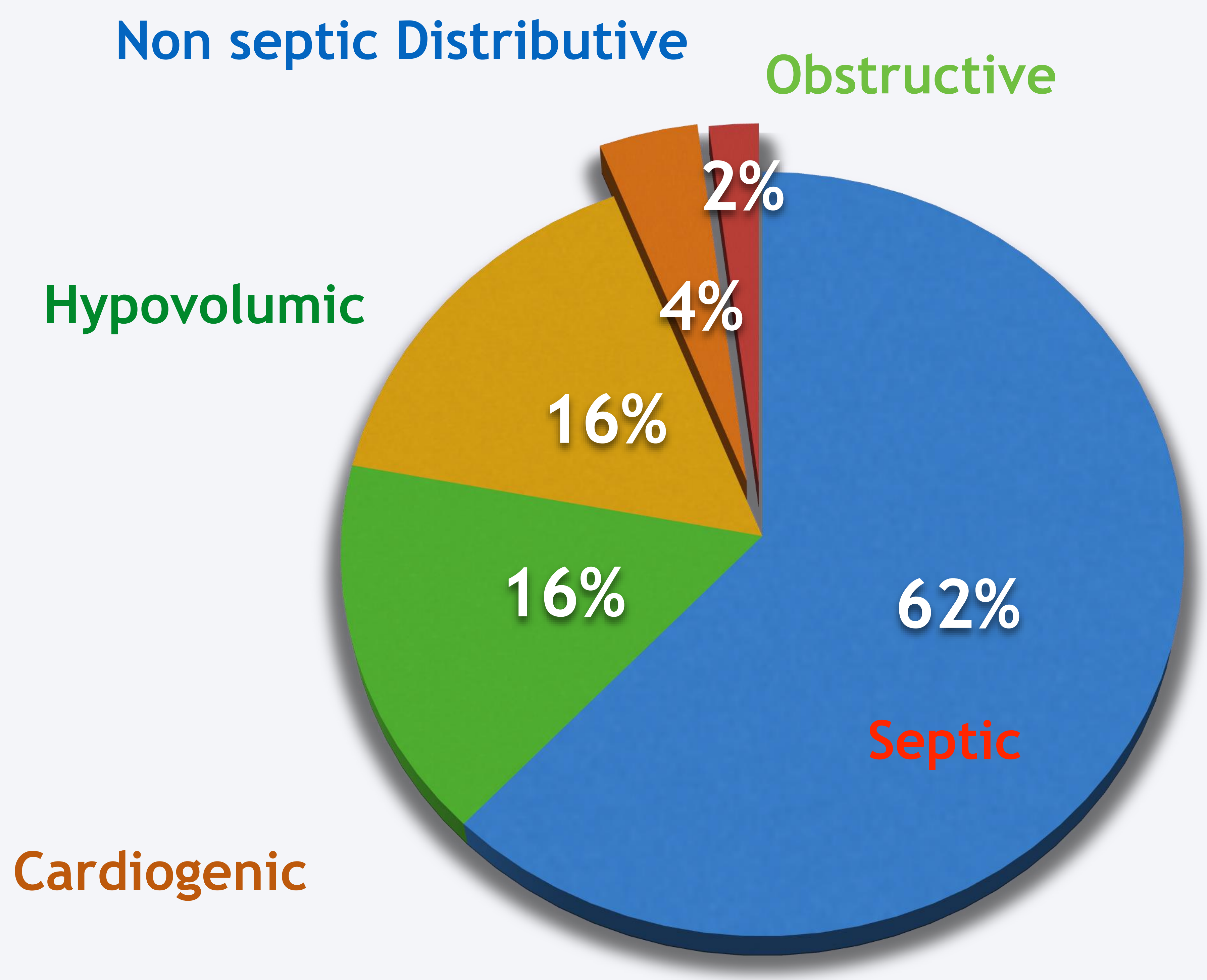
Two Days 2020



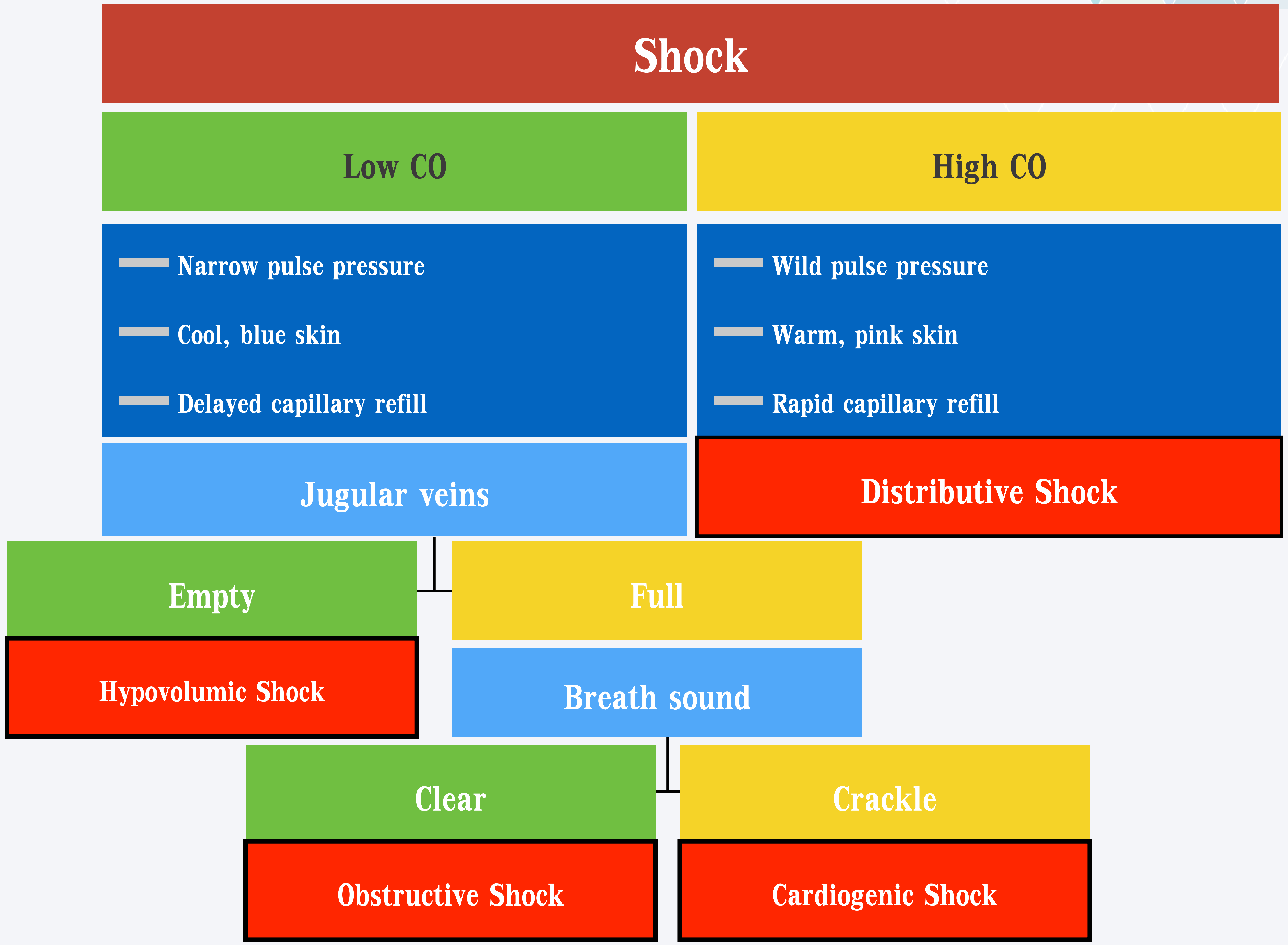
Shock

1. **Systemic arterial hypotension:** Typically, in adults, SBP < 90 mm Hg or MAP < 70 mmHg, with associated tachycardia.
2. **Clinical signs of tissue hypoperfusion:** Cutaneous (skin that is cold and clammy, with vasoconstriction and cyanosis)
   - Renal (urine output of <0.5 ml/kg/hr)
   - Neurologic (altered mental state, disorientation)
3. **Hyperlactatemia**: >1.5 mmol/L

Type to Shock



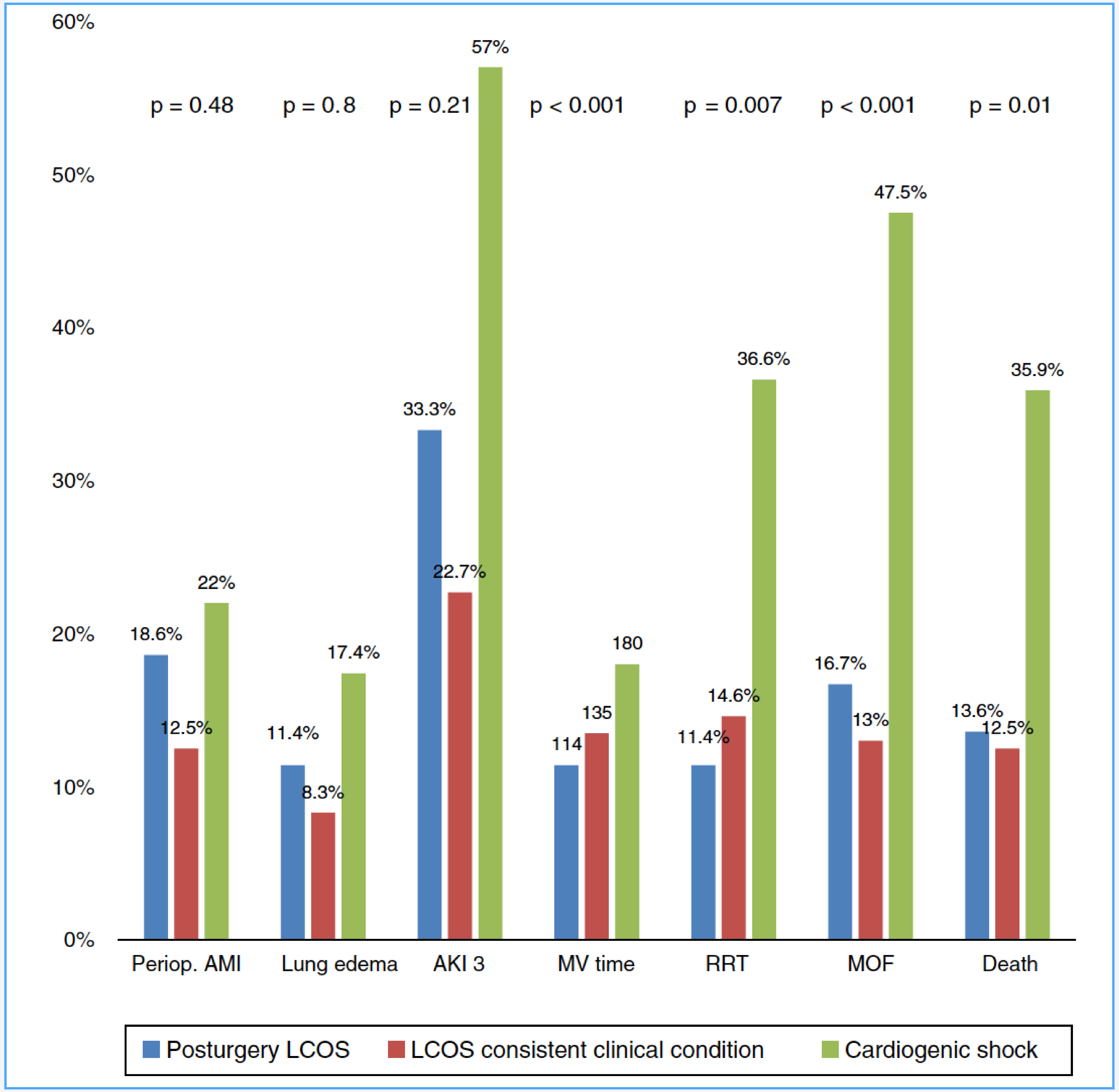
De Backer D, N EngJ Med. 2010;362:779-89



Principle of critical care. 4th ed. New York: McGraw-Hill, 2015:249-62.

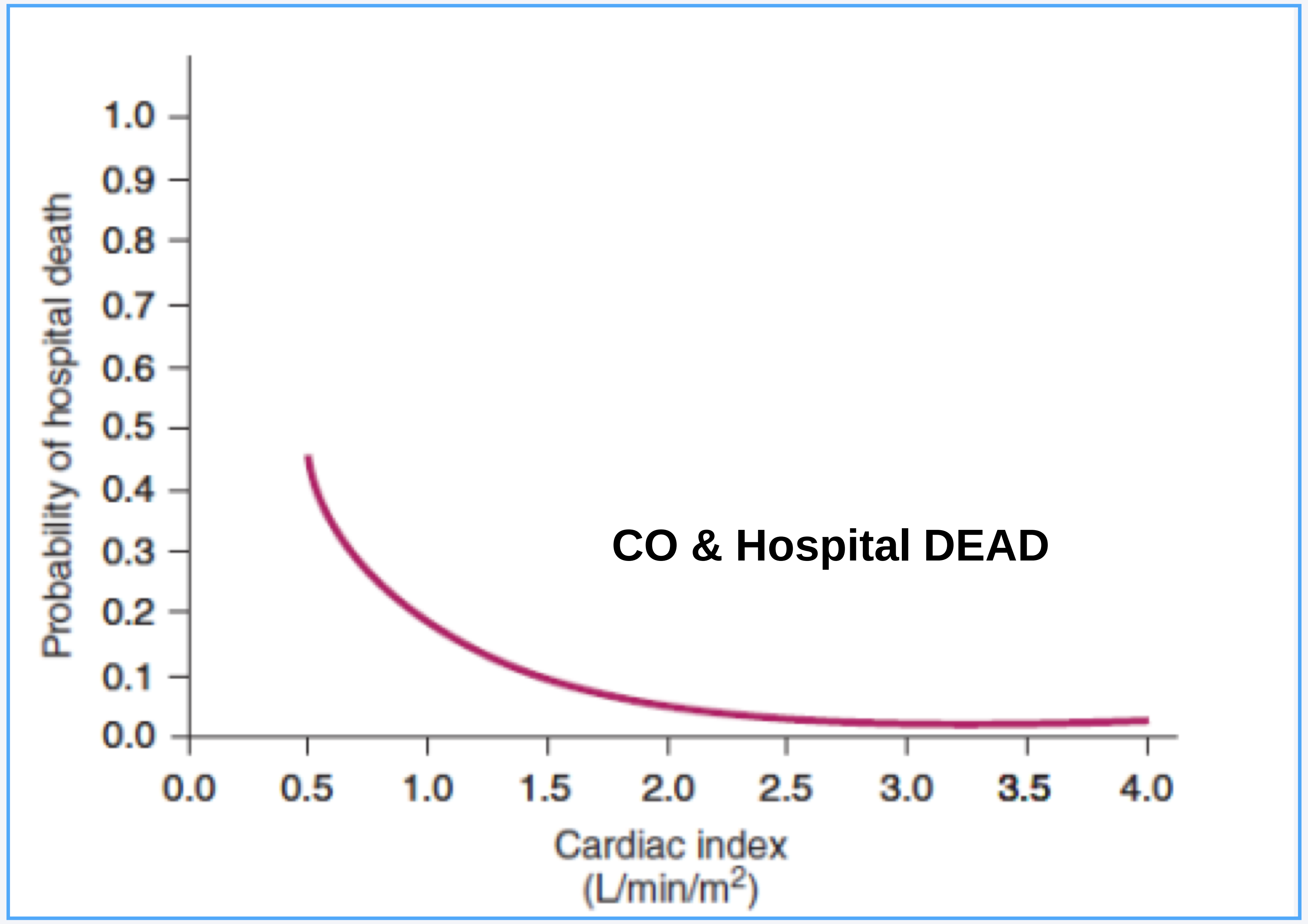
Low Cardiac Output Syndrome : Definition

LCOS	Cardiac index <2.2 l/min/m
	without relative hypovolemia
	secondary to LV, RV failure
	+/− systemic, pulmonary congest
↓	
Clinical malperfusion	Oliguria (diuresis <0.5 ml/kg/h)
	SvO2 <60%
	Lactate >3 mmol/l
↓	
Cardiogenic shock	Cardiac index <2.0 l/min/m
	SBP <90 mmHg



Complication

Relate LCOS



Med Intensiva. 2018;42(3):159—167

Cardiac Surgery risk for LCOS

Incident：3 - 5 %, Post Adult Cardiac Operation

25 %, Post Congenital Cardiac Operation

LCOS

- Decrease Oxygen delivery
- Reduce Cardiac output
- Organ/Tissue poor perfusion

- Surgical manipulations
- Arrhythmias / Valvular dysfunction
- Impaired preload
- Myocardial depression / dysfunction
- Vascular resistance / tone

Preoperative factors	-Age>65 years / Female -LVEF<50% -On-pump CABG -Recent MI -Complex congenital Surgery -Severe PHT -DM and CKD -Malnutrition
Laboratory predictors	-Hemoglobin -TLC 2,000 cells per microliter -NT-proBNP -BNP -hFABP

Peri-operative factors	-CPB duration -Emergency / Redo surgery -Incomplete revascularization
Multifactorial cause	-CPB with cardioplegic arrest: myocardial dysfunction -Inadequate myocardial protection -Systemic inflammatory responses -ischemic / Reperfusion injury -Alteration in signal transduction system -Uncorrected pre-existing cardiac conditions

* TCL : Total lymphocyte count , hFABP : Heart fatty acid binding protein

LCOS Risk factors

Table 3 Distribution of patients according to diagnostic subgroup and pre- and intraoperative variables.

	LCOS (n = 46)	LCOS consistent clinical condition (n = 50)	Cardiogenic shock (n = 41)	Total	*p*-Value
Female gender, n (%)	15 (32.6)	17 (34)	15 (36.6)	47 (34.3)	0.925
Male gender, n (%)	31 (67.4)	33 (66)	26 (63.4)	90 (65.7)	
Mean age in years	68.7	67.96	68.59	68.26	0.91
EuroSCORE II, mean	5.58	9.33	15.83	9.99	0.001
Arterial hypertension, n (%)	33 (72)	35 (70)	29 (71)	97 (70.8)	0.98
Dyslipidemia, n (%)	28 (61)	29 (58)	30 (73)	87 (63.5)	0.29
Diabetes, n (%)	17 (37)	21 (42)	8 (20)	46 (33.6)	0.06
Smoker, n (%)	11 (24)	14 (28)	11 (27)	36 (26.3)	0.89
COPD, n (%)	4 (8.7)	6 (12)	5 (12)	15 (10.9)	0.83
Recent ischemic damage (3 months), n (%)	10 (22)	10 (20)	13 (32)	33(24.6)	0.43
Previous myocardial infarction, n (%)	14 (30)	13 (26)	17 (41)	44 (32.1)	0.27
Resting angina (CCS class 4), n (%)	1 (2.2)	3 (6)	7 (17)	11 (8)	0.03
Previous PTCA/stent, n (%)	7 (15)	7 (14)	9 (22)	23 (16.8)	0.56
Depressed LVEF (<35%), n (%)	14 (30)	16 (32)	15 (37)	45 (33.8)	0.89
Presurgical NYHA III and IV, n (%)	20 (43)	24 (48)	27 (66)	71 (52.6)	0.04
Preoperative critical condition, n (%)	6 (13)	7 (14)	13 (32)	26 (19)	0.04
Valve disease with previous moderate-severe pulmonary hypertension, n (%)	15 (33)	17 (34)	12 (29)	44 (33.1)	0.82
Previous cardiac surgery, n (%)	4 (8.7)	9 (18)	11 (27)	24 (17.5)	0.08
Active endocarditis, n (%)	2 (4.3)	6 (12)	3 (7.3)	11 (8)	0.37
Emergency surgery, n (%)	2 (4.3)	0 (0)	7 (17)	9 (6.6)	0.002
ECC > 120 min, n (%)	23 (50)	30 (60)	32 (78)	85 (62.5)	0.03
ECC reentry, n (%)	5 (10.9)	6 (12)	11 (26.8)	22 (16)	0.05

Cardiogenic Shock
Patient risk factors

- EuroSCORE II
- Diabetes
- CCS class 4
- Preoperative critical condition
- Previous cardiac surgery
- Emergency surgery
- CPB > 120 min
- CPB re-entry

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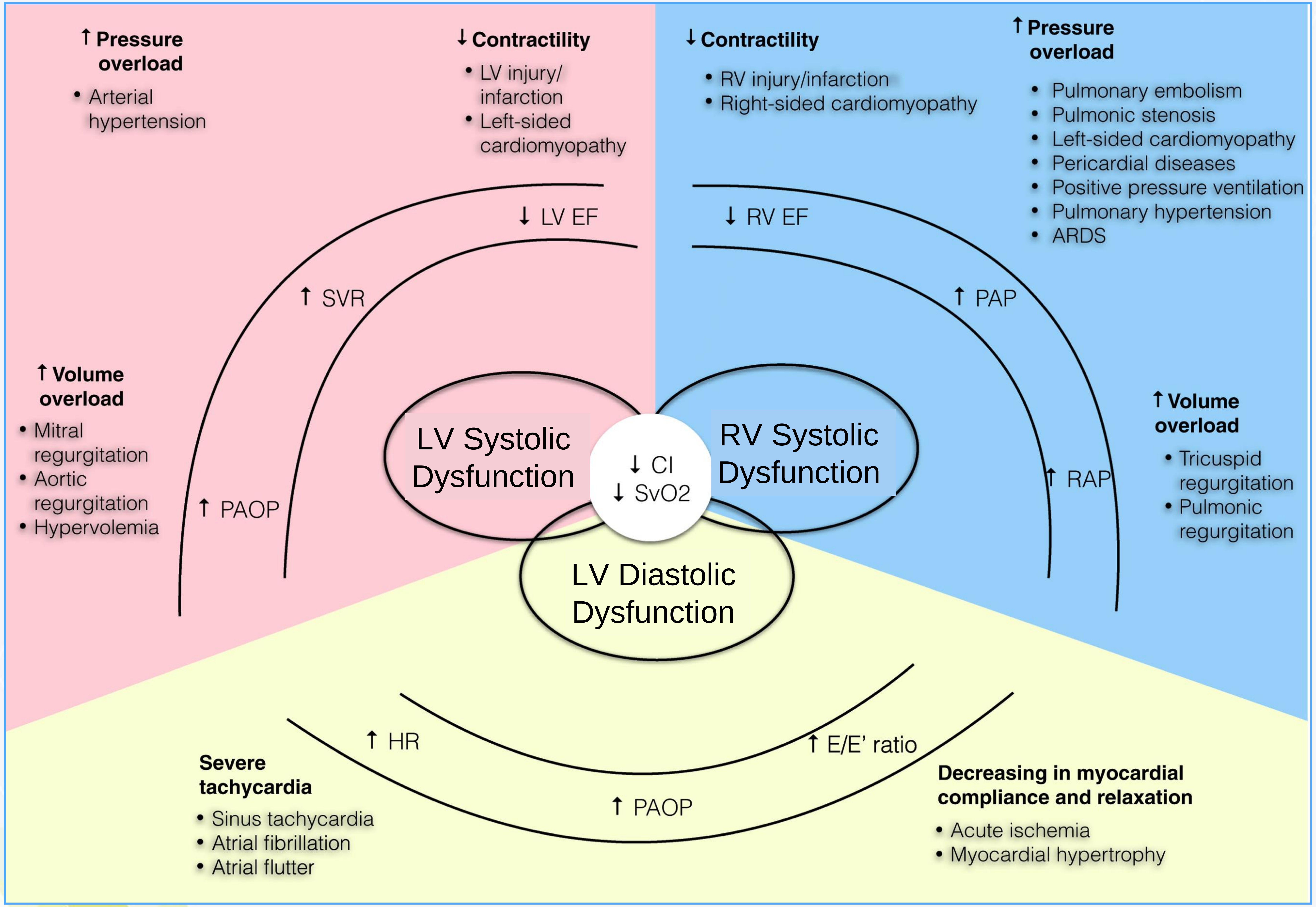
Common Cardiac pathophysiologic mechanism



