SEPTIC CARDIOMYOPATHY: WHERE WE ARE & WHERE WE'RE GOING

ROBERT R. EHRMAN, MD, MS, FACEP, FASE

MARCH 31, 2022

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LECTURE OBJECTIVES

1. TO DESCRIBE THE CURRENT UNDERSTANDING OF CARDIAC DYSFUNCTION IN SEPSIS
2. TO REVIEW THE CURRENT LITERATURE ON SEPTIC CARDIOMYOPATHY AND DISCUSS ITS MAJOR LIMITATIONS
3. TO PRESENT NOVEL DIAGNOSTIC APPROACHES TO SEPTIC CARDIOMYOPATHY USING ADVANCED ECHOCARDIOGRAPHY AND EMERGING BIOMARKERS
4. WHY SHOULD I CARE ABOUT SEPTIC CARDIOMYOPATHY?

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WHAT IS SEPTIC CARDIOMYOPATHY?

- TRANSIENT AND REVERSIBLE MYOCARDIAL DEPRESSION THAT OCCURS IN THE SETTING OF SYSTEMIC INFECTION

Annals of Internal Medicine®

LATEST ISSUES CHANNELS CME/MOC IN THE CLINIC JOURNAL CLUB WEB EXCLUSIVES AUTHOR INFO

‹ PREV ARTICLE | THIS ISSUE | NEXT ARTICLE ›

ORIGINAL RESEARCH | 1 APRIL 1984

Profound but Reversible Myocardial Depression in Patients with Septic Shock

MARGARET M. PARKER, M.D.; JAMES H. SHELHAMER, M.D.; STEPHEN L. BACHARACH, Ph.D.; MICHAEL V. GREEN, M.S.; CHARLES NATANSON, M.D.; TERRI M. FREDERICK, B.S.N.; BARBARA A. DAMSKE, R.N.; JOSEPH E. PARRILLO, M.D.

Article, Author, and Disclosure Information

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WHAT IS SEPTIC CARDIOMYOPATHY?

ELSEVIER

The American Journal of Surgery

Volume 133, Issue 4, April 1977, Pages 512-521

Scientific paper

Myocardial depression during sepsis ☆ ☆☆

Richard D. Weisel MD [1], Louis Vito MD [1], Richard C. Dennis MD [1], C.Robert Valeri (Capt MC USNR) [1], Herbert B. Hechtman MD [1]

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https://doi.org/10.1016/0002-9610(77)90141-6 Get rights and content

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WHAT IS SEPTIC CARDIOMYOPATHY (SCM)?

- TRANSIENT AND REVERSIBLE MYOCARDIAL DEPRESSION THAT OCCURS IN THE SETTING OF SYSTEMIC INFECTION
  - DEFINITION IS NEARLY 40 YEARS OLD!
- DESPITE EXTENSIVE STUDY, A THOROUGH UNDERSTANDING OF SCM REMAINS ELUSIVE
- IF WE DON'T UNDERSTAND SCM, HOW CAN WE TREAT IT?

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WHAT IS SEPTIC CARDIOMYOPATHY (SCM)?

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SCM: WHY SHOULD I CARE?

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SCM: WHY SHOULD I CARE?

- You treat sepsis!
- You want to do it well
  - Treating individual patient, not the disease…

Surviving Sepsis Campaign®

- Mandated and/or generic treatments
  - 30cc/kg IVF
  - Static MAP goals

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SCM & FLUID LOADING

- TOO MUCH FLUID CAN BE HARMFUL
  - PULMONARY EDEMA, HYPOXIA
  - SYSTEMIC EFFECTS BEYOND THE LUNGS
- TOO LITTLE FLUID CAN ALSO BE HARMFUL
  - HYPOTENSION/HYPOPERFUSION→ RENAL FAILURE, AMS, TACHYCARDIA…

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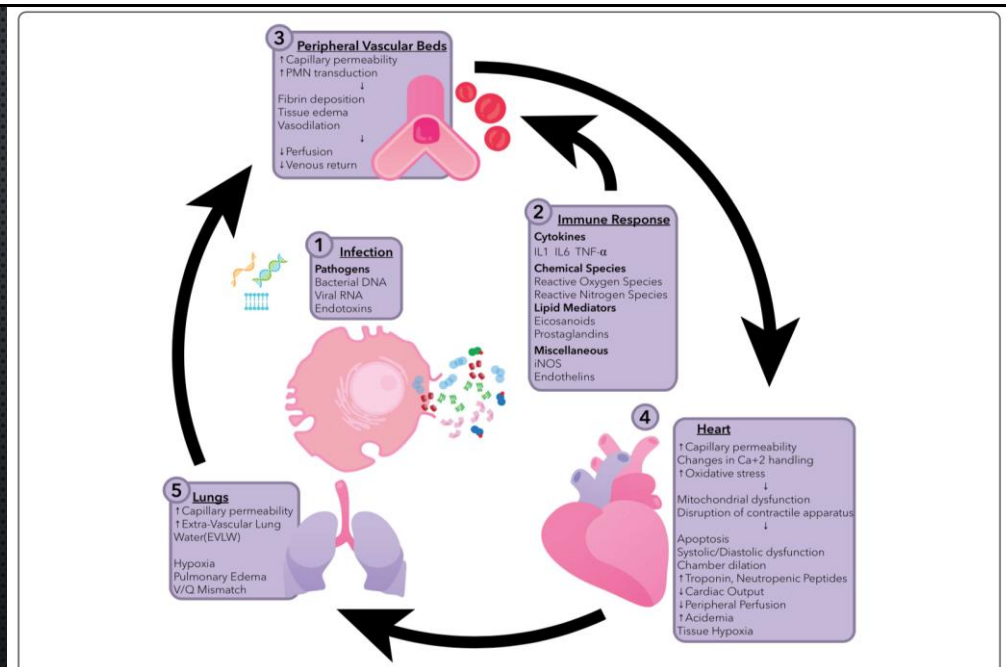

