

# Hemodynamics Value in Critical Care Fluid, Inotrope or Vasopressors

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### Hemodynamics

- Achieving <u>circulatory stability</u> is a real challenge sometimes specially in critical patients due to the complex physiology of the cardiovascular system.
- The clinical assessment of <u>systemic blood flow</u> (SBF) and <u>tissue perfusion</u> by indirect parameters, such as "<u>blood pressure</u>, <u>capillary refill time</u>, <u>heart rate</u>, <u>urine output</u>, <u>and CVP</u>".. is **inaccurate**

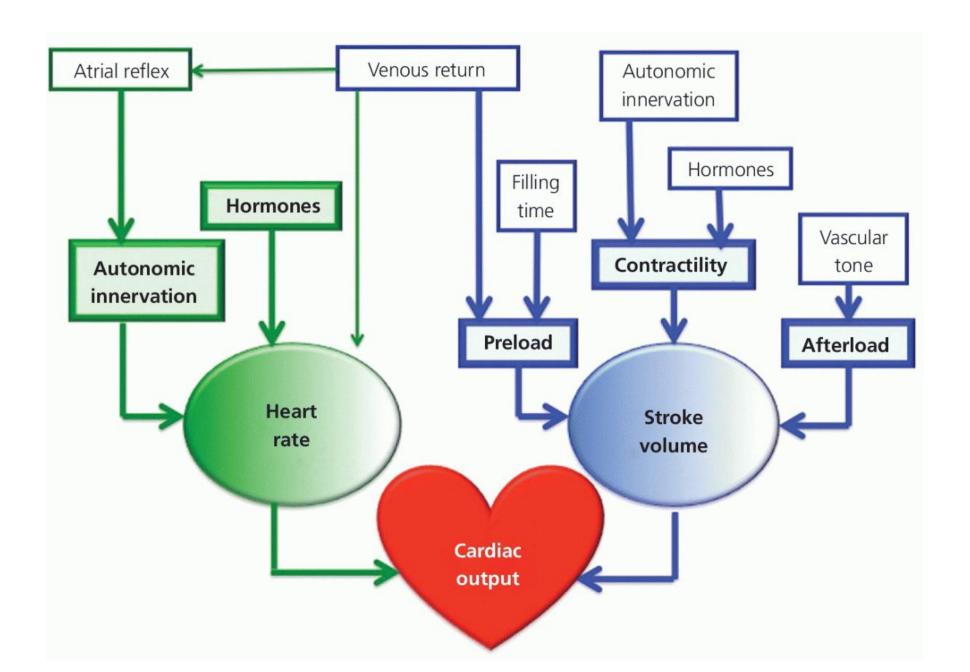
### Vital Sign Monitors.. Lack of knowledge

- Vital signs tell us <u>end-of-equation result</u>
- NIBP will start to show changes <u>10:15 minutes later</u> than the actual time when the changes started within the patient's cardiovascular system.
- If BP gets high (suppose we know on time), what shall we do?
- If BP dropped (suppose we knew early enough)
- What options are we going to use?
- Will it work in all patients?

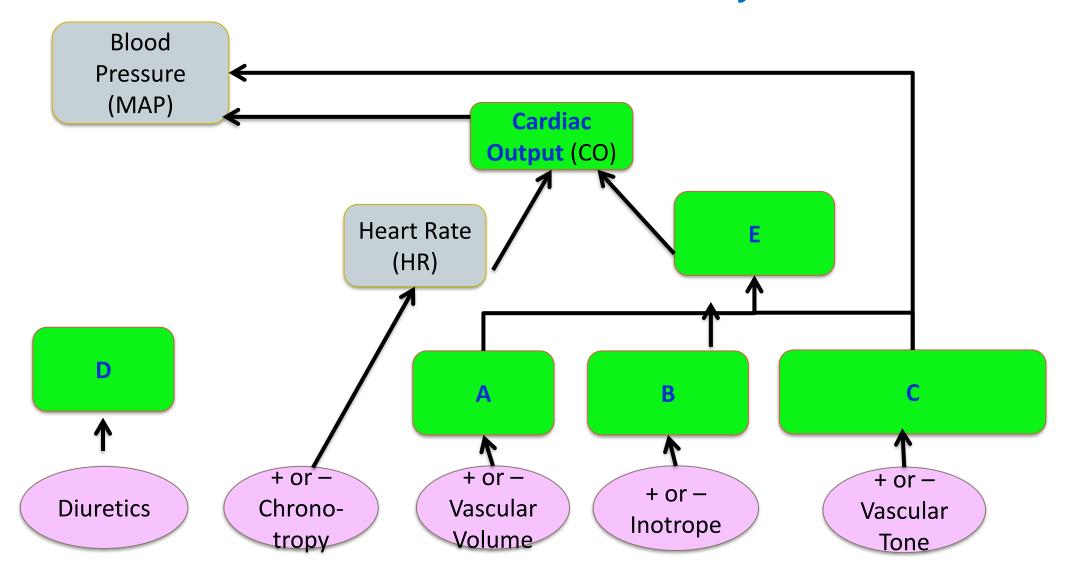


### Hemodynamics.. Why?

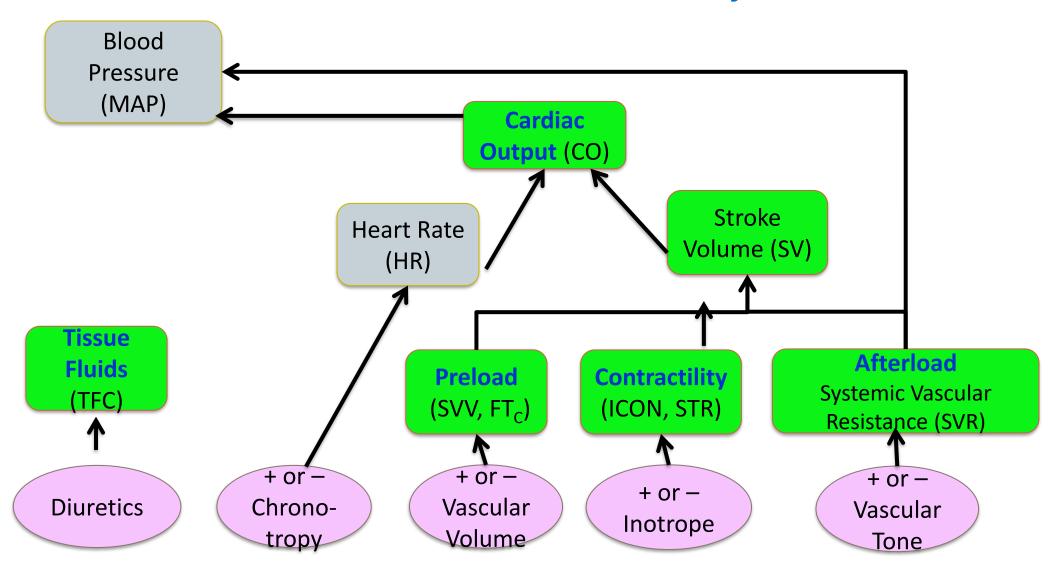
- Information on Cardiac output, Systemic vascular resistance,
   Preload and end organ perfusion should be obtained to detect inadequate tissue perfusion and oxygenation at early stage...
   Why?
- <u>To decide to initiate</u> <u>treatment</u> (<u>should every patient with</u> <u>hypotension be treated the same way</u>?) and if so, which therapy is the best for this individual patient (inotropes, vasopressors or fluid)?
- To detect the hemodynamic response to the initiated treatment regularly and modify dose accordingly, as changes in cardiovascular function can happen quickly.

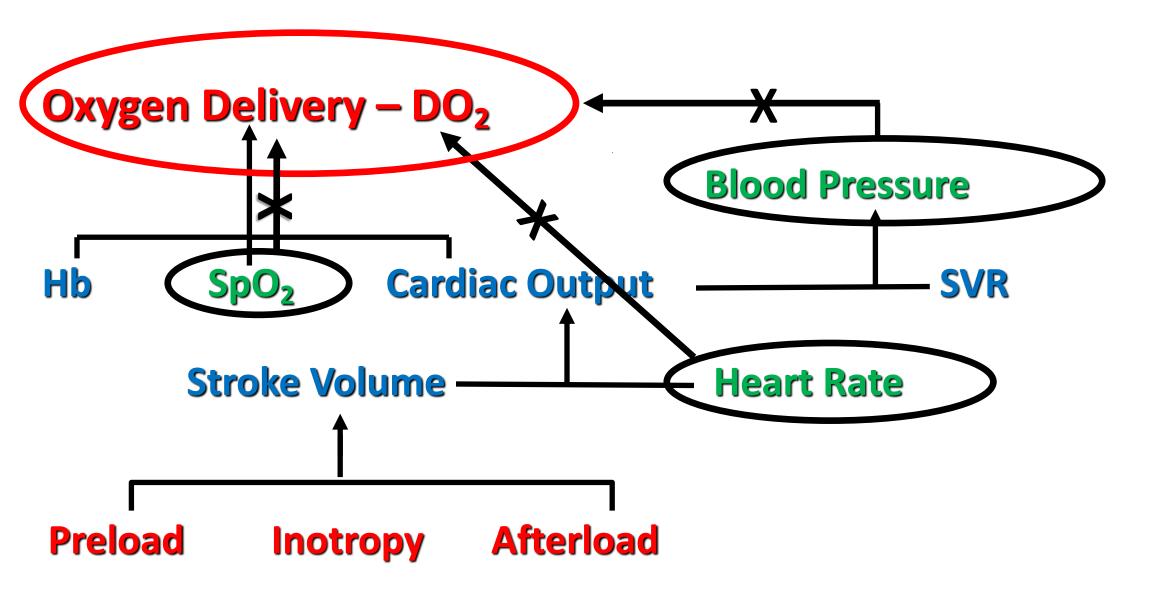


# Getting The Full Hemodynamic Picture BP – HR – CVP are not real hemodynamics!



# Getting The Full Hemodynamic Picture BP – HR – CVP are not real hemodynamics!





BP – HR – CO are not the real hemodynamics.. They are just nice to know

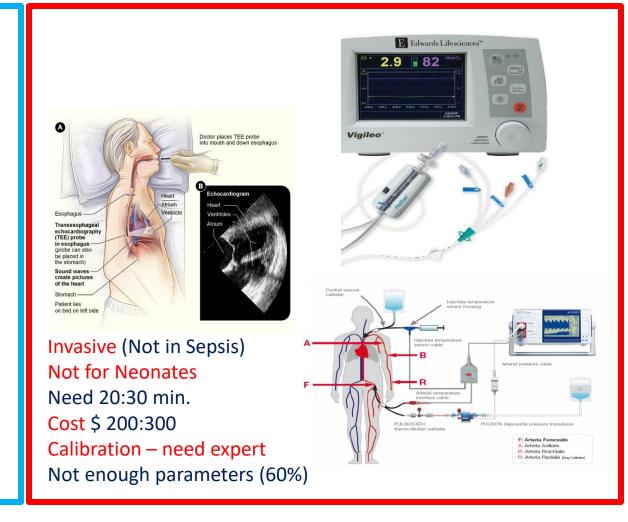
### Reliable Hemodynamics Technologies



Non-Invasive
Continuous (BBB)
User Independent
Validated
Full parameters
Quick and easy



Non-Invasive
Not Continuous
Need expert
No running cost
80% of parameters



### Fluid Management

#### Many critical conditions need proper fluid management:

- Neonates, CoVid-19, Sepsis, Burn, Shock
- <u>Dehydration</u>, <u>Pneumonia</u>, Pancreatitis,
   Bleeding,...etc

How do we know which patient needs fluid?

NIBP (late to show), CVP? Weight? Urine?

What type of fluid to give?
How much shall I give?
And when shall I stop?



review of

Dynamic Table 2. Ability of dynamic and static hemodyresponsiv namic variables to predict volume responsiveness: pooled data with 95% confidence intervals

Data Sources: trolled Trials an

Objectives: A s the ability of dyna ables to predict static indices of 1 tient's intravascul critical care med ables have proven ness. Dynamic chastroke volume in pemerged as useful Data Sources:         PPV (78 (.7482) (.72 (.6577) (.6577) (.86 (0.82-0.90) (.82-0.90) (.82-0.90) (.84 (0.78-0.88) (.72 (.6678) (.72 (.6678) (.72 (.6678) (.73 (.73 (.73 (.7482) (.7482) (.7482) (.7482) (.75 (.7482) (.7482) (.75 (.7482) (.75 (.7482) (.75 (.7482) (.75 (.7482) (.75 (.7482) (.75 (.7482) (.75 (.75 (.7482) (.75 (.75 (.75 (.75 (.75 (.75 (.75 (.75	Paul E. Marik,		Correlation (r)	AUC
	the ability of dyna ables to predict of static indices of filent's intravascul critical care med ables have proven ness. Dynamic chastroke volume in pemerged as usefu	SPV SVV LVEDAI GEDVI	.72 (.65–.77) .72 (.66–.78) —	0.86 (0.82–0.90) 0.84 (0.78–0.88) 0.64 (0.53–0.74) 0.56 (0.37–0.67)

nty-nine studies (which criteria. Overall, 56% of The pooled correlation ressure variation, stroke tion, and the change in d 0.72, respectively. The eristic curves were 0.94. vith 0.55 for the central diastolic volume index, astolic area index. The 6 for the pulse pressure

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Conclusions: Dynamic changes of arterial waveform-derived variables during mechanical ventilation are highly accurate in predicting volume responsiveness in critically ill patients with an accuracy greater than that of traditional static indices of volume responsiveness. This technique, however, is limited to patients who receive controlled ventilation and who are not breathing spontaneously. (Crit Care Med 2009; 37:2642–2647)

# Predicting fluid responsiveness in cardiac postoperative children, Electrical Cardiometry Hemodynamics

- Postoperative <u>fluid management is a milestone of post</u> <u>operative period</u> concerning neonates with congenital heart disease <u>and critical patients</u>.
- Electrical Cardiometry (EC), (ICON®) is a continuous noninvasive hemodynamic monitor used routinely in our unit.
- Objective: This study try to evaluate reliability stroke volume variation (SVV), to predict fluid responsiveness of our patients compared to classical parameters.



## Predicting fluid responsiveness in cardiac postoperative children, Electrical Cardiometry Hemodynamics

- Patients were prospectively included in postoperative period.
- Stroke volume (SV), (SVV) on EC & cardiac output, central venous pressure, left auricular (LA) pressure, invasive blood pressure, saturation, echo velocity time variation were noted by Echo
- 90 patients were included (Ave. 6.5 months, 6.4 kg)
- Responders to volume expansion (VE) had an increase in SV of at least 15% after Volume given.





# Predicting fluid responsiveness in cardiac postoperative children, Electrical Cardiometry Hemodynamics

- SVV and SV were the only parameter which is significantly different with area under curve (19% versus 13%) between patients with or without VE (Volume Expansion)
- All other parameters (clinical, echo. or invasive measures) have no significant difference and too low AUC, including delta aortic peak flow velocity on echo. and delta invasive blood pressure.
- These preliminary results (of electrical cardiometry) confirms the reliability of SVV, which seems to be more practical and reliable than classical and invasive parameters

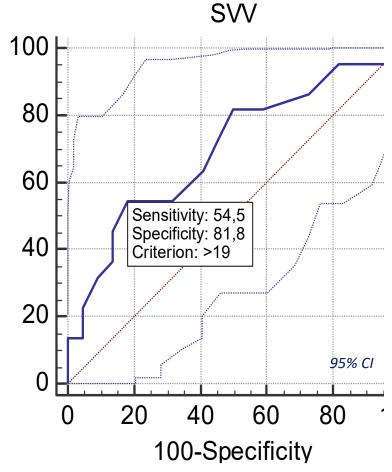
Conclusions: Noninvasive measures of SVV and SV using ICON seems to give reliable data to guide fluid management in postoperative period.



