FILLING PRESSURE:
CENTRAL VENOUS PRESSURE AND PULMONARY WEDGE PRESSURE

UTILITY AND CURRENT EVIDENCE

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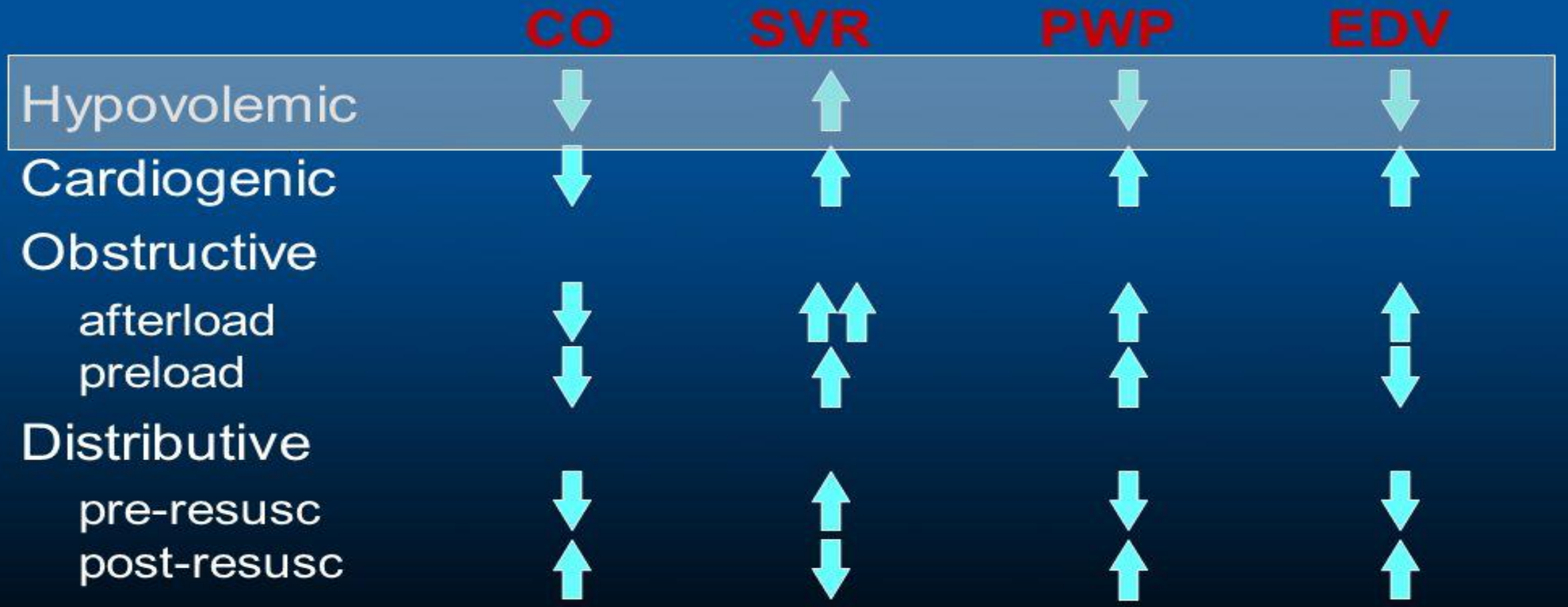
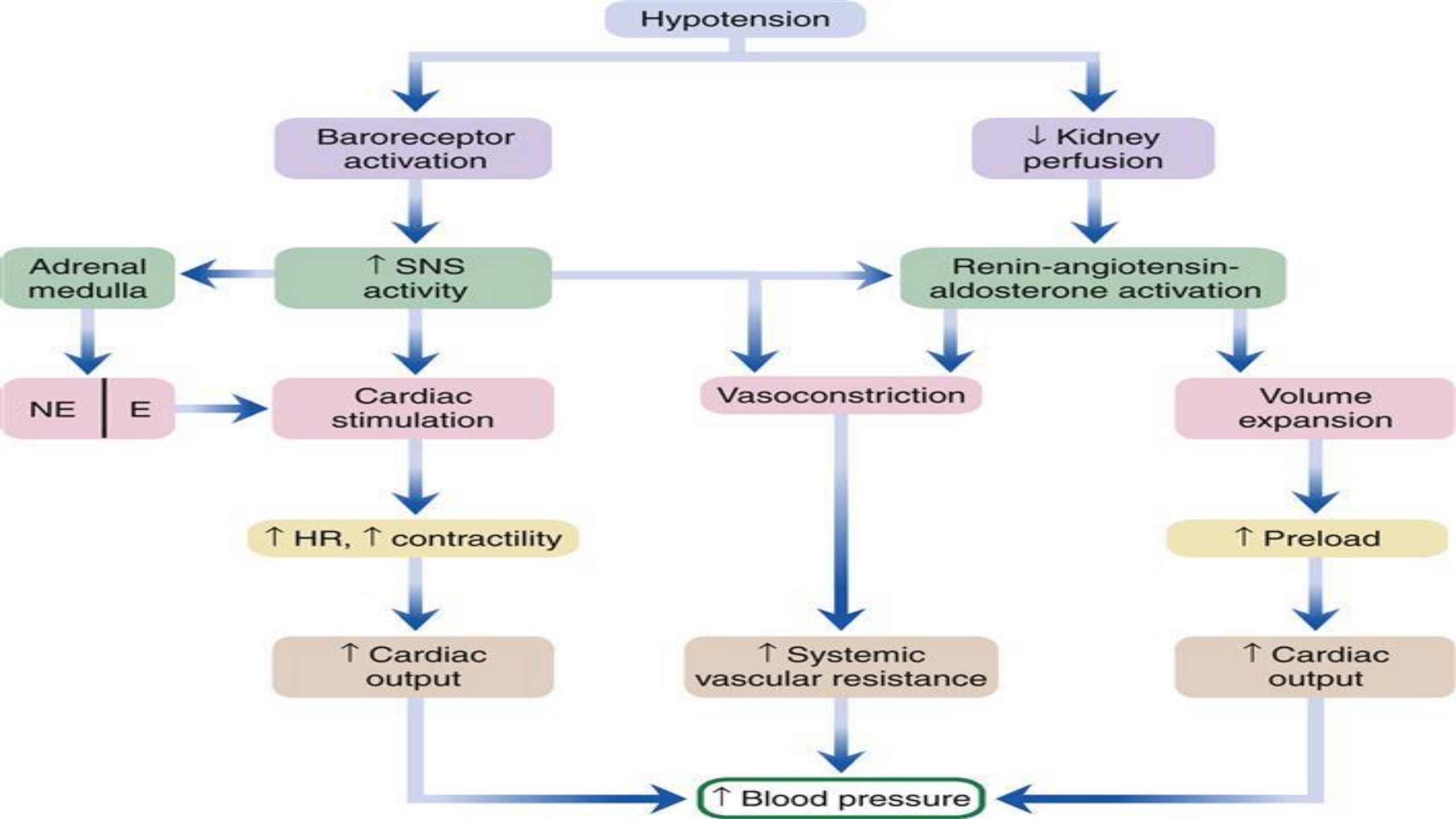
Hypovolemic Shock Hemodynamics