Fig. 1 Pathophysiology of septic myocardial dysfunction. IL interleukin, iNOS induced nitric oxide synthase, *PMN* polymorphonuclear cell, *TNF* tumor necrosis factor

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SCM & FLUID LOADING

- EACH INDIVIDUAL HEART WILL BE DIFFERENT!

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SCM & FLUID LOADING

- EACH INDIVIDUAL HEART WILL BE DIFFERENT!
- STARLING CURVE

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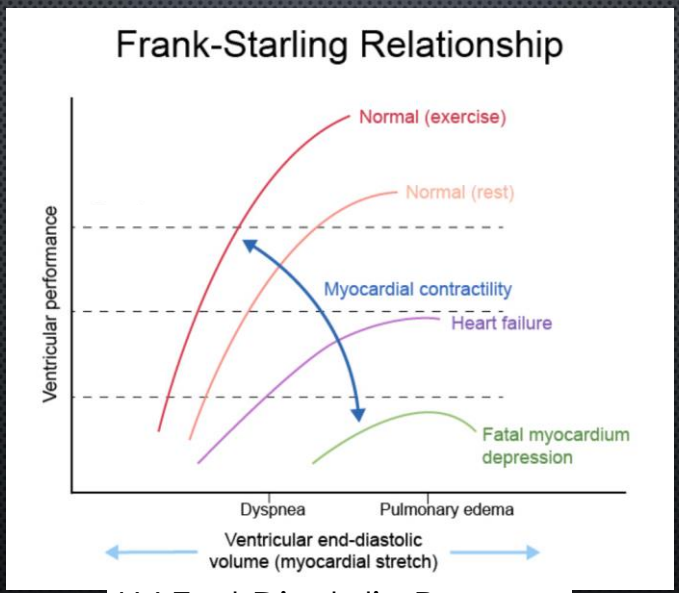

LV End-Diastolic Pressure

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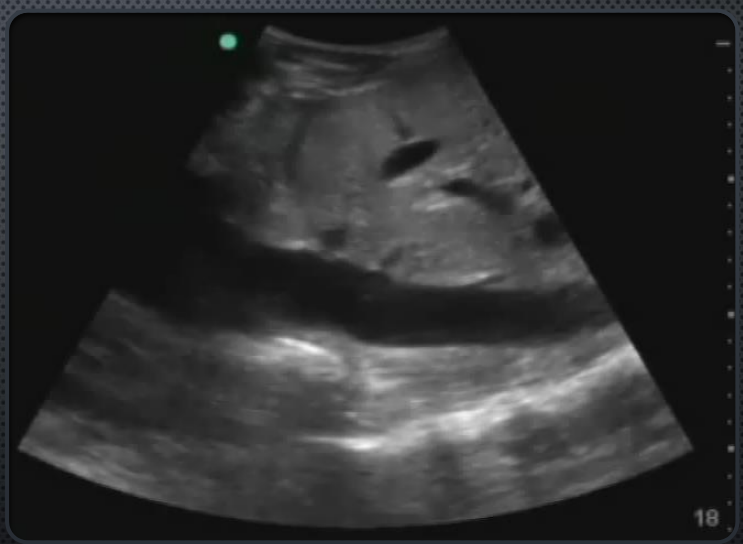
WHAT ABOUT IVC ASSESSMENT?

15

WHAT ABOUT IVC ASSESSMENT?

- INDIRECT INFORMATION ABOUT CARDIAC PERFORMANCE…



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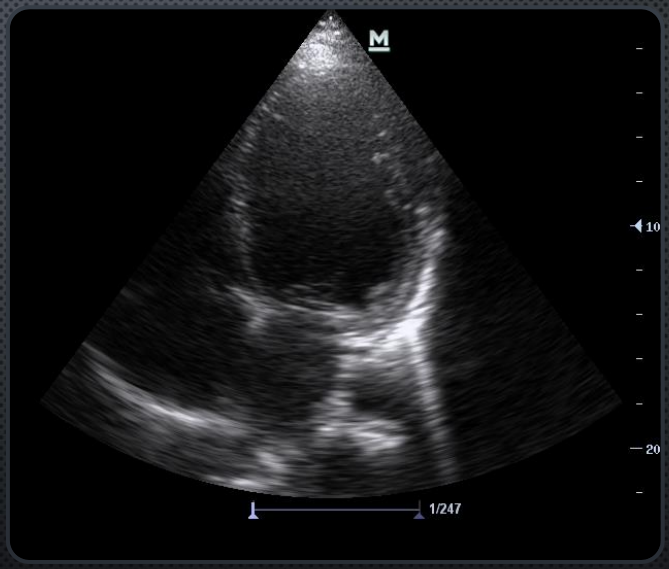
WHAT ABOUT IVC ASSESSMENT?