### Responders versus Non Responders

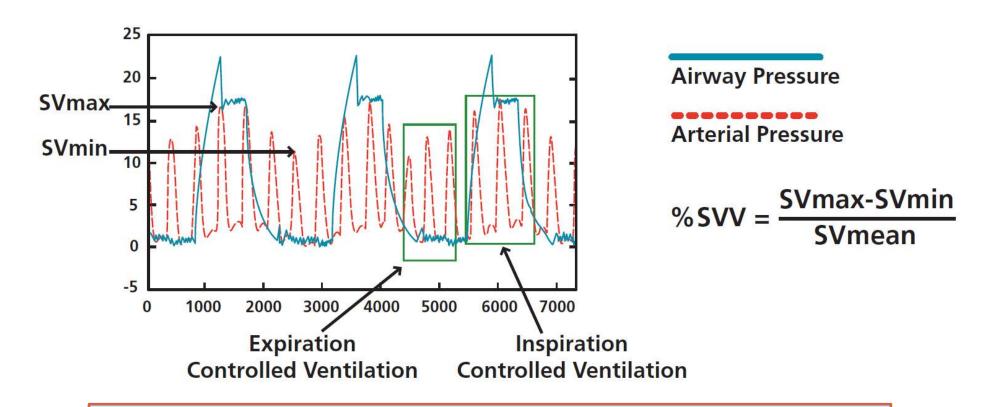
	Responders (n=40)	Non Responders (n=41)	р
SI (ml/kg)	0.79 (0.67-0.91)	1.15 (1.01-1.29)	0.0001
SVV (%)	<b>19.4</b> (17.2-21.6)	<b>13</b> (11-15)	0.0001
ICON (UI)	<b>33</b> (23-43)	<b>56</b> (48-64)	0.021

No Significant Difference in Other Parameters or Tools CVP, LAP, DBP, VT<sub>us</sub>

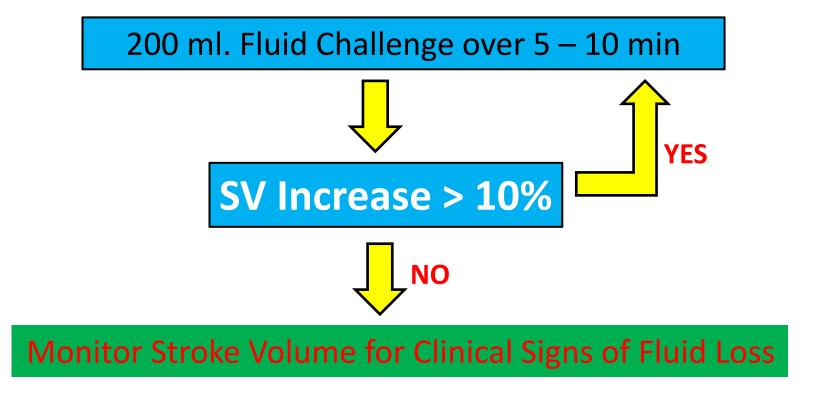




# SVV is a reliable indicator of preload responsiveness on control-ventilated and regularly breathing patients

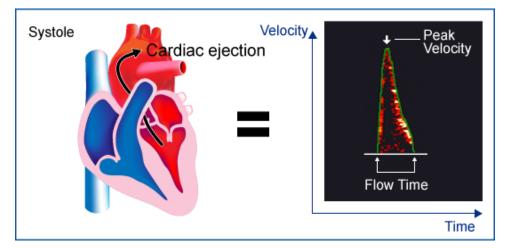


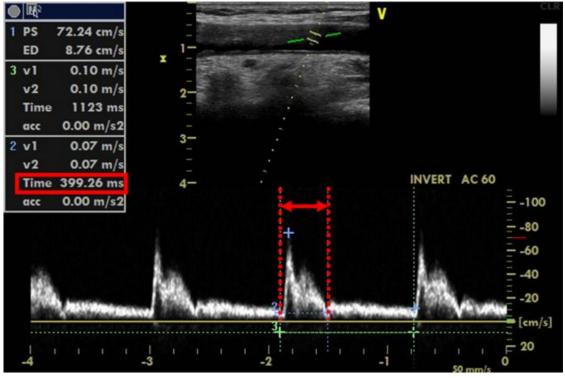
### Stroke Volume Optimization



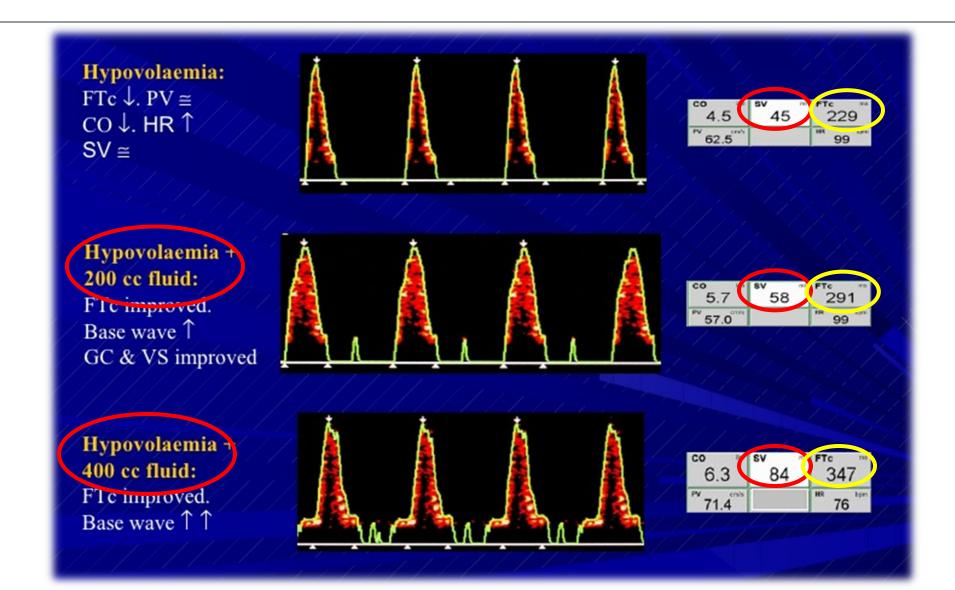
% Change in Stroke Volume (ΔSV) is a sensitive method for assessing preload responsiveness on all patients.

### **Corrected Flow Time**

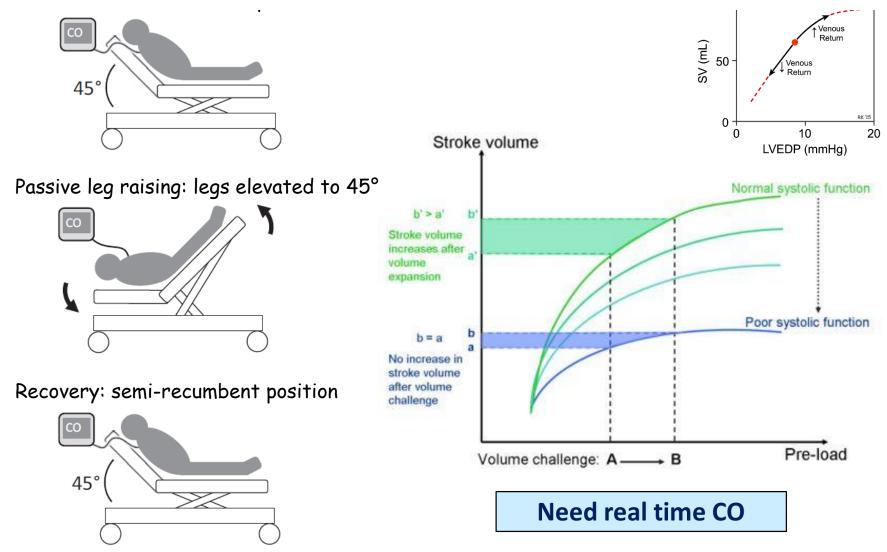




#### Corrected Flow Time



# Passive Leg Raising Test for fluid responsiveness in patient with spontaneous breathing



### Definition, Classification, Etiology of shock

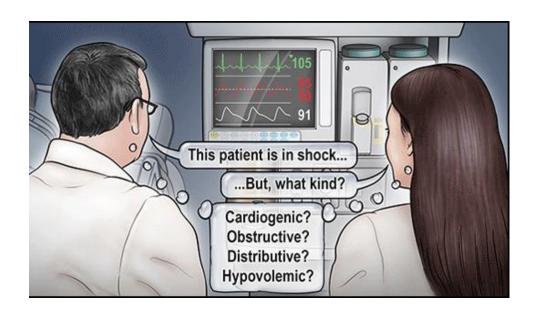
- Shock is a <u>life-threatening</u> condition of circulatory failure, causing <u>inadequate oxygen delivery</u> to meet body needs, producing <u>cellular and tissue hypoxia</u>.
- The effects of shock are <u>initially **reversible**</u>, but rapidly become <u>irreversible</u>, resulting in multi-organ failure (MOF) and death.
- When a patient presents with <u>undifferentiated shock</u>, it is important that we immediately <u>initiate therapy</u> while <u>rapidly</u> <u>identifying the type of shock</u> so that <u>exact therapy</u> can be administered to reverse shock and <u>prevent MOF</u> and death.

### Treatment / Management of Shock

- Maintain airway and breathing with oxygen and oral mechanical ventilation when needed.
- <u>Peripheral IV access</u> or intraosseous infusion (IO) access should be obtained.
- Central venous access may be required if there is difficulty securing peripheral venous access, or the patient needs prolonged vasopressor therapy or large-volume resuscitation.
- Immediate treatment with intravenous (IV) fluid should be initiated, followed by vasopressor therapy, if needed, to maintain tissue perfusion. Depending on the underlying etiology of shock, specific therapies might also be needed.

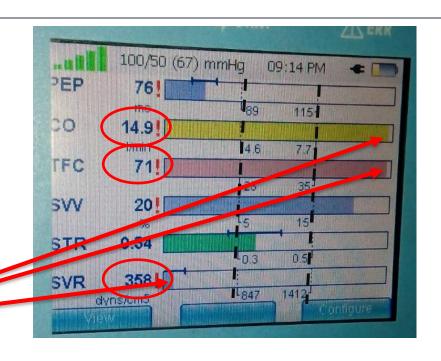
#### What we need to do ASAP?

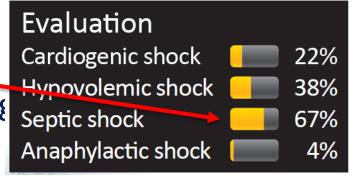
- <u>Identify the type of shock</u> (Cardiogenic, Septic, Hypovolemic,...etc.)
- <u>Determine and start the appropriate treatment</u> before irreversible damage
- Fluid (what type, how much & response)
- Vasopressor or Inotrope?
   which of them or both?
   How much?... Response?
- Continuous follow up and do drug titration
- See response and modify dose when needed



#### A Shock case

- 60 Years old Male
   80Kg, 170 cm, <u>BP 100/50, 39.4C</u>,
   <u>HR 116, RR 23</u>, semi-conscious, <u>no</u>
   <u>Urine for one day</u>
- Hemodynamics: Hyperdynamic State.
- Very Low SVR, very high CO
- Chest Congestion
- Sepsis primarily diagnosed based on
- Shock DDx. Algorithm, confirmed later by very hig Lactate



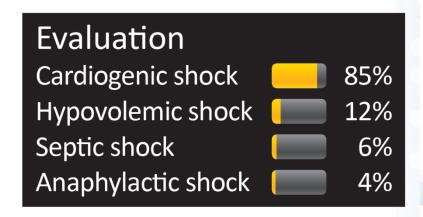


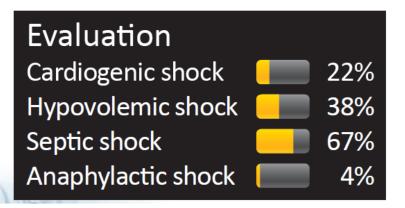
### New Application – Shock DDx



# ShockDDXTM

Shock Differential Diagnosis





This study aims to evaluate the effect of an early fluid bolus administered to children with septic shock on the cardiac index and mean arterial pressure, as well as on the hemodynamic response and its relationship with outcome.

We prospectively collected hemodynamic data from **children with septic shock** presenting to the emergency department or the PICU **who received a fluid bolus** (10 mL/kg of Ringer's Lactate over 30 min).

A <u>clinically significant response in cardiac index-responder and mean</u> <u>arterial pressure-responder</u> was defined as <u>an increase of greater than</u> <u>or equal to 10%, 10 minutes after a fluid bolus</u>.

Suchitra Ranjit, MD, FCCM1 Rajeswari Natraj, DNB, IDPCCM1 Niranjan Kissoon, MB BS, FRCP(C), FAAP, MCCM, FACPE2 Ravi R. Thiagarajan, MB BS, MPH3,4 Balasubramaniam Ramakrishnan, MSc Biostatistics5 M. Ignacio Monge García, MD6 © 2021 by the Society of Critical Care Medicine and the World Federation of Pediatric Intensive and Critical Care Societies

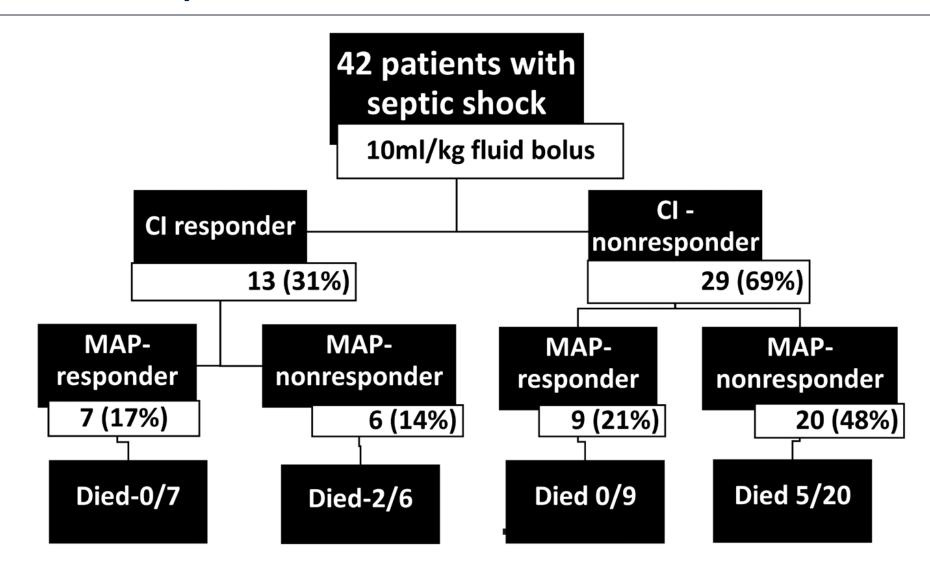
42 children with septic shock, <u>1 month to 16 years old</u>, of whom <u>66%</u> were hypotensive and received a fluid bolus within the first hour of <u>shock recognition</u>.

Cardiac index and mean arterial pressure-responsiveness rates were 31% and 38%, respectively.

Cardiac function was similar in mean arterial pressure- and cardiac index-responders and non-responders.

Mean arterial pressure responders increased systolic, diastolic, and perfusion pressures after a fluid bolus due to higher indexed systemic vascular resistance and arterial elastance index.

Mean arterial pressure-non-responders required greater vasoactive-inotrope support and had higher mortality.



#### TABLE 2.

Hemodynamic Changes According to Cardiac Index Responsiveness

Variables	Baseline	Post Fluid Bolus		
CI, L/min/m <sup>2</sup>				
CI-responders	4.3 ± 1.1	$5.3 \pm 1.2^{b}$		
Cl-nonresponders	$4.9 \pm 1.4$	$4.5 \pm 1.4^{b}$		
Stroke volume index, mL/m <sup>2</sup>				
CI-responders	35 ± 10	$40 \pm 10^{b}$		
Cl-nonresponders	36 ± 14	$34 \pm 13$		
CVP <sup>a</sup> , mm Hg				
CI-responders	7 ± 3	7 ± 3		
Cl-nonresponders	6 ± 3	7 ± 4 <sup>b</sup>		
Heart rate, beats/min				
Cl-responders	127 ± 25	$135 \pm 23$		
Cl-nonresponders	144 ± 32	$139 \pm 28^{b}$		
Systemic vascular resistance index, dynes-sec-cm <sup>-5</sup> /m <sup>2</sup>				

#### TABLE 3.

Hemodynamic Changes According to Mean Arterial Pressure-Responsiveness

Variables	Baseline	Post Fluid Bolus		
Cardiac index, L/min/m <sup>2</sup>				
MAP-responders	5.1 ± 1.5	5.4 ± 1.4		
MAP-nonresponders	$4.4 \pm 1.2$	$4.3 \pm 1.2$		
Stroke volume index, mL/m <sup>2</sup>				
MAP-responders	$38 \pm 12$	40 ± 12		
MAP-nonresponders	$34 \pm 13$	$33 \pm 12$		
CVPa, mm Hg				
MAP-responders	6 ± 3	6 ± 2		
MAP-nonresponders	6 ± 3	8 ± 4 <sup>b</sup>		
Heart rate, beats/min				
MAP-responders	137 ± 26	$139 \pm 25$		
MAP-nonresponders	139 ± 34	137 ± 28		

CONCLUSIONS: The hemodynamic response to a fluid bolus in pediatric septic shock was <u>variable and unpredictable</u>.

We failed to find a relationship between mean arterial pressure & CVP and cardiac index changes.

The adverse effects of fluid bolus extended beyond fluid overload and, in some cases, were associated with reduced mean arterial pressure, perfusion pressures, and higher vasoactive support.

Non-responders to CI and MAP had increased mortality.

The response to the initial fluid bolus may help understand each patient's individualized physiologic response and guide continued hemodynamic management and better outcomes.