LCOS Management

Early recognition / monitor

Treatment

Prevention

Diagnosis LCOS

- Signs / Symptoms / Clinical assessment
- Hemodynamic monitoring
- Diagnostic studies / Laboratory tests / Serologic variables

Early recognition

Timely intervention / Management

Hemodynamic Monitor and Early Detection

Principle of Hemodynamic Monitor

Balance Oxygen delivery (DO2) vs Oxygen consumption (VO2)

Real time measurement

Less invasive

Monitor Depend on

- Type of Surgery
- Patients relative risks

Methods of Hemodynamic Monitoring

Non invasive Technique

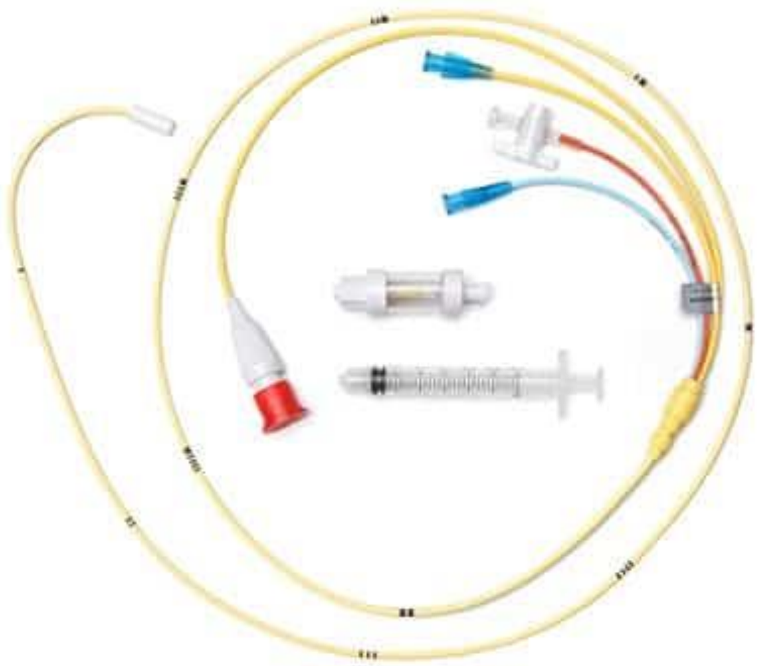
- TTE
- Non invasive PC system: Nexfin, ClearSight
- Bioimpedance
- Estimated CCO: esCCO
- Ultrasound CO monitoring: USCOM

Less invasive Technique

- Transpulmonary thermodilution: PiCCO, VolumeView/EV1000
- Transpulmonary dye dilution: LiDCO Plus
- Ultrasound flow dilution: COstatus
- Pulse contour and Pulse pressure analysis: FloTrac/Vigileo/EV1000, ProAQT/Pulsioflex, LiDCO Rapid system, Most Care
- Respiratory derived CO monitoring system: NiCO
- TEE
- Esophageal Doppler: CardioQ

Invasive Technique

PAC



Advantages and Disadvantages of Different Hemodynamic Monitoring Devices in Cardiac Surgery

	Invasiveness	Reliability in Cardiac Surgery Patients	Ease of Use	Ability to Monitor CO in Real Time	Ability to Measure Variables Other Than CO
Ultrasound techniques*	+	++	+	+	+++
Pulmonary artery catheter	+++	+++	+	++†	++
Transpulmonary thermodilution	++	+++	++	+++	+++
Lithium dilution	++	+++	+	+++	++
Uncalibrated pulse contour analysis	+	+	++	+++	+
Applanation tonometry	0	+	+++	+++	+
Estimated continuous cardiac output	0	+/ -	+++	+++	+
Bioreactance	0	+/ -	++	+++	+

Curr Opin Crit Care 21:395–401, 2015

Hemodynamic Monitor

BASIC

ECG MONITORING

HR, Rhythm , Arrhythmia , Ischemic pattern

BLOOD PRESSURE MONITORING

MAP, SVR : CO

Cuff (sphygmomanometer), Catheter

SPO_2 MONITORING

Peripheral skin perfusion, O_2 dissociation curve

Oxygen saturation, SpO_2 should be maintained >92%

SERUM LACTATE

resting humans ~1 mmol/L (0.7-2.0)

^ serum lactate : Poor tissue perfusion; circulatory failure, anaerobic metabolism and tissue hypoxia

PRELOAD MEASUREMENT

STATIC

Pressure

CVP
PAOP

Measurement devices:
- **CVP:** CVC
- **PAOP:** PAC

Volume

GEDV
LVEDV

Measurement device:
- **GEDV** (Transpulmonary thermodilution): PiCCO, VolumeView
- **LVEDV:** Echocardiography

DYNAMIC

SPV
PPV
SVV
IVC/SVC collapsibility

Measurement device:
- **PPV:** PiCCO, LiDCO, Most Care
- **SPV:** PiCCO, LiDCO, Most Care
- **SVV:** PiCCO, LiDCO, FloTrac/Vigileo/EV1000, Most Care, Volume clamp method (Finapres, Nexfin), Esophageal Doppler, Ultrasound Doppler
- **IVC/SVC:** Echocardiography

Static Measurement

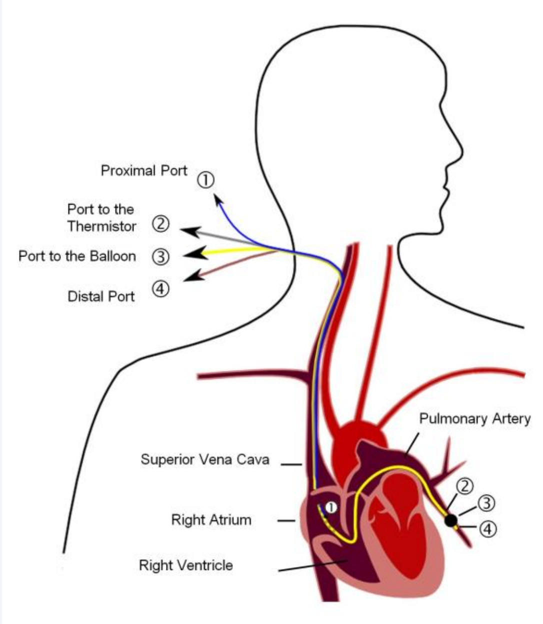
CVP / RAP

- Assume right ventricular output is proportionate to left ventricular preload
- Inaccurate and unreliable predictor of fluid responsiveness

- RAP is a measure pressure not volume
- Variation in vascular tone, intra-thoracic, and cardiac function directly affect RAP, without change of preload

PAP / PAOP

- Measure LV preload Equivalent to LVEDP
- myocardial compliance (sepsis, myocardial ischemia),Right ventricular overload, Pericardial disease, Increase intrathoracic pressure



Critical Care Reviews

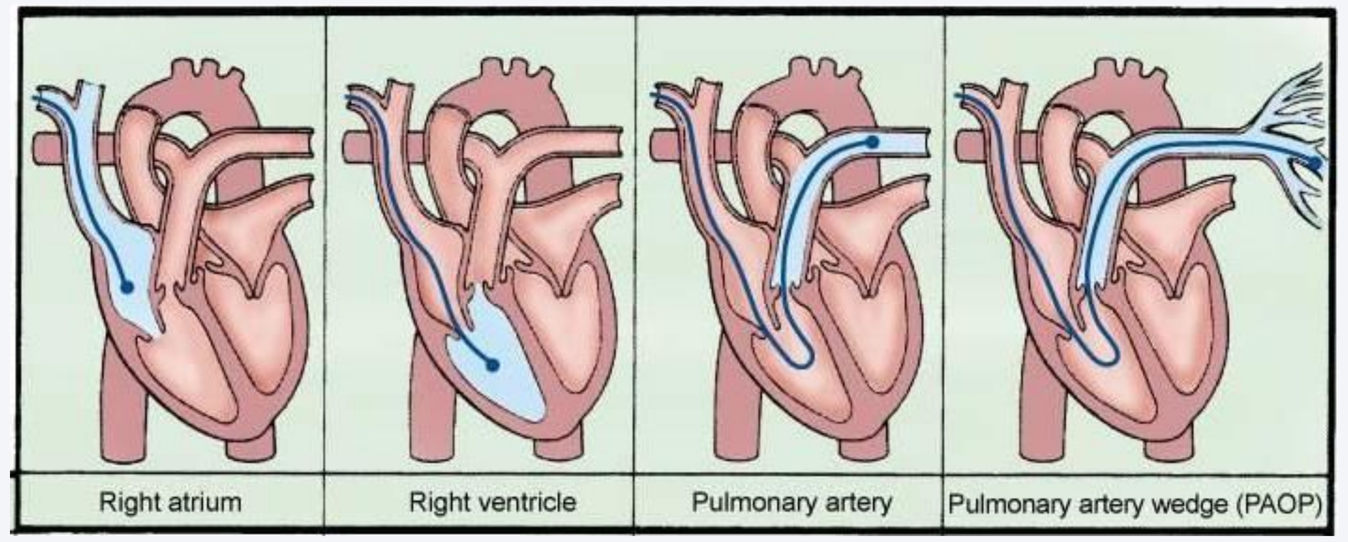
Predicting Fluid Responsiveness in ICU Patients: A Critical Analysis of the Evidence Ann Intensive Care. 2016; 6: 111.

-RAP, PAOP, RVEDV, and LVEDA were not significantly lower in responders than in non-responders

- no threshold value could discriminate

Does the Central Venous Pressure Predict Fluid Responsiveness? An Updated Meta-Analysis and a Plea for Some Common Sense*

Data Synthesis: Overall 57% ± 13% of patients were fluid responders. The summary AUC was 0.56 (95% CI, 0.54–0.58) **Conclusions:** There are no data to support the widespread practice of using central venous pressure to guide fluid therapy. This approach to fluid resuscitation should be abandoned. (*Crit Care Med* 2013; 41:1774–1781) N=2015, 20 studies



Conclusion

Dynamic parameters should be used preferentially to static parameters to predict fluid responsiveness in ICU patients.

Correlation PAOP and CO , AUC 0.63

Osman D. Crit Care Med. 2007

Dynamic Hemodynamic Monitor

A. mechanical ventilation (MV) induced cyclic variation

- SPV
- PPV
- SVV
- Aortic blood flow

B. hemodynamic parameters based on MV

- Vena Cava Diameter
- Ventricular-pre-ejection period

C. hemodynamic parameters based on preload redistribution manoeuvres

- Passive leg raising
- Valsalva manoeuvre

HOW DO I CHOOSE THE APPROPRIATE HAEMODYNAMIC MONITORING?

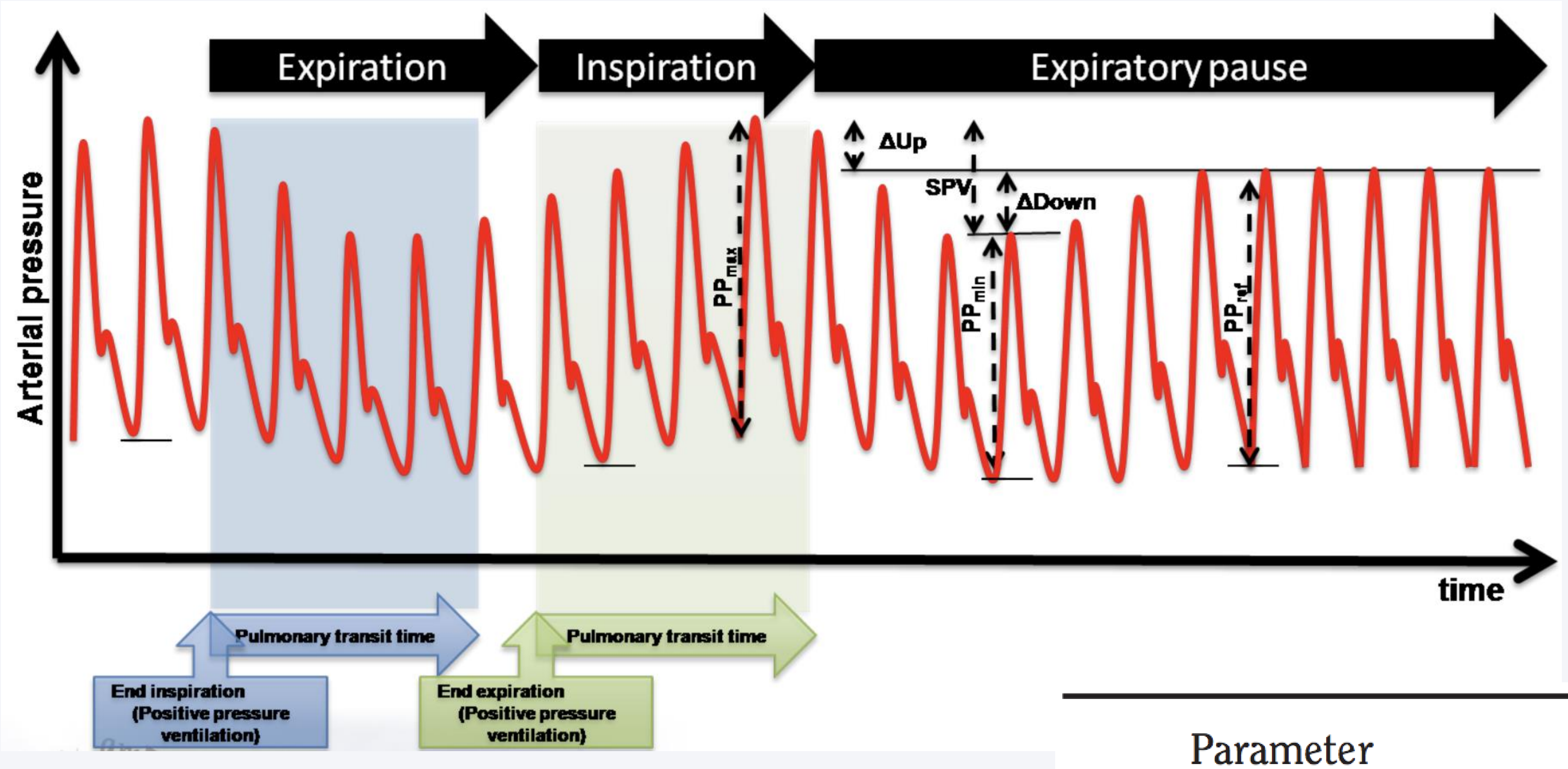
- Haemodynamic monitoring per se has no favourable impact on outcome.
- **Only the interventions based on haemodynamic data will impact outcome.**

Decision matrix for intraoperative hemodynamic monitoring

Patient's Individual Risk	Surgical Risk: Low	Surgical Risk: Intermediate	Surgical Risk: High
High	**Basic monitoring** Minimal-invasive, cont. AP, SV/CO + PPV/SVV, PAC	**Basic monitoring** Minimal-invasive, cont. AP, SV/CO + PPV/SVV, PAC/TEE	**Basic monitoring** CVC, minimal-invasive, cont. AP, SV/CO + PPV/SVV, PAC/TEE
Intermediate	**Basic monitoring** Non-invasive, cont. arterial pressure (AP), SV/CO + PPV/SVV	**Basic monitoring** Minimal-invasive, cont. AP, SV/CO + PPV/SVV	**Basic monitoring** CVC, minimal-invasive, cont. AP, SV/CO + PPV/SVV
Low	**Basic monitoring** Non-invasive, intermittent arterial pressure (AP)	**Basic monitoring** Non-invasive, cont. arterial AP, SV/CO + PPV/SVV	**Basic monitoring** CVC, minimal-invasive, cont. AP, SV/CO + PPV/SVV

J Renner et al. Best Practice & Research Clinical Anaesthesiology. 2016;30:201-216

Pulse pressure, Stroke volume Variation



predict volume responsiveness

	Correlation (r)	AUC
PPV	.78 (.74–.82)	0.94 (0.93–0.95)
SPV	.72 (.65–.77)	0.86 (0.82–0.90)
SVV	.72 (.66–.78)	0.84 (0.78–0.88)
LVEDAI	—	0.64 (0.53–0.74)
GEDVI	—	0.56 (0.37–0.67)
CVP	.13 (−.01–.28)	0.55 (0.48–0.62)

PPV (%) = (PPmax −PPmin)/ (Ppmax+Ppmin)/2

$$SVV = \frac{SV\ max - SV\ min}{SV\ mean}$$

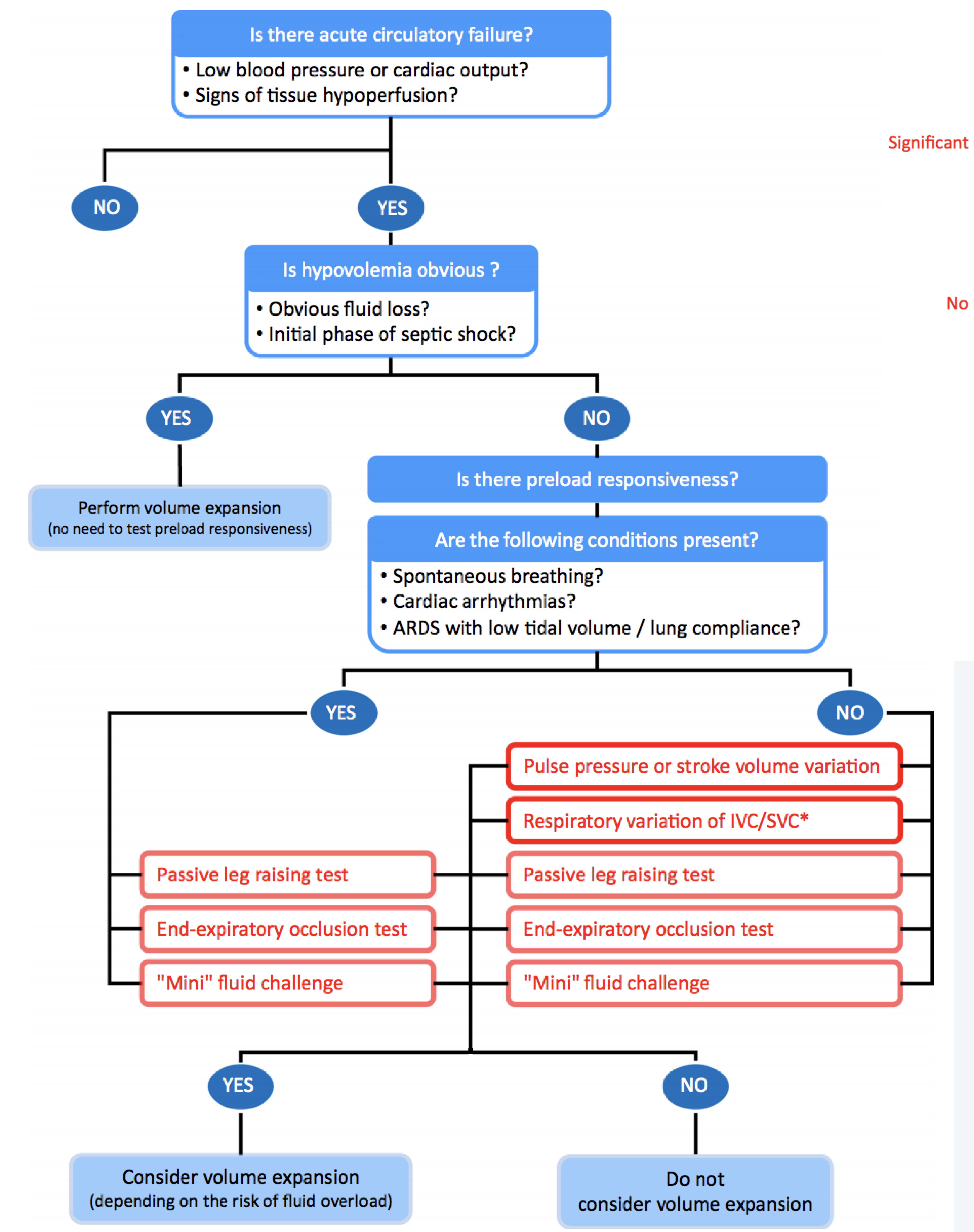
Parameter	PPV (n = 14)	SVV (n = 5)
ROC area	0.94 (0.92–0.96)	0.84 (0.81–0.87)
Sensitivity	0.89 (0.82–0.94)	0.82 (0.75–0.98)
Specificity	0.88 (0.81–0.92)	0.86 (0.77–0.92)
Positive likelihood ratio	7.26 (4.46–11.80)	5.77 (3.43–9.72)
Negative likelihood ratio	0.12 (0.07–0.21)	0.21 (0.15–0.30)
Diagnostic odds ratio	59.86 (23.88–150.05)	27.34 (13.46–55.53)

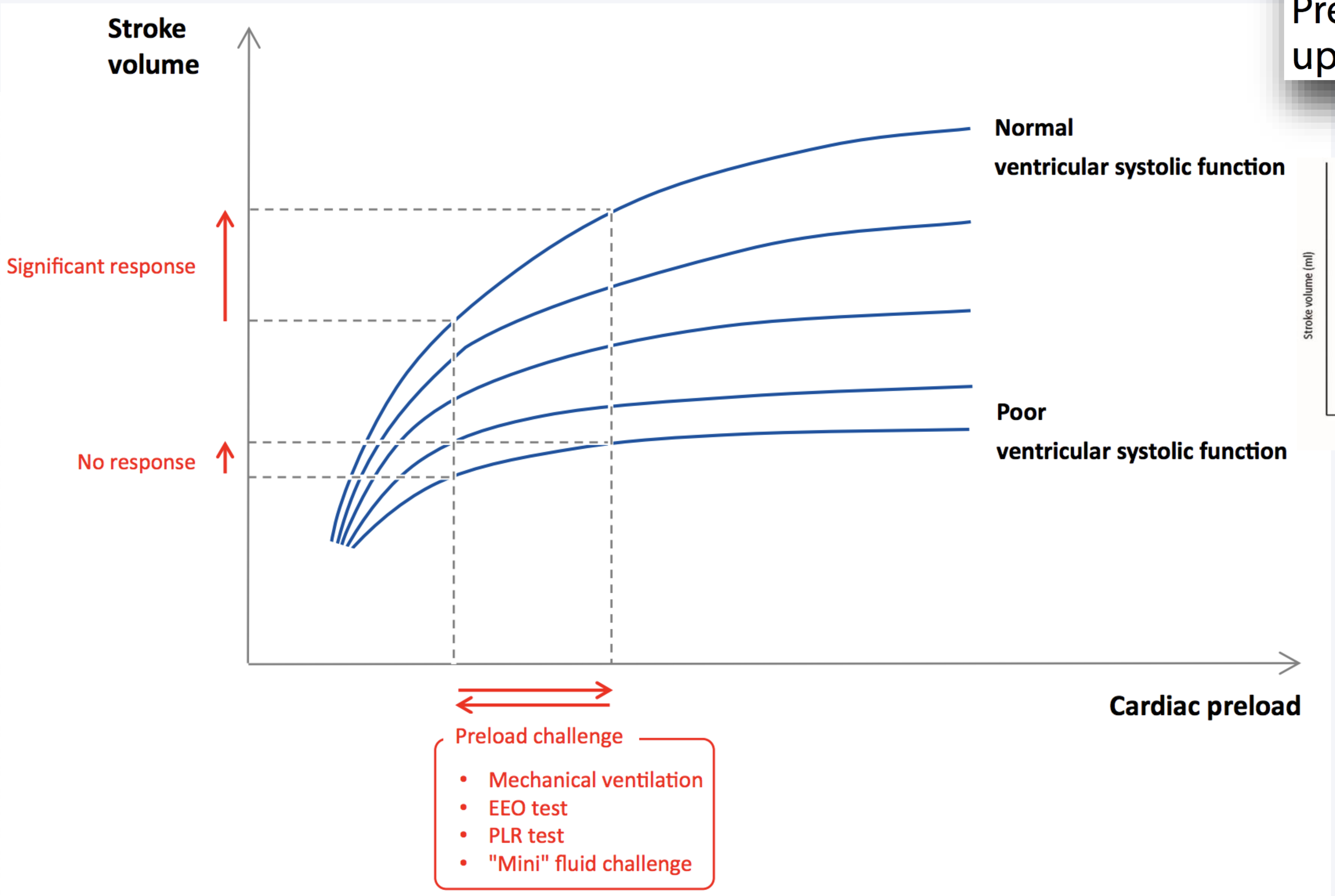
Crit Care Med 2009 Vol. 37, No. 9

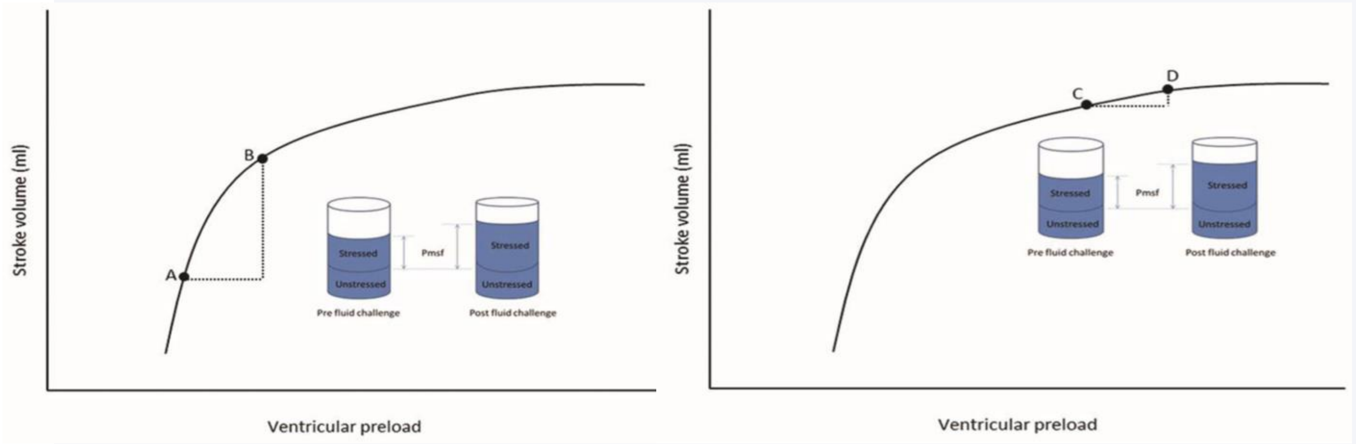
REVIEW Open Access

Prediction of fluid responsiveness: an update

Ann Intensive Care. 2016; 6: 111.