- INDIRECT INFORMATION ABOUT CARDIAC PERFORMANCE…



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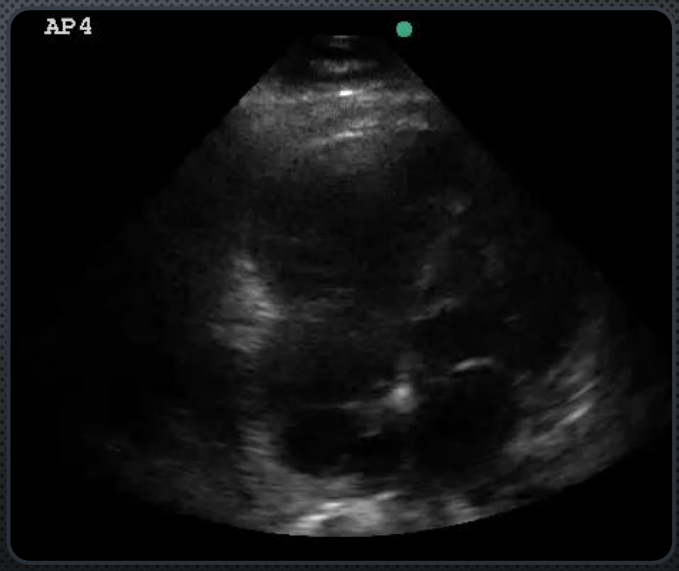
WHAT ABOUT IVC ASSESSMENT?

- INDIRECT INFORMATION ABOUT CARDIAC PERFORMANCE…
- WHICH CAN BE MISLEADING!



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WHAT ABOUT VOLUME RESPONSIVENESS?

- INCREASE IN CO >10% AFTER 500CC FLUID CHALLENGE

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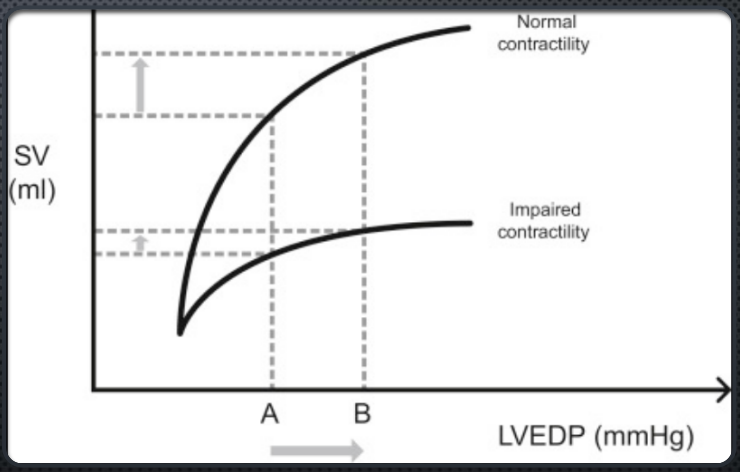
WHAT ABOUT VOLUME RESPONSIVENESS?

- INDIRECT INFO THAT DOESN'T TELL THE WHOLE STORY



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SCM LITERATURE: WHAT'S OUT THERE

- LEFT VENTRICULAR SYSTOLIC FUNCTION: EJECTION FRACTION (LVEF)
  - MEASURE OF LV PERFORMANCE NOT CONTRACTILITY
  - INFLUENCED BY MANY FACTORS: AFTERLOAD, PRELOAD, ETC