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**Review Article: Special Edition** 

# Hemodynamic monitoring and management of pediatric septic shock

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#### Hemodynamic monitoring and management of pediatric septic shock

- Sepsis remains a major cause of morbidity and mortality among children worldwide.
- Furthermore, refractory septic shock and multiple organ dysfunction syndrome are the most critical groups which account for a high mortality rate in pediatric sepsis, and their clinical course often deteriorates rapidly.
- Resuscitation based on hemodynamics can provide objective values for identifying the severity of sepsis and monitoring the treatment response.

#### Hemodynamic monitoring and management of pediatric septic shock

- Hemodynamics in sepsis can be divided into 2 groups (<u>basic and</u> <u>advanced hemodynamic parameters</u>).
- Previous therapeutic guidance of early-goal directed therapy
   (EGDT), which resuscitated based on the basic hemodynamics
   (central venous pressure and central venous oxygen saturation
   (ScvO2)) has lost its advantage compared with "usual care".
- Optimization of advanced hemodynamics, such as cardiac output, SVV, and systemic vascular resistance, has now been endorsed as better therapeutic guidance for sepsis.

#### Hemodynamic monitoring and management of pediatric septic shock

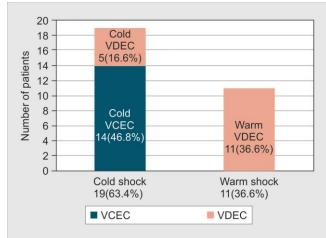
- Early recognition, resuscitation and initial management of pediatric septic shock can improve outcomes.
- When septic shock is recognized, crystalloid challenge is recommended after a <u>rapid evaluation of the hemodynamics</u>, such as the HR, SV, CO, SVV, SVR level, and lactate level.
- Evaluation of Hemodynamics is suggested to continue beat by beat to manage inotrope and fluid administration.
- Assessment of fluid responsiveness should be conducted to decide whether or not to continue volume expansion, then vasoactiveinotropic agents should be administered based on the CI, SVV and SVRI hemodynamics.
- Clinicians should monitor the dynamic changes in these hemodynamic parameters continuously until they are optimized.

# Hemodynamic Categorization and Assessment of Fluid Responsiveness in Ped. Septic Shock by E. Cardiometry

- 30 children were enrolled over 6 months with a median age of 87 months and pediatric risk of mortality (PRISM) III score of 6.75.
- Clinically, 19 (63.3%) children had <u>cold shock</u> and 11(36.7%) had <u>warm shock</u>; however, 16 (<u>53.3%</u>) children had <u>VDEC</u> (including five with clinical cold shock) and 14 (<u>46.7%</u>) had <u>VCEC</u> using electrocardiometry.

• Fluid responsiveness was seen in 16(53.3%) children, 10 in the VCEC group and 6 in the VDEC group.

- In the VCEC group, the responders had a significant rise in CI and a fall in SVRI.
- While the responders in the VDEC group had a significant rise in CI and SVRI.



# Hemodynamic Categorization and Assessment of Fluid Responsiveness in Ped. Septic Shock by E. Cardiometry

• Fluid responders, compared to non-responders, had a significantly higher stroke volume variation (SVV) before fluid bolus (24.1 ± 5.2% vs. 18.2 ± 3.5%, p < 0.001) and a higher reduction in SVV after fluid bolus (10.0 ± 2.8% vs. 6.0 ± 4.5%, p = 0.006).

#### Conclusions and clinical significance:

- Continuous, noninvasive hemodynamic monitoring using electrocardiometry permits hemodynamic categorization and assessment of fluid responsiveness in pediatric septic shock.
- This may provide real-time guidance for optimal interventions, and thus, improve the outcomes.

# Starting IV fluid to Non-Fluid responders, will lead to chest congestion

Table 2: Comparison between fluid respo	onders and no	nresponders				
Parameter	Total $(n = 30)$	Responders	(n = 16) Noni	responders (n =	= 14)	p value
Severity illness scores						
PRISM III scorea	6.75 (1.5, 8.2	5) 6 (2, 8)	13.8 (2.8,16.	5)		0.05
Clinical and biochemical parameters						
Heart rate reduction postbolus (/min)b	15.6 ± 4.5	18.4 ± 4.5	14.0 ± 9.4			0.24
MAP improvement postbolus (mm Hg)b	11.8 ± 9	13.5 ± 8	$8.7 \pm 5.4$			0.23
Lactate prebolus (mmol/L)b		$4.2 \pm 3.3$	$5.5 \pm 2.0$	4.6 ± 3.5		0.82
Electrocardiometry parameters						
SVV before fluid bolus (%)	22.3 ± 5.6	24.1 ± 5.2	18.2 ± 3.5			<0.001
Reduction in SVV after fluid bolus (%	) 8.1 ± 5.1	10.9 ± 2.8	6.0 ± 4.5			0.006
TFC before fluid bolus (k/ohm)b	34.2 ± 12.9	34.2 + 8.2	33.6 + 16.5			0.54
Increase in TFC after fluid bolus (k/ol	nm) 4.2 ± 4.2	2.3 ± 1.8	8.2 ± 3.4			<0.001
Outcome parameters						
Lactate clearance at 6 hrsa (%)	33 (27, 60)	55 (28.6, 58.6	5)	25.4 (6.5, 35.8)	)	0.03
VIS at 6 hrsa	30 (20, 120)	30 (25.5, 50.5	5)	82.5 (20, 120)		0.043
Clinical resolution of shock at 6 hrsc	18 (60)	13 (81)	5(36)			0.01
Clinical resolution of shock at 12 hrsc	21 (70)	15 (93.7)	6 (42.8)			0.01
Need for mechanical ventilation	8 (26.6)	4 (25)	4 (28.4)			0.87
Duration of mechanical ventilation (days	s)8 (2, 10)	3.5 (1.3, 3.8)	5.5 (1.5, 7)		0.34	
Length of PICU stay in survivors (days)a	5 (3, 8)	4 (2, 5)	9.6 (7.3, 17)			0.35
Mortalityc	4 (13.3)	0 (0)	4 (28.5)			0.002

### Case Study – Fluid Management Challenge

- A 77-year-old man is admitted to the ICU with septic shock BP is 88/52 mm Hg with marked respiratory variation (high SVV) which indicates low intravascular volume, on mechanical ventilation, received 4 liters of crystalloids the heart rate is 120 BPM in sinus rhythm, CVP is 6 mm Hg, and the temperature is 35.6°C. He is peripherally cool, with prolonged capillary refill. Arterial blood gas results with a fraction of inspired oxygen of 0.4 are as follows: pH, 7.32; and lactate 13.0 mmol per liter.
- Sodium, 142 mmol per liter; potassium, 4.4 mmol per liter; chloride, 109 mmol per liter; urea, 22.0 mg per deciliter (7.9 mmol per liter); creatinine, 2.3 mg per deciliter (203 µmol per liter); and albumin, 23 g per liter. The urine output during the last 2 hours in the operating room was 28 ml.

### What Fluid to Give?

- BP 88/52
- SVV 23 (low intravascular volume)
- Received 4 Liters of Saline
- Mechanical Ventilation
- HR 120
- CVP 6
- Temp. **35.6**
- Lactate 13
- Urine output: 28ml/2 hours (N. 2 per kg per hour)

What fluid shall we give?

- 1) Saline
- 2) Ringers
- 3) Starch
- 4) Albumin



# How our opinions vary without hemodynamics

#### Poll

After an assessment showing that the patient has inadequate intravascular volume, which one of the following options for fluid resuscitation would you choose to be administered over the next 30 minutes to 1 hour?

A total of 1 liter of normal saline (0.9% sodium chloride).	36%
A total of 1 liter of Ringer's lactate (Hartmann's solution).	26%
A total of 500 ml of 6% hydroxyethyl starch (130/0.42).	6%
A total of 500 ml of 4% human albumin solution.	30%

Hypotension 90/54 .. How shall we manage ? Low intravascular volume ... <u>congested chest</u> → fluid.. What type

Good contractility and heart function & Low SVR → Pressor



# Hemodynamics Inotrope Titration & misleading stable BP

Clinical Diagnosis: Pulmonary Hypertension And PDA

On Dopamine + Adrenaline 0.3 HR170b/m

**BP 70:77/55:60 (Remember this)** 

Fluid: 5ml/kg/h

Urine Output 4.59 ml/kg/h

Vital Signe... All seems OK, but Hemodynamics?



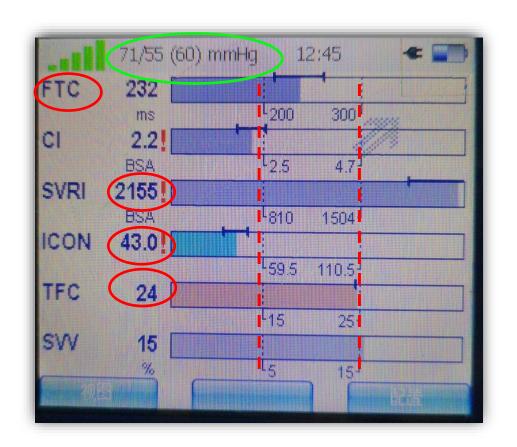
# Hemodynamics Inotrope Titration & misleading stable BP

#### **Hemodynamics?**

Very High SVRI 2155
Low CI 2.2
Low Contractility 43
Normal SVV FTc (Normal Preload)
High Normal Chest Fluid
BP is Normal!!

So, you won't see changes outside

What about adjusting vasoactive drugs? Add Dobutamine?



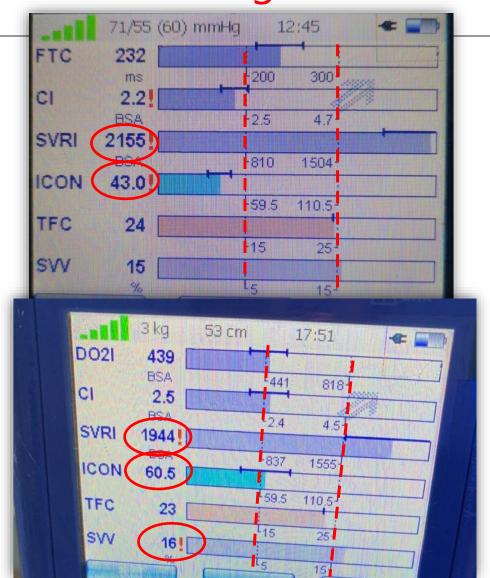
# Hemodynamics Neonatal Case – misleading stable BP

Dobutamine 8ug/kg added 5 hours later:

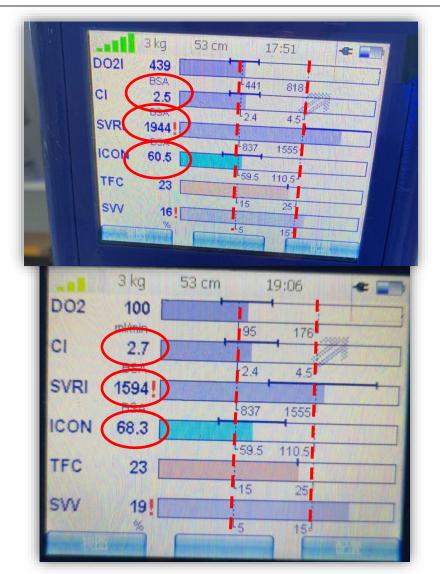
Better SVRI 1950 (from 2155)
Low Normal CI (2.5 instead of 2.2)
Mild High SVV (Low Preload)
Improved Contractility (60 instead of 44)

#### **BP** is still Normal!!

What about adjust vasoactive drugs? Add Fluid (Albumin)?



# Hemodynamics Neonatal Case – misleading stable BP



Increase Dobutamine to 12,
Adrenaline to 0.9

17:50 till 19:06 (75 minutes)
Notice the changes



年龄: 44 代 号	項目	結果	单位	参考值	代号	項目	结果	单位	参考值
FIO2	吸氧浓度	40.0	%	21100	Het	红细胞压积	42.2	%	30-50
T	体温	37.0			t02	总血氧浓度	19.2	ml/dL	1622
pH(T)	酸碱度	7, 372		7. 35-7. 45	tCO2	二氧化碳总量	17.8 4	mmo1/L	24-32
	二氧化碳分压	35, 4	mmHg	3545	SBC	标准碳酸氢根	21.1 4	mmo1/L	22-28
p02(T)	氧分压	113 t	mmHg	83108	HC03-	实际碳酸氢根	20.0 1	mmol/L	2228
tHb	总血红蛋白	138	g/L	120-160	SBE	标准剩余碱	-4.3 1	mmol/L	-33
	总血氧饱和度	99.61	%	9398	ABE	实际剩余碱	-4.0	mmol/L	-33
K+	钾	4.0	mol/I	3.55	p02(a)/F02(	氧合指数	283 4		400500
Na+	钠	139	mol/I	136146					
Ca2+	钙	1.24	mol/I	1. 15-1. 29					
Glu	血糖	6.5 t	mol/I	3.95.8					
Lac	乳酸	1.71	mol/I	0.51.6					
送检日期	2019/09/05 报1	告日期 20	019/09/05		检验员	梁燕桂		送检医生	谢友军

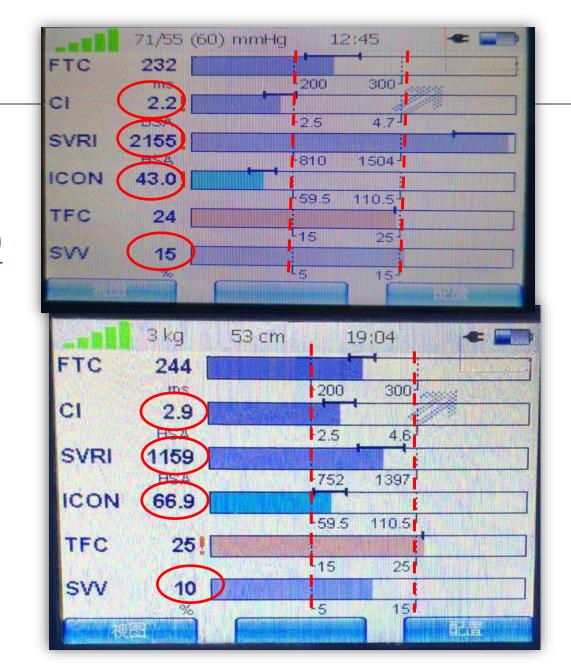
# Admission to Discharge How easy to manage

@ 19:04 pm next day

Normal SVRI 1159 (from 2155)
Normal CI (2.9 instead of 2.2)
Normal SVV/FTc (Normal
Preload)

Normal Contractility (61 instead of 44)

BP is still 70/55!!!





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# Fluids and Early Vasopressors in the Management of Septic Shock: Do We Have the Right Answers Yet?

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# Fluids and Early Vasopressors in the Management of Septic Shock: Do We Have the Right Answers Yet?

Septic shock is a common condition associated with <u>hypotension and organ</u> <u>dysfunction</u>.

It is associated with <u>high mortality rates</u> despite the best recommended resuscitation strategies in international guidelines.

Patients with septic shock have <u>hypotension which is the most important</u> <u>determinant of mortality among this group of patients.</u>

The extent and duration of hypotension are important.

The two initial options that we have are

- 1) administration of intravenous (IV) fluids and
- 2) vasopressors,

# Fluids and Early Vasopressors in the Management of Septic Shock: Do We Have the Right Answers Yet?

The current recommendation of the Surviving Sepsis Campaign guidelines to administer 30 ml/kg fluid (10 ml/kg for children) cannot be applied to all patients.

<u>Complications of fluid over-resuscitation further delay organ recovery, prolong ICU and hospital length of stay, and increase mortality.</u>

The only reason for administering intravenous fluids in a patient with circulatory shock is to increase the mean systemic filling pressure in a patient who is volume-responsive, such that cardiac output also increases.

The use of vasopressors seems to be a more appropriate strategy, the very early administration of vasopressors, preferably during the first hour after diagnosis of septic shock, may have a multimodal action and potential advantages, leading to lower morbidity and mortality in the management of septic patients.