BLOOD PRESSURE

BP = CO x SVR

BLOOD PRESSURE = CARDIAC OUTPUT x SYSTEMIC VASCULAR RESISTANCE


Hypotension
Baroreceptor activation
↓ Kidney perfusion
Adrenal medulla
↑ SNS activity
Renin-angiotensin-aldosterone activation
NE
E
Cardiac stimulation
Vasoconstriction
Volume expansion
↑ HR, ↑ contractility
↑ Preload
↑ Cardiac output
↑ Systemic vascular resistance
↑ Cardiac output
↑ Blood pressure

BP = CO x SVR

↓

CO = SV x HR

↓

Stroke Volume

↙ ↓ ↘

Preload

↓

Volume of blood at end-diastole

Afterload

↓

Resistance the heart must overcome to eject blood

Contractility

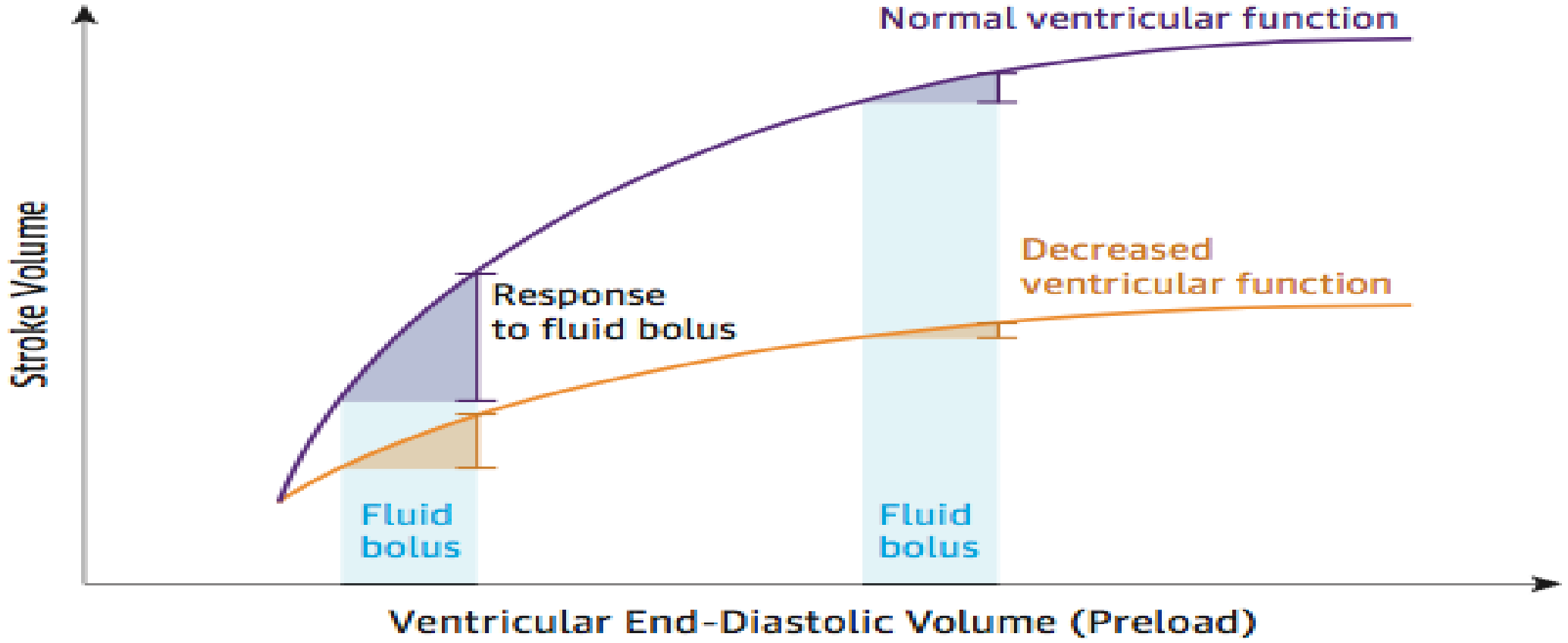
↓

The heart **Strength** with pumping and its **Force**

PRELOAD

- CORNERSTONE OF TREATING HYPOTENSION , HYPOPERFUSION AND SHOCK REMAINS TO BE FLUIDS
- FLUID OPTIMIZATION PROTOCOLS ARE BASED ON MAXIMIZING STROKE VOLUME AND CARDIAC OUTPUT
- SHOWN TO REDUCE POSTOPERATIVE COMPLICATIONS AND LENGTH OF STAY AT HOSPITAL
- EARLY AGGRESSIVE RESUSCITATION OF CRITICALLY ILL PATIENT MAY LIMIT AND REVERSE TISSUE HYPOXIA AND PROGRESSION TO ORGAN FAILURE

Figure 1. Effect of Increase in Preload on Stroke Volume of Ventricles With Normal and Decreased Contractility



Frank-Starling curves illustrate that the effect of a given increase in preload on stroke volume is dependent both on ventricular contractility and on baseline preload.

PROBLEM WITH FLUID RESUSCITATION ???

- OVER ZEALOUS FLUID RESUSCITATION HAS BEEN ASSOCIATED WITH INCREASED COMPLICATIONS, INCREASED LENGTH OF ICU AND HOSPITAL STAY AS WELL AS MORTALITY
- THE ONLY REASON TO GIVE FLUIDS IS TO INCREASE SV (PRELOAD) WITH AN INCREASE CARDIAC OUTPUT AND OXYGEN DELIVERY