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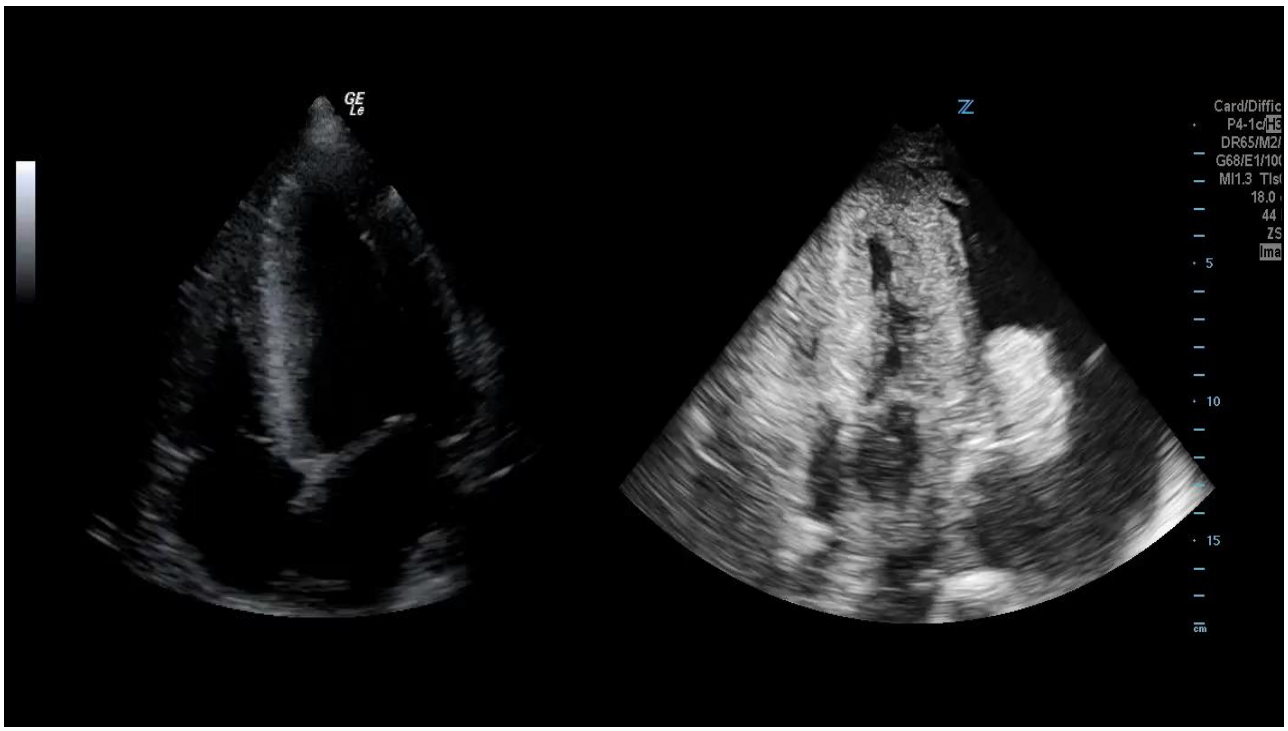

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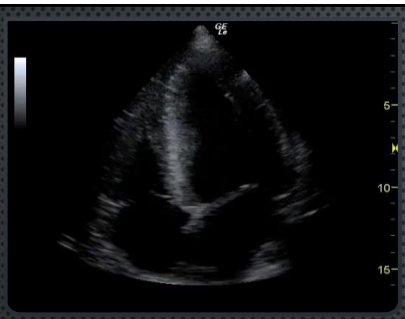
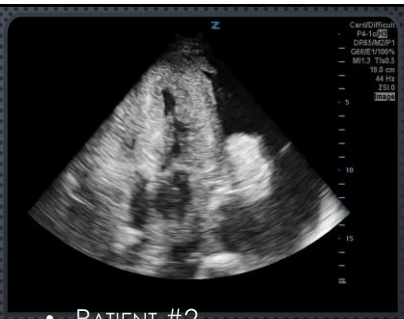


- Patient #1
- LVEDV 180mL
- SV 50mL
- **LVEF**=50/180 x100%
  - 28%
- **CO**= 50 x 90
  - 4.5 L/min



- Patient #2
- LVEDV 42mL
- SV 30mL
- **LVEF**=30/38x100%
  - – 71%
- **CO**= 30x90
  - – 2.7 L/min

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SCM LITERATURE: LVEF

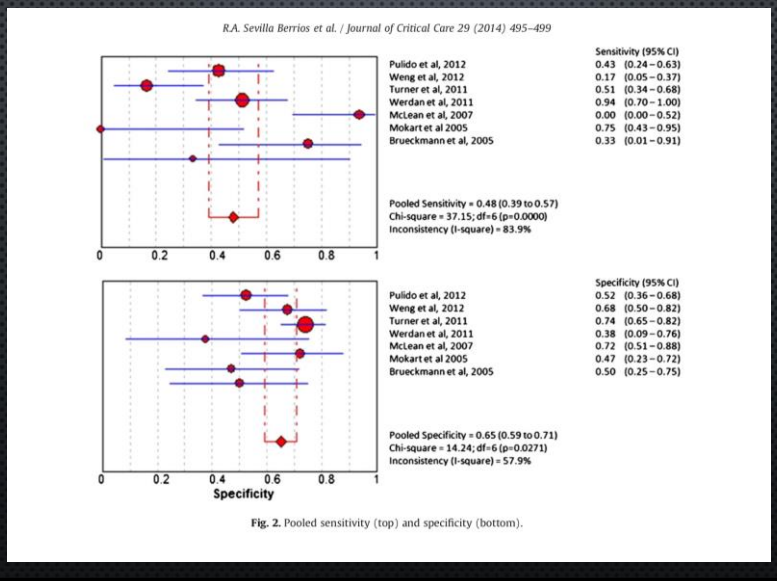

Fig. 2. Pooled sensitivity (top) and specificity (bottom).