PPV, SVV limitation

Spontaneous breathing	False+
Cardiac arrhythmias	False+
Low Vt/low lung compliance	False−
Open chest	False−
Increased intra-abdominal pressure	False+
Very high respiratory rate (HR/RR < 3.6)	False−
Right heart failure*	False+

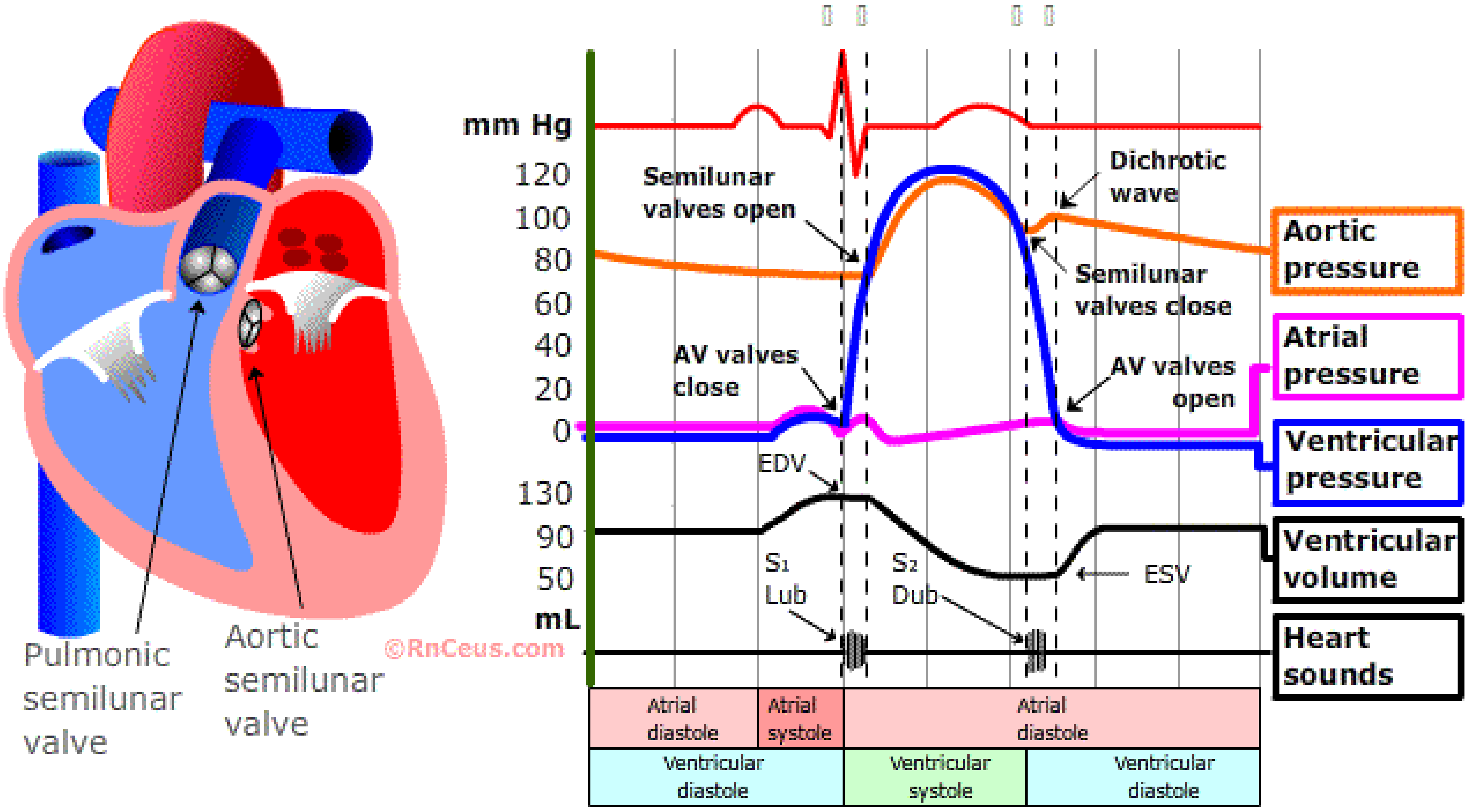
Table 1 Summary of methods predicting preload responsiveness with diagnostic threshold and limitations

Method	Threshold	Main limitations
Pulse pressure/stroke volume variations [22]	12%	Cannot be used in case of spontaneous breathing, cardiac arrhythmias, low tidal volume/ lung compliance
Inferior vena cava diameter variations [44]	12%	Cannot be used in case of spontaneous breathing, low tidal volume/lung compliance
Superior vena caval diameter variations [44]	36%*	Requires performing transesophageal Doppler Cannot be used in case of spontaneous breathing, low tidal volume/lung compliance
Passive leg raising [55]	10%	Requires a direct measurement of cardiac output
End-expiratory occlusion test [75]	5%	Cannot be used in non-intubated patients Cannot be used in patients who interrupt a 15-s respiratory hold
"Mini"-fluid challenge (100 mL) [84]	6%**	Requires a precise technique for measuring cardiac output
"Conventional" fluid challenge (500 mL) [81]	15%	Requires a direct measurement of cardiac output Induces fluid overload if repeated

* Thresholds from 12 to 40% have been reported

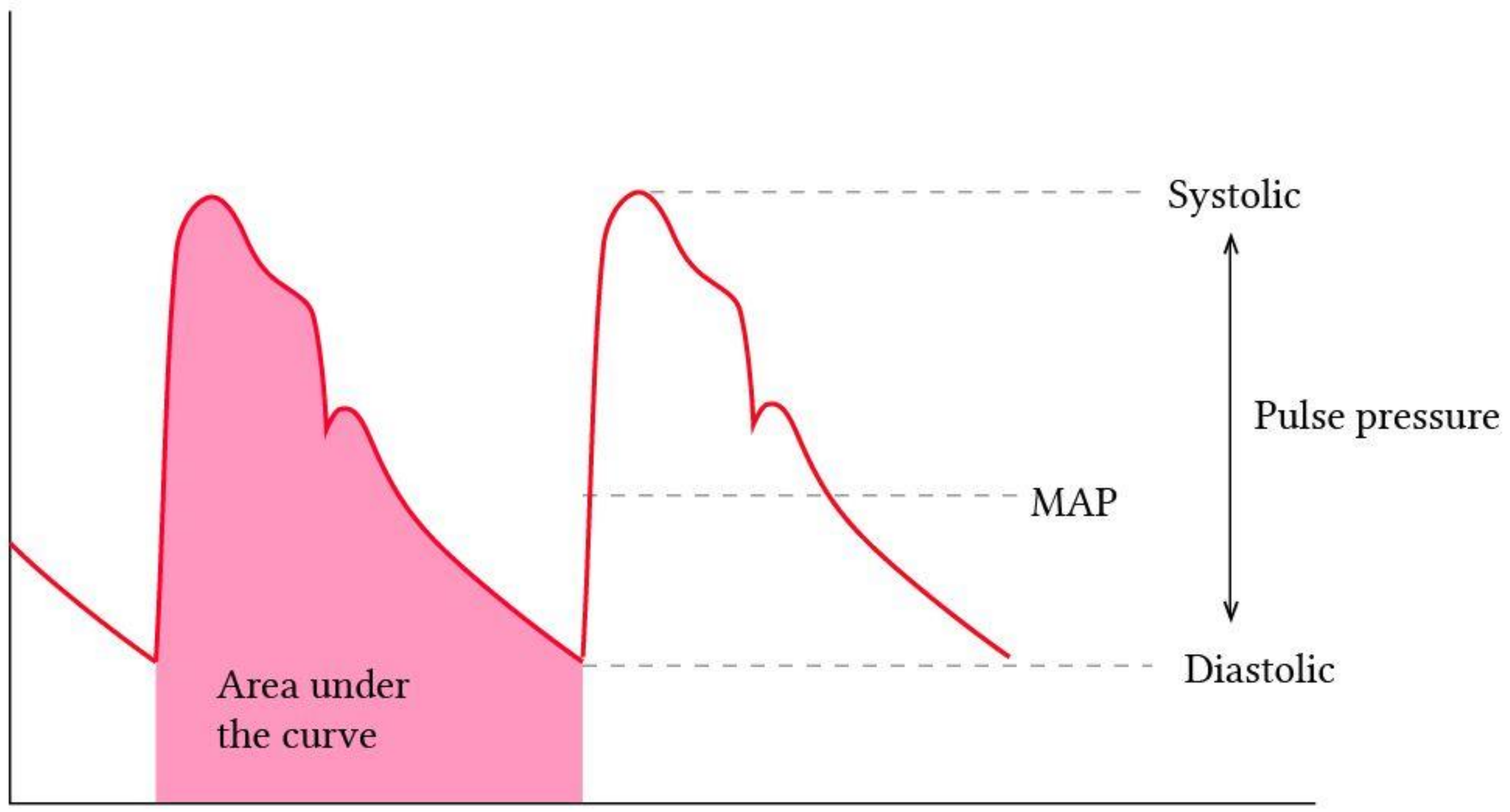
** 10% is more compatible with echography precision. Citations indicate the most important reference regarding the test

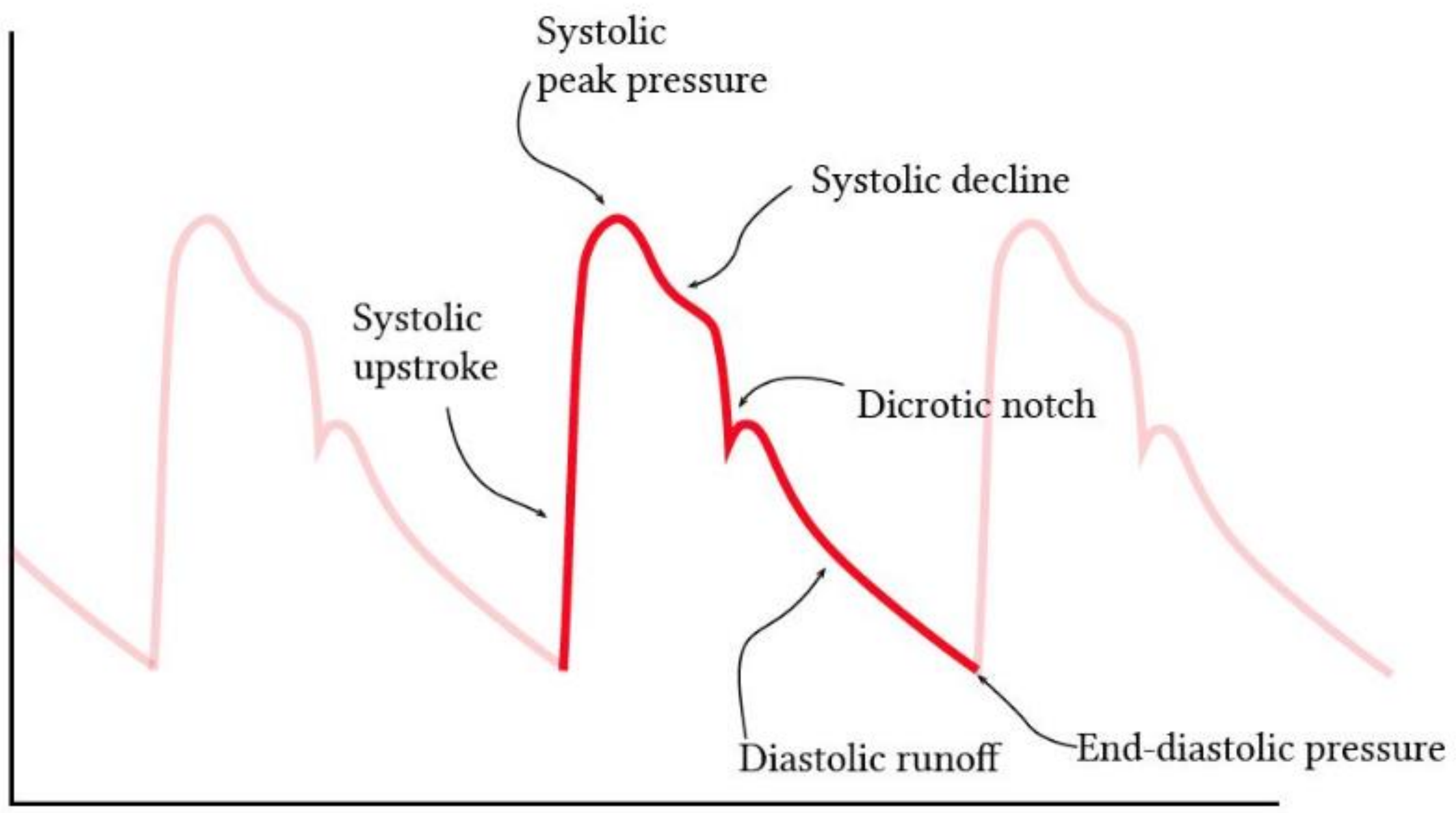
Pulse pressure Analysis



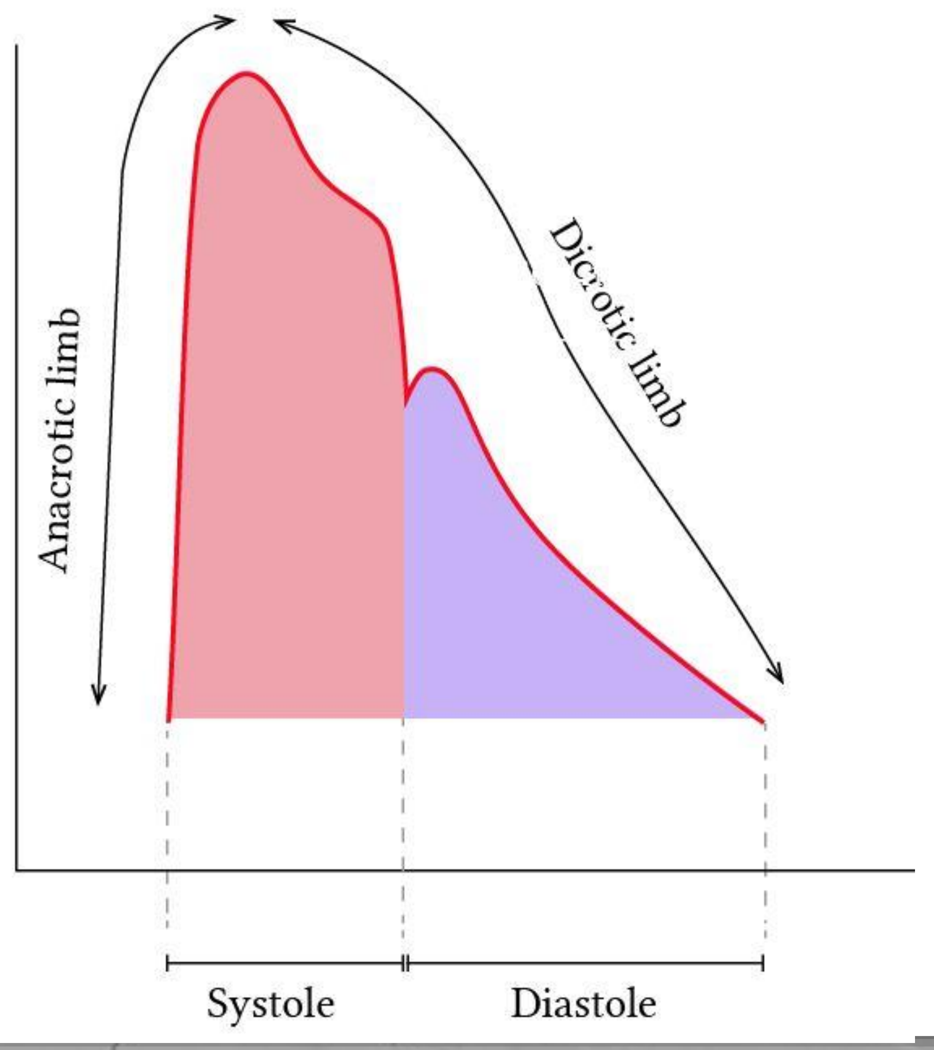
Cardiac Cycle

Arterial Wave form

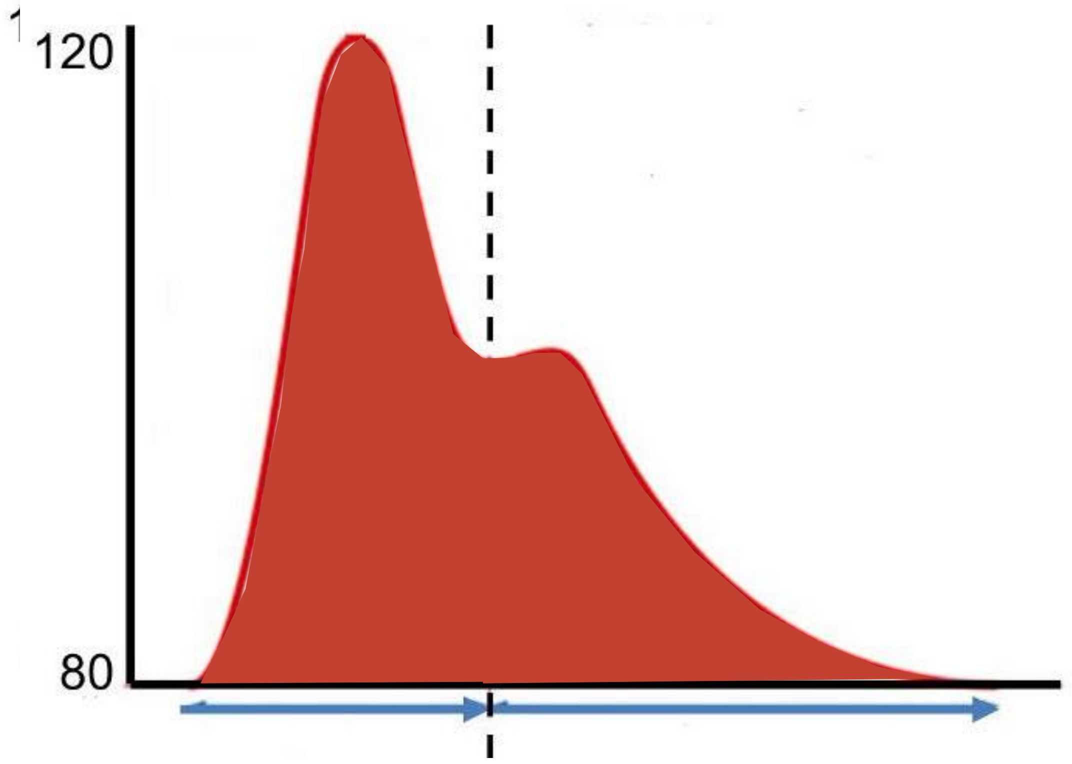




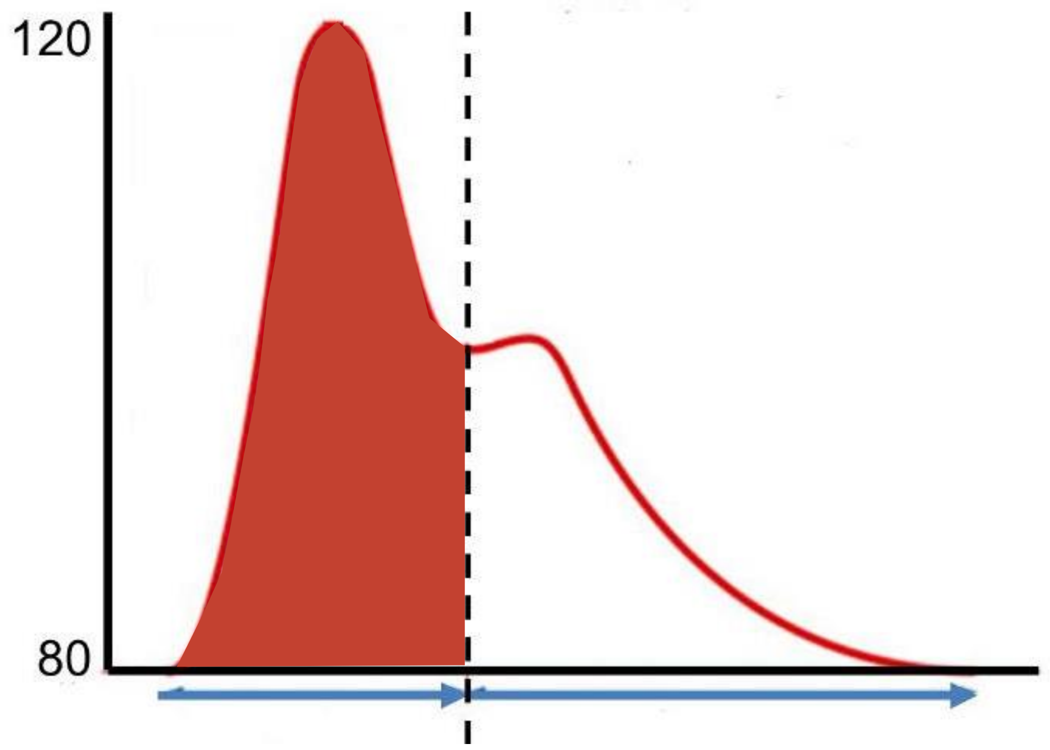
- Systolic upstroke
- Systolic peak pressure
- Systolic decline
- Dicrotic notch
- Diastolic runoff
- End-diastolic pressure