- IF YOUR FLUIDS DOES NOT INCREASE STROKE VOLUME THEN YOUR VOLUME LOADING SERVES NO USEFUL BENEFIT AND MAY CAUSE MORE HARM

CENTRAL VENOUS PRESSURE

- INTRAVASCULAR PRESSURE IN THE GREAT THORACIC VEINS MEASURED RELATIVE TO ATMOSPHERIC PRESSURE
- MEASURED AT THE JUNCTION OF THE SUPERIOR VENA CAVA AND RIGHT ATRIUM
- PROVIDES AN ESTIMATE OF THE RIGHT ATRIAL PRESSURE

CENTRAL VENOUS PRESSURE

- THE USE OF CVP AS AN INDEX OF PRELOAD RELIES ON TWO AASUMPTION
    - CVP IS EQUIVALENT TO THE FILLING PRESSURE OF THE HEART
    - MYOFIBRIL LENGTH IS PROPORTIONAL TO THE CARDIAC FILLING PRESSURE
- ISSUES :
    - MEASURED CVP DOES NOT TRULLY CORRESPOND TO THE PRESSURE DISTENDING THE RIGHT ATRIUM AT THE END OF DIASTOLE
    - THE PRESSURE THAT DILATES THE VENTRICLES IS NOT THE INTRAVASCULAR PRESSURE BUT THE TRANSMURAL PRESSURE (INTRAVASCULAR PRESSURE – INTRATHORACIC PRESSURE

LIMITATIONS

Table 1. Factors affecting the measured CVP

Central venous blood volume	• Venous return/cardiac output • Total blood volume • Regional vascular tone
Compliance of central compartment	• Vascular tone • Right ventricular compliance – Myocardial disease – Pericardial disease – Tamponade
Tricuspid valve disease	• Stenosis • Regurgitation
Cardiac rhythm	• Junctional rhythm • AF • A-V dissociation
Reference level of transducer	• Positioning of patient
Intrathoracic pressure	• Respiration • Intermittent positive pressure ventilation (IPPV) • Positive end-expiratory pressure (PEEP) • Tension pneumothorax

CLINICAL EVIDENCE
(CVP AND CARDIAC OUTPUT

CHEST.2003 Nov;124(5):1900-8.
Global end-diastolic volume as an indicator of cardiac preload in patients with septic shock.
Michard F1, Alaya S, Zarka V, Bahloul M, Richard C, Teboul JL.