<u>Vasopressor therapy should be initiated as soon as possible in patients with septic shock while monitoring hemodynamics regularly.</u>

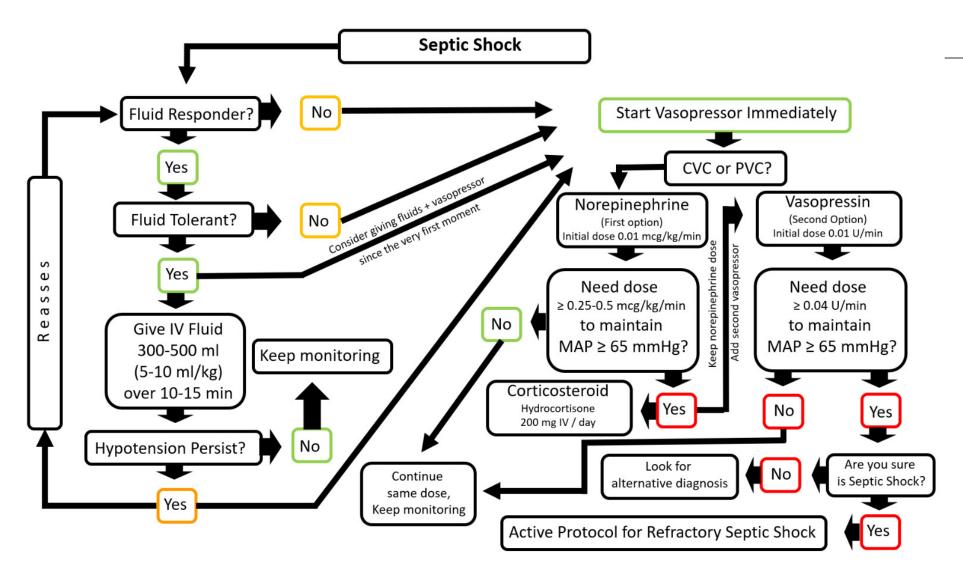


Fig. 1. Rational approach and management of septic shock with intravenous fluids and early vasopressors based on current evidence. IV: Intravenous, CVC: Central Venous Catheter, PVC: Peripheral Venous Catheter.





# Noninvasive cardiac output monitoring in septic shock patients

#### A retrospective study on hemodynamic status and outcomes

Yu-Jang Su, MDa,b,c,d,\*, Sheng-Teck Tan, MDa and Yasser Nassef, MD, PhDe

#### Abstract

Septic shock is a frequent condition in emergency departments, requiring rapid hemodynamic assessment. Noninvasive cardiac output monitoring (NICOM) offers a convenient method for evaluating these patients. In this study, we retrospectively analyzed 50 septic shock patients (34 males, 16 females) from a cohort of 627 NICOM cases in northern Taiwan emergency department between January 2020 and December 2021. Patients were classified into normal and high stroke volume variation percentage groups, and survivors versus non-survivors. The high stroke volume variation percentage group had an older average age (72.1 vs 59.5, P = .004) and required more fluid resuscitation before inotropic agents (1322 mL vs 864 mL, P = .043). Non-survivors were older (77.6 vs 64.7 years, P = .013), had higher NT-proBNP levels (655 vs 307, P = .029), and longer ICU stays (3.7 vs 1.2 days, P = .007). The overall mortality rate was 22%. NICOM is a valuable tool for guiding fluid resuscitation in septic shock patients. Further studies are recommended to refine its application.

**Abbreviations:** BNP = B-type natriuretic peptide, CHF = Congestive heart failure, CO = cardiac output, COPD = chronic obstructive pulmonary disease, EC = electrical cardiometry, ED = emergency department, FTC = corrected flow time, HIV = human immunodeficiency, HR = heart rate, ICON = index of contractility, NICOM = noninvasive cardiac output monitoring, NT-proBNP = N-terminal prohormone of brain natriuretic peptide, PAC = pulmonary artery catheters, SBP = systolic blood pressure, STR = systolic time ratio, SV = stroke volume, SvO2 = central venous oxygen saturation, SVR/SVRI = systemic vascular resistance, SVV = stroke volume variation, TFC = thoracic fluid content, WBC = white blood cell count.

**Keywords:** inotropic agent, outcome, sepsis, shock

#### Non-invasive cardiac output monitoring From Jan. 1, 2020 to Dec. 31, 2021 627 cases

276 cases: Not infectious diseases

172 cases: Missing parameters

58 cases : High values of NTproBNP

34 cases : High values of BNP

14 cases : WBC not above 12000/  $\mu$  L

7 cases: Referral from other facilities

6 cases: Young cases with normal variant hypotension

4 cases : Pancreatitis

4 cases: Transferred to other hospitals

2 cases : Against advice discharged

Normal values of SVV% 18 cases High values of SVV% 32 cases

#### Noninvasive cardiac output monitoring in septic shock patients

Table 2 - Comparisons are made via SVV% normal and high value.

	SVV normal	SVV high
Lactate (mg/dL)	23.5 ± 21.6	28.8 ± 27.5

P = .007). The overall mortality rate was 22%. NICOM is a valuable tool for guiding fluid resuscitation in septic shock patients. Further studies are recommended to refine its application.

Albumin (bottle)	1.2 ± 1.4	2.0 ± 1.3
Time to stable (min)	428.5 ± 280.4	560.1 ± 506.2
ICU stay (d)	1.9 ± 5.0	1.6 ± 4.0
LOS (d)	15.7 ± 12.7	14.5 ± 11.2
CO (L/min)	5.0 ± 1.1	5.6 ± 2.1
TFC (1/Zo)	20.7 ± 4.3	23.1 ± 7.0
Icon	39.6 ± 15.0	41.0 ± 19.6
SVV%	10.6 ± 2.9	24.0 ± 12.0
SVRI (BSA)	1465.9 ± 612.9	1701.4 ± 1852.2
CHF	1/18	2/32

# Association between chest congestion and mortality in ICU patients

#### Yasser Nassef<sup>1\*</sup>, Dung-Hung Chiang<sup>2\*</sup>, Ming-Chih Chou<sup>1</sup>

**Purpose:** Recent years have seen significant progress in hemodynamic monitoring and management. There has been an evolution from invasive to less invasive technologies. This study evaluated the relationship between chest congestion via thoracic fluid content (TFC) and mortality among critically ill patients in the Intensive Care Unit (ICU).

**Methods:** This retrospective case-control study assessed 373 patients admitted to the ICU of Taipei Veterans General Hospital from December 2012 to June 2013. In total, 149 individuals (n = 149) were excluded due to incomplete or missing information. Patients who died during the study interval were selected as the case group while the surviving participants represented the control group. The (TFC) thoracic fluid content was collected by an ICON Electrical Cardiometry device in all patients. Mortality odds ratios (ORs) and 95% confidence intervals (CI) were estimated using multiple logistic regression models.

**Results:** A total of 224 patients (84 who died in the ICU and 140 who remained alive) were included in the final analysis. The aOR for mortality was significantly higher in patients with abnormal or high TFC ( $\geq$ 50 kΩ<sup>2</sup>) than in those with 1FC <50 kΩ<sup>2</sup> (aOR, 2.2/8; 95% CI 1.1216-4.268). Results from the sex stratified analysis model showed that the aOR for mortality was significantly higher among men (aOR, 2.209; 95% CI, 1.006-4.848; P=0.0482) but not women (aOR, 2.085; 95% CI, 0.631-6.890; P=0.2284) with TFC  $\geq$ 50 kΩ<sup>-1</sup> compared to TFC <50 kΩ<sup>-1</sup>.

**Conclusions:** In this study, high TFC was associated with a higher mortality rate in critically ill ICU patients. The mortality risk was more pronounced in male patients.

<sup>&</sup>lt;sup>1</sup>Nnstitute of Medicine, Chung Shan Medical University, Taichung City, Taiwan

<sup>&</sup>lt;sup>2</sup> Department of Critical Care Medicine, Taipei Veterans General Hospital, Taipei City, Taiwan.

#### Association between Chest Congestion and ICU Mortality in ICU Patients

- A retrospective case-control study assessed <u>373 patients</u> admitted to the ICU of <u>Taipei Veterans General Hospital</u>.
- 149 individuals (n = 149) were excluded due to incomplete or missing information.
- Patients who <u>died</u> during the study interval were selected as <u>the case</u> group, while the <u>surviving participants</u> represented the <u>control group</u>.
- This study evaluated the relationship between chest congestion via thoracic fluid content (TFC) and mortality among critically ill patients in the Intensive Care Unit (ICU).
- The (TFC) thoracic fluid content was collected by an ICON Electrical Cardiometry device in all patients.
- Mortality odds ratios (ORs) and 95% (CI) were estimated using multiple logistic regression models.

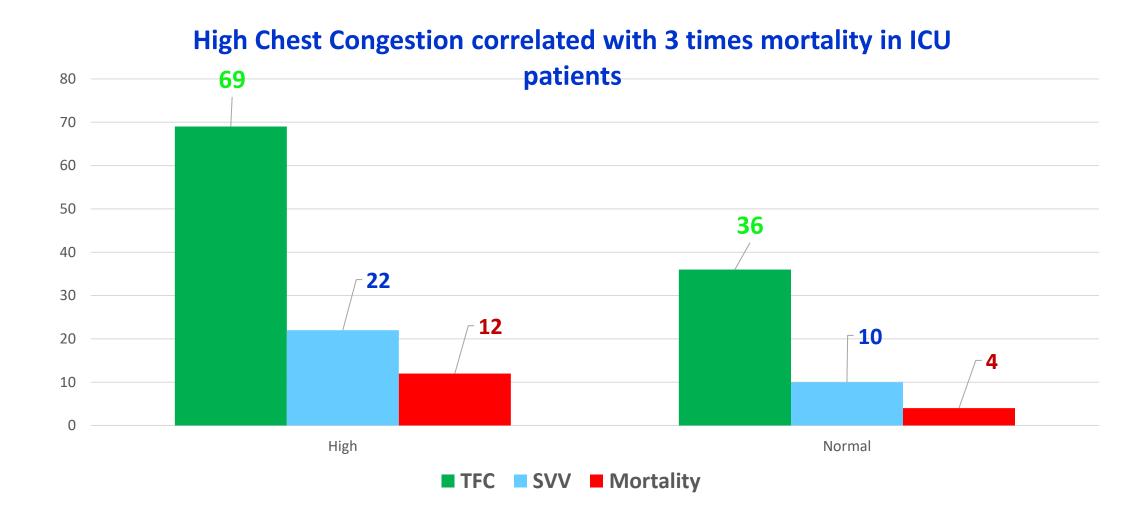
#### Association between Chest Congestion and ICU Mortality in ICU Patients

- After exclusions, <u>224 critically ill patients were recruited</u>.
   Among those selected, <u>84 patients who died during the study period constituted the study group while the rest (n = 140) were enrolled into the control group (Table 1).</u>
- The mean age (±SD) was 66.821 (19.303) years for the study group and 66.814 (17.421) years for the control group.
- The TFC, SVV, weight, DM, and hypertension were significantly different between the two groups while other indexes remained similar

Table 2. Multiple logistic regression analysis showing the association between ICU mortality and potential risk factors

Risk factors	aOR	p-value	95% CI
TFC (ref: <50)			
≥50	2.278	0.0101*	1.216-4.268
Sex (ref: Female)			
Male	1.459	0.3450	0.666-3.196
Age	1.007	0.4274	0.989-1.025
Height	1.026	0.3285	0.975-1.079
Weight	0.975	0.0470	0.951-1.000
DM (ref: No)			
Yes	1.129	0.7655	0.509-2.504
CAD (ref: No)			
Yes	0.681	0.5049	0.220-2.107
Vasopressor agent (ref: No)			
Yes	1.428	0.2589	0.769-2.653

#### Association between Chest Congestion and ICU Mortality in ICU Patients

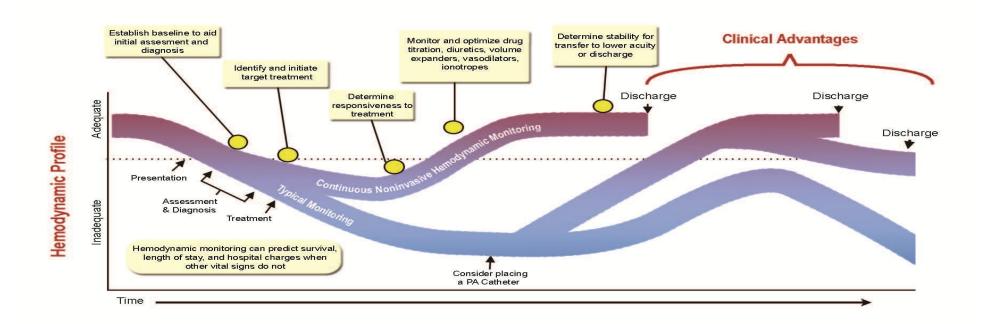


# Non-Invasive Hemodynamic Benefits

- Do Not manage patients without Hemodynamics (EC & FE)
- Look at the whole picture, not single or couple of parameters
- Use Non-invasive, if possible, (No Complications), no Trial and Error
- Continuous (excellent guide for medication titration) is preferred
- Chose easy to use, accurate and reliable (validated from neonates to obese)
- Cost effective
- Valuable additional parameters for your daily practice:
  - Cardiac Contractility, Pre-load (SVV, FTC), After-load (SVR), EF (STR),
  - Tissue Edema (TFC), DO2, CPI, PEP, LVET, LCW, VIC, HRV & HRC

### Cost Effective – Reduce LOS with NIHD

# Continuous, Noninvasive Hemodynamic Monitoring for Cost-Effective, Quality Patient Care



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# Thank You







Yasser Nassef MD PhD
Pediatric Cardiology – Emergency Medicine

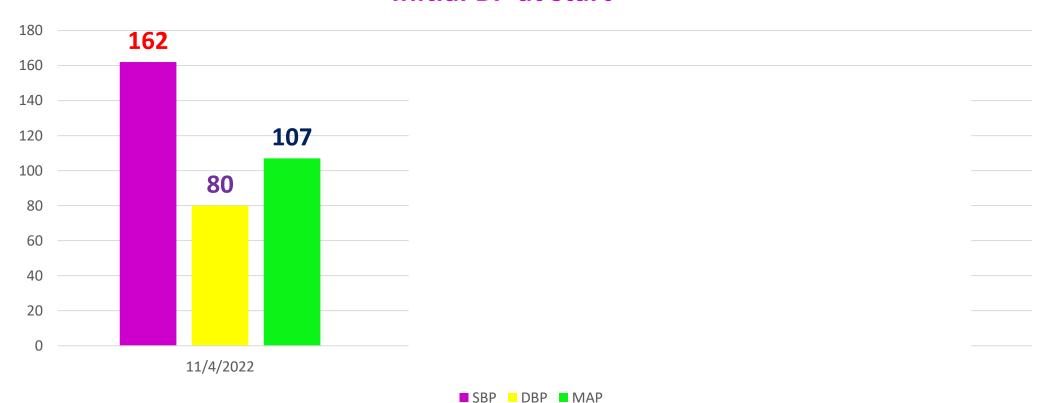
Assistant Professor Chung Shan Medical, NYMCT, Hung Kuang Universities Adjunct Assistant Professor, San Jose & Fu Ren Universities Vice President International Society of Hemodynamics

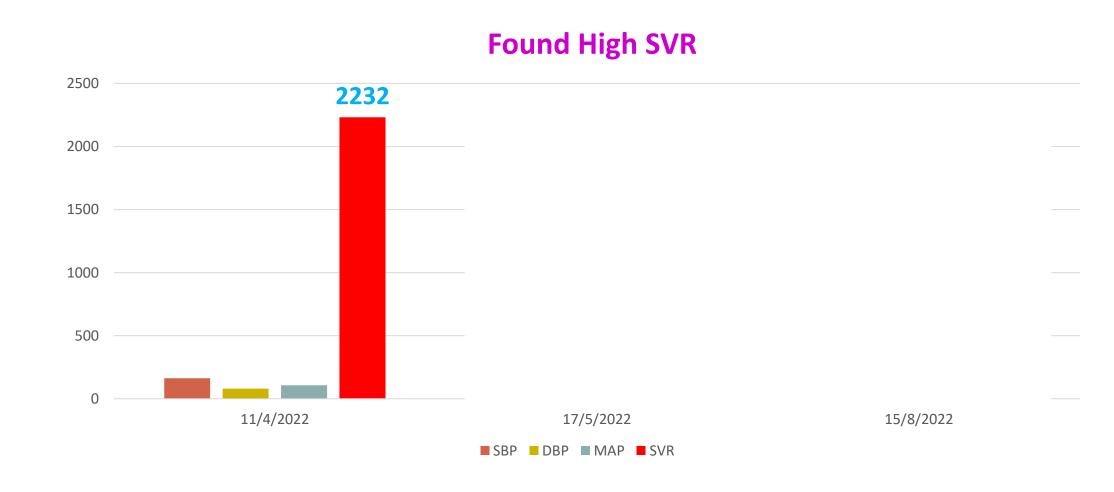


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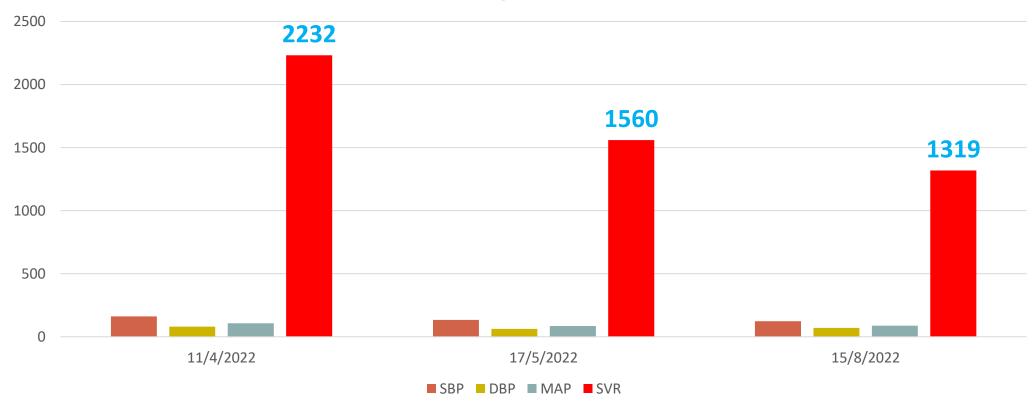
М	2691639	74y/o								
	SBP	DBP	MAP	FTC	SVV	TFC	ICON	CI	SVR	HR
				(300- 500ms)	(5-10)	(25-35)	(40-60)	(3-5)	(900- 1200)	(54-90)
4/11/2022	162	80	107	315	5	20	41.5	2.2	2232	67
5/17/2022	133	62	86	328	9	21	45.6	2.7	1560	73
8/15/2022	123	70	88	348	9	19	41.3	3	1319	79