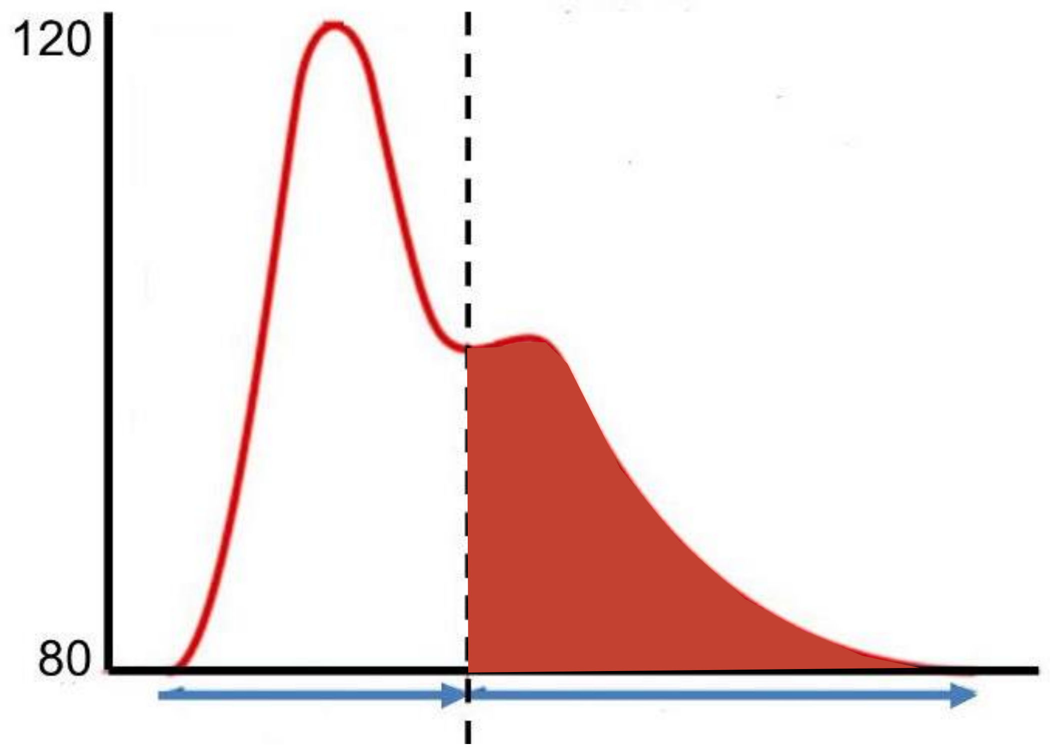
Arterial wave form



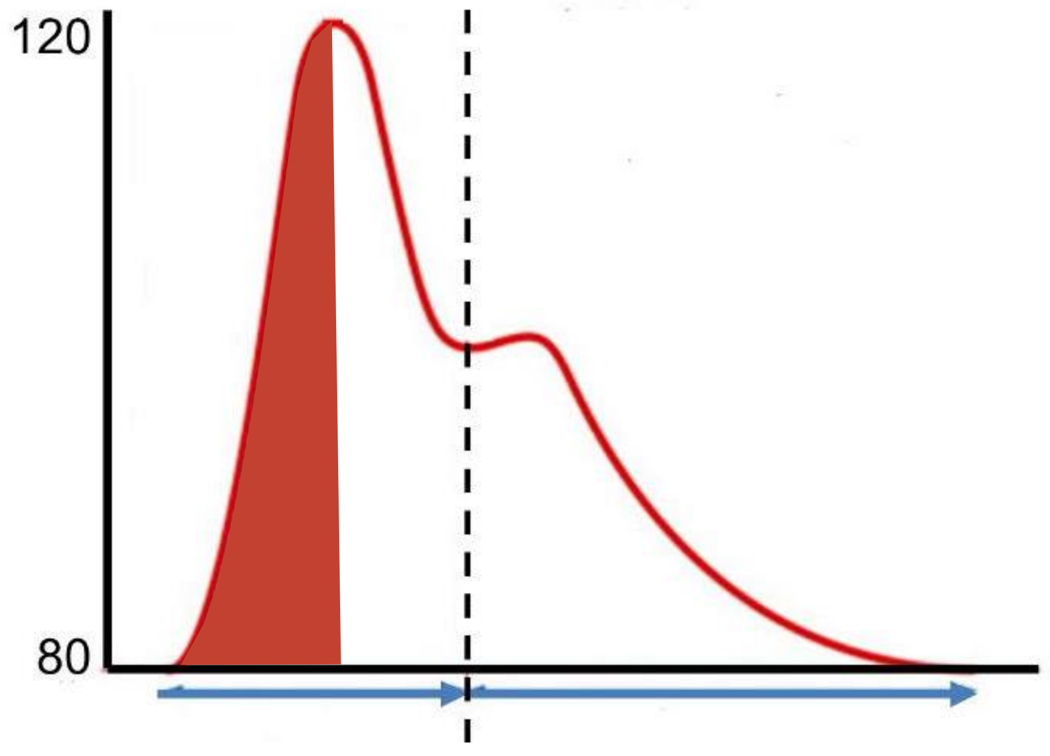
Full cardiac cycle



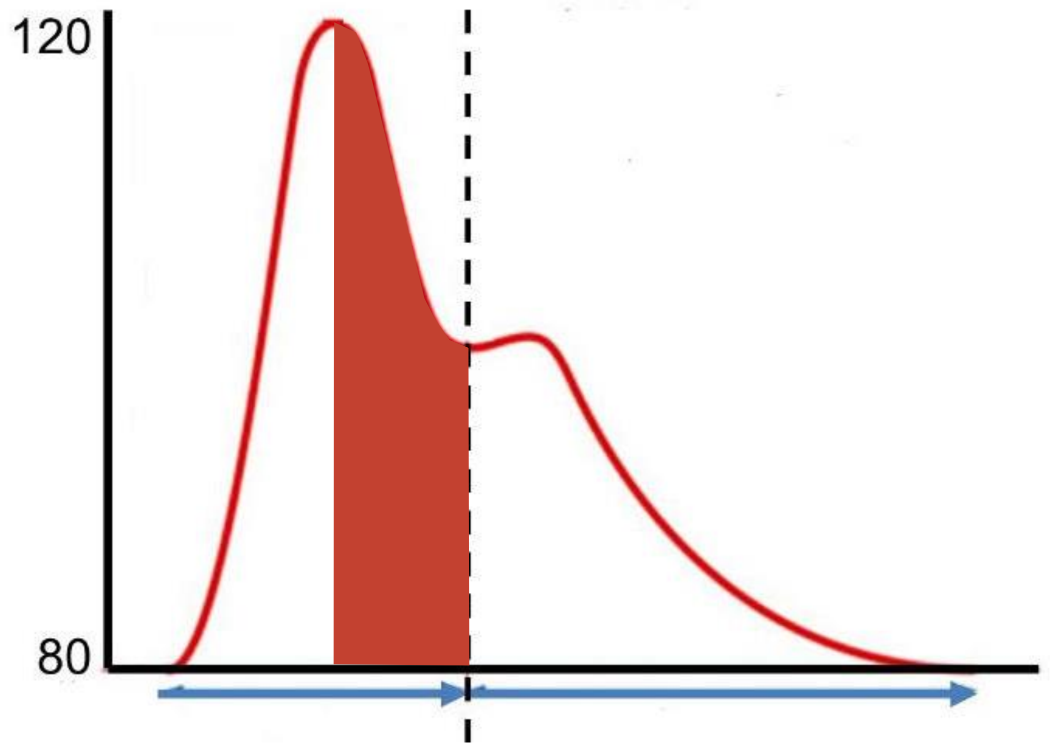
Stroke volume



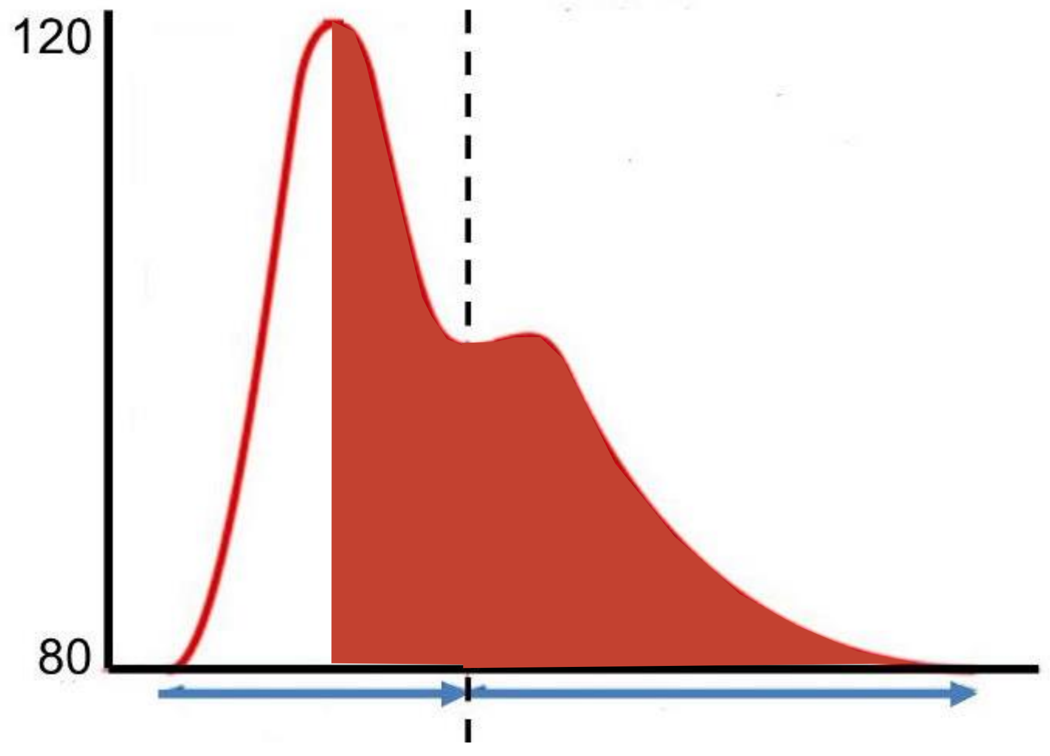
Vascular tone



Contractility

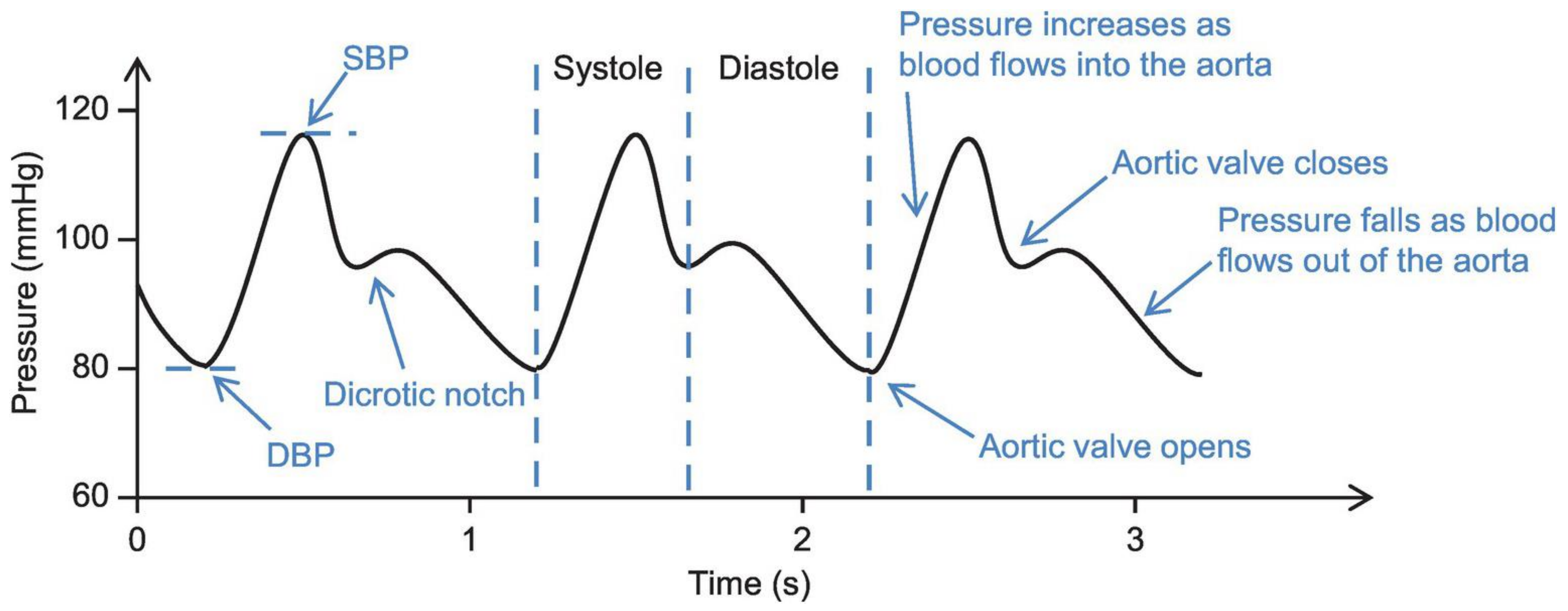


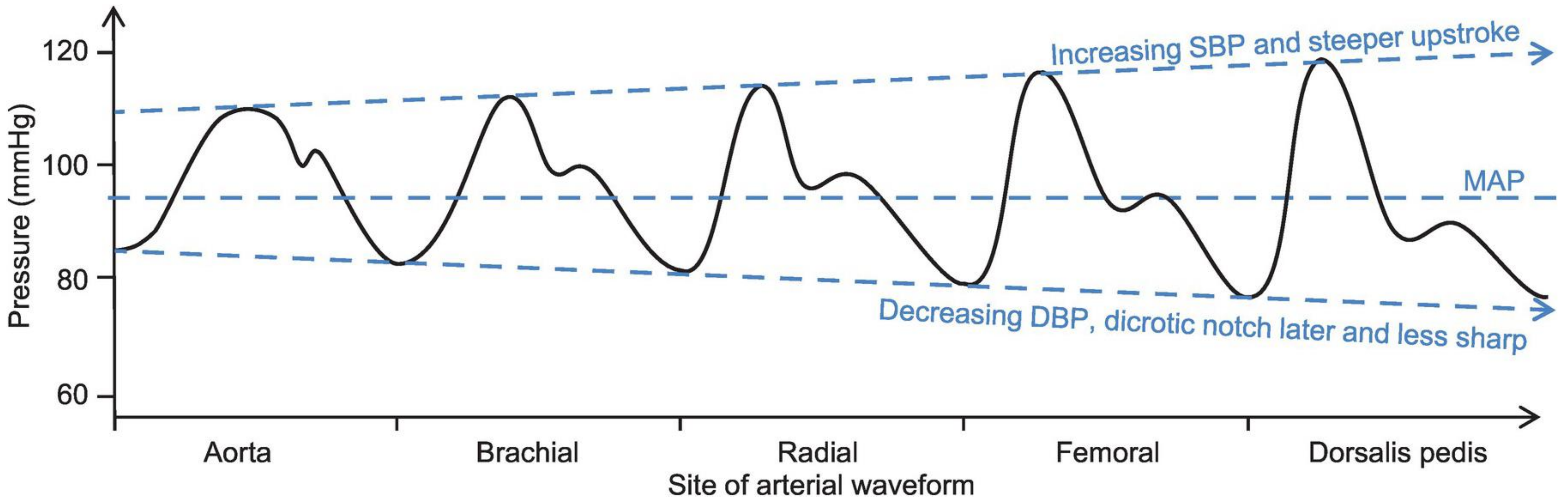
Aortic compliance

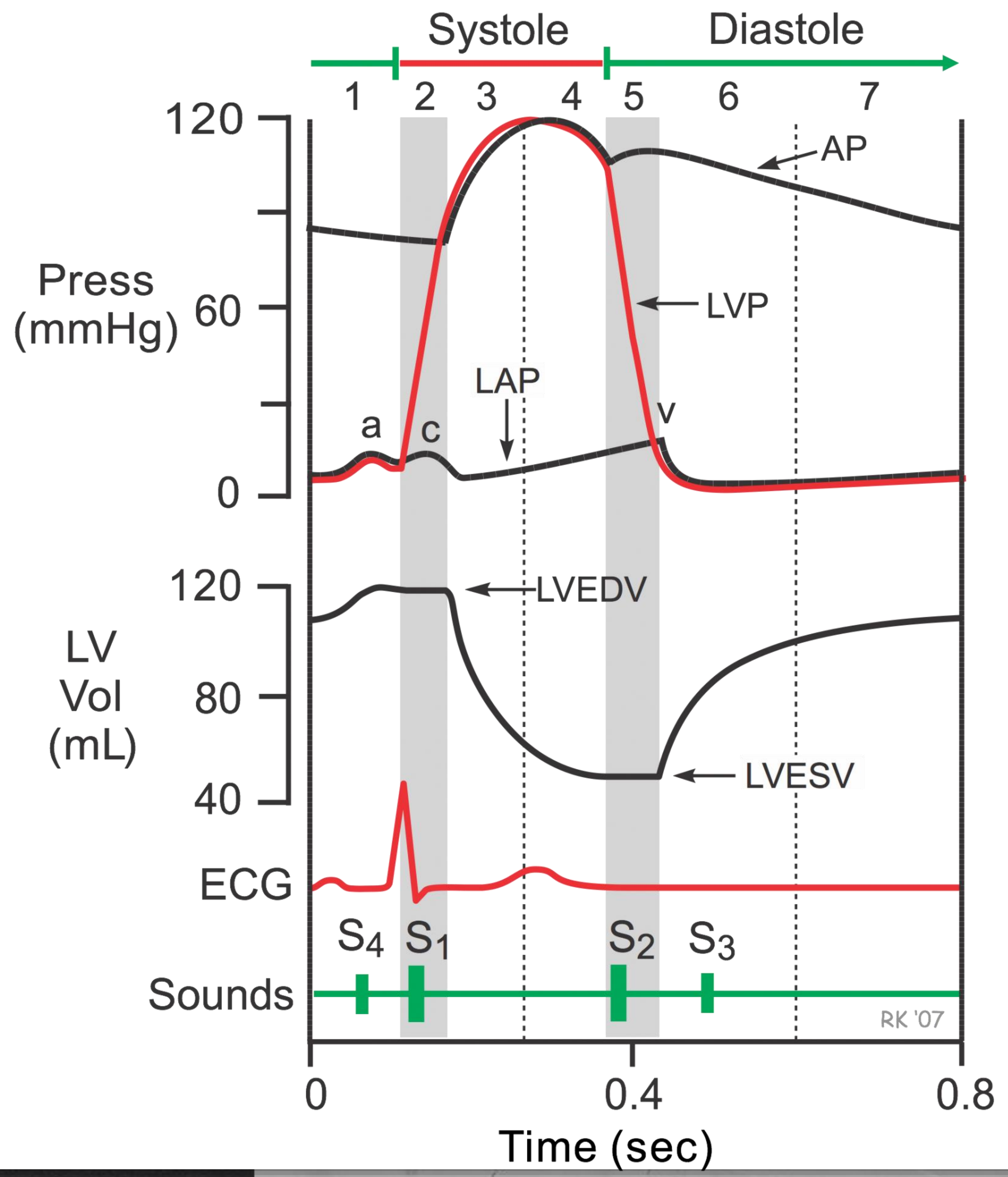


Afterload

Arterial Waveform







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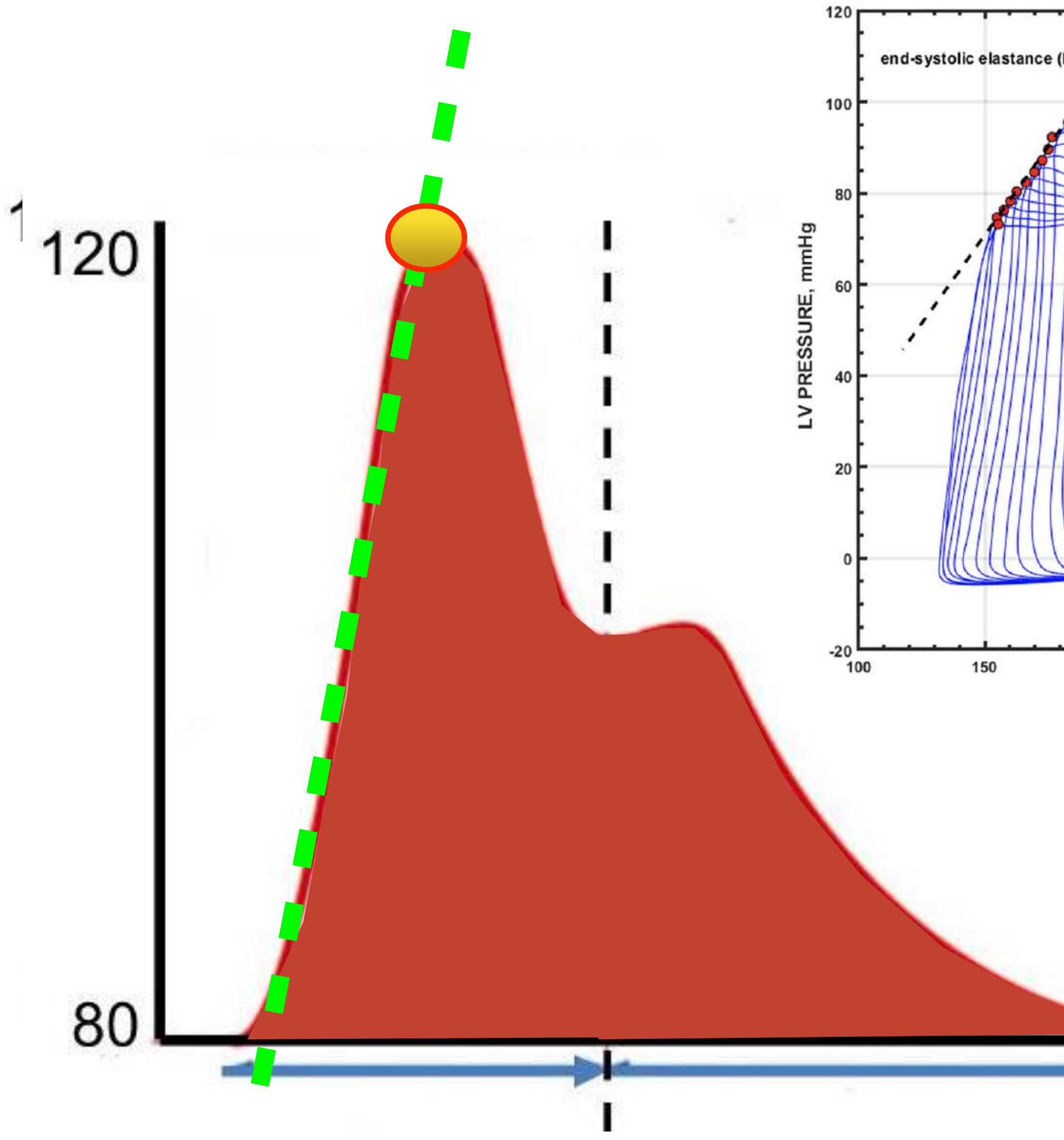
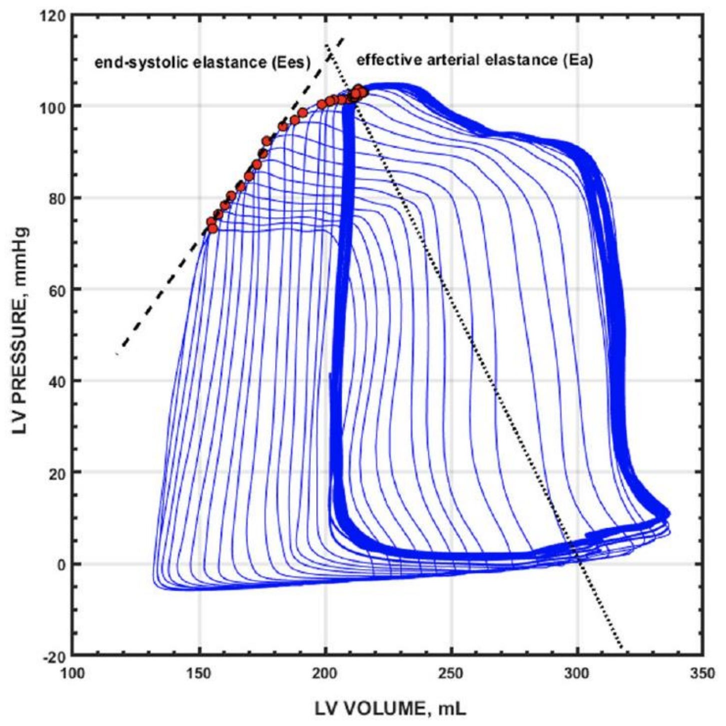
Advance Arterial wave form