- CVP has been shown to correlate poorly with cardiac index
- CVP correlates poorly with stroke volume index
- Lack of correlation of measured CVP with cardiac output

CLINICAL EVIDENCE
(CVP AND CARDIAC OUTPUT)

Hinder F, Poelaert JI, Schmidt C, et al (1998) Assessment of cardiovascular volume status by transoesophageal echocardiography and dye dilution during cardiac surgery. Eur J Anaesthesiol 15:633-640

- CVP correlates poorly with other indices of cardiac preload that have been demonstrated to correlate well with cardiac output or cardiac index including the intrathoracic blood volume index and left ventricular end diastolic volume index and right ventricular end diastolic volume index

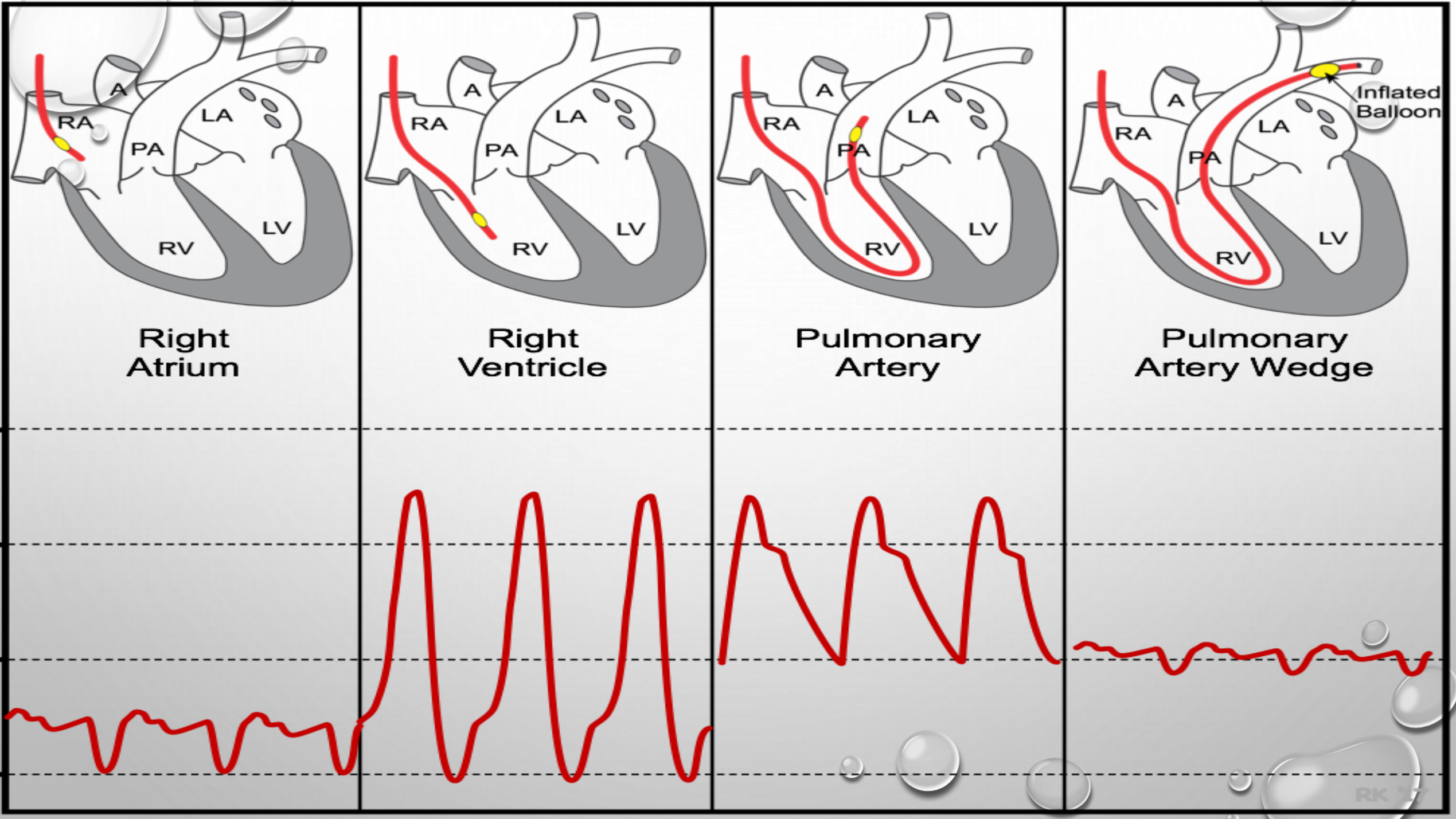
CLINICAL EVIDENCE
(CVP AND FLUID RESPONSIVENESS)