#### **Initial BP at start**

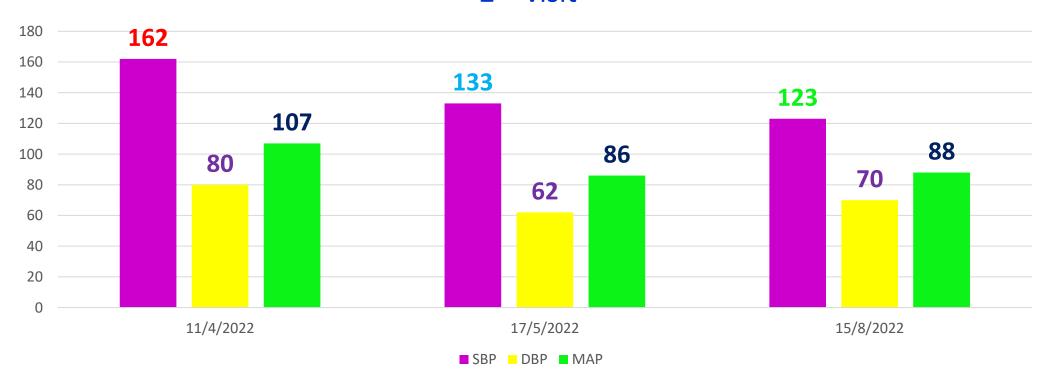




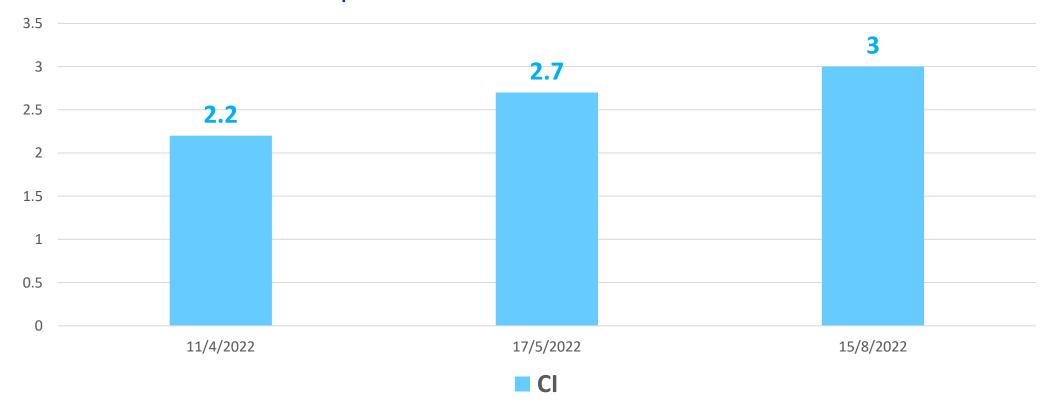




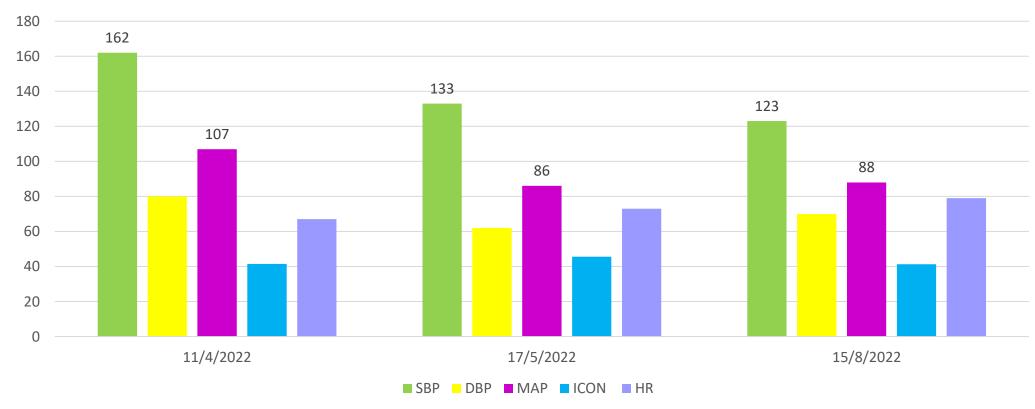
Changes in BP over 3 visits to OPD.. Targeted BP achieved from 2<sup>nd</sup> visit



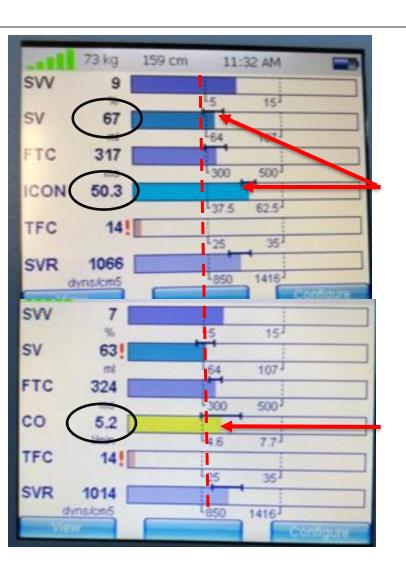
#### Improvement in CI over the 3 visits

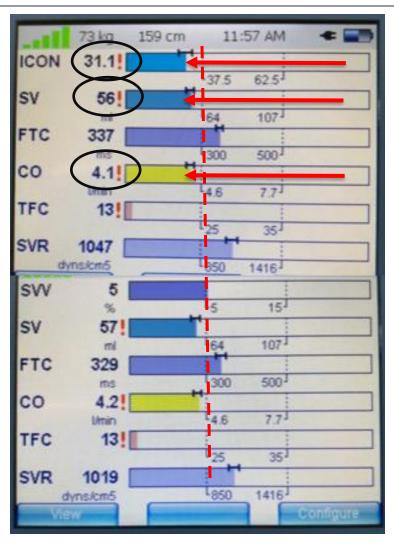






## Can We Predict Sudden Drop of BP 15 Min earlier?





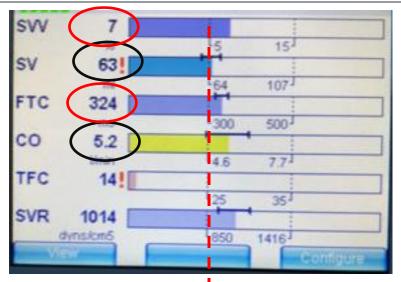
11.32 AM till 12.00

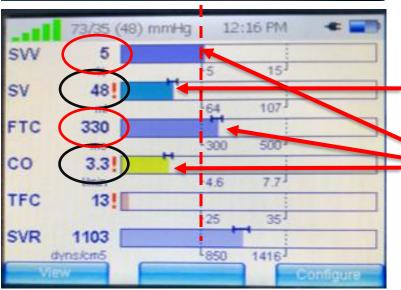
**BP 110/67 BP 105/73**Nothing wrong?

First change to notice: **Decreased CO, Contractility** 

IV fluid is normal So? Shall we wait? As per old school

## Hypotension.. Fluid or Inotrope?





12.16 PM CO and SV keep decreasing

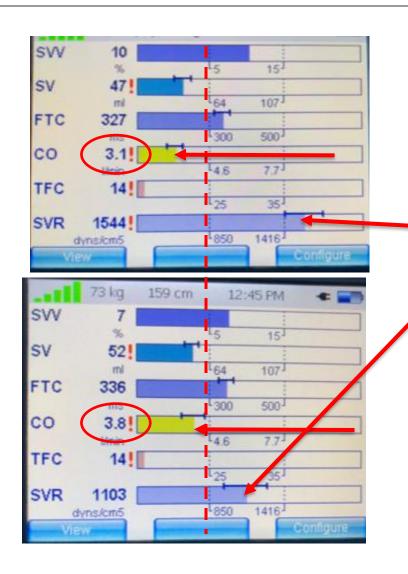
BP suddenly drop to 73/35 !! (12:16)

20 min. <u>after SV, CO, Contractility</u> decreased, and 45 min. <u>from the</u> start

Old school: give IV fluid immediately But IV fluid is still **normal (FTc & SVV)** 

12.42 Inotrope started

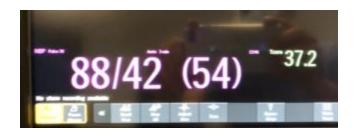
## Beauty of Hemodynamics



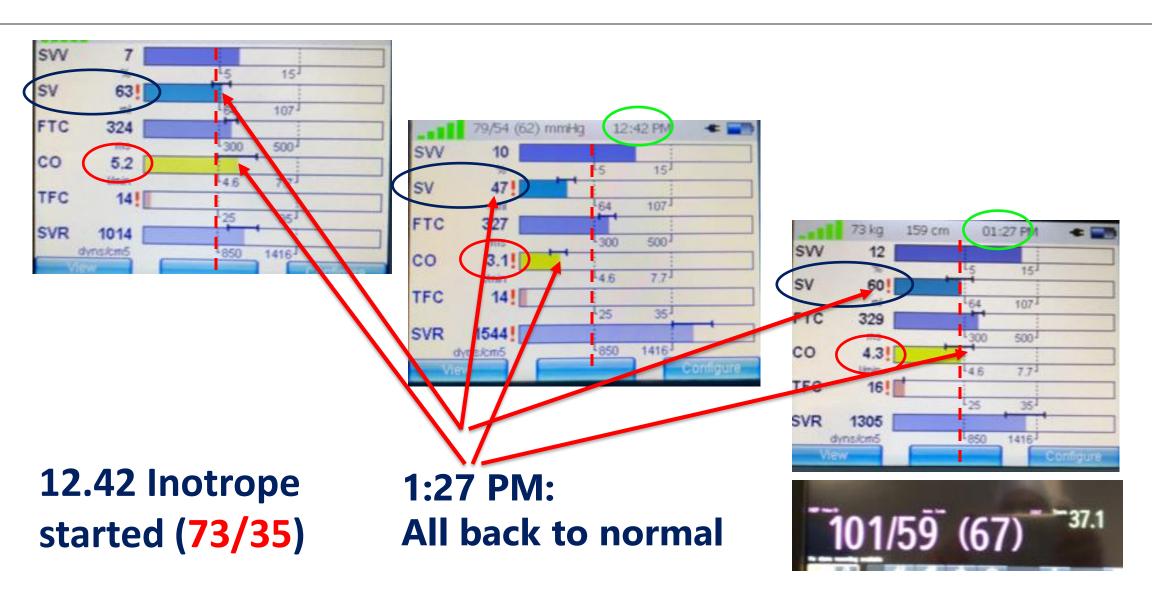
12.42 Inotrope started,

12.45 CO increased by 10% and SVR decreased by 20%

**BP** increased by 10%



### CO recovered and BP Recovered 15 min. later



## Continuous noninvasive cardiac output in Neonates & children Massachusetts G. Hospital Children, Boston, MA, USA

- Initial experience in 402 pediatric/Neonatal patients
- Objective: To determine whether <u>continuous cardiac output</u>
   (CO) <u>data provide additional information to current monitors</u>
   that is useful to <u>practitioners</u>.
- Data from 374 children/Neonates were in the final cohort
- 292,012 measurements during 58,049 min. of anesthesia were made in these children (1 day to 19 years and 1 to 107 kg).

Division of Pediatric Anesthesia, Department of Anesthesia, Critical and Pain Management, Mass General Hospital for Children, Massachusetts General Hospital, Boston, MA, USA



#### **Continuous noninvasive cardiac output in Neonates & children**

Massachusetts G. Hospital Children, Boston, MA, USA

15 : 18		143		1.905	13.37
15 : 19		145	56	1.848	13.06
15:20		141		1.870	13.20
15:21	Caudal injection	144		1.993	13.84
15:22		146	60	2.560	17.58
15 : 23		145		2.482	17.14
15:24		146		2.380	16.42
15:25		149	65	M*	M*
15 : 26		151		3.440	23.06
15 : 27		148		3.523	23.67
15:28		151	48	3.552	23.55   rejidsoH
15:29		152		3.510	22.96
15:31		152	54	3.537	23.24
15:34		155	61	3.723	
15:37		165	63	4.197	25.01
15:40		175	81	4.447	25.61
15:43		159	82	4.227	26.49
					24.06 25.01 25.61 26.49 28Seneral
					_

This was a 2-month-old 4-kg female undergoing bilateral inguinal hernia repair. 1 ml·kg<sup>-1</sup> of 1 : 10 000 epinephrine was administered rather than 1 ml·kg<sup>-1</sup> of 0.125% bupivacaine with epinephrine 1 : 200 000. Cl ↑~37% within 1 min of administration and ↑Cl ~87% 4 min after injection with minimal early changes in other vital signs. Similar changes in other cardiac parameters were noted. This case will be reported in greater detail elsewhere. The error was recognized ~ 5 min later, but in retrospect, the monitor indicated a change several minutes before the error was realized.

√assGeneral H for Children"

## Continuous noninvasive cardiac output in Neonates & children Massachusetts G. Hospital Children, Boston, MA, USA

- Conclusions: <u>EC Hemodynamics provides real-time</u>
   cardiovascular information regarding developing hemodynamic
   events and successfully tracked the rapid response to
   interventions in children of all sizes. Intervention decisions must
   be based on the combined data from all monitors and the
   clinical situation
- Our experience suggests that this type of monitor may be an important addition to real-time hemodynamic monitoring.

 Division of Pediatric Anesthesia, Department of Anesthesia, Critical and Pain Management, Mass General Hospital for Children, Massachusetts General Hospital, Boston, MA, USA Initial experience in 402 pediatric patients



### Hemodynamics vs. Normal Monitor in Shock

Male patient admitted represented with:

39.3 C - 94 Kg - MAP 68 mmHg

HR: 118 - RR 22 - Spo2 87%

Leg swelling, drowsiness,

shortness of breath, confusion

History of repeated renal stones & Infections

(Acute Renal Impairment)??

Would Hemodynamic add any value?



Possible Sepsis with Renal Impairment?

Lactate Clearance came later 26.3 – WBC

# Thank You





International Society of Hemodynamics

Know Early \_ Manage Better

Yasser Nassef MD PhD

Pediatric Cardiology – Emergency Medicine Hemodynamic Clinical Lecturer Assistant Professor Chung Shan Medical University

<u>yasser@nassef.org</u>
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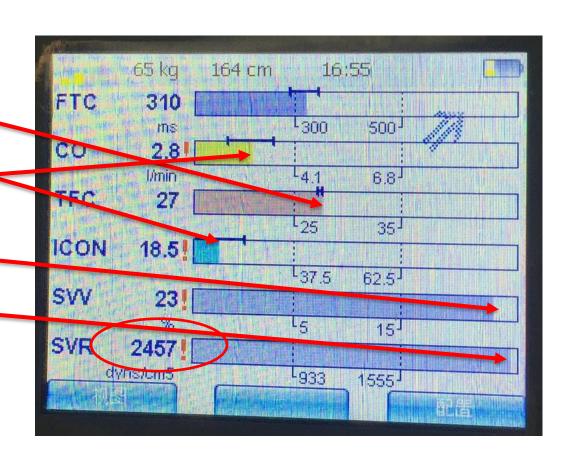
## Hypertension Case

- At admission to ER <u>17.00 pm</u>, Patient adult 65kg., height 164 cm, <u>BP 210/100</u>, headache, <u>Tachycardia</u>, vital signs (ECG, Temp., RR) are normal
- What possible diagnosis?
- What other investigation?