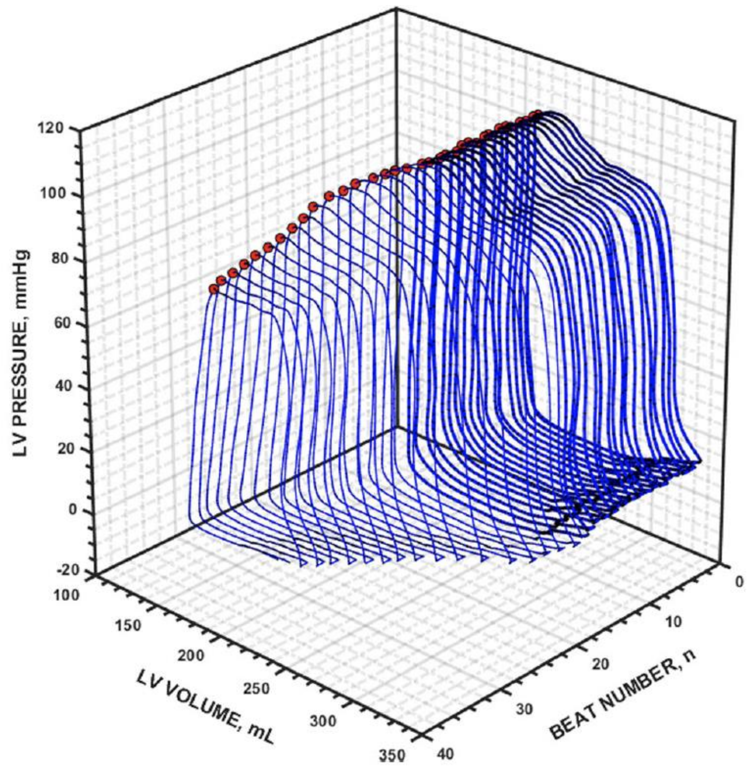
Performance comparison of ventricular and arterial dP/dt$_{max}$ for assessing left ventricular systolic function during different experimental loading and contractile conditions

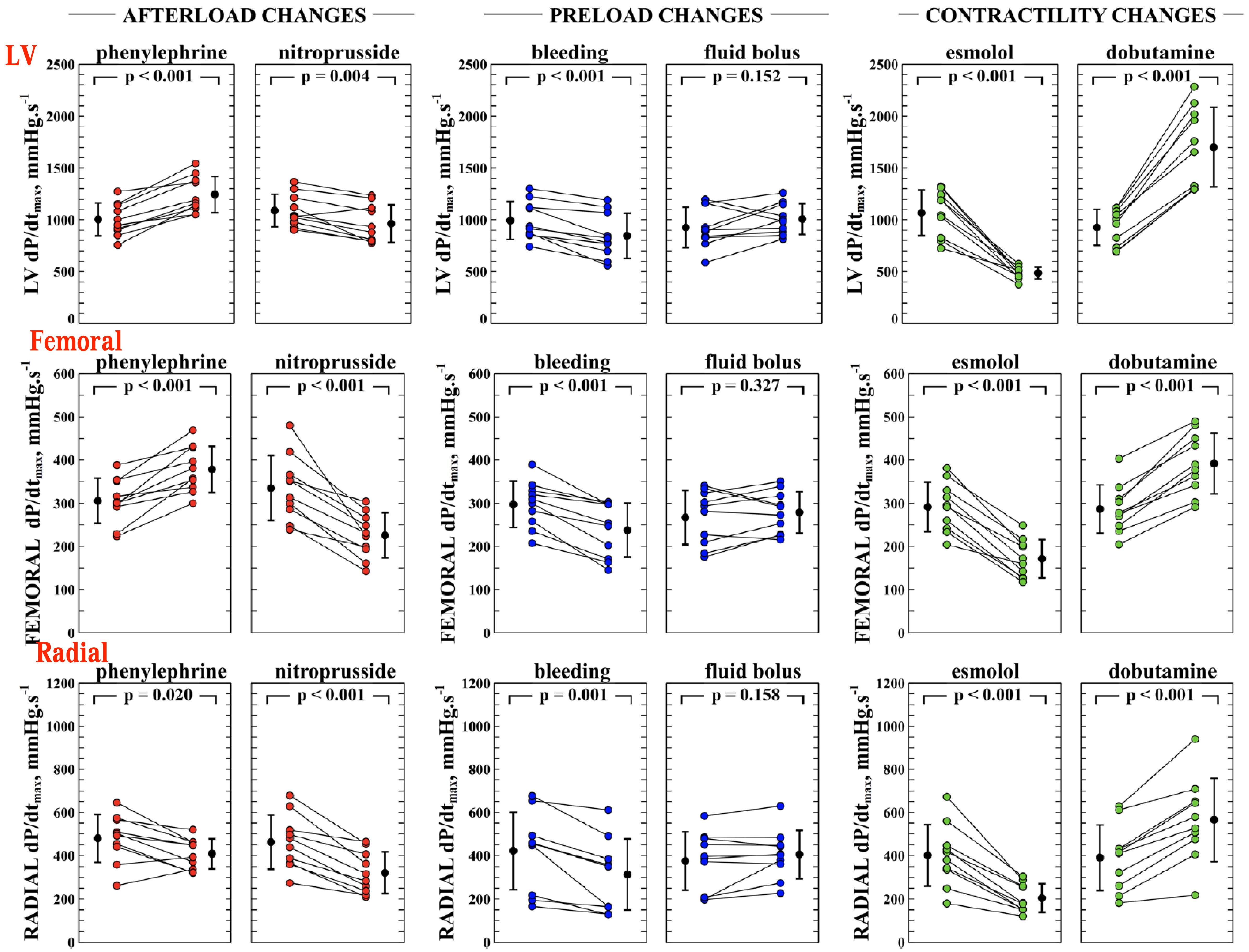
Manuel Ignacio Monge Garcia[1*], Zhongping Jian[2], Jos J. Settels[2], Charles Hunley[3], Maurizio Cecconi[4], Feras Hatib[2] and Michael R. Pinsky[5]

Critical Care (2018) 22:325









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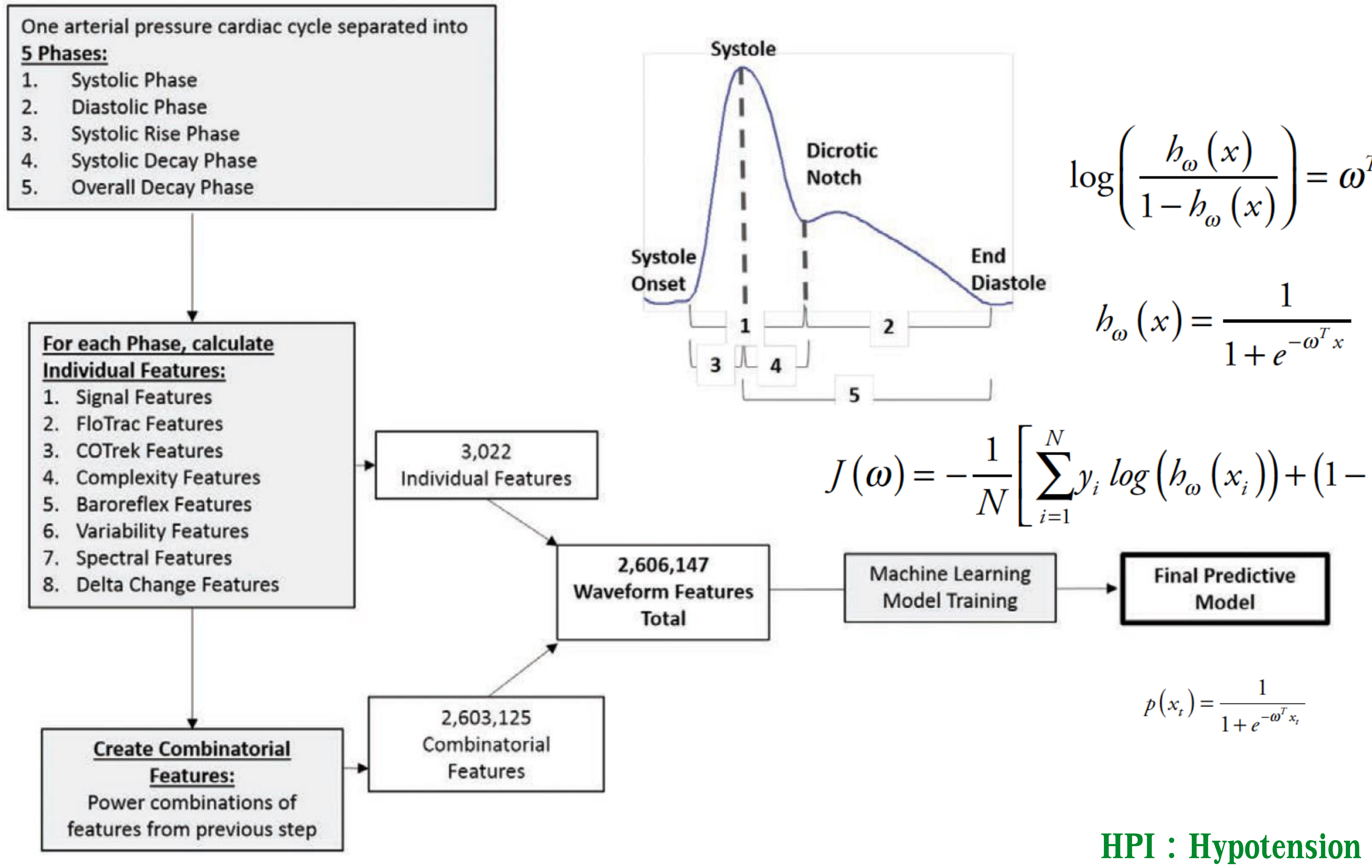
Maximal left ventricular (LV) pressure rise (LV dP/dtmax) : marker of LV systolic function

AI Analysis

Machine-learning Algorithm to Predict Hypotension Based on High-fidelity Arterial Pressure Waveform Analysis

Feras Hatib, Ph.D., Zhongping Jian, Ph.D., Sai Buddi, Ph.D., Christine Lee, M.S., Jos Settels, M.S., Karen Sibert, M.D., F.A.S.A., Joseph Rinehart, M.D., Maxime Cannesson, M.D., Ph.D.

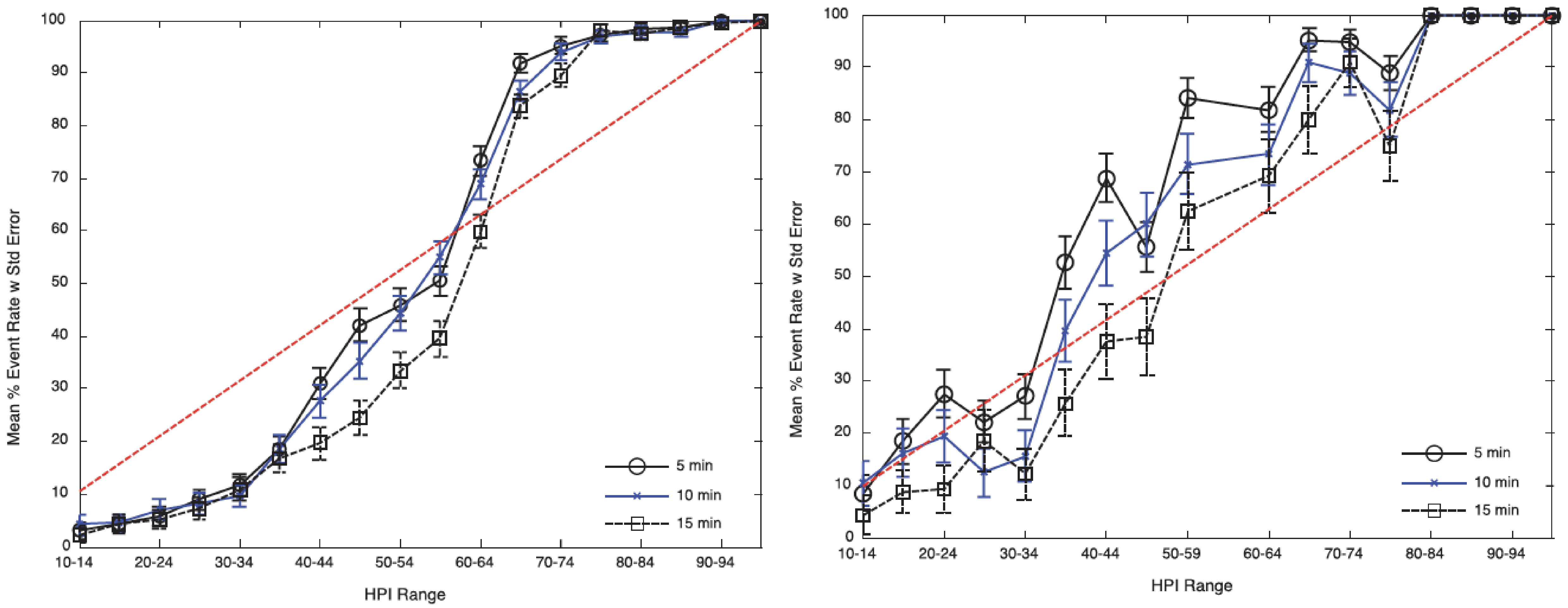


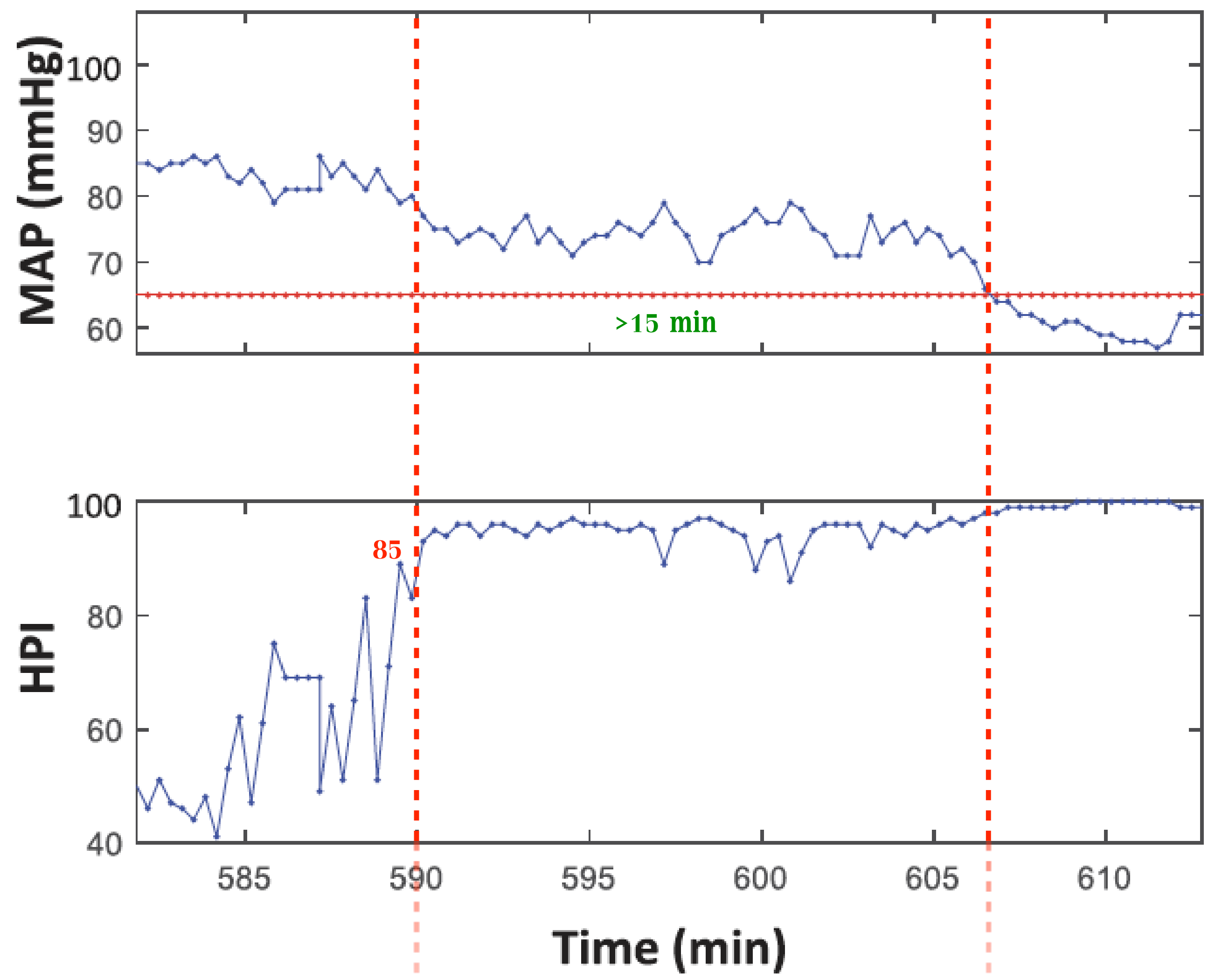
$$\log\left(\frac{h_\omega(x)}{1-h_\omega(x)}\right)=\omega^T x$$

$$h_\omega(x)=\frac{1}{1+e^{-\omega^T x}}$$

$$J(\omega)=-\frac{1}{N}\left[\sum_{i=1}^{N} y_i \log\left(h_\omega(x_i)\right)+(1-y_i)\log\left(1-h_\omega(x_i)\right)\right]$$

$$p(x_i)=\frac{1}{1+e^{-\omega^T x_i}}$$





HPI : Hypotension prediction index

Anesthesiology 2018; XXX:00–00

Preventive LCOS
Monitor Application

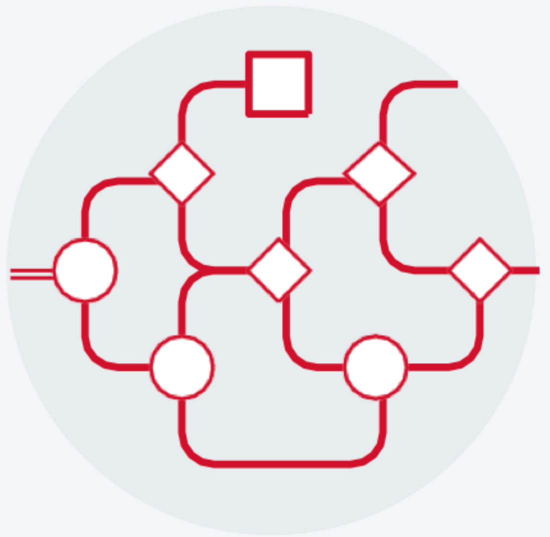
Hypotension Prediction Index software algorithm

Past Events

Machine learning based on arterial blood pressure waveform from development dataset

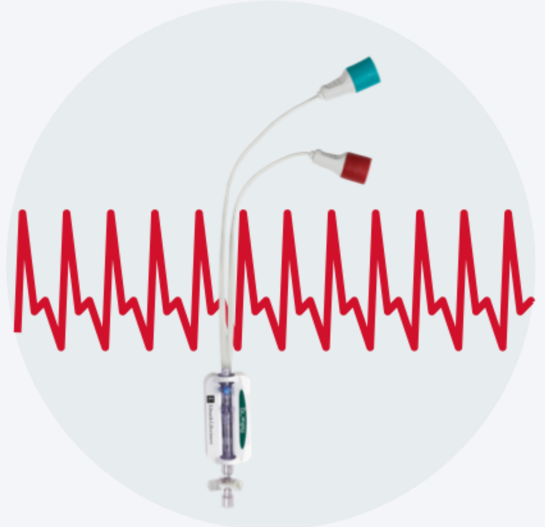
200,000 hypotensive and non-hypotensive events*