1. CalvinJF,DriedgerAA,SibbaldWJ(1981)Thehemodynamiceffectofrapidfluidinfusionin critically ill patients. Surgery 90:61-76
2. Reuse C, Vincent JL, Pinsky MR (1990) Measurements of right ventricular fluid volumes during fluid challenge. Chest 98:1450-1454
3. MichardF,BoussatS,ChemlaD,etal(2000)Relationbetweenrespiratorychangesinarterial pulse pressure and fluid responsiveness in septic patients with acute circulatory failure. Am J Respir Crit Care Med 162:134-138

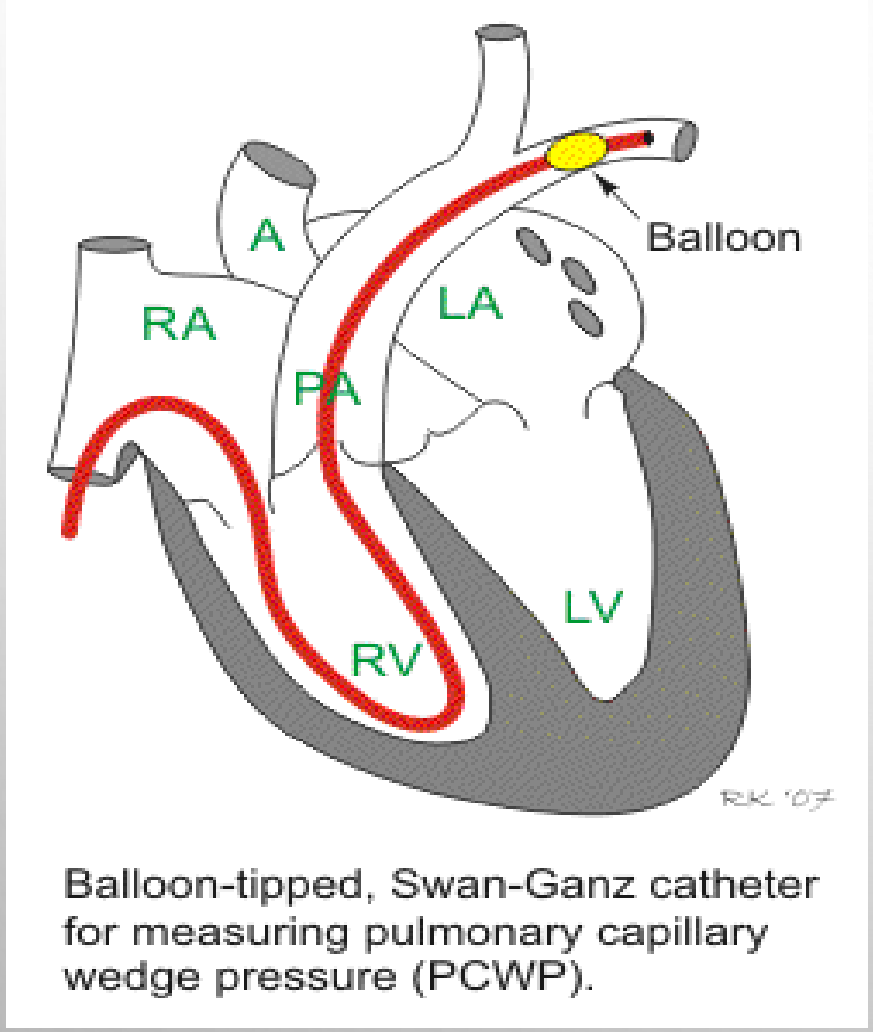
- predictive value of CVP for fluid responsiveness have been unable to demonstrate a relationship between the baseline CVP and the response to filling
- overlap of CVP values between the responder and non-responder groups that no threshold value which would discriminate between the two groups could be determined
- CVP measurements to assess whether or not a patient's cardiac output will increase significantly in response to an infusion of intravenous fluid cannot therefore be recommended.