- Initial suggested treatment: <u>Diuretics</u>
- Let us see the hemodynamics

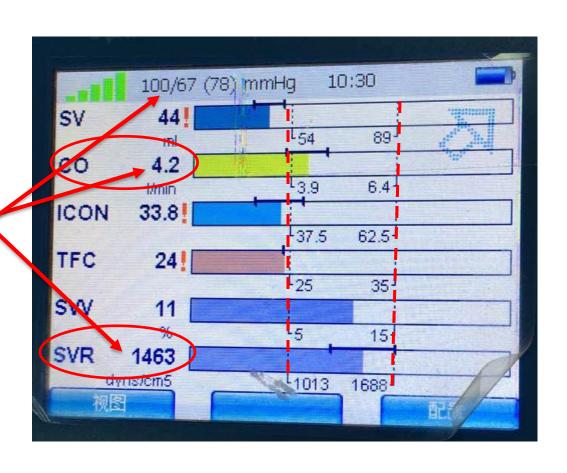
## Hypertension case – Hemodynamic help

- EC ICON Report differential diagnosis:
- Normal body fluid.
- Low contractility
- Low CO
- Low pre-load
- High SVR
- <u>Diuretic was not the best choice</u>



## Hypertension Case – Hemodynamic help

- Started <u>IV fluid</u> .. And <u>vasodilator</u>
- 18 hour later, SVR came down from 2457 to 1463
- CO becomes <u>4.2</u>
   (initially was <u>2.8</u>)
- Preload back to normal
- BP <u>100/67</u>
- Goal achieved



## Hypotension.. Standard monitor .. Will It help?

### **Case Study 3**

A 71-year-old man in the ER with sudden hypotension of unknown origin

He is unresponsive with a Glasgow coma scale of 4.

Vital sigs: **BP 92/63 mmHg** 

Pulse 101 beats/min

RR 26/m

Shall we start IV fluids as per the guidelines?

Can hemodynamics help in making better decisions?

## Getting Hemodynamics. Goal achieved

```
Case Study 3 (continued)

CO
3.9 L/min

CI
2.3 L/min/m <sup>2</sup>

SVR

SVV

1850 dynes/sec/cm

11%
```

CI and CO are low. HR, and SVR are elevated.

- <u>Dobutamine</u> is started to the patient
- One hour after the Dobutamine, hemodynamics reveals the following:

## Titrating Medications..Goal achievdd

**Case Study 3 (continued)** 

#### **After and Before 1-hour treatment (Dobutamine)**

```
CO 4.4 L/mi
Cl 2.6 L/min/m<sup>2</sup>
Cl 2.3 L/min/m<sup>2</sup>
SVR 1530 dynes/sec/cm
NIBP 110/74 mmHg
NIBP 92/63 mmHg
Pulse 92 beats/min
CO 3.9 L/mi
Cl 2.3 L/min/m<sup>2</sup>
SVR 1850 dynes/sec/cm
NIBP 92/63 mmHg
Pulse 101 beats/min
```

## MI with Heart Failure follow up on OPD

#### Titration of an ACE Inhibitor

Visit	Signs/Symptoms	CI	SVRI	TFC
#1	SOB, Inability to sleep   HR 81   BP 112/88	2.3	3136	32.5

**Patient**: 54 year old male.

**History:** Heart failure with previous CABG surgery.

**Current** ACE inhibitor (Enalapril 10 mg bid), inotrope (Digoxin

**Therapy**: 0.125 mg qd), diuretic (Furosemide 20 mg qd).

- NIHD Interpretation: High SVRI indicates vasoconstriction.
- Treatment Decision: Increase Enalapril to 20 mg bid, increase Furosemide to 40 mg qd.

## MI with Heart Failure follow up on OPD - Cont.

Visit	Signs/Symptoms	CI	SVRI	TFC
#1	SOB, Inability to sleep HR 81 BP 112/88	2.3	3136	32.5
#2 (1 Week)	SOB resolved HR 97 BP 98/72	3.5	<u>1277</u>	30.3

#### NIHD Interpretation:

- Up titration of Enalapril reduced SVRI and increased CI.
- Decrease in TFC indicates responsiveness to Furosemide.
- Hemodynamic improvement provided objective confirmation of resolution of symptoms.

#### **Treatment Decision:**

Re-evaluate in two weeks for consideration of initiation of beta-blocker (Metoprolol or Carvedilal)

## Utility of ICG to determine cardiac vs. noncardiac cause of dyspnea in ER

Table 1. Summary of Hemodynamic Statistics (N=38)*				
CHARACTERISTIC	Cardiac Final Dx (n=12)	NONCARDIAC FINAL DX (N=26)	P Value	
SI (mL/m²)	31.8 (25.3-38.4)	35.9 (22.9-38.9)	0.24	
CI (L/min/m²)	2.2 (1) 9–2.5)	3.1 (2.9–3.3)	< 0.0001	
SVRI (dyne × s × cm <sup>-5</sup> × m <sup>2</sup> )	2742 (2066-3420)	2106 (1903-2309)	< 0.05	
VI (/1000 s)	32.9 (25.9-39.9)	42.7 (28.4-47.1)	< 0.01	
STR	0.52 (0.37-0.68)	0.37 (0.33-0.41	< 0.01	
Dx=diagnosis; SI=stroke index; CI=cardiac index; SVRI=systemic vascular resistance index; VI=velocity index; STR=systolic time ratio;				
*values are expressed as mean (95%	confidence interval)			

Table II. Summary Diagnosis Statistics (N=38)				
Метнов	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
ICG	92	88	79	96
ED physician	83	77	63	91

Sensitivity=probability that a person with a true cardiac cause will test cardiac by the method; specificity=probability that a patient with a true noncardiac cause will test noncardiac by the method; PPV=positive predictive value, the probability that a person who tests cardiac is truly a cardiac cause; NPV=negative predictive value, the probability that a person who tests noncardiac is truly a noncardiac cause

Charles L Springfield 1, Frank Sebat, David Johnson, Steven Lengle, Christian Sebat Congest Heart Fail - 2004 Mar-Apr;10(2 Suppl 2):14-6

## Heart Rate Variability Analysis is More Sensitive at Identifying Neonatal Sepsis than Conventional Vital Signs

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## Dyspnea D.D.

#### Cardiac or Pulmonary Cause?

- Patient: 81 year old female.
- History: Hypertension, left ventricular hypertrophy, mitral regurgitation, atrial fibrillation, chronic obstructive pulmonary disease.
- Current therapy Inotrope (Digoxin 0.125 mg qd), ACE inhibitor (Enalapril 5 mg bid).

Exam / Symptoms	CI	SVRI	TFC
Rales, ronchi, Shortness of breath	<b>2.8</b> (2.5 – 4.2)	<b>2400</b> (1337 – 2483)	<b>23.2</b> (21 – 37)

NIHD Interpretation: Normal CI, SVRI, and TFC do not indicate decompensated HF or pulmonary edema. Likely cause of symptoms is primary pulmonary disease.

Treatment Decision: Initiate antibiotics, bronchodilator.

From John Strobeck, MD, PhD, FACC, Heart Lung Center, Hawthorne, NJ

# Impact of NIHD on Diagnosis and Therapy of Emergent Dyspnea: the ED-IMPACT Trial

# ED-IMPACT

- Peacock WF, Summers RF, Vogel J, Emerman C. Impact of impedance cardiography on diagnosis and therapy of emergent dyspnea: the ED-IMPACT trial. *Acad Emerg Med.* 2006; 13(4):365-371.
- Department of Emergency Medicine, <u>Cleveland Clinic</u>, Cleveland, OH, USA. peacocw@ccf.org

### **ED-IMPACT: Study Design**

Setting: (Cleveland Clinic)

**Dyspnea pts. >65 y.o. (89 Pt.)** 

**History & Examination** 

Dx. and Tx. Plan

Physician review of NIHD data

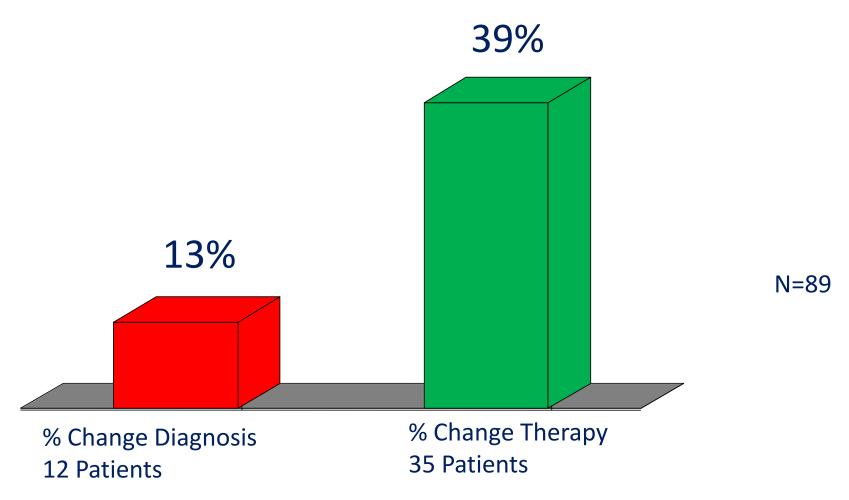
**New Dx and Tx Plan** 

% change Dx??

% change Tx ??

Peacock WF, et al. Acad. Emerg. Med. 2006; 13(4):365-371.

### NIHD Changes Diagnosis and Therapy Decisions in Dyspnea



Peacock WF, et al. Acad. Emerg. Med. 2006; 13(4):365-371.

## The Role of Electrical Cardiometry in Paediatrics and Neonatal Anaesthesia and Intensive Care: A Narrative Review

Khaled A Yassen<sup>1\*</sup>, Zahra Al Ghadeer<sup>1</sup>, Fatimah AlHejji<sup>1</sup>, Fatimah Alothman<sup>1</sup>, Huda Alethan<sup>1</sup>, Danah AlAli<sup>1</sup>, Lamis AlJamaan<sup>1</sup>, Dur E Shahwar<sup>1</sup>, Lien Reyin<sup>2</sup>, Yasser Nassef<sup>3</sup>

<sup>1</sup>Department of Anaesthesia Unit / Surgery, College of Medicine, King Faisal University, Al-Ahsa, Hofuf City, Saudi Arabia

<sup>2</sup>Department of Pediatrics, Chang Gung Memorial Hospital Linkou, Taoyuan, Taiwan

<sup>3</sup>Institute of Medicine, Chung Shan Medical University, Taiwan

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Anesth Crit Care 2022; 4 (2): 73-83

Invasive hemodynamic monitoring could be challenging among pediatrics, This current narrative review aims to discuss and present the published literature that addresses the optimal use and validity of the electrical cardiometry (EC) device among pediatrics.

Literature search focused on PubMed (MEDLINE), Saudi Digital library (SDL), and Google Scholar database between 2010 and 2022.

This review provided evidence that EC monitoring among the pediatrics age group was able to track CO changes over the time (Trend).

The EC technology is easy to use and is an addition for future developments in monitoring, particularly for the pediatric age group.

### Conclusion

This review provided evidence that <u>EC can track CO changes</u> with time and warn against significant hemodynamic changes, but more in the future are still required to improve the precision of the absolute EC CO measurements as presented in several studies.

In specific clinical scenarios, the EC CO was found to be of extreme help as during transportations of neonates and infants, in operating rooms during PDA ligation, and in NICU and PICU.

### Application of Electrical Cardiometry in Postoperative Children with Total Anomalous Pulmonary Venous Connection

Methods: We examined 24 radical surgical correction of TAPVC conducted in the Children Heart Center, Beijing between August 2015 and January 2016.

The median age of the patients was 3m.(2-3.75)m., and the mean body weight was  $5.26 \pm 1.44kg.$ 

We used the EC implemented in ICON® to monitor the patients' postoperative hemodynamics. Sampling time points were:

At ICU admission At 12 hours, 24 H., - 48 H., - and 72 H. after surgery.

We monitored heart rate (HR), mean arterial pressure (MAP), left atrial pressure (LAP), stroke volume variation (SVV), thoracic fluid content (TFC), cardiac index (CI), stroke volume index (SVI), cardiac muscle contractility (ICON), left ventricular systolic time ratio (STR), and systemic vascular resistance index (SVRI).

### Application of Electrical Cardiometry in Postoperative Children with Total Anomalous Pulmonary Venous Connection

Results: The postoperative HR, LAP, TFC, and STR all decreased significantly from 0 to 72 h (p<0.01); the postoperative SVV, SVI, ICON all increased significantly from 0 to 72 h (p<0.01); the postoperative changes of MAP, CI and SVRI did not reach statistical significance.

Conclusion: TAPVC infants had the smallest systolic volume right after surgery, which may be related to their low cardiac contractility. The compensatory increase of HR could be for stabilizing and maintaining balanced blood pressure, cardiac output and blood vessel resistance.

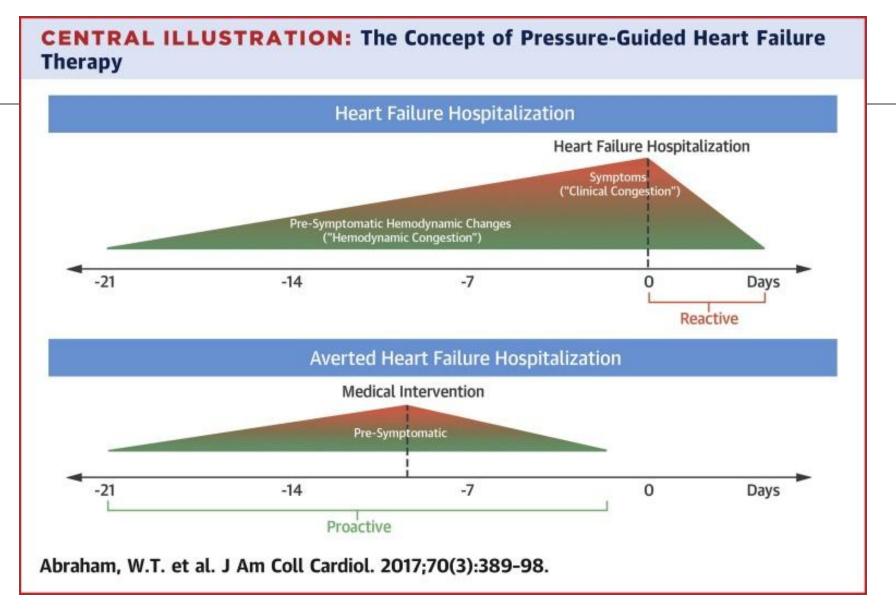
The cardiac contractility was improved within 72 h. after surgery, with the systolic volume gradually increasing and HR decreasing.

EC, can provide continuous non-invasive hemodynamic monitoring. Its application in pediatric and neonatal congenital heart diseases during the perioperative and postoperative periods is promising

He Yan, Liu Ying Long, Yasser Nassef, Su Jun Wu, Cheng Pei, Chen Yan, Zhu Yan Pediatric Intensive Care Unit, Children Heart Center, Beijing Anzhen hospital University of Medical Science No. 2 Anzhen Road, Chaoyang District, Beijing

## Heart Rate Variability Analysis is More Sensitive at Identifying Neonatal Sepsis than Conventional Vital Signs

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William T. Abraham MD, 2017 Departments of Medicine, Division of Cardiovascular Medicine, and the Davis Heart & Lung Research Institute, The Ohio State University, Cardiology Department, Rabin Medical Center, Petah Tikva, Israel, Sackler Faculty of Medicine, Tel Aviv University, Tel Aviv, Israel

Division of Cardiovascular Medicine, Stanford University Medical Center, Stanford, California

### Investigations to Differentiate.. Time Consuming

Resuscitation should not delay while investigating the etiology of undifferentiated shock

- A 12-lead electrocardiogram: ECGs <u>might</u> show evidence of acute coronary syndrome, arrhythmias, or provide diagnostic clues suggestive of pericardial effusion or pulmonary embolism.
- 2) Laboratory tests should include:
- 3) CBC and differential
- 4) Renal and liver function tests
- 5) Serum lactate level
- 6) Cardiac biomarkers, D-dimer level, coagulation profile
- 7) Blood and urine cultures
- 8) Blood gas analysis
- 9) Initial imaging include chest <u>x-rays or US</u> to look for the source of infection such as pneumonia, or complications of shock

## Case Study 1.. Shock DD

Shock patients need immediate action and it is a challenge to differentially diagnose shock. Is it Cardiogenic or non-Cardiogenic, and if non-Cardiogenic, is it Hypovolemic, Septic, or Obstructive? Diagnosis helps us easily decide the treatment (fluids, Inotropes, or Vasopressors).