+130M cardiac cycles

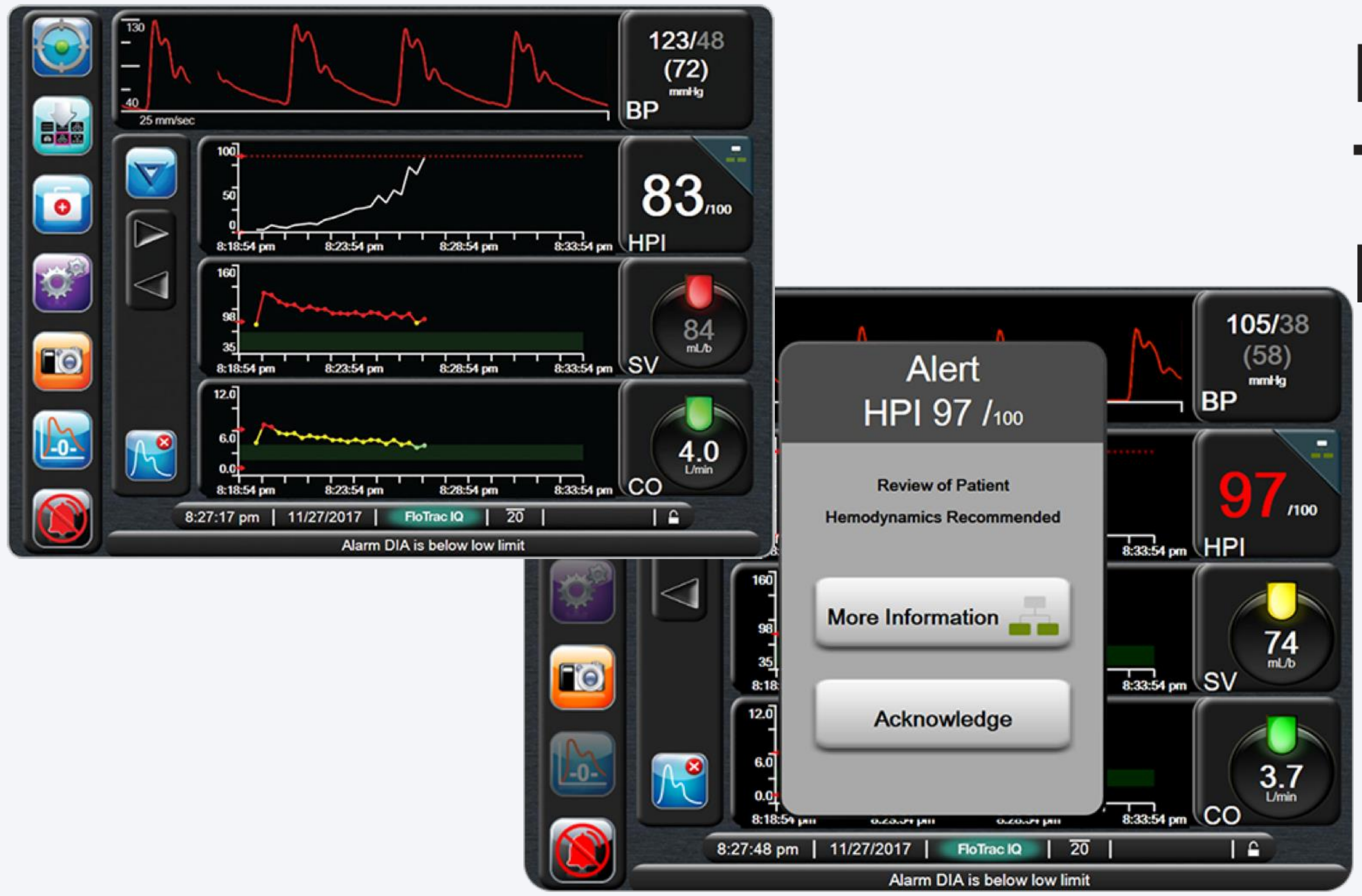


Algorithm with 23 predictive features

Real Time Input

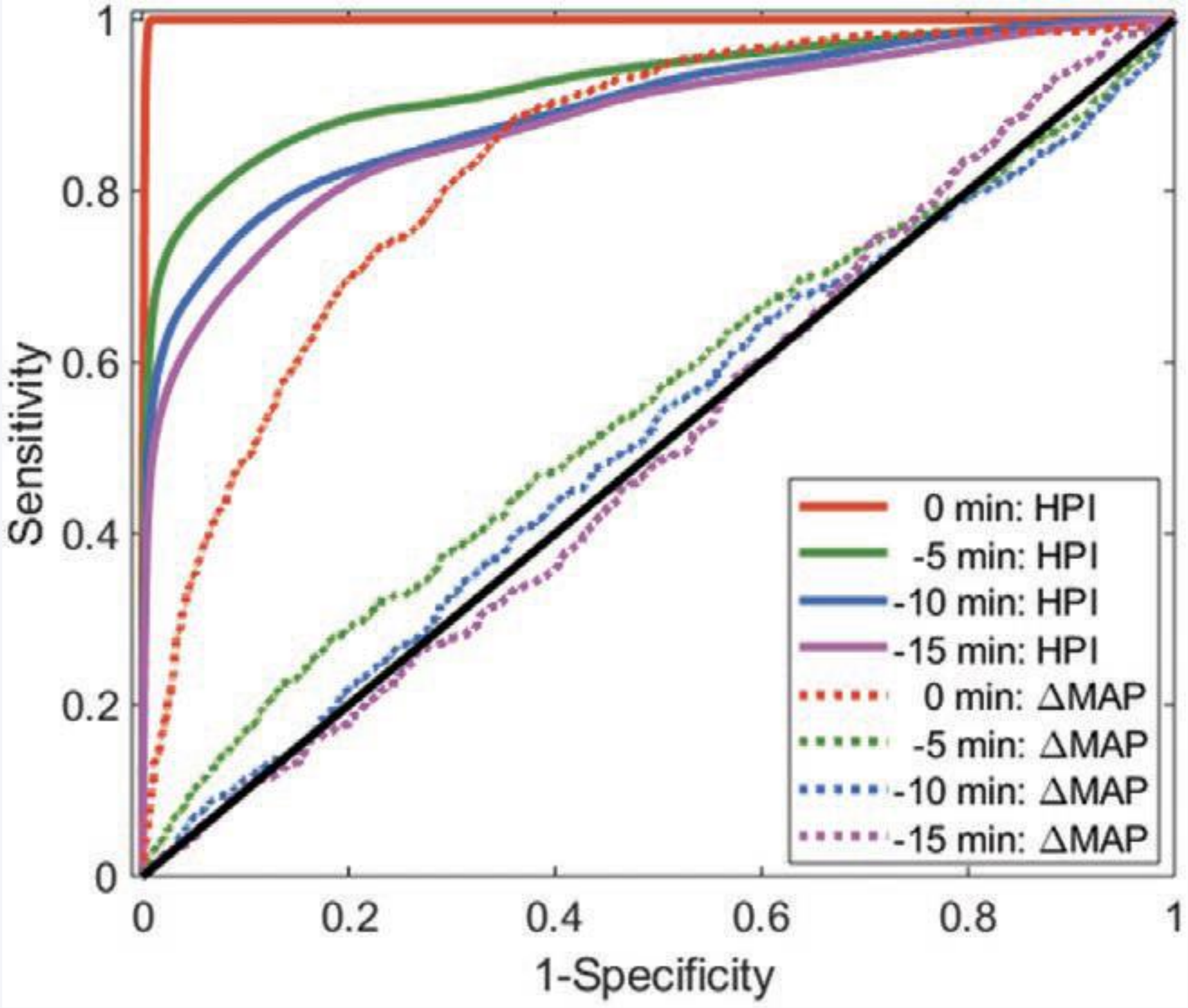


Patient waveform from FloTrac IQ sensor



Predictive Output

HPI parameter value



Ability of an Arterial Waveform Analysis–Derived Hypotension Prediction Index to Predict Future Hypotensive Events in Surgical Patients

Simon James Davies, MD, PhD,* Simon Tilma Vistisen, PhD,† Zhongping Jian, PhD,‡ Feras Hatib, PhD,‡ and Thomas W. L. Scheeren, PhD§

JAMA | Preliminary Communication | CARING FOR THE CRITICALLY ILL PATIENT February 17, 2020.

Effect of a Machine Learning–Derived Early Warning System for Intraoperative Hypotension vs Standard Care on Depth and Duration of Intraoperative Hypotension During Elective Noncardiac Surgery

Table 2. Primary and Secondary End Points

	Median (Interquartile Range)[a]			
	Intervention (n = 31)	Control (n = 29)	Median Difference (95% CI)[b]	*P* Value[c]
Primary End Point				
Time-weighted average of hypotension, mm Hg	0.10 (0.01-0.43)	0.44 (0.23-0.72)	0.38 (0.14 to 0.43)	.001
Secondary End Points				
Hypotension				
Area under the threshold, mm Hg/min[d]	20.0 (2.2-148.3)	142.2 (64.67-258.92)	74.0 (33.0 to 137.7)	.002
Incidence	3.0 (1.0-8.0)	8.0 (3.5-12.0)	4.0 (1.0 to 7.0)	.004
Total time, min	8.0 (1.3-26.0)	32.7 (11.5-59.7)	16.7 (7.7 to 31.0)	.001
Surgery time, %	2.8 (0.8-6.6)	10.3 (4.6-15.6)	5.6 (3.0 to 9.4)	<.001
Hypertension				
Time-weighted average, mm Hg	0.09 (0.00-0.21)	0.05 (0.00-0.13)	0.00 (−0.85 to 0.17)	.47
Area above the threshold, mm Hg/min[d]	33.3 (0.0-88.0)	13.3 (0.0-44.3)	−3.5 (−29.0 to 5.5)	.40
Incidence	2.0 (0.0-3.0)	1.0 (0.0-2.0)	0.0 (−1.0 to 0.0)	.23
Total time, min	4.0 (0.0-10.7)	3.0 (0.0-6.8)	−0.7 (−4.3 to 0.7)	.40
Surgery time, %	1.5 (0.0-3.3)	0.9 (0.0-1.9)	−0.2 (−1.4 to 0.3)	.40
Treatment behavior				
Reaction time, s[e]	53.0 (24.0-99.0)	87.3 (53.0-172.5)	34.3 (22.8 to 47.3)	<.001
Post Hoc End Points				
Treatment behavior				
No. of treatments per patient[f]	15.0 (5.0-29.0)	9.0 (3.5-13.0)	−6.0 (−13.0 to −1.0)	.02
Early warning system alarms				
Time-weighted average, Hypotension Prediction Index[g]	1.99 (1.12-3.17)	4.31 (2.50-5.79)	1.79 (0.74 to 2.95)	.001
Area above the threshold, Hypotension Prediction Index/min[d]	529.7 (196.3-1315.0)	1231.0 (701.5-1966.3)	629.3 (229.3 to 1012.3)	.002
Incidence	11.0 (7.0-16.0)	11.0 (8.0-14.5)	0.0 (−4.0 to 3.0)	.84
Total time, min	56.7 (21.7-122.7)	116.3 (68.3-170.3)	51.7 (20.7 to 91.0)	.002
Time, %	20.9 (14.5-35.6)	41.1 (23.9-56.4)	15.8 (5.8 to 25.9)	.002

JACS Journal of the American College of Surgeons

CARDIOTHORACIC SURGERY | VOLUME 229, ISSUE 4, SUPPLEMENT 1, S48, OCTOBER 01, 2019

Predictive Hemodynamic Monitoring in Cardiac Surgery: An Observational Validation Study of the Hypotension Prediction Index

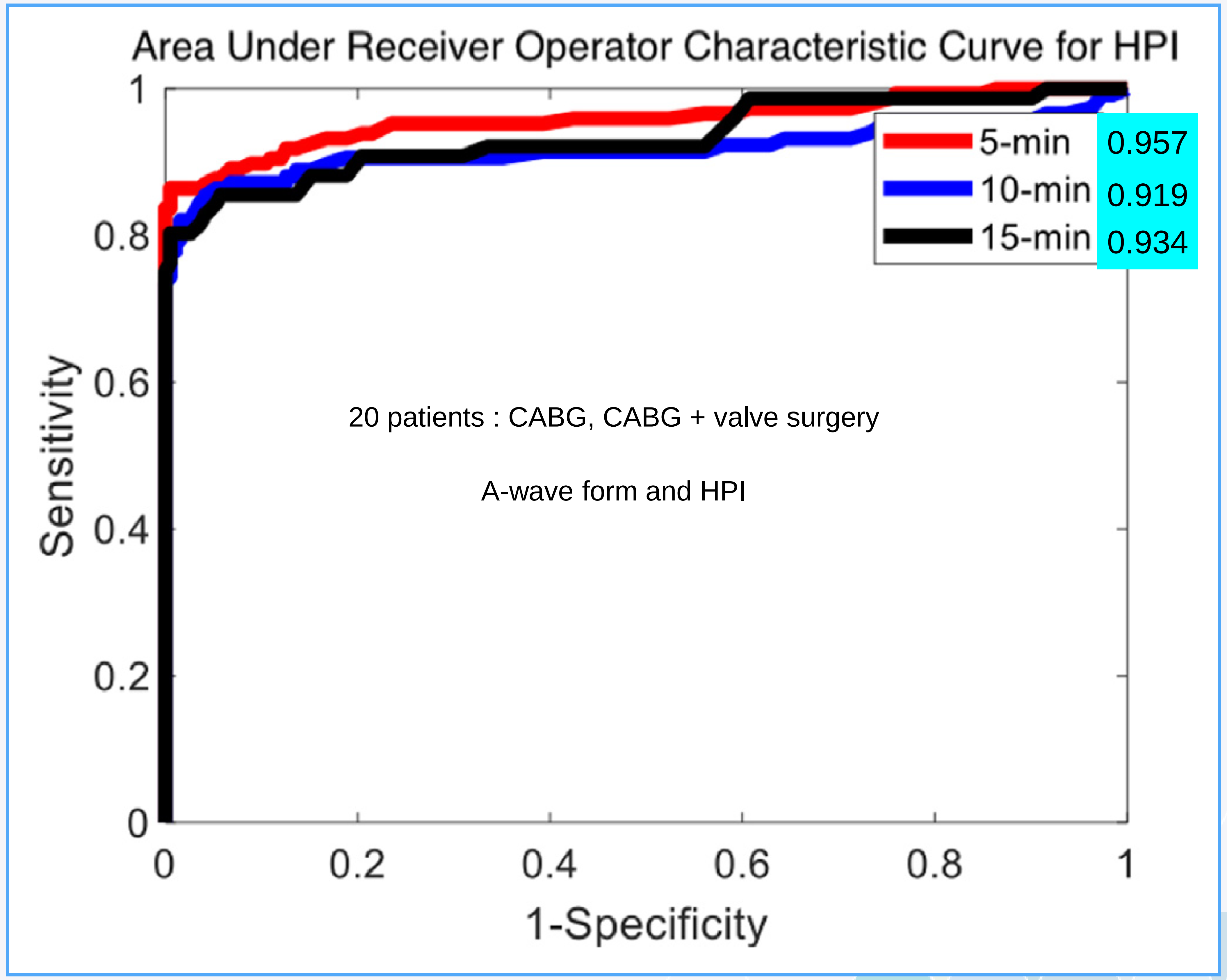
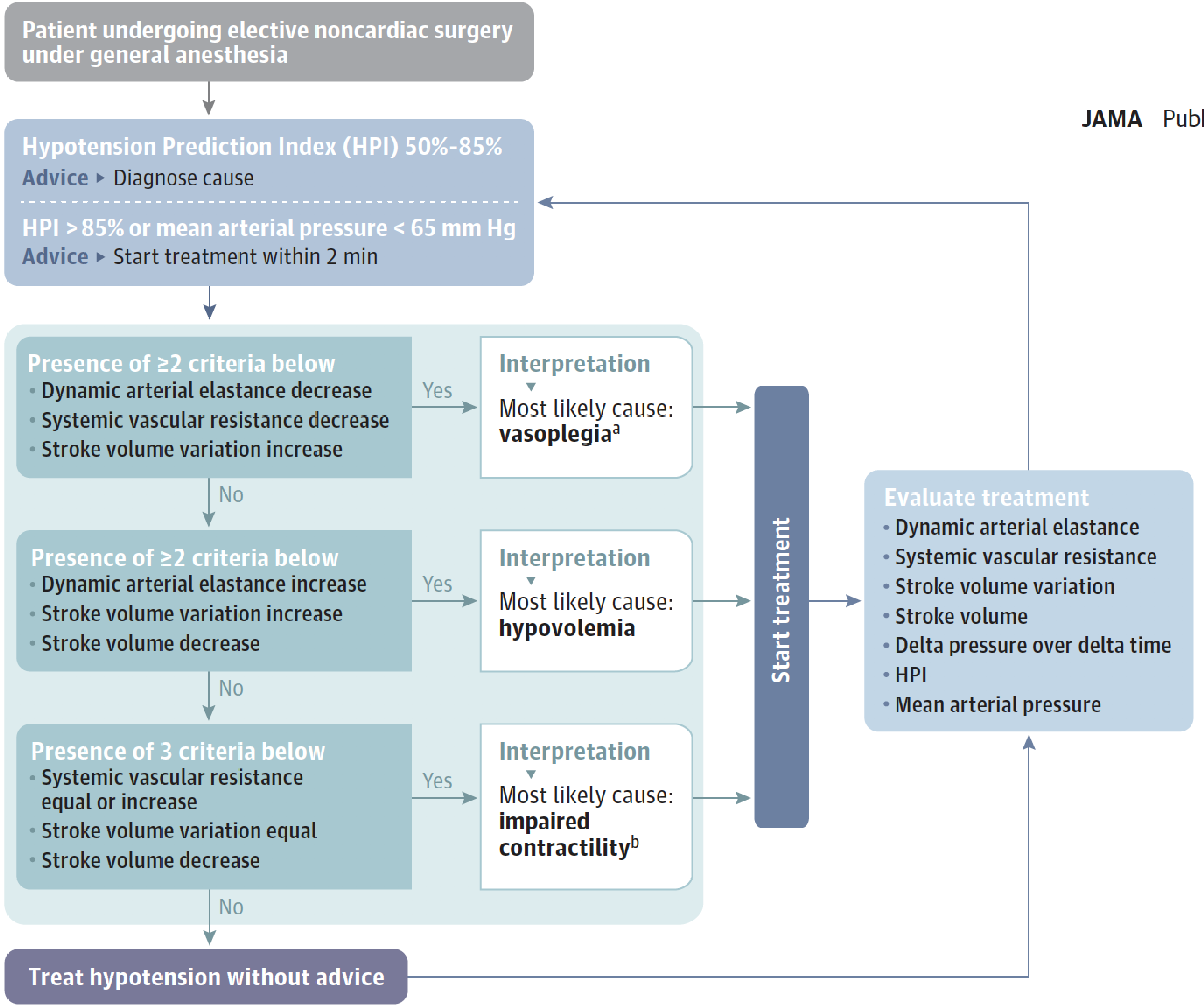


Figure 2. Hemodynamic Diagnostic Guidance and Treatment Protocol



JAMA Published online February 17, 2020

[a] Vasoplegia indicates decreased systemic vascular resistance.

[b] Impaired left ventricular contractility.

Treatment LCOS

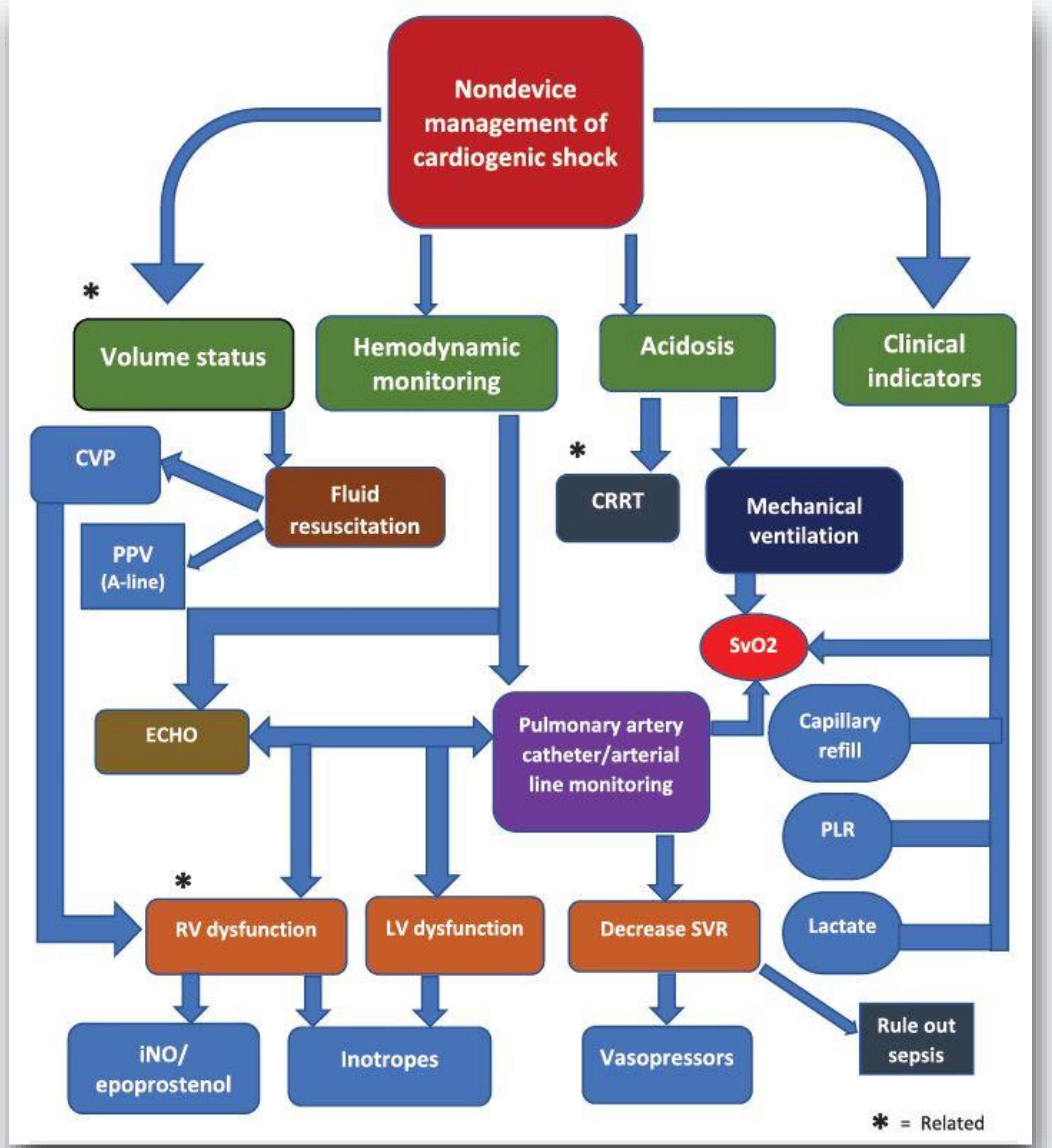
Initial Management

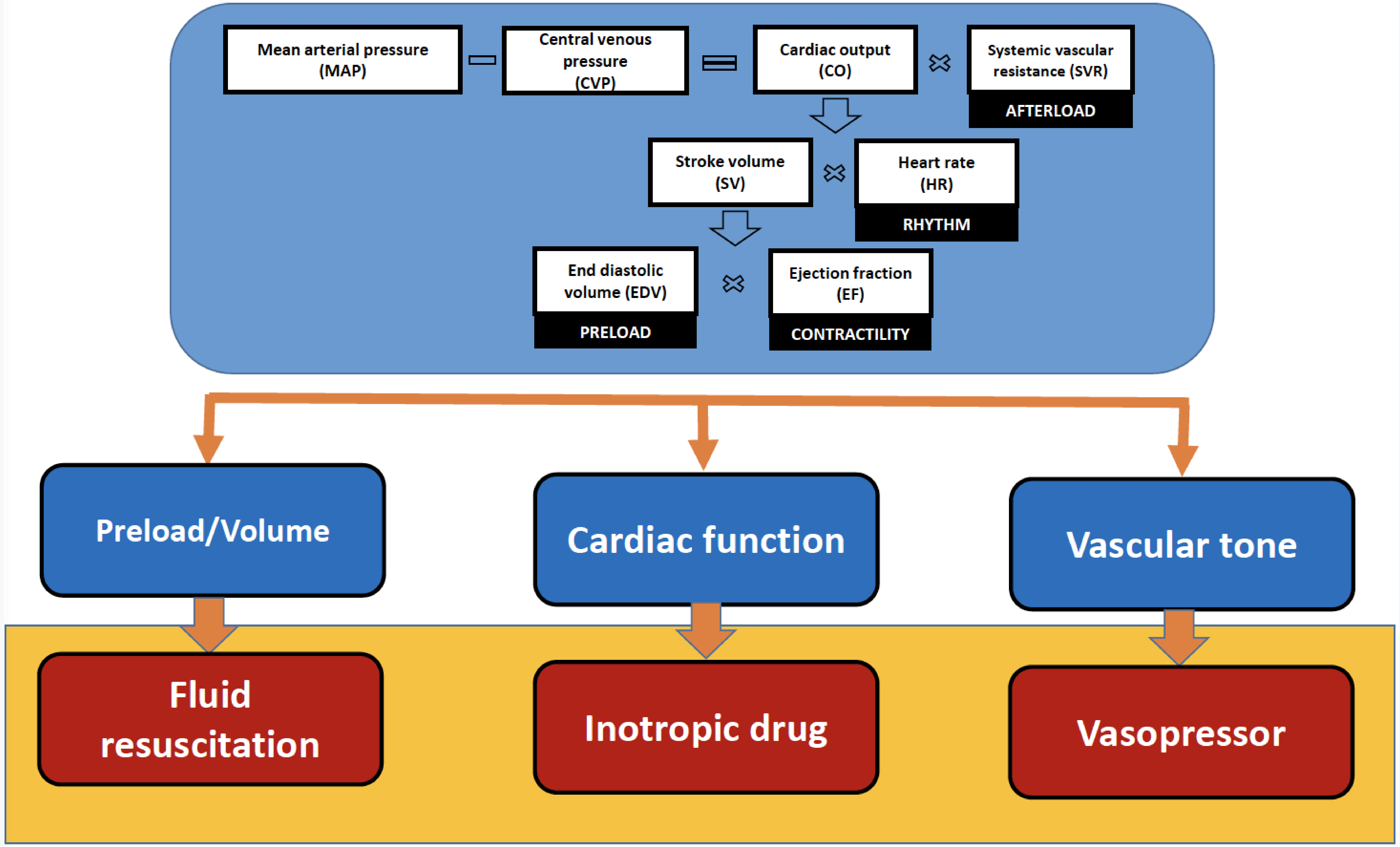
Identify correctable causes

- Graft dysfunction
- Valvular incompetence
- Pericardial tamponade
- Pneumothorax