PULMONARY WEDGE PRESSURE

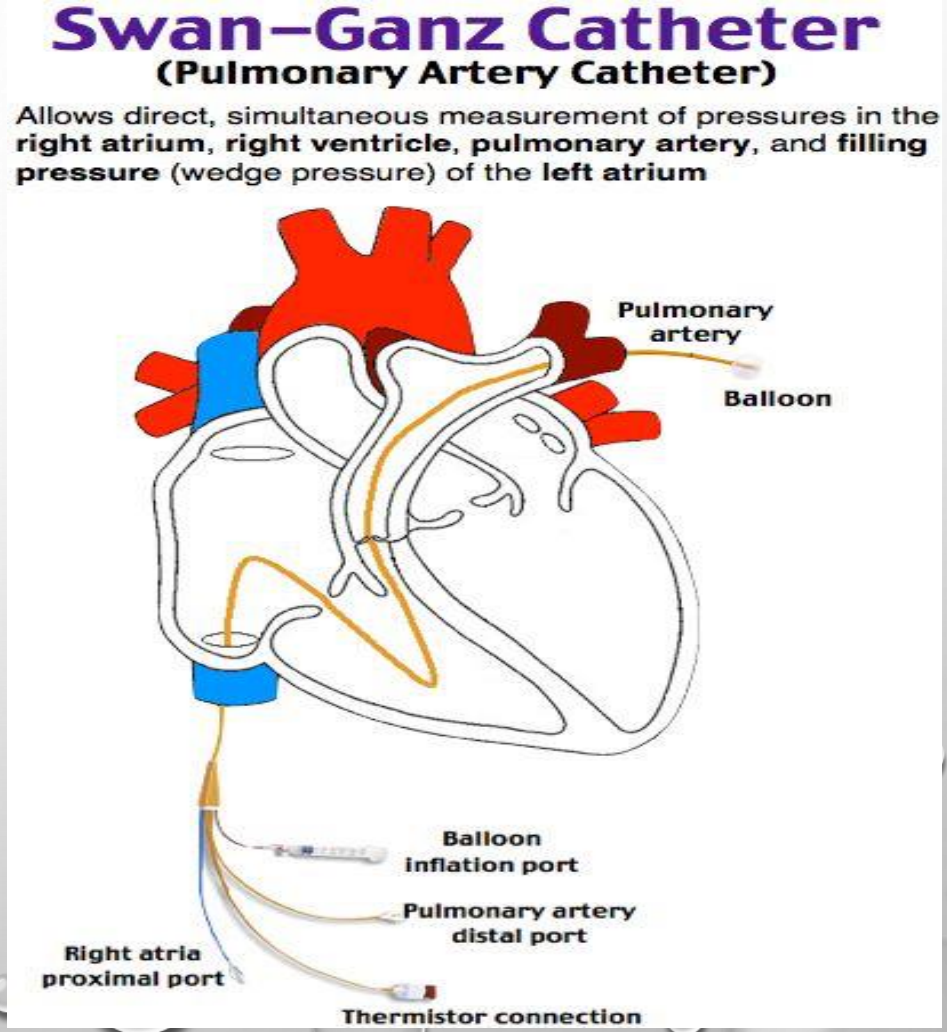
- PULMONARY CAPILLARY WEDGE PRESSURE IS A PHASE-DELAYED, AMPLITUDE-DAMPED VERSION OF LA PRESSURE.
- THE PCWP IS THEN REFLECTIVE OF THE LV DIASTOLIC PRESSURE OR LV FILLING PRESSURE
- PROVIDES THE MEASURE OF HYDROSTATIC PRESSURE THAT IS RESPONSIBLE FOR FORCING FLUID OUT OF THE PULMONARY VASCULAR SPACE. I
- DIRECTLY RELATED TO DIASTOLIC FIBER STRETCH ACCORDING TO STARLING'S PRINCIPLE, WHICH STATES THAT THE STRENGTH OF CONTRACTION IS PROPORTIONAL TO MYOCARDIAL FIBER LENGTH/LV VOLUME


A
RA
LA
PA
RV
LV
Right Atrium
Right Ventricle
Pulmonary Artery
Inflated Balloon
Pulmonary Artery Wedge

PCWP



Balloon-tipped, Swan-Ganz catheter for measuring pulmonary capillary wedge pressure (PCWP).



CLINICAL EVIDENCE
PCWP AND VOLUME RESPONSIVENESS

1. Diebel LN, Wilson RF, Tagett MG et al. End-diastolic volume: a better indicator of pre-load in the critically ill. Arch Surg 1992; 127: 817–822.
2. Wagner JG, Leatherman JW. Right ventricular end-diastolic volume as a predictor of the hemodynamic response to a fluid challenge. Chest 1998; 113: 1048–1054.
3. Tousignant CP, Walsh F, Mazer CD. The use of transesophageal echocardiography for preload assessment in critically ill patients. Anesth Analg 2000; 90: 351–355.
4. Osman D, Ridel C, Ray P et al. Cardiac Filling Pressures are not appropriate to predict hemodynamic response to volume challenges. Crit Care Med 2007; 35: 64–68.

- demonstrated a poor correlation between PCWP, volume status, and responsiveness to fluid resuscitation.

CLINICAL EVIDENCE

Table 1—*Main Characteristics of Clinical Studies Investigating the Predictive Factors of Fluid Responsiveness in ICU Patients

Source	Patients, No.	FC, No.	Fluid Infused	Volume Infused, mL	Speed of FC, min	Definition of Response	Rate of Response, %	Parameters Tested
Calvin et al[2]	28	28	5% Alb	250	20–30	ΔSV > 0%	71	RAP, PAOP, RVEDV
Schneider et al[3]	18	18	FFP	500	30	ΔSV > 0%	72	RAP, PAOP, RVEDV
Reuse et al[4]	41	41	4.5% Alb	300	30	ΔCO > 0%	63	RAP, PAOP, RVEDV
Magder et al[5]	33	33	9% NaCl	100–950		ΔCO > 250 mL/min	52	ΔRAP
Diebel et al[6]	15	22	R. lactate Colloids	300–500 500		ΔCO > 10%	59	PAOP, RVEDV
Diebel et al[7]	32	65	R. lactate	300–500		ΔCO > 20%	40	PAOP, RVEDV
Wagner and Leatherman[8]	25	36	9% NaCl 5% Alb, FFP	938 ± 480 574 ± 187	7–120	ΔSV > 10%	56	RAP, PAOP, RVEDV
Tavernier et al[9]	15	35	HES	500	30	ΔSV > 15%	60	PAOP, LVEDA, Δdown
Magder and Lagonidis[10]	29	29	25% Alb 9% NaCl	100 150–400	15	ΔCO > 250 mL/min	45	ΔRAP
Tousignant et al[11]	40	40	HES	500	15	ΔSV > 20%	40	PAOP, LVEDA
Michard et al[12]	40	40	HES	500	30	ΔCO > 15%	40	RAP, PAOP, ΔPP
Feissel et al[13]	19	19	HES	8 mL/kg	30	ΔCO > 15%	53	LVEDA, ΔVpeak
Total	334	406					52	