Sample Case

Low Normal High

HR 95
55 88

MAP 65
mmHg 70 105

Patient: 84-year-old female, History: Diabetic, hypertensive

**Vital Signs:** BP 85/50, HR 95

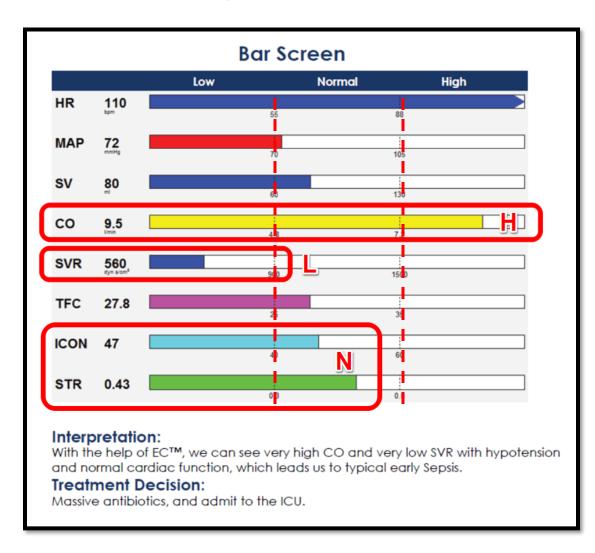
Exam: Low blood pressure, tachycardia

**Current Therapy:** Diuretics

PS: Sepsis (alone)cause more death than cancer colon + cancer beast together

## Septic Shock DD with Hemodynamic

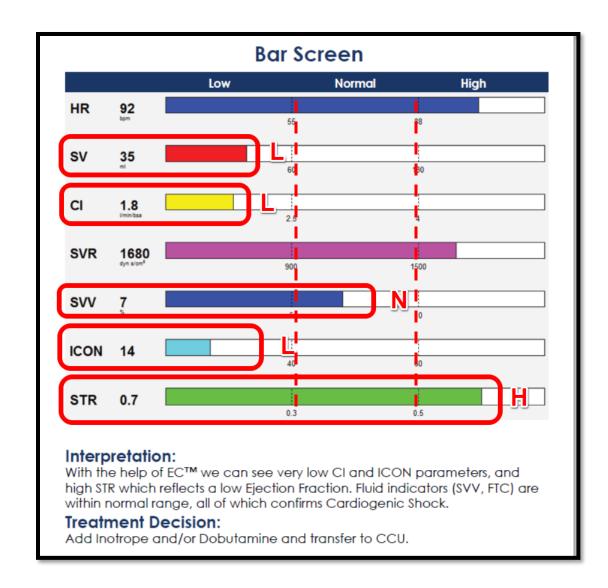
Hyperdynamic circulation, while cardiac parameters are normal and fluid status normal, so early Sepsis is confirmed



### Cardiogenic Shock DD with Hemodynamics

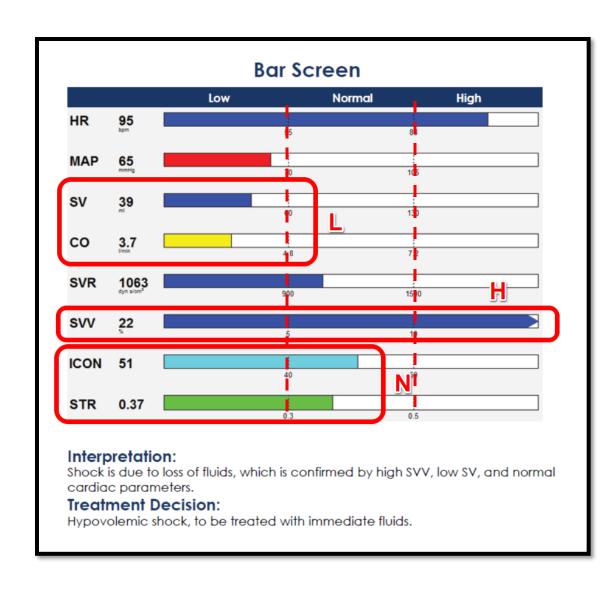
Low contractility
Low Ejection fraction.

Fluid level is almost normal Not hyperdynamic (so hypovolemic and Septic are excluded) so Cardiogenic Shock is confirmed

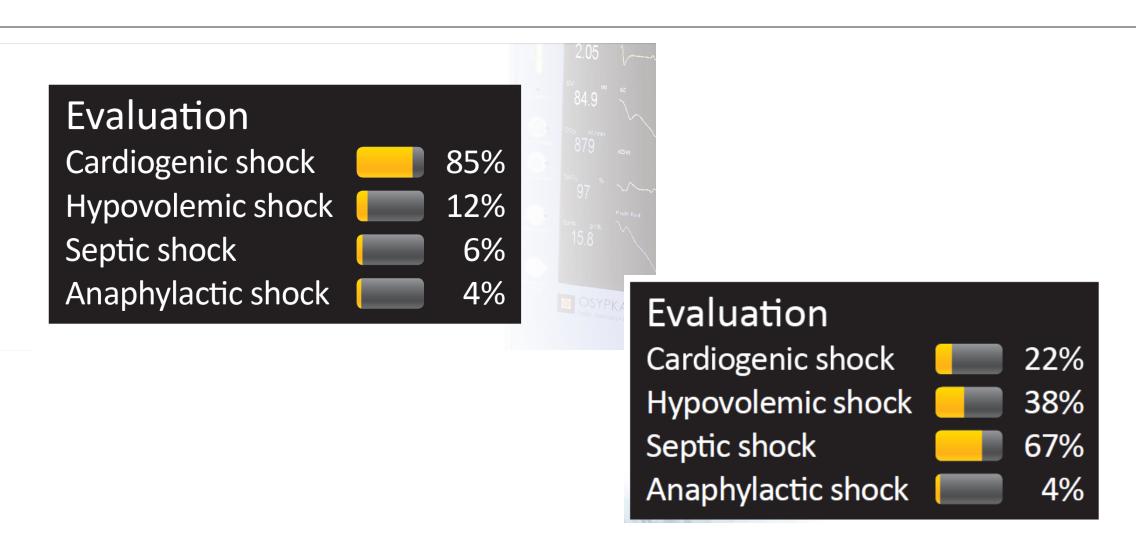


## Hypovolemic Shock DD with ICON

Very high SVV which indicates very low pre-load and intravascular volume, while cardiac parameters are normal and not Hyperdynamic status, so Hypovolemic shock



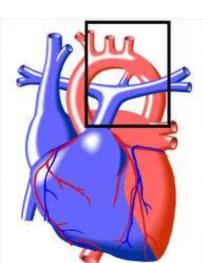
## New Application – Shock DDx

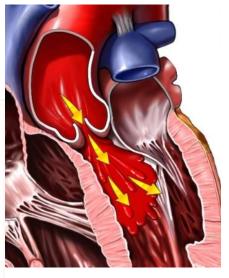


#### Limitations for EC

We still can use it, but the accuracy drop by 10:20%.. So count on the trends over time

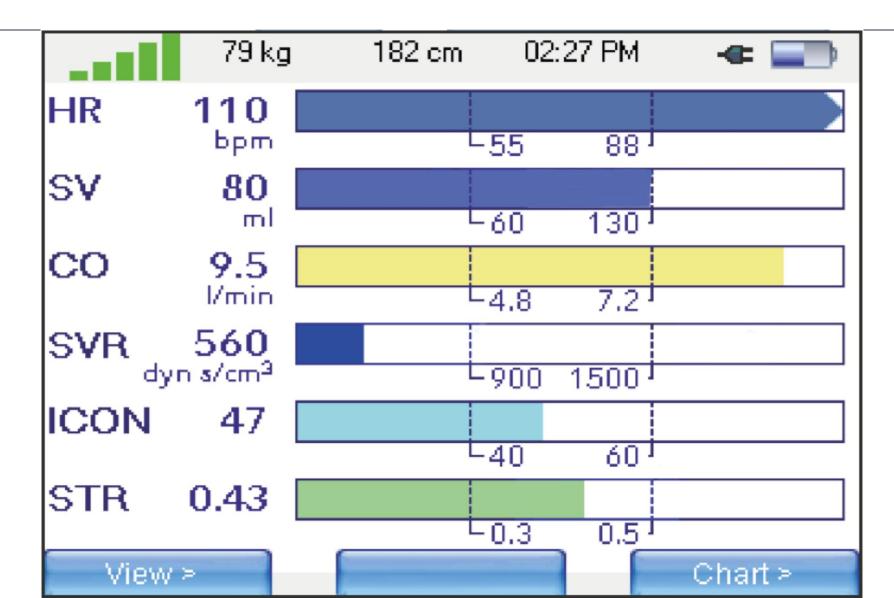
- Moderate to Severe Aortic Valve Regurge
- Massive Chest <u>Wall</u> edema (PE)
- Patent Ductus Artriosus (PDA) Severe cases
- Arrhythmia (only for SVV)







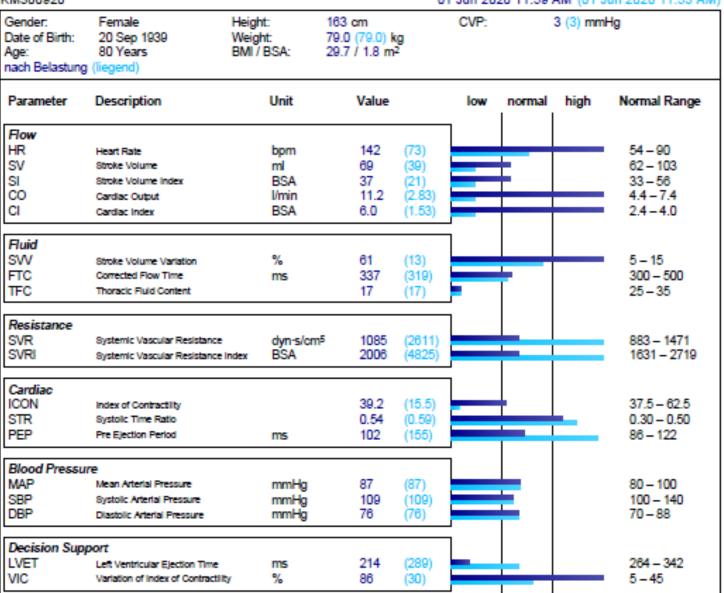
## Easy (diagnostic) screen



#### Hemodynamic Status Report

KM300920

01 Jun 2020 11:59 AM (01 Jun 2020 11:53 AM)





# Hypotension.. What fluid to give? Chest congested, Low intravascular Good contractility and heart function & Low SVR



### **Cardiac Contractility**

<u>Contractility changes</u> due to changes in <u>ventricular pressure and stroke volume</u>,

This can occur as a result of <u>changes in EDV or</u> <u>arterial properties alone</u>.

So, stroke volume (SV) and pressure (MAP &CVP) would not be reliable indices of contractility.

It turns out that we can look towards <u>changes</u> <u>in the ESPVR</u> (End Systolic Pressure-Volume Relation) to indicate changes in contractility

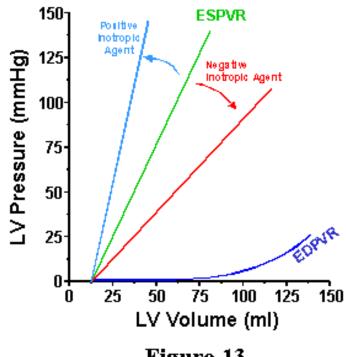


Figure 13

## Noninvasive hemodynamic monitoring of septic shock in children

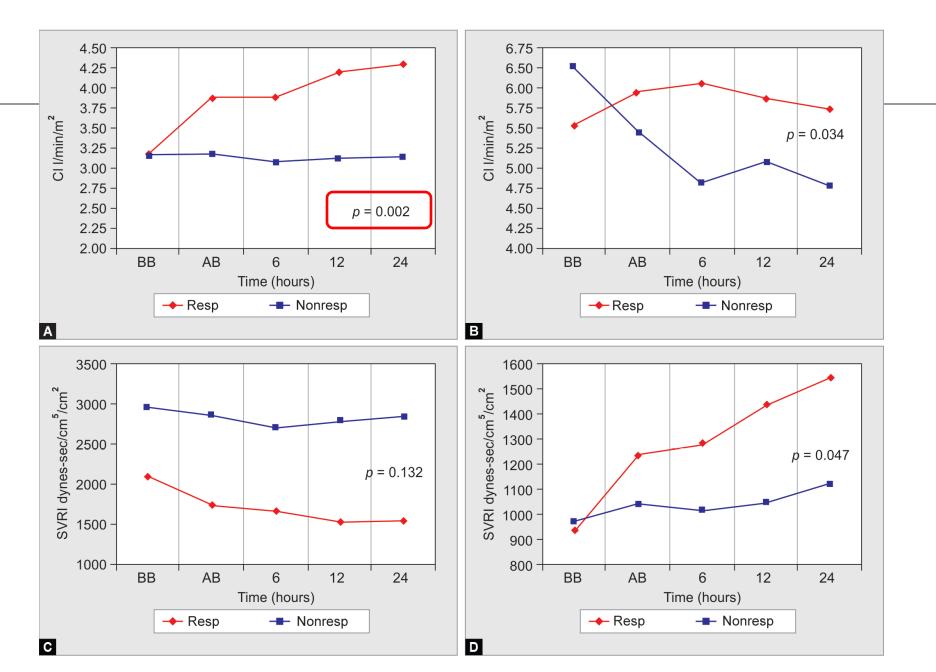
Septic shock in children is associated with high mortality and morbidity. Its management is time-sensitive and must be aggressive and target oriented.

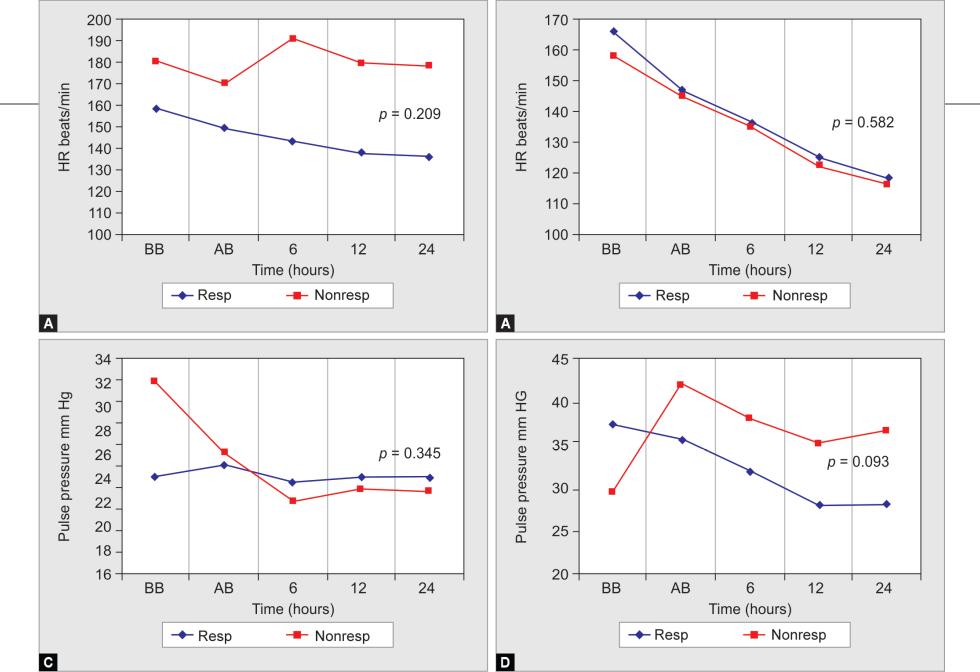
The use of clinical assessment alone to differentiate between cold and warm shock and to select the appropriate inotropic and vasoactive medications is fraught with errors.

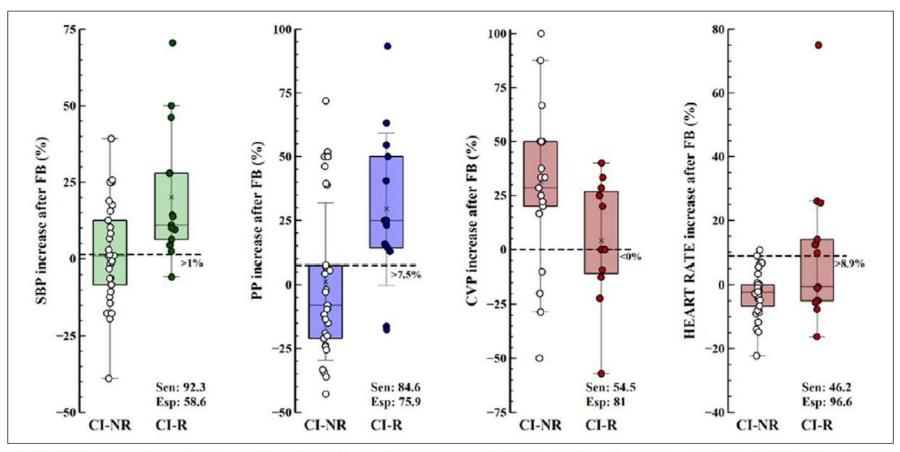
Assessment of the preload, contractility, and afterload using non-invasive tools has been suggested, in conjunction with clinical and laboratory assessment, to direct shock management and select between vasopressors, vasodilators, inotropes and other drugs.