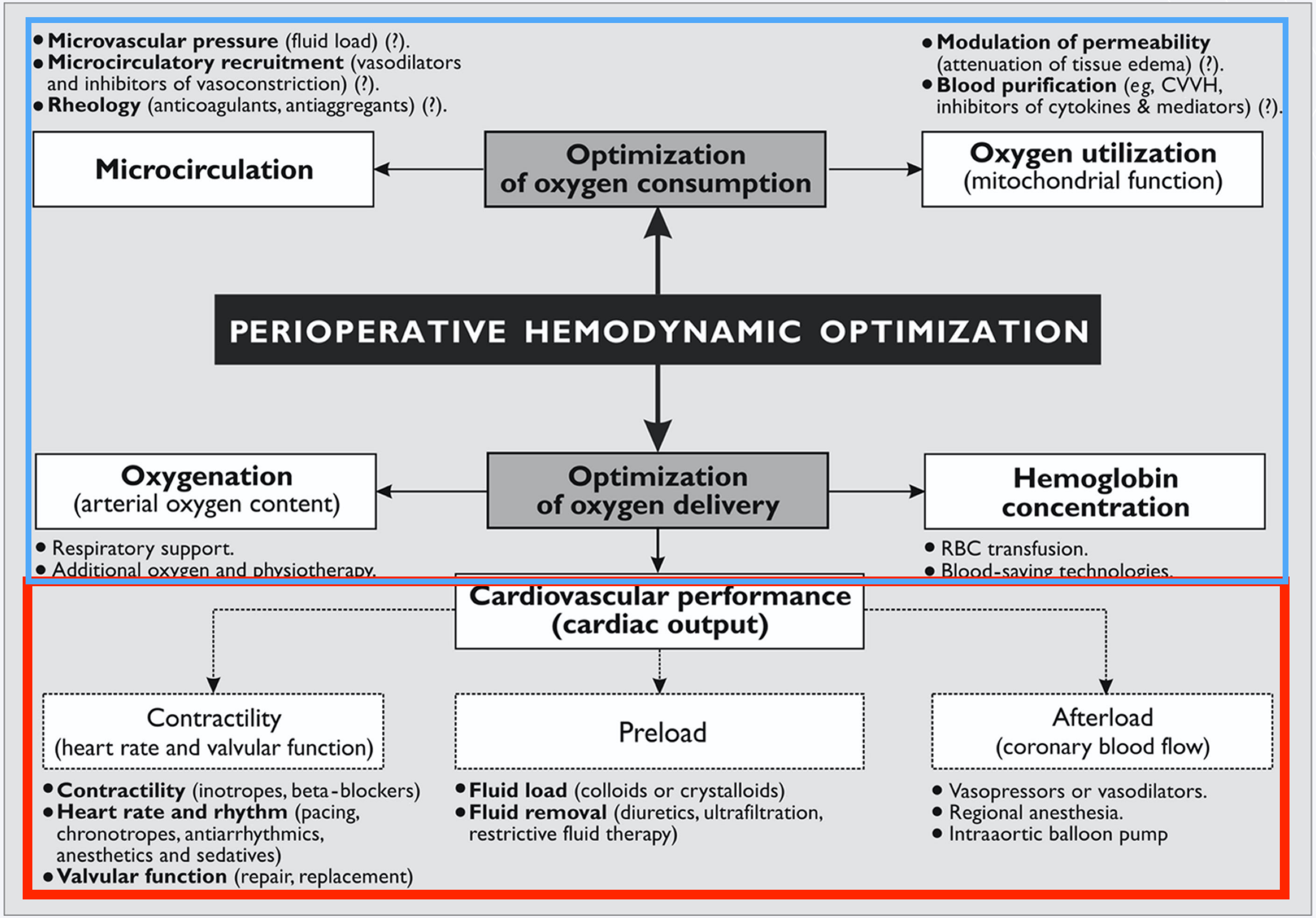
- ❑ Adequate ventilation management
- ❑ Normothermia
- ❑ Maintain acid-base balance
- ❑ Correct electrolyte abnormalities

LCOS: Treatments

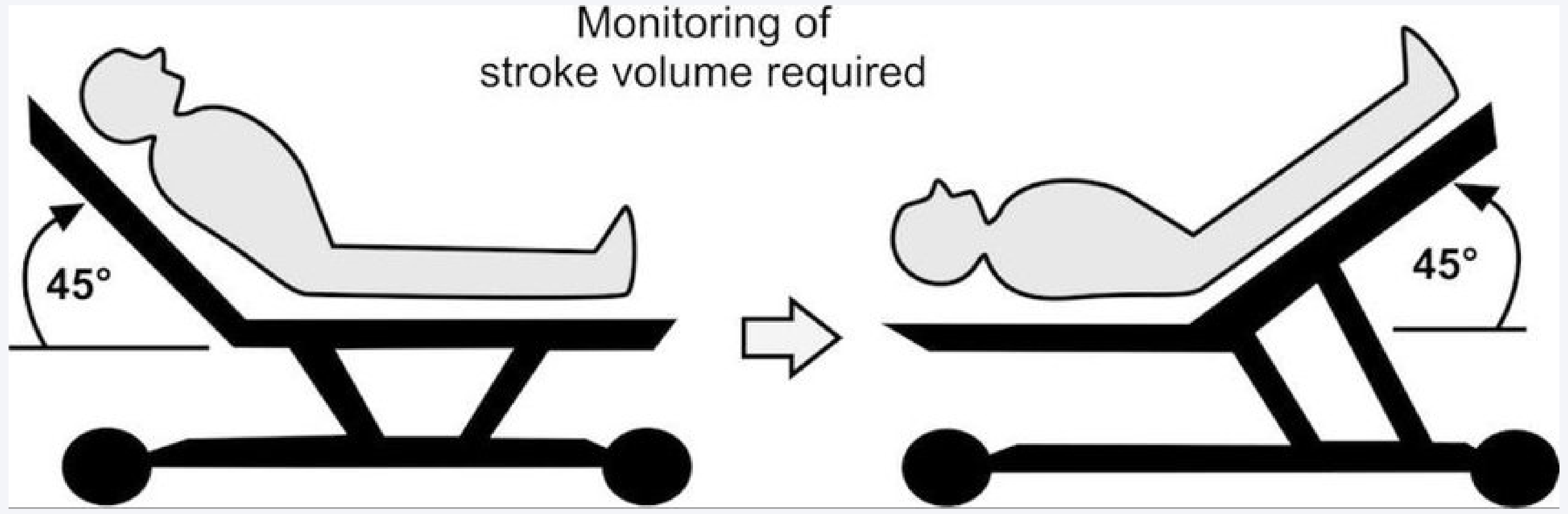


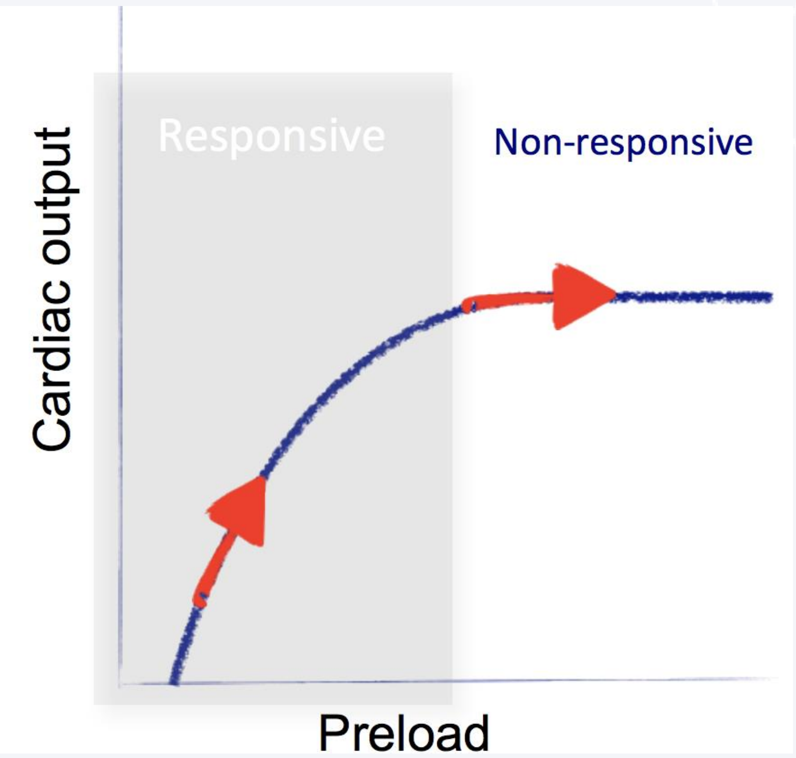


LCOS Treatment Guide



Preload : Passive leg raising





Diagnostic accuracy of passive leg raising for prediction of fluid responsiveness in adults: systematic review and meta-analysis of clinical studies

Subgroup	Correlation *r*	*p**	AUC	*p**
Ventilation				
Adapted	0.81 (0.53–0.93)	0.97	0.94 (0.87–1.00)	0.74
Inspiratory efforts	0.81 (0.74–0.87)		0.95 (0.91–0.99)	
Cardiac rhythm				
Sinus rhythm	0.73 (0.58–0.84)	0.15	0.96 (0.92–0.99)	0.94
Arrhythmias	0.83 (0.75–0.89)		0.96 (0.89–1.03)	
Starting position				
Supine	0.78 (0.64–0.87)	0.39	0.93 (0.87–1.00)	0.62
Semirecumbent	0.83 (0.75–0.89)		0.95 (0.92–0.97)	

* Test for interaction

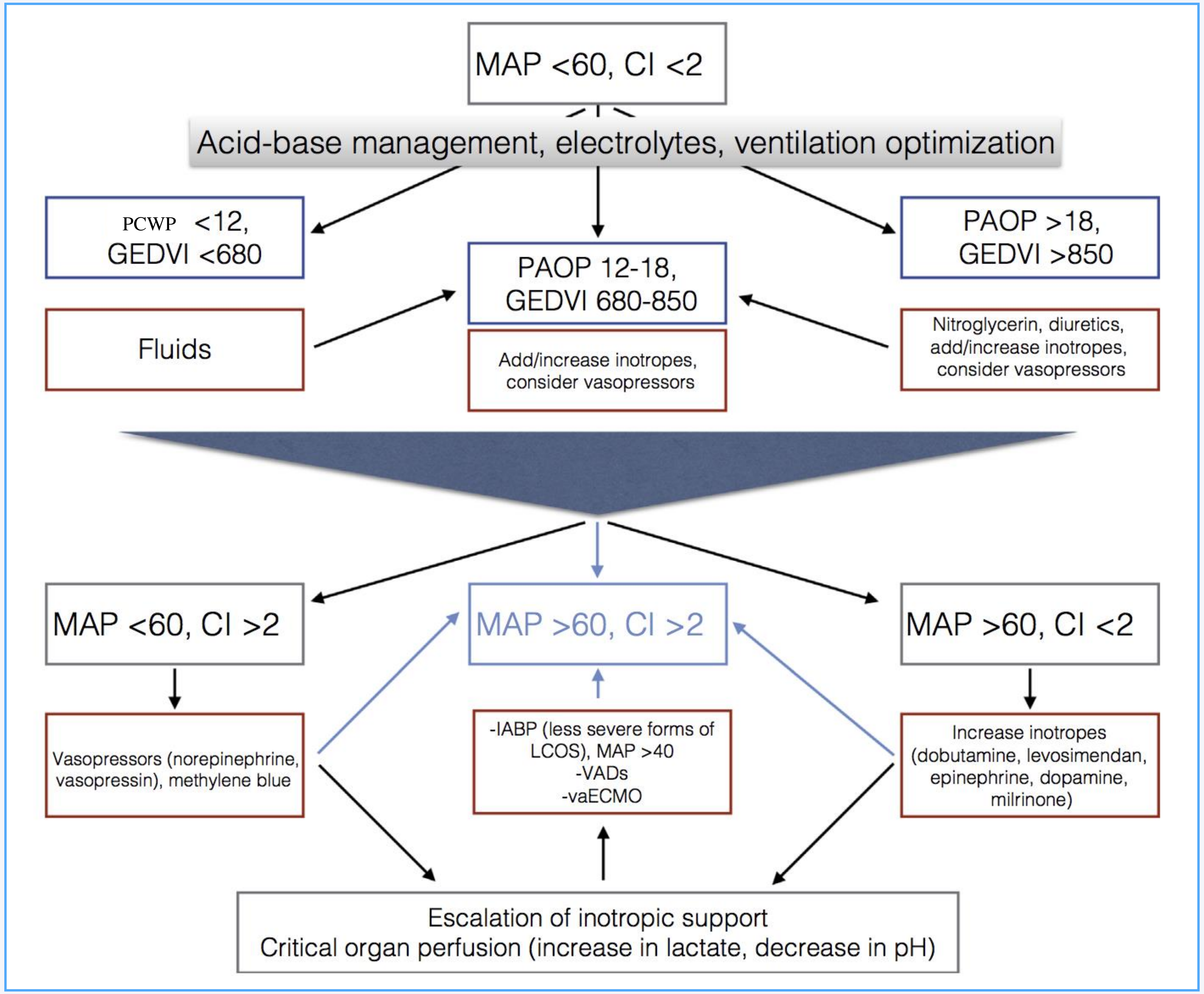
Intensive Care Med (2010) 36:1475–1483

Suitable for the critically ill ICU population

Limitation

- Need to assess real time direct measurement of CO
- Increase in risk of aspiration
- Increased Intracranial pressure (limit use in traumatic brain injury)
- Fracture of lower extremities (painful)
- Limit use in operating room

Preload：PGDT



Perioperative Goal-Directed Therapy (PGDT)

- **GDT**：**hemodynamic optimization algorithm** based on the **use of fluids, inotropes, and/or vasopressors** to **achieve normal or supranormal hemodynamic values** (hemodynamic goals)”.
- **PGDT** -> GDT initiated in the **intraoperative period** and **maintained in the immediate postoperative period**

PGDT Benefit - Cardiac Surgery

Goal-directed therapy in cardiac surgery: a systematic review and meta-analysis

H. D. Aya, M. Cecconi*, M. Hamilton and A. Rhodes

British Journal of Anaesthesia 110 (4): 510–17 (2013)

- PGDT protocol 24Hr preoperative
- RCT 5 studies 722 patients
- Adult (>18y) cardiac surgery

Post operative complication

Study or subgroup	EGDT Events	EGDT Total	Standard Events	Standard Total	Weight	Odds ratio M-H, Random, 95% CI	Year
Mythen and webb[21]	0	30	6	30	6.6%	0.66 [0.00, 1.15]	1995
Polonen and colleagues[20]	2	196	13	197	20.9%	0.15 [0.03, 0.66]	2000
McKendry et al.[22]	17	89	26	90	53.7%	0.58 [0.29, 1.17]	2004
Kapoor et al.[23]	1	13	2	14	8.6%	0.50 [0.04, 6.28]	2008
Smetkin et al.[24]	1	20	4	20	10.3%	0.21 [0.02, 2.08]	2009
Total (95% CI)		**348**		**351**	**100.0%**	**0.33 [0.15, 0.73]**	
Total events	21		51				

Heterogeneity: τ^2=0.17; χ^2=4.93, df=4 (P=0.29); I^2=19%
Test for overall effect: Z=2.76 (P=0.006)

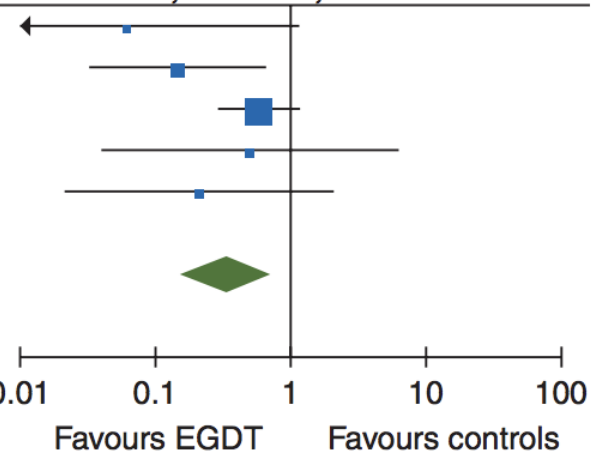


Fig 3 Forest plot showing the effect of early goal-directed therapy (EGDT) on postoperative complications rate *vs* control group. M-H, Mantel–Haenszel.

Mortality

Study or subgroup	EGDT Events	EGDT Total	Standard Events	Standard Total	Weight	Odds ratio M-H, Random, 95% CI	Year
Mythen and webb[21]	0	30	1	30	14.6%	0.32 [0.01, 8.24]	1995
Polonen and collegues[20]	2	196	6	197	44.7%	0.33 [0.07, 1.65]	2000
McKendry et al.[22]	4	89	2	90	40.7%	2.07 [0.37, 11.60]	2004
Kapoor et al.[23]	0	13	0	14		Not estimable	2008
Smetkin et al.[24]	0	20	0	20		Not estimable	2009
Total (95% CI)		**348**		**351**	**100.0%**	**0.69 [0.19, 2.56]**	
Total events	6		9				

Heterogeneity: τ^2=0.32; χ^2=2.59, df=2 (P=0.27); I^2=23%
Test for overall effect: Z=0.55 (P=0.58)

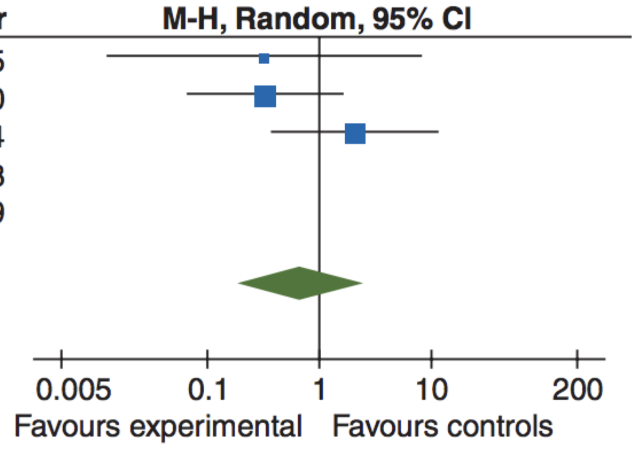


Fig 2 Forest plot showing the effect of early goal-directed therapy (EGDT) on mortality rate *vs* control group. M-H, Mantel–Haenszel.

Hospital length of Stay

Study or subgroup	EGDT Mean	EGDT SD	EGDT Total	Standard Mean	Standard SD	Standard Total	Weight	Mean difference IV, Random, 95% CI
Kapoor and colleagues[23]	5.8	1.2	13	8.8	2.1	14	34.2%	−3.00 [−4.28, −1.72]
McKendry and colleagues[22]	11.4	13.2	89	13.9	15	90	11.1%	−2.50 [−6.64, 1.64]
Mythen and webb[21]	6.4	1.1	30	10.1	9.4	30	14.7%	−3.70 [−7.09, −0.31]
Polonen and colleagues[20]	7.3	6.6	196	17.9	7.5	197	32.8%	−0.60 [−2.00, 0.80]
Smetkin and colleagues[24]	14	7.1	20	19.4	10	20	7.3%	−5.40 [−10.77, −0.03]
Total (95% CI)			**348**			**351**	**100.0%**	**−2.44 [−4.03, −0.84]**

Heterogeneity: τ^2=1.51; χ^2=8.66, df=4 (P=0.07); I^2=54%
Test for overall effect: Z=3.00 (P=0.003)

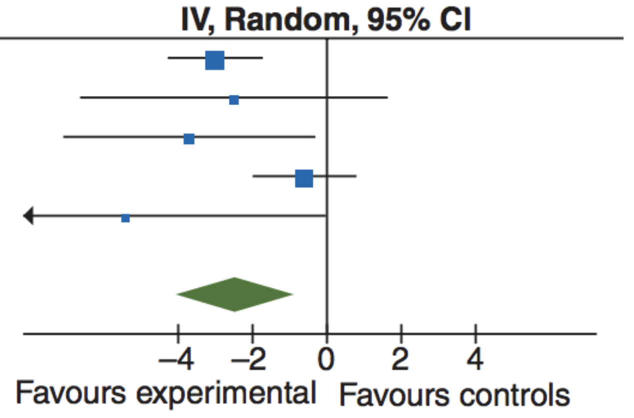


Fig 4 Forest plot showing the effect of early goal-directed therapy (EGDT) on hospital length of stay (LOS) *vs* control group. IV, inverse of variance. Data obtained by direct contact with author.

Medication Treatment : Inotropic

Drugs Used for the Treatment of LCOS

Intervention	Indications	Dosages	Receptors and Effects	Side Effects
Dobutamine	LCOS treatment	1-20 μg/kg/min	β-adrenergic, ↑inotropy	Arrhythmia (less than dopamine)
Dopamine	LCOS treatment	0.5-2 μg/kg/min	DA, vasodilation	Worsens renal injury in states of LCOS
		2-5 μg/kg/min	β-adrenergic, ↑inotropy	Tachycardia
		5-20 μg/kg/min	α- and β-adrenergic, vasoconstriction, ↑inotropy	Arrhythmia, tachycardia
Epinephrine	LCOS treatment, anaphylaxis	0.01-0.03 μg/kg/min	β-adrenergic, ↑inotropy	Lactic acidosis, hyperglycemia, mesenteric ischemia
		0.03-0.1 μg/kg/min	α- and β-adrenergic, vasoconstriction, ↑inotropy	
Norepinephrine	Decreased SVR, vasoplegic syndrome, septic shock	0.01-0.1 μg/kg/min	α- and β-adrenergic, vasoconstriction, ↑inotropy (less pronounced)	Arrhythmia, tachycardia
Vasopressin	Decreased SVR, vasoplegic syndrome	0.01-0.1 IU/min	V1 stimulation, vasoconstriction	Myocardial ischemia, ventricular arrhythmia
Milrinone	LCOS treatment	0.5-1 μg/kg/min	Phosphodiesterase inhibitor, ↑inotropy, vasodilation	Thrombocytopenia, arrhythmia, tachycardia
Levosimendan	LCOS treatment and prophylaxis	10 μg/kg loading dose, 0.1 μg/kg/min infusion	Increasing myofilament sensitivity to calcium	Arrhythmia, hypotension

Lovosimendan vs Dobutamine

Hemodynamic Effects of Levosimendan Compared With Dobutamine in Patients With Low Cardiac Output After Cardiac Surgery

Rev Esp Cardiol. 2006;59(4):338-45

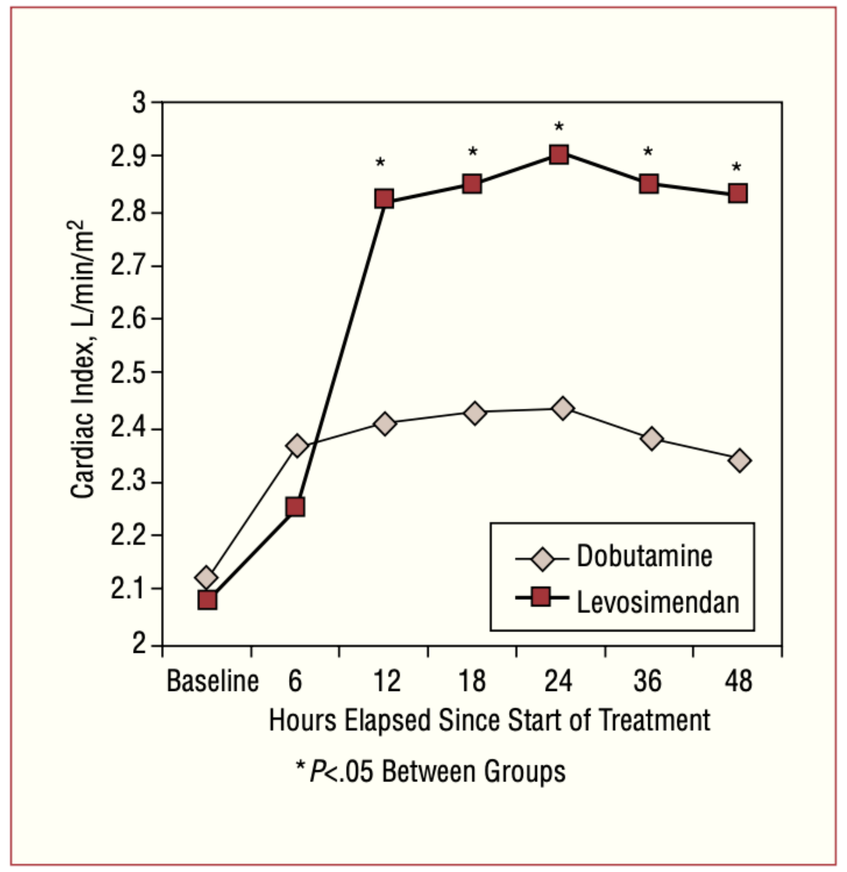


Figure 1. Changes in the cardiac index over the study period. Dobutamine: dobutamine group; levosimendan: levosimendan group. *Significant difference between treatment groups.