*FC = fluid challenge; Alb = serum albumin; FFP = fresh frozen plasma; NaCl = serum saline solution; R. lactate = Ringer's lactate; HES = hydroxyethylstarch; ΔSV = volume expansion-induced changes in stroke volume; ΔCO = volume expansion-induced changes in cardiac output.

Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121: 2000–2008.

CLINICAL EVIDENCE

Table 2—*Criteria Used to Decide Volume Expansion

Source	Criteria
Calvin et al[2]	Cardiac index < 3.5 L/min/m^2 and PAOP < 12 mm Hg in septic and trauma patients Cardiac index < 2.5 L/min/m^2 and PAOP < 20 mm Hg in acutely ill patients with a defined cardiac cause
Schneider et al[3]	Systematic infusion in patients with septic shock
Reuse et al[4]	Systolic BP < 90 mm Hg or cardiac index < 2.5 L/min/m^2 or heart rate > 120/min or decreased urine output (< 25 mL/h)
Magder et al[5]	Clinical impression that the cardiac output was inadequate for tissue needs and would respond to volume loading
Diebel et al[6]	Oliguria (urine output < 30 mL/h) or hypotension or in an attempt to optimize oxygen delivery
Diebel et al[7]	In an attempt to increase oxygen delivery to > 600 mL/min/m^2 or to reach a plateau in the oxygen consumption-delivery relationship
Wagner and Leatherman[8]	One or more clinical conditions that suggested the possibility of inadequate preload: hypotension (< 100 mm Hg), oliguria (< 30 mL/h), tachycardia (> 100/min), lactic acidosis, cool extremities, vasopressor wean, azotemia
Tavernier et al[9]	Sepsis-induced hypotension (systolic BP < 90 mm Hg or its reduction by ≥ 40 mm Hg from usual values)
Magder and Lagonidis[10]	As part of routine testing to assess cardiac filling status if PAOP ≤ 18 mm Hg
Tousignant et al[11]	PAOP < 20 mm Hg or inotropic support or low urine output and adequate gas exchange
Michard et al[12]	Systolic BP < 90 mm Hg or the need of vasoactive drugs (dopamine > 5 μg/kg/min or norepinephrine) and PAOP < 18 mm Hg and PaO_2/FIO_2 > 100 mm Hg
Feissel et al[13]	Systematic infusion in septic shock patients with preserved left ventricular systolic function and PaO_2/FIO_2 > 100 mm Hg

*FIO_2 = fraction of inspired oxygen.

Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. Chest 2002; 121: 2000–2008.

CLINICAL EVIDENCE

Poor Agreement between Pulmonary Capillary Wedge Pressure and Left Ventricular End-Diastolic Pressure in a Veteran Population