Pooled Sn: 0.48

Pooled Sp: 0.65

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SCM LITERATURE: LVEF

Study or Subgroup	Survivors Mean	SD	Total	Non Survivors Mean	SD	Total	Weight	Std. Mean Difference IV, Random, 95% CI
Boissier et al, Int Care Med 2017	54	13.19	44	54	18.3	34	11.7%	0.00 [−0.45, 0.45]
Chang et al, Int Care Med 2015	65	15.19	72	64.5	8.89	39	15.3%	0.04 [−0.35, 0.43]
De Geer et al, Crit Care 2014	50	10.37	33	47	14.81	17	6.9%	0.25 [−0.34, 0.83]
Innocenti et al, Intern Emerg Med 2016	51	14	43	39	21	13	5.9%	0.75 [0.11, 1.38]
Landesberg et al, Crit Care Med 2014	56.2	7.8	65	56.9	12.3	41	15.2%	−0.07 [−0.46, 0.32]
Lanspa et al, Ann Intensive Care 2017	59.66	12.83	229	60.09	15.75	69	30.9%	−0.03 [−0.30, 0.24]
Orde et al, Crit Care 2014	55	15	31	59	16	29	9.1%	−0.25 [−0.76, 0.25]
Shahul et al, Anaesth Analg 2015	50.8	19.22	23	53.02	19	12	4.9%	−0.11 [−0.81, 0.59]
Total (95% CI)			**540**			**254**	**100.0%**	**0.02 [−0.14, 0.17]**

Heterogeneity: $Tau^2 = 0.00$; $Chi^2 = 7.21$, df = 7 (P = 0.41); $I^2 = 3\%$
Test for overall effect: Z = 0.22 (P = 0.83)

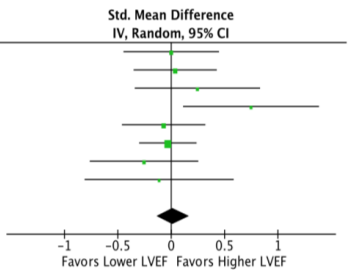

Fig. 4 Comparison of left ventricular global ejection fraction (LVEF) values between survivors and non-survivors among patients with severe sepsis and/or septic shock, in studies also reporting global longitudinal strain (GLS)

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SCM LITERATURE: WHAT'S OUT THERE

- LEFT VENTRICULAR EF*

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SCM LITERATURE: WHAT'S OUT THERE

- LEFT VENTRICULAR EF*
- LV DIASTOLIC FUNCTION

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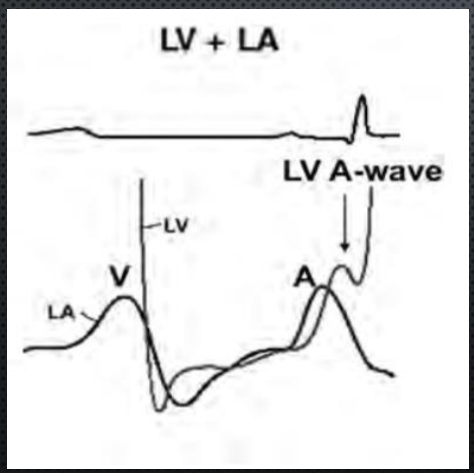
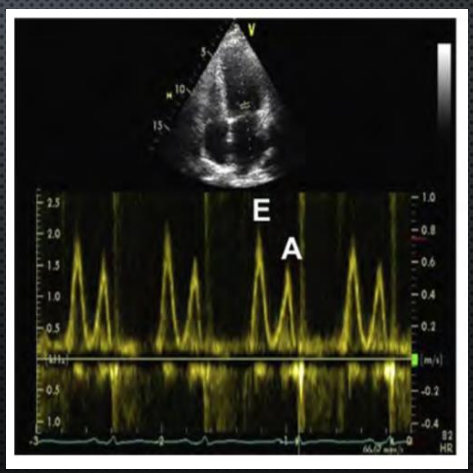
LV DIASTOLIC FUNCTION

- DIASTOLE IS ACTIVE LV RELAXATION





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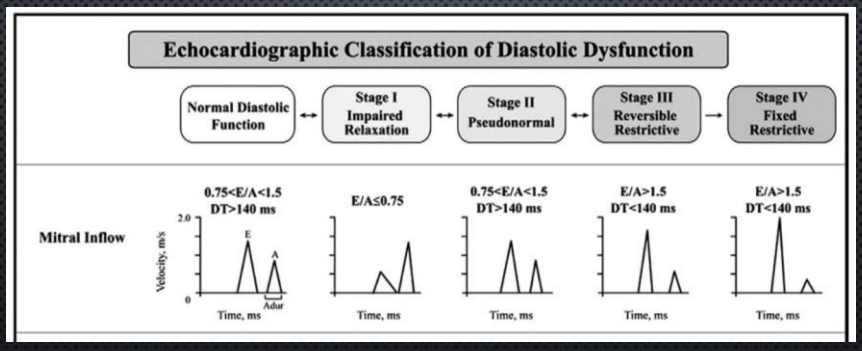
LV DIASTOLIC FUNCTION