Septic shock is a dynamic condition that changes markedly over time; frequent or continuous measurement of (CO), (SVR), and other hemodynamic parameters using EC is essential to personalize and adapt the treatment over time.

The different combinations of hemodynamics serve as a pathophysiological framework to manage fluid therapy and titrate inotropic and vasoactive drugs.







**Figure 2.** Change in hemodynamic variables in cardiac index-responders (CI-R) and cardiac index-nonresponders (CI-NR). CVP = central venous pressure, Esp = specificity, FB = fluid bolus, PP = arterial pulse pressure, SBP = systolic blood pressure, Sen = sensitivity.

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### STR as indication of Ejection Fraction

- Systolic Time Ratio a Useful Method to Determine Left Ventricular Systolic Dysfunction in Heart Failure
- 52 patients of HF in ER mixed sex and race
- The overall <u>correlation</u> and accuracy between EF and STR was <u>90.4%</u>
- demonstrated a sensitivity of 92%, specificity of 85%,
- and positive and negative predictive values of 95% and
- 79%, respectively.
- Overall accuracy was 90.4%.

- University of Texas Annual Scientific Meeting of the Heart Failure Society of America, September 14, 2004.
- Abstract published in the Journal of Cardiac Failure, 2004;10(suppl 4):S38.

## Electrocardiometry Fluid Responsiveness in Pediatric Septic Shock

Hemodynamic monitoring and categorization of patients based on fluid responsiveness is the key to decisions prompting the use of fluids and vasoactive agents in septic shock.

Distinguishing patients who are going to benefit from fluids from those who will not is of great importance as large amounts of fluids used conventionally based on surviving sepsis guidelines may be detrimental.

Noninvasive monitoring techniques for the assessment of various cardiovascular parameters are increasingly accepted as the current medical practice. Electrical cardiometry (EC) is one such method for the determination of SV, CO, and SVR

It has been validated against gold standard methods such as thermodilution [Malik V, Subramanian A, Chauhan S, et al. World J 2014;4(7):101–108] and is being used more often as a point-of-care noninvasive technique for hemodynamic monitoring.

EC is validated for use in neonates, children, and adults.

A metanalysis in 2016, including 20 studies and 624 patients comparing the accuracy of CO measurement by using EC with other noninvasive technologies, demonstrated that is Cowasiating device that a Command Hospital, Chandimandir, Haryana, India - 2021

The article in the current issue of IJCCM by Rao et al. (2020) has extended the use of EC to categorize pediatric patients with septic shock into vasodilated and vasoconstricted states based on systemic vascular resistance and correlate the categorization clinically.

They also studied the changes in hemodynamic parameters after an isotonic fluid bolus of 20 mL/kg was administered. This is a pilot prospective observational study of 30 patients, which has given an insight into physiological rearrangements following fluid administration in patients with septic shock.

This study endorses the need for continuous preferably noninvasive hemodynamic monitoring for initial classification of vasodilated vs. vasoconstricted states with IBP-derived pulse pressure or SVR as the additional parameters to clinical monitoring. Utility of functional echocardiography in identifying

patients with SMD who may not benefit from further fluid therapy is also highlighted. Though EC has not been used for guiding interventions, it provides an insight into the physiological aspects of hemodynamic monitoring of children with septic shock.

## Hemodynamic monitoring and management of pediatric septic shock

Sepsis remains a major cause of morbidity and mortality among children worldwide.

Furthermore, refractory septic shock and multiple organ dysfunction syndrome are the most critical groups which account for a high mortality rate in pediatric sepsis, and their clinical course often deteriorates rapidly.

Resuscitation based on hemodynamics can provide objective values for identifying the severity of sepsis and monitoring the treatment response.

Initial volume expansion with 10e20 ml/kg per bolus (up to 40e60 ml/kg within 1 h) with frequent assessment of hemodynamics (mainly CO) was recommended as first-line management in children with septic shock [7].

Clinical markers for considering fluid bolus included HR, BP, urine output, and blood lactate are not of high sensitivity.

Clinical signs of overdosed fluid included new onset acute pulmonary edema and hepatomegaly, which indicated that fluid bolus was no longer recommended.

Overdosed fluid is associated with poor prognosis in both adult and pediatric septic shock

#### Conclusion

Early recognition, resuscitation and initial management of pediatric septic shock can improve outcomes.

When septic shock is recognized, crystalloid challenge is recommended after a rapid evaluation of the basic hemodynamics, such as the HR, SBP, SV, CO, SVR, MAP-CVP, ScvO2 and lactate level.

Evaluation of advanced hemodynamics is suggested in critical conditions, such as persistent hypotension despite initial crystalloid volume expansion. Assessment of fluid responsiveness should be conducted to decide whether or not to continue volume expansion.

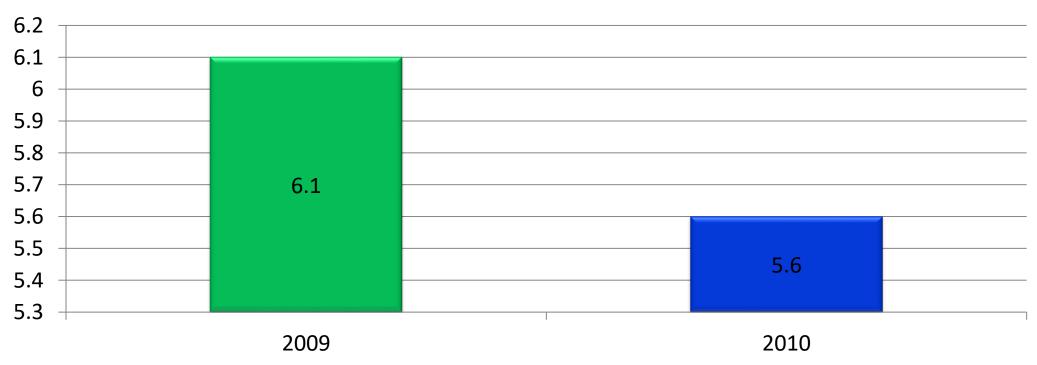
Then vasoactive-inotropic agents should be administered based on the CI and SVRI hemodynamics.

Clinicians should monitor the dynamic changes in these hemodynamic parameters continuously until they are optimized. Because recent studies report that hemodynamic management based on EGDT did not lead to a better prognosis and has lost its advantage

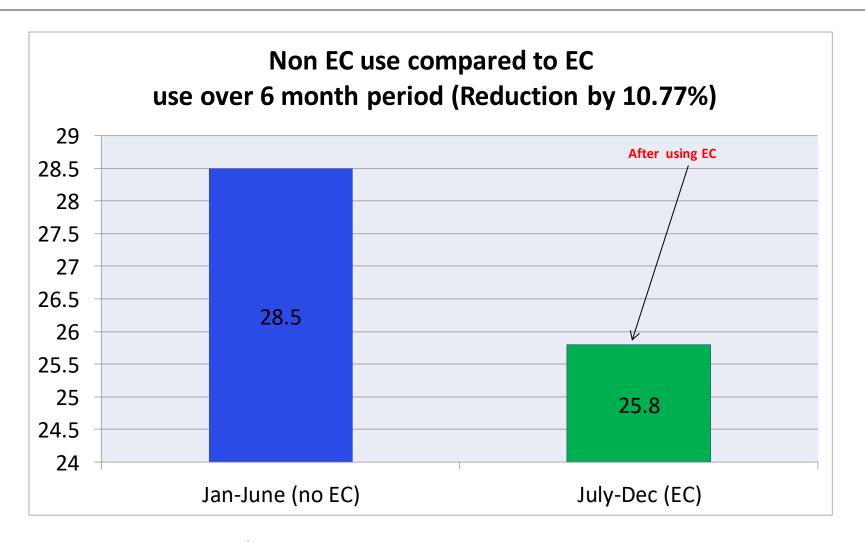
## Reduction in mortality after using EC in Tawam Hospital ICU (A Johns Hopkins Subsidiary)

Non EC use compared to EC use over 12 month period (Mortality

**Reduction by 8.20%)** 



## Reduction in LOS after using EC in Tawam Hospital ICU (A Johns Hopkins Subsidiary)



### Hemodynamics

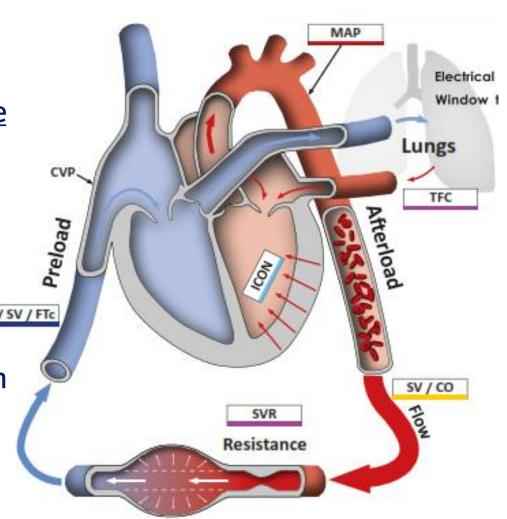
#### **Definition:**

 Measuring the <u>factors</u> that <u>influence</u> the flow of blood, <u>Inotropy</u>, <u>Resistance</u> and fluids in the body

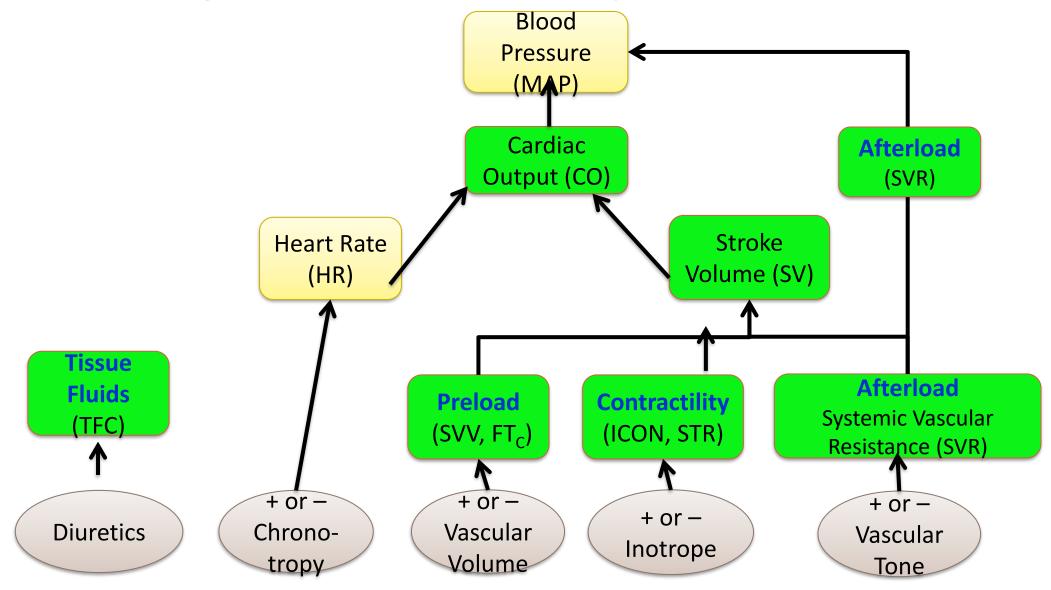
#### **Purpose:**

To help in <u>diagnosis</u>, <u>differential</u>
 <u>diagnosis</u>, <u>Monitoring</u>, <u>treatment and</u>
 <u>drug titration</u> of critically ill patients

 If we can know hemodynamics, we can detect the initial changes within the cardiovascular system very early, which makes the rest easy to manage



## Getting The Full Hemodynamic Picture



### Reliable Hemodynamics Technologies



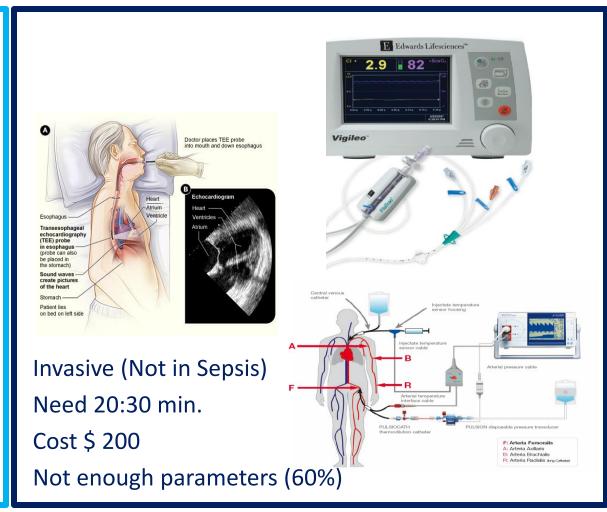
No need for expert

1/20 running cost

Full parameters

Continuous

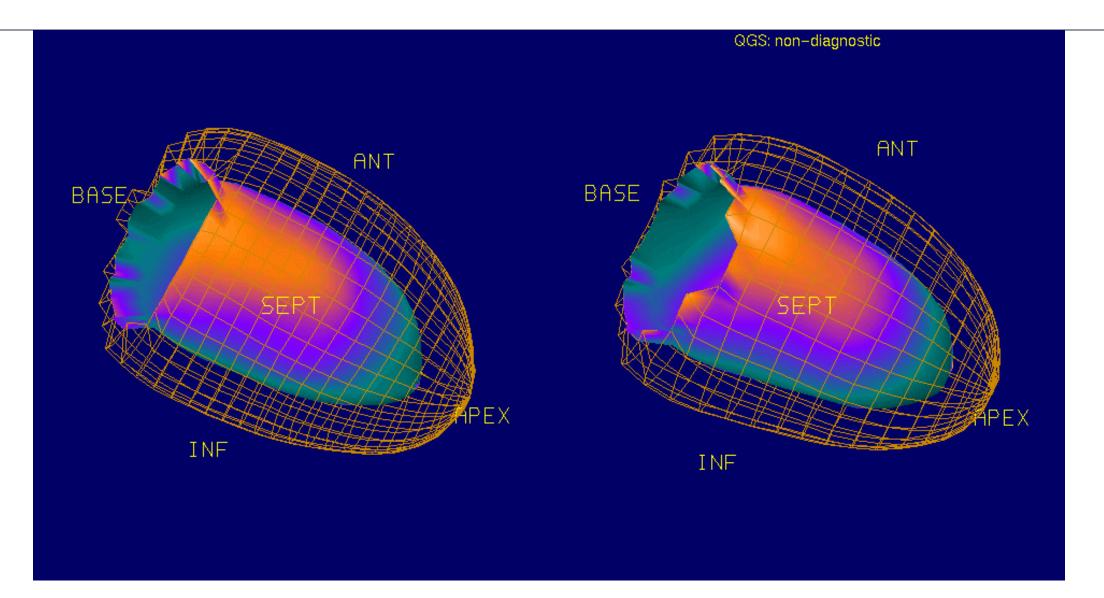




## Hemodynamic parameters for assessing volume status and fluid responsiveness

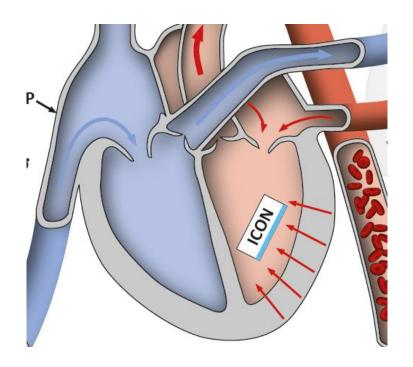
Static Parameters	Dynamic parameters
Arterial pulse pressure(NIBP	Systolic pressure variation (SPV)
Mean arterial pressure	Arterial pulse pressure variation (PPV)
Central venous pare (CVP)	Stoke volume variation (SVV)
Pulmonary occlusion pressure P)	cardiac output (CO)
Heart	central venous oxygen saturation (ScvO2)
Un e Output	Delivered Oxygen (DO2)
Intrathoracic blood volume (ITBV	Stroke Volume (SV)

## **Ejection Fraction**



### Index of LV contractility (ICON)

- It represent the power of left ventricular contraction
- It is measured based on changes in blood speed and acceleration in the Aorta every beat
- It is very good index of left ventricular contractility
- It is of great value to easily titrate Inotrope and to improve CO and contractility for HF patients



#### Hemodynamics...



#### **Great Value in Cardiac Patient Management**

TAMIS - GAMI - Taipei, Taiwan, Feb. 2024

#### **Yasser Nassef MD PhD**

Pediatric Cardiology – Emergency Medicine Hemodynamic Clinical Lecturer Assistant Professor CS Medical University

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#### Hemodynamics...

#### **Great Value in Cardiac Patient Management**

#### APSC – Singapore, July 2023 APAC Taiwan Feb. 2024



#### **Yasser Nassef MD PhD**

Pediatric Cardiology – Pediatric Emergency
Hemodynamic Clinical Lecturer
Assistant Professor CS Medical University
Vice President International Hemodynamic Society