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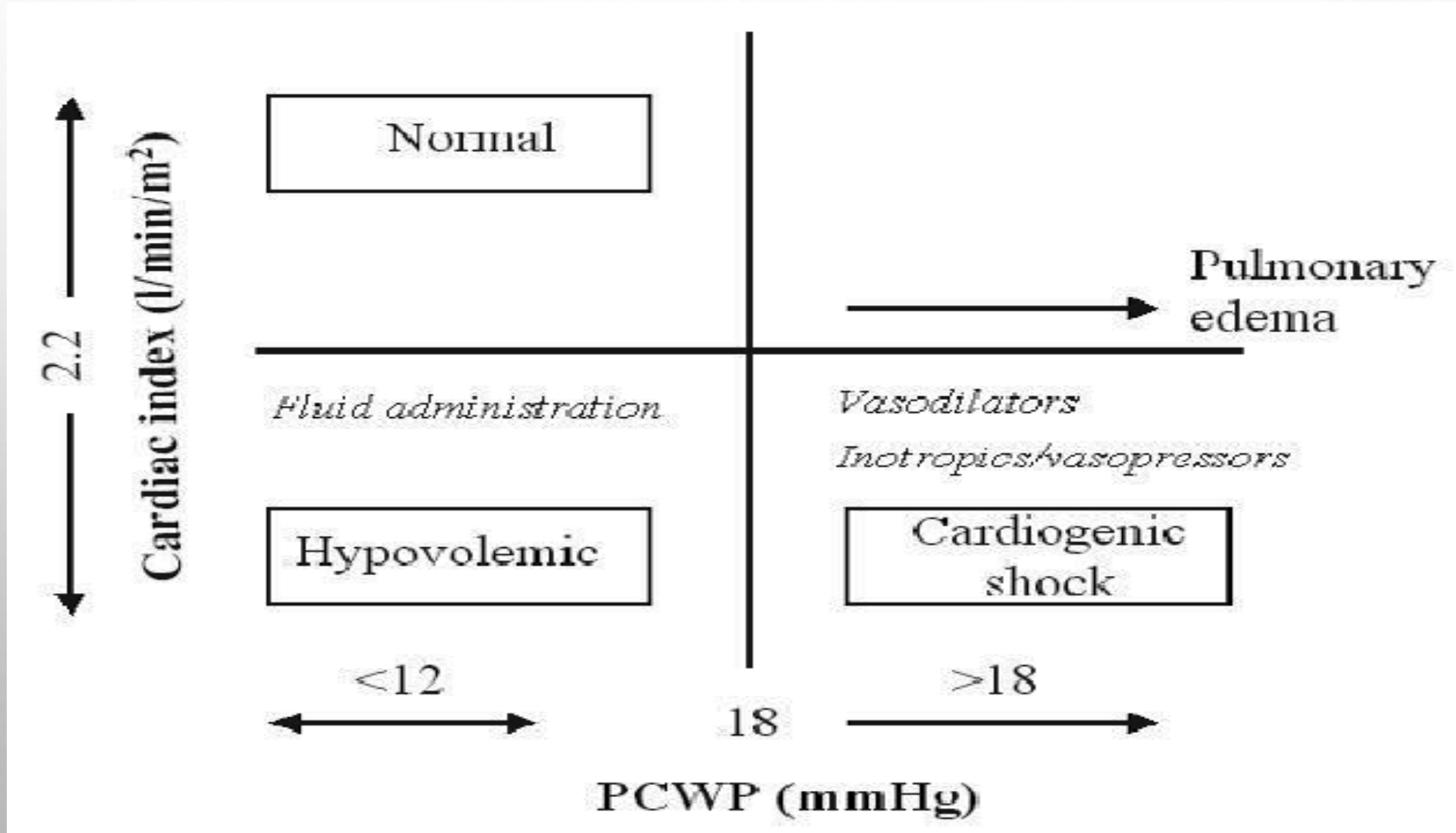
PCWP CAN STILL BE USE ???



SUMMARY (CVP)

- CVP DOES NOT TELL YOU VOLUME STATUS OR GIVE ESTIMATE OF THE MEAN CIRCULATORY FILLING PRESSURE
- CVP GIVES AN INDICATION OF THE INTERACTION OF PUMP FUNCTION AND RETURN FUNCTION
- THERE IS NO UNIVERSAL VALUE THAT CAN BE GIVEN FOR THE RIGHT ATRIAL PRESSURE AT WHICH THE PLATEAU OF THE FUNCTION CURVE OCCURS
- SHOULD NOT BE INTERPRETED AS A SINGLE PARAMETER BUT SHOULD BE ASSESSED TOGETHER WITH DYNAMIC TEST SUCH AS PULSE PRESSURE VIARIABILITY OR DOPPLER FLOW STUDIES
- IT IS NOT THE ACTUAL CVP THAT COUNTS BUT THE HEMODYNAMIC CONSEQUENCES OF THE CHANGE IN CVP

SUMMARY (PCWP)

- THERE IS POOR ASSOCIATION BETWEEN PCWP AND LEFT VENTRICULAR END DIASTOLIC PRESSURE
- CAN NOT DETECT WHO ARE FLUID RESPONDERS OR NOT
- SHOULD BE INTERTPRETED TOGETHER WITH OTHER DYNAMIC PARAMETERS

MIXED VENOUS AND CO2 GAP

CRIT CARE RES PRACT. 2013;2013:583598. DOI: 10.1155/2013/583598. EPUB 2013 AUG 29.

CENTRAL VENOUS-TO-ARTERIAL CO2 GAP IS A USEFUL PARAMETER IN MONITORING HYPOVOLEMIA-CAUSED ALTERED OXYGEN BALANCE: ANIMAL STUDY.

KOCSI S1, DEMETER G, ERCES D, NAGY E, KASZAKI J, MOLNAR Z.

SCVO2 < 73% AND CO2 GAP >6 MMHG VALUES TOGETHER TO PREDICT AN OXYGEN EXTRACTION >30%, THE POSITIVE PREDICTIVE VALUE IS 100%; NEGATIVE PREDICTED VALUE IS 72%

MIXED VENOUS AND CO2 GAP

Shock type	Lactate	O2ER	ScvO2	cvaCO2gap
Cardiogenic or hypovolemic	HIGH	HIGH	LOW	HIGH
Anemic or hypoxemic	HIGH	HIGH	LOW	LOW
Distributive	HIGH	LOW	HIGH	HIGH
Cytopathic	HIGH	LOW	HIGH	LOW


Thank you