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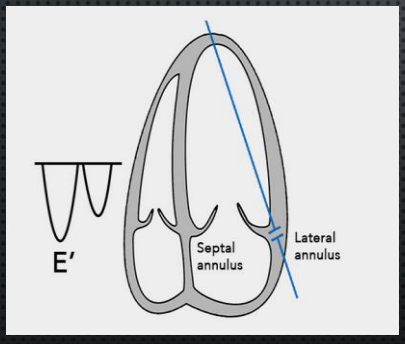
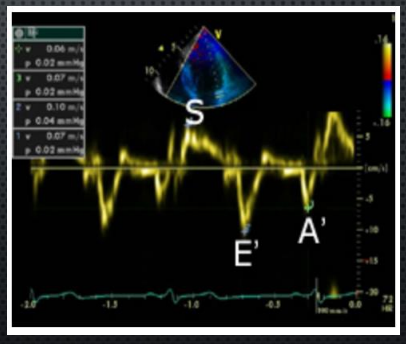
LV DIASTOLIC FUNCTION: TDI





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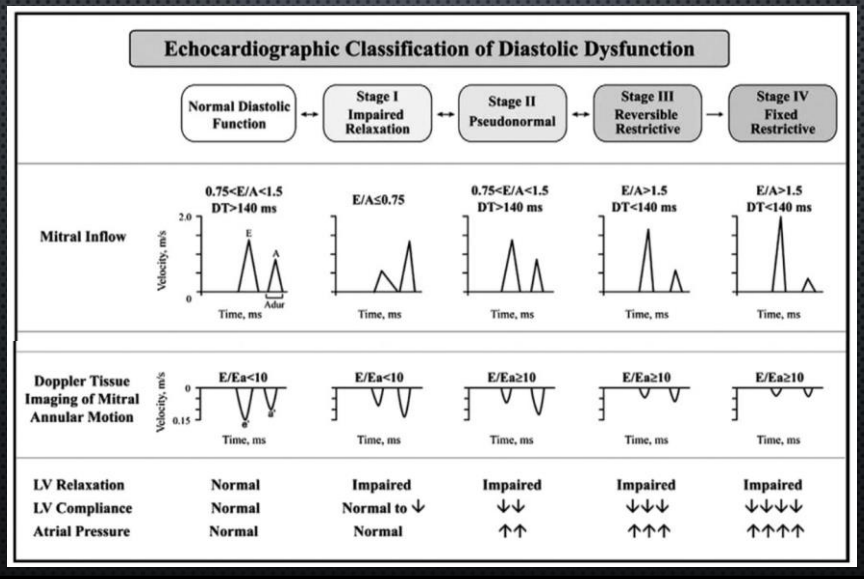
LV DIASTOLIC FUNCTION



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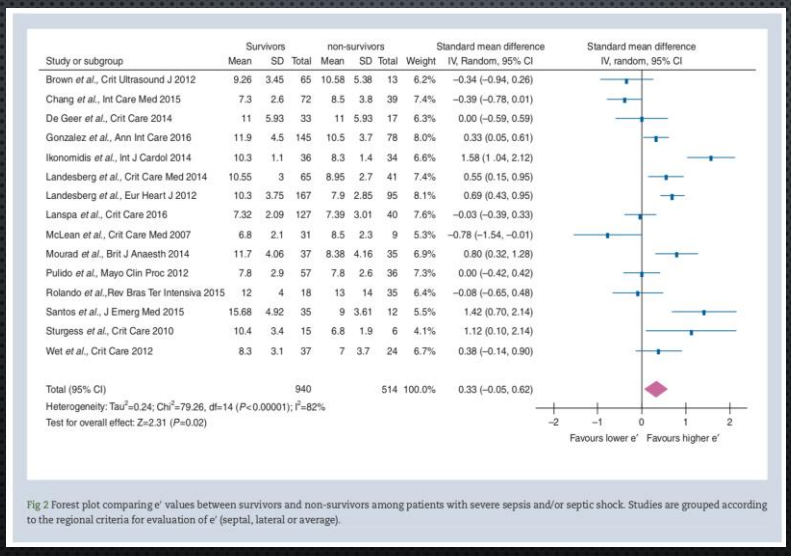
SCM LITERATURE: DIASTOLIC DYSFUNCTION (E')