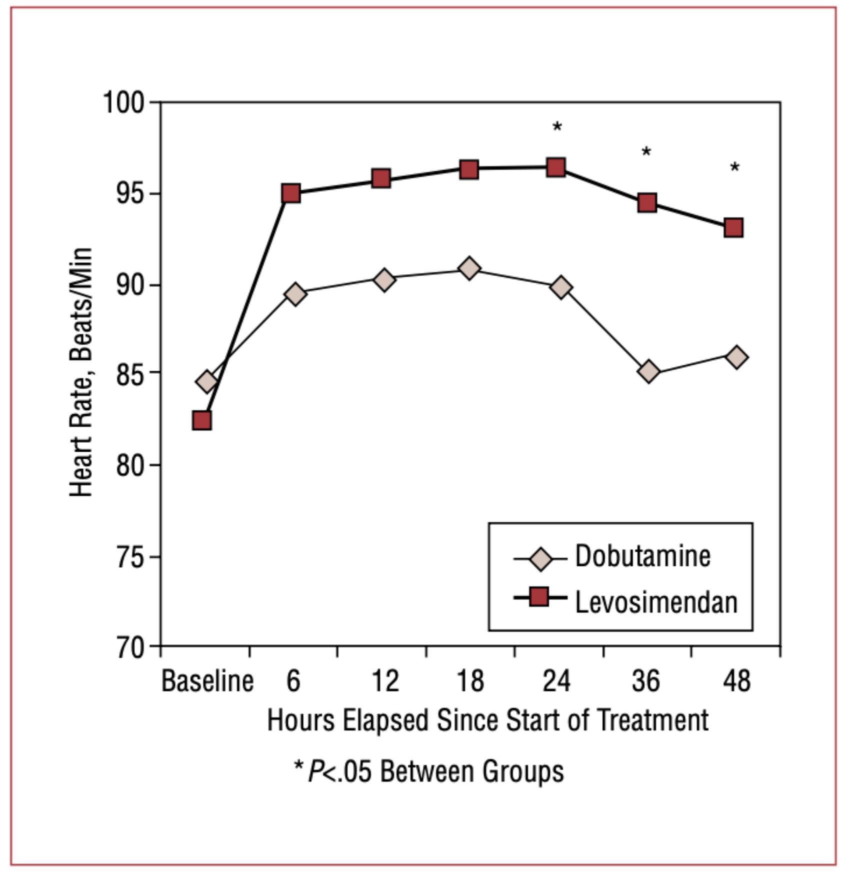


Figure 2. Change in heart rate over the study period. Dobutamine: dobutamine group; levosimendan: levosimendan group. *Significant difference between treatment groups.

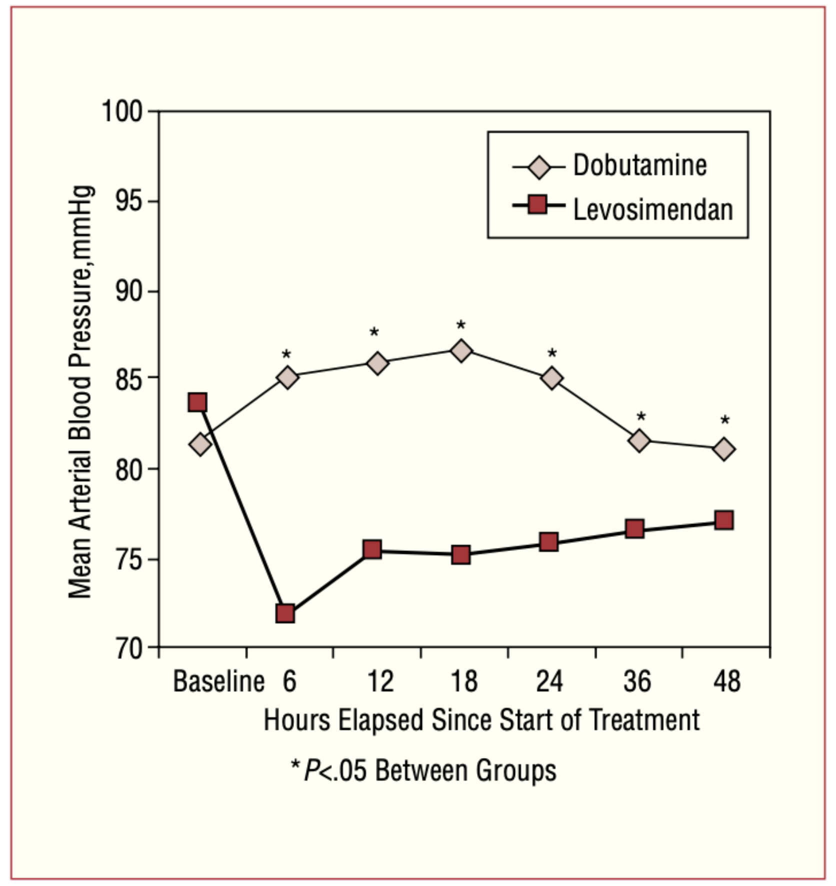
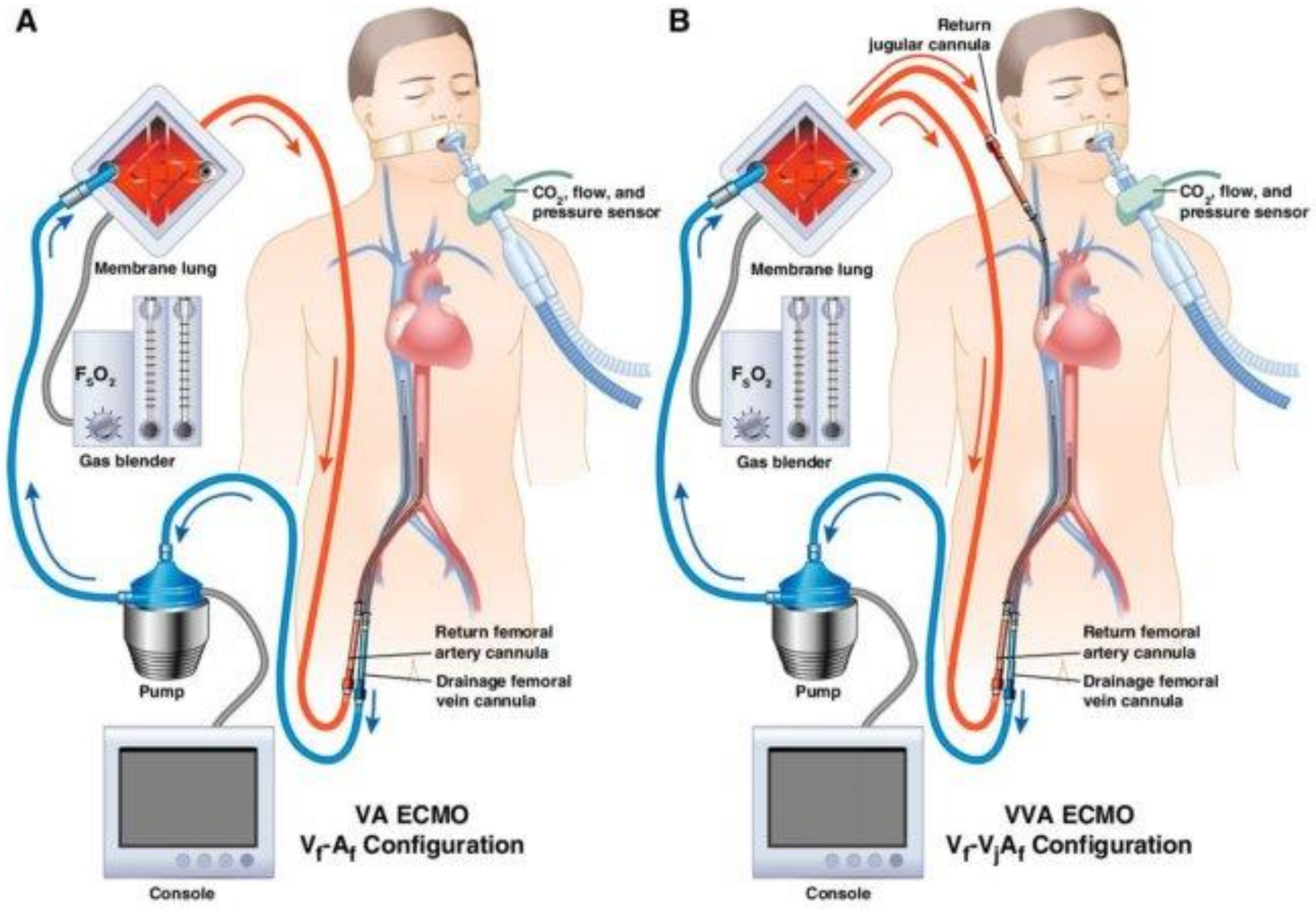
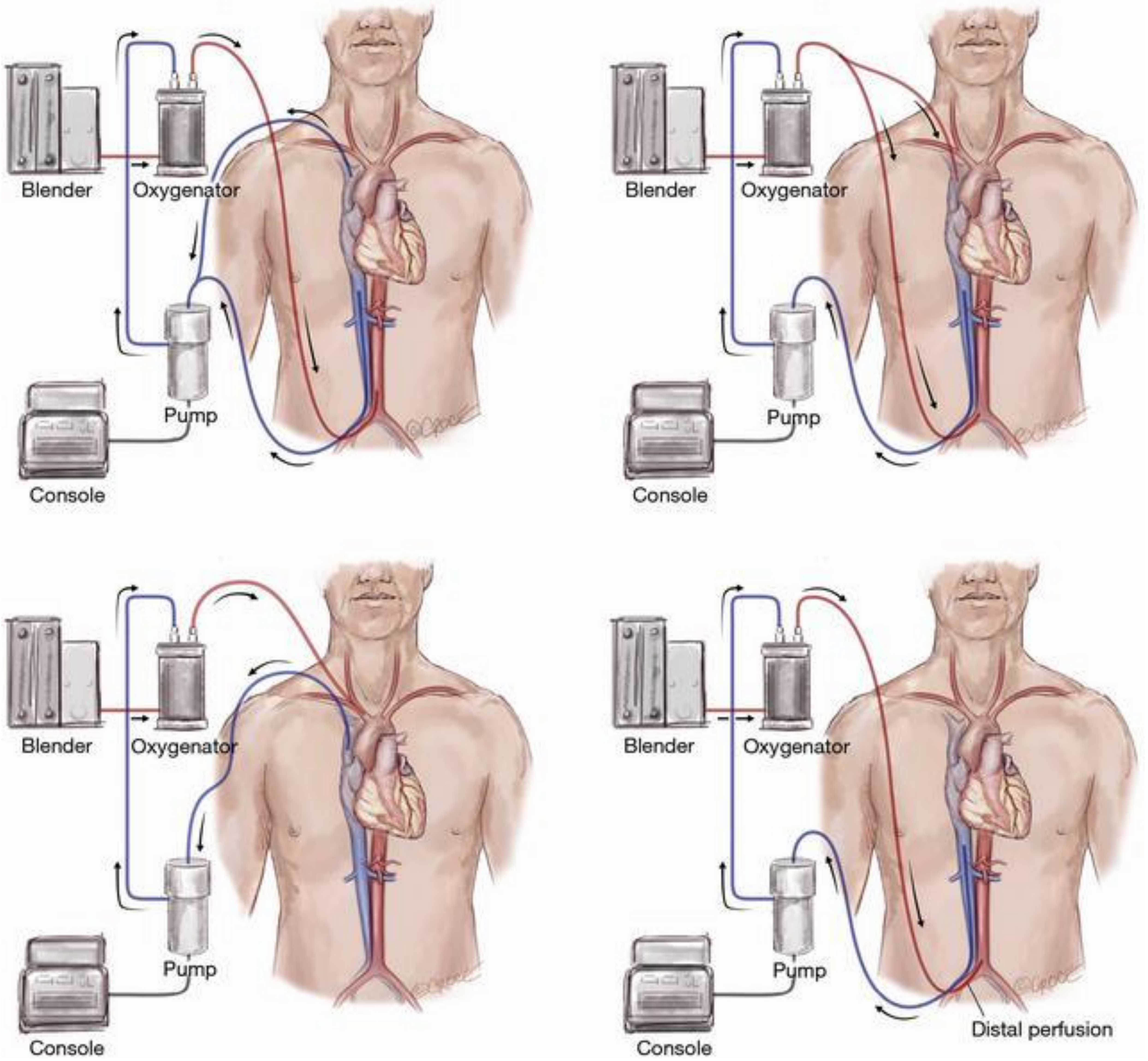


Figure 3. Change in mean arterial blood pressure over the study period. Dobutamine: dobutamine group; levosimendan: levosimendan group. *Significant difference between treatment groups.

Cochrane Library

	Anticipated absolute effects (95% CI)		Relative effect (95% CI)	No of participants (studies)	Quality of the evidence (GRADE)	Comments
	Risk with dobutamine	Risk with levosimendan				
All-cause short-term mortality: range 15 to 31 days	148 per 1000[1]	**89 per 1000** (53 to 152)	**RR 0.60** (0.36 to 1.03)	1701 (4 studies)	⊕⊕⊖⊖ **low**[2]	Studies included participants with LCOS or CS due to cardiac surgery or HF.
All-cause long-term mortality: range 4 to 12 months	288 per 1000[1]	**242 per 1000** (181 to 325)	**RR 0.84** (0.63 to 1.13)	1591 (4 studies)	⊕⊕⊖⊖ **low**[2]	Studies included participants with LCOS or CS due to HF or AMI.

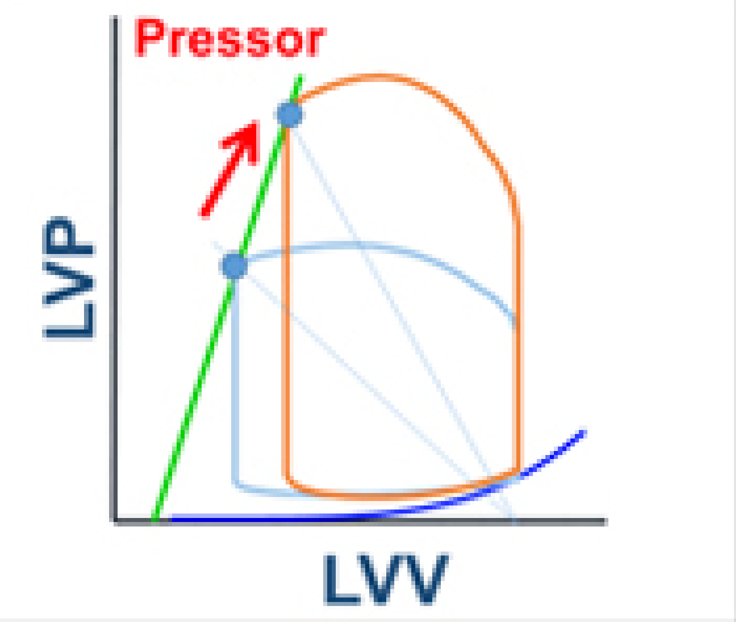


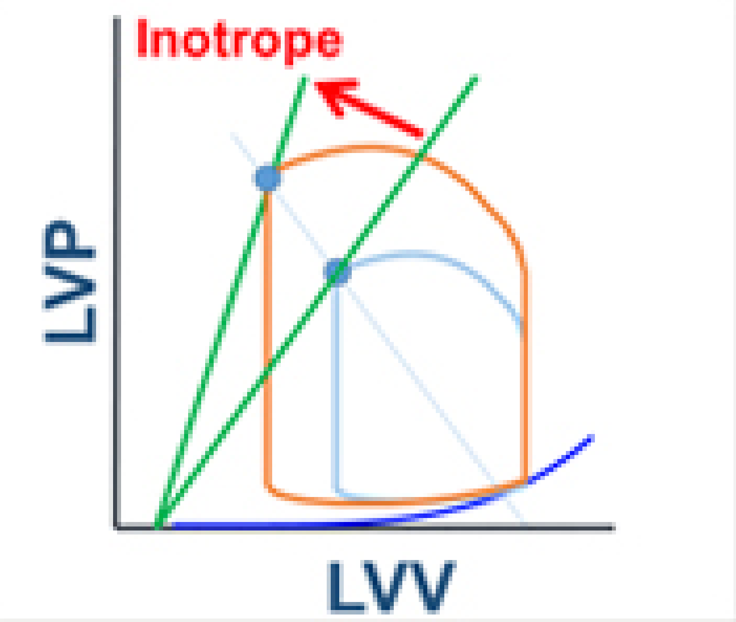


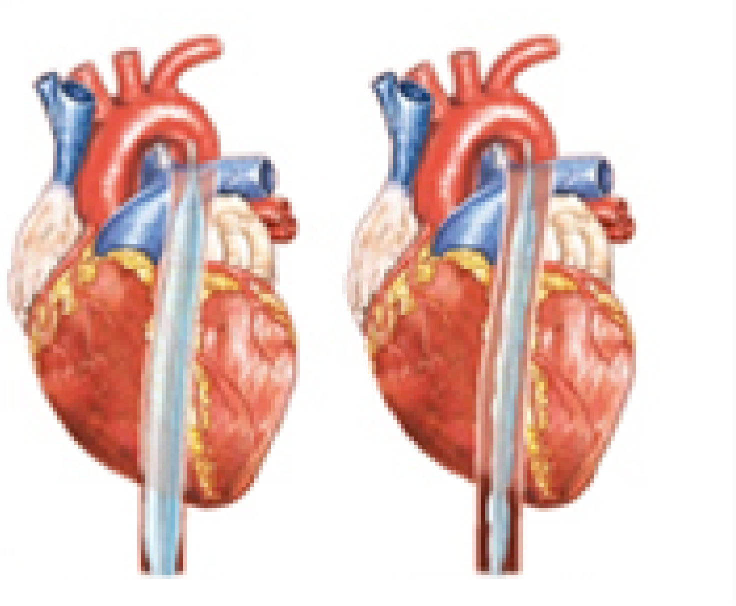
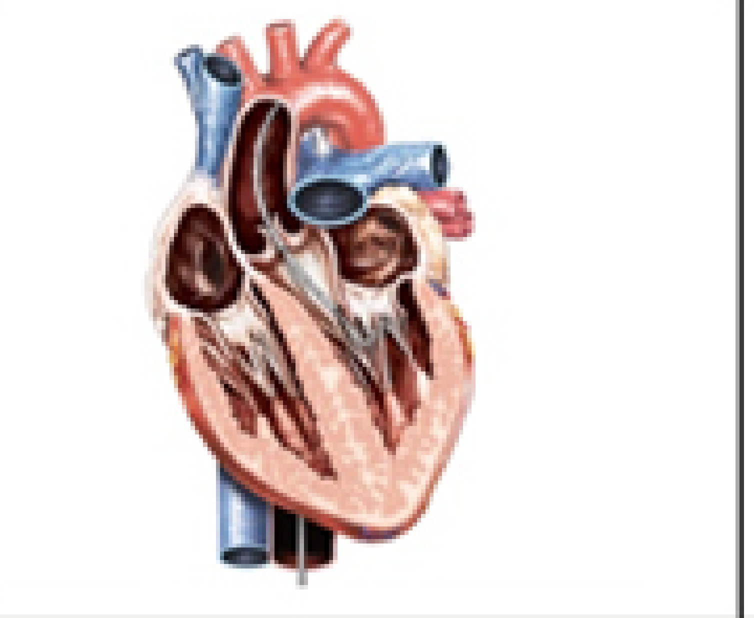
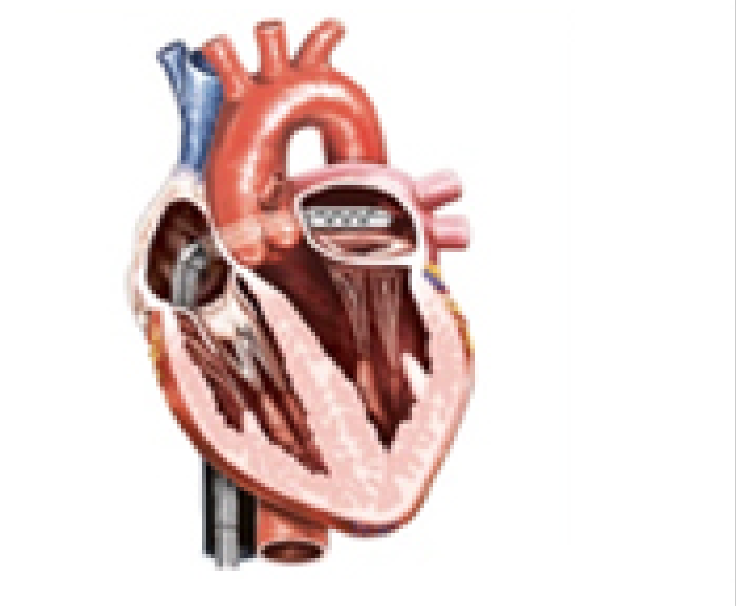
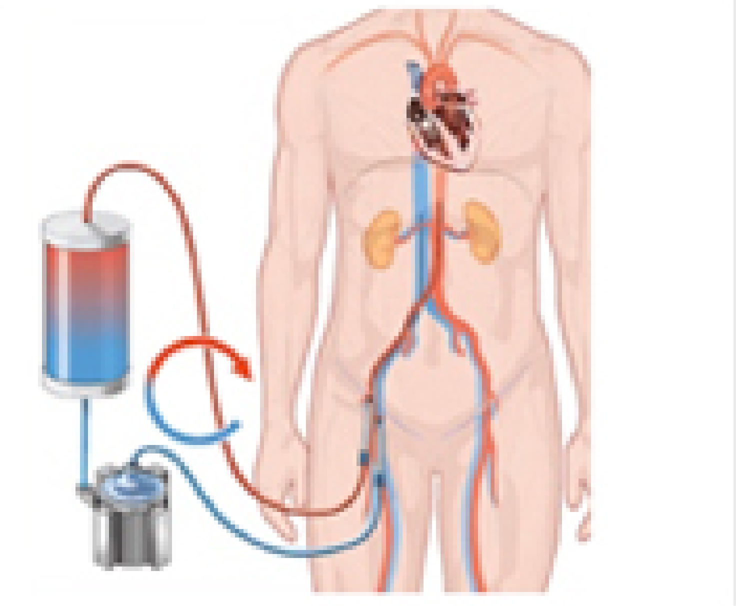
ECMO

Hemodynamic

Medication / Mechanical Support & Hemodynamic





	Vasopressor	Inotrope	IABP	IMPELLA	TANDEMHEART	VA-ECMO
Mechanism	Peripheral vasoconstriction	Increased myocyte calcium cycling	Aortic counter-pulsation	Left ventricle (LV) to aorta transvalvular circulatory support (LVADs)	Left atrium (LA) to arterial circulatory support	Right atrium (RA) to peripheral artery circulatory support with gas exchange unit
LV Contractility	↔ or ↑	↑	↔	↔	↔	↔
TPR	↑	↔ or ↑ or ↓	↔	↔	↔	↔
LV Flow	↓	↑	↑	↓	↓	↓
Total CO	↓	↑	↑	↑↑	↑↑	↑↑↑
CVP	↔ or ↑	↔	↔ or ↓	↔ or ↓	↔ or ↓	↓
PCWP	↔ or ↑	↔ or ↓	↔ or ↓	↓	↓	↔ or ↑
MAP	↑	↑	↑	↑↑	↑↑	↑↑
Total CPO	↔ or ↑	↑	↑	↑↑	↑↑	↑↑
PVA	↑	↑	↔ or ↓	↓↓	↔ or ↓	↑↑
MVO2	↑	↑	↓	↓↓	↔ or ↓	↑↑
Sheath size	NA	NA	7-8 French arterial	14 French arterial	15-17 French arterial 21 French Venous	15 – 17 French arterial 21-23 French venous

Take Home message

- Low cardiac output syndrome make severe complication after cardiac surgery
- Hemodynamic monitor in high risk patients and and high risk procedure
- Peri-operative Goal-Directed Therapy (PGDT) is very important to management patient to prevent low cardiac output syndrome
- Early recognition , Timely intervention / treatment, less sequence LCOS complications.

Management
Post operative
Low Cardiac Output Syndrome

Bundit Mokarat, MD

Queen Sirikit Heart centre, Khonkaen University

Two Days 2020