Study or subgroup	Survivors Mean	SD	Total	non-survivors Mean	SD	Total	Weight	Standard mean difference IV, Random, 95% CI
Brown *et al.*, Crit Ultrasound J 2012	9.26	3.45	65	10.58	5.38	13	6.2%	−0.34 (−0.94, 0.26)
Chang *et al.*, Int Care Med 2015	7.3	2.6	72	8.5	3.8	39	7.4%	−0.39 (−0.78, 0.01)
De Geer *et al.*, Crit Care 2014	11	5.93	33	11	5.93	17	6.3%	0.00 (−0.59, 0.59)
Gonzalez *et al.*, Ann Int Care 2016	11.9	4.5	145	10.5	3.7	78	8.0%	0.33 (0.05, 0.61)
Ikonomidis *et al.*, Int J Cardol 2014	10.3	1.1	36	8.3	1.4	34	6.6%	1.58 (1 .04, 2.12)
Landesberg *et al.*, Crit Care Med 2014	10.55	3	65	8.95	2.7	41	7.4%	0.55 (0.15, 0.95)
Landesberg *et al.*, Eur Heart J 2012	10.3	3.75	167	7.9	2.85	95	8.1%	0.69 (0.43, 0.95)
Lanspa *et al.*, Crit Care 2016	7.32	2.09	127	7.39	3.01	40	7.6%	−0.03 (−0.39, 0.33)
McLean *et al.*, Crit Care Med 2007	6.8	2.1	31	8.5	2.3	9	5.3%	−0.78 (−1.54, −0.01)
Mourad *et al.*, Brit J Anaesth 2014	11.7	4.06	37	8.38	4.16	35	6.9%	0.80 (0.32, 1.28)
Pulido *et al.*, Mayo Clin Proc 2012	7.8	2.9	57	7.8	2.6	36	7.3%	0.00 (−0.42, 0.42)
Rolando *et al.*,Rev Bras Ter Intensiva 2015	12	4	18	13	14	35	6.4%	−0.08 (−0.65, 0.48)
Santos *et al.*, J Emerg Med 2015	15.68	4.92	35	9	3.61	12	5.5%	1.42 (0.70, 2.14)
Sturgess *et al.*, Crit Care 2010	10.4	3.4	15	6.8	1.9	6	4.1%	1.12 (0.10, 2.14)
Wet *et al.*, Crit Care 2012	8.3	3.1	37	7	3.7	24	6.7%	0.38 (−0.14, 0.90)
Total (95% CI)			940			514	100.0%	0.33 (−0.05, 0.62)

Heterogeneity: Tau^2=0.24; Chi^2=79.26, df=14 (P<0.00001); I^2=82%
Test for overall effect: Z=2.31 (P=0.02)

Fig 2 Forest plot comparing e' values between survivors and non-survivors among patients with severe sepsis and/or septic shock. Studies are grouped according to the regional criteria for evaluation of e' (septal, lateral or average).

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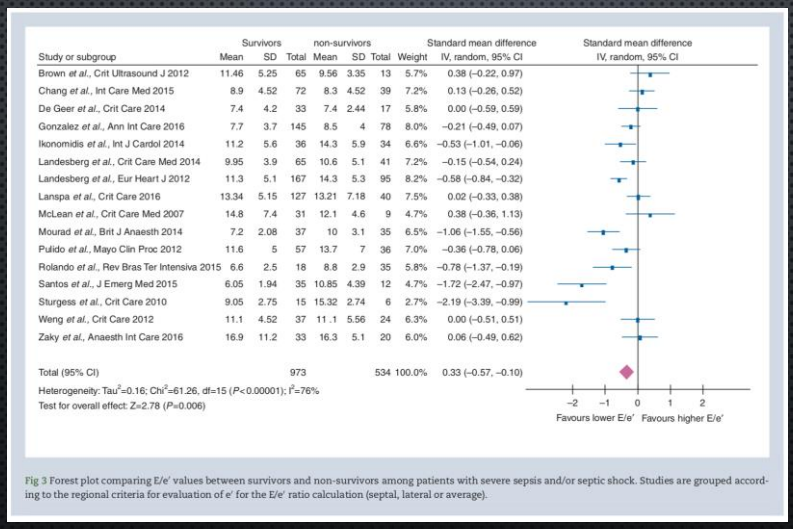
SCM LITERATURE: DIASTOLIC DYSFUNCTION (E/E')



Study or subgroup	Survivors Mean	SD	Total	non-survivors Mean	SD	Total	Weight	Standard mean difference IV, random, 95% CI
Brown *et al.*, Crit Ultrasound J 2012	11.46	5.25	65	9.56	3.35	13	5.7%	0.38 (−0.22, 0.97)
Chang *et al.*, Int Care Med 2015	8.9	4.52	72	8.3	4.52	39	7.2%	0.13 (−0.26, 0.52)
De Geer *et al.*, Crit Care 2014	7.4	4.2	33	7.4	2.44	17	5.8%	0.00 (−0.59, 0.59)
Gonzalez *et al.*, Ann Int Care 2016	7.7	3.7	145	8.5	4	78	8.0%	−0.21 (−0.49, 0.07)
Ikonomidis *et al.*, Int J Cardiol 2014	11.2	5.6	36	14.3	5.9	34	6.6%	−0.53 (−1.01, −0.06)
Landesberg *et al.*, Crit Care Med 2014	9.95	3.9	65	10.6	5.1	41	7.2%	−0.15 (−0.54, 0.24)
Landesberg *et al.*, Eur Heart J 2012	11.3	5.1	167	14.3	5.3	95	8.2%	−0.58 (−0.84, −0.32)
Lanspa *et al.*, Crit Care 2016	13.34	5.15	127	13.21	7.18	40	7.5%	0.02 (−0.33, 0.38)
McLean *et al.*, Crit Care Med 2007	14.8	7.4	31	12.1	4.6	9	4.7%	0.38 (−0.36, 1.13)
Mourad *et al.*, Brit J Anaesth 2014	7.2	2.08	37	10	3.1	35	6.5%	−1.06 (−1.55, −0.56)
Pulido *et al.*, Mayo Clin Proc 2012	11.6	5	57	13.7	7	36	7.0%	−0.36 (−0.78, 0.06)
Rolando *et al.*, Rev Bras Ter Intensiva 2015	6.6	2.5	18	8.8	2.9	35	5.8%	−0.78 (−1.37, −0.19)
Santos *et al.*, J Emerg Med 2015	6.05	1.94	35	10.85	4.39	12	4.7%	−1.72 (−2.47, −0.97)
Sturgess *et al.*, Crit Care 2010	9.05	2.75	15	15.32	2.74	6	2.7%	−2.19 (−3.39, −0.99)
Weng *et al.*, Crit Care 2012	11.1	4.52	37	11 .1	5.56	24	6.3%	0.00 (−0.51, 0.51)
Zaky *et al.*, Anaesth Int Care 2016	16.9	11.2	33	16.3	5.1	20	6.0%	0.06 (−0.49, 0.62)
Total (95% CI)			973			534	100.0%	0.33 (−0.57, −0.10)

Heterogeneity: Tau^2=0.16; Chi^2=61.26, df=15 (P<0.00001); I^2=76%
Test for overall effect: Z=2.78 (P=0.006)

Standard mean difference
IV, random, 95% CI

−2 −1 0 1 2
Favours lower E/e' Favours higher E/e'

Fig 3 Forest plot comparing E/e' values between survivors and non-survivors among patients with severe sepsis and/or septic shock. Studies are grouped according to the regional criteria for evaluation of e' for the E/e' ratio calculation (septal, lateral or average).

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SCM LITERATURE: WHAT'S OUT THERE

- LEFT V[illegible]RICULAR EF*
- LV D[illegible]LIC FUNCTION*
- GLOBAL LONGITUDINAL STRAIN (GLS)?

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WHAT IS GLS?

Speckle-Tracking Echocardiography (STE)

Gather optimal two-dimensional digital echocardiograms

↓

Use image-processing algorithms to identify small stable myocardial footprints ('speckles')

↓

Tracked over the cardiac cycle, the displacement of speckles can provide information about myocardial deformation

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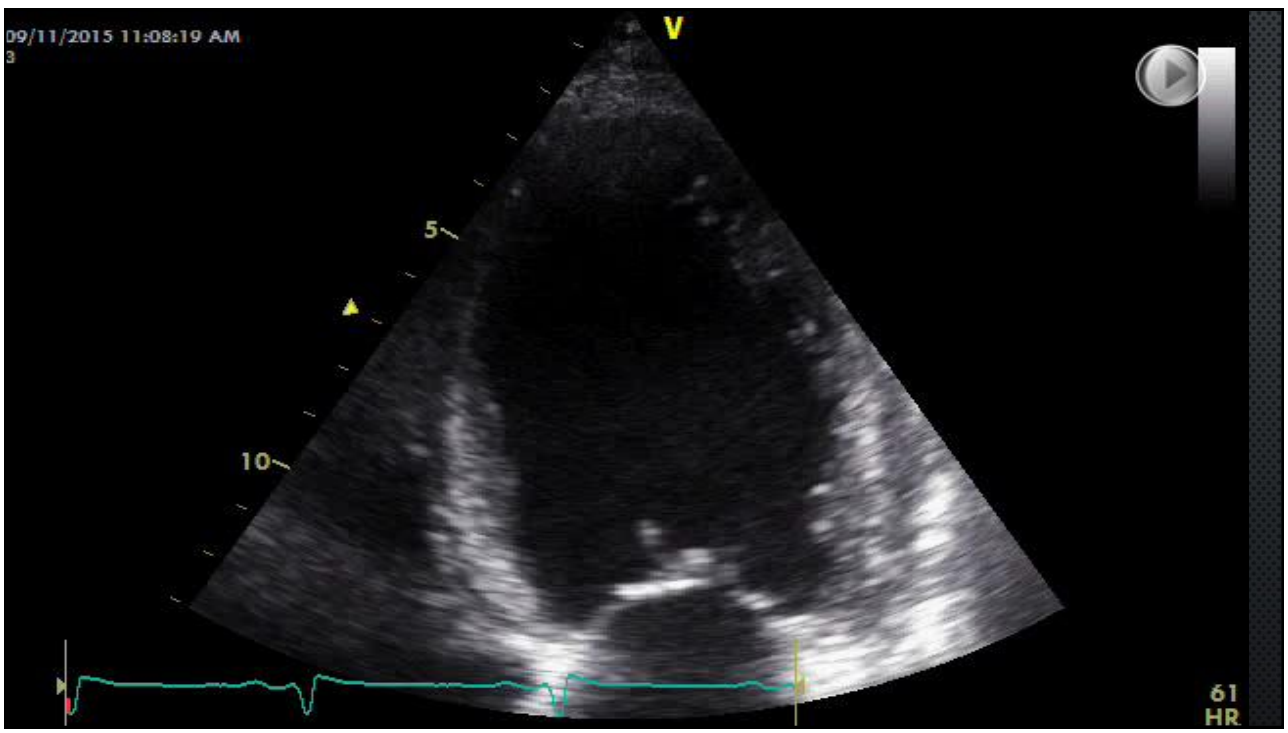

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WHAT IS GLS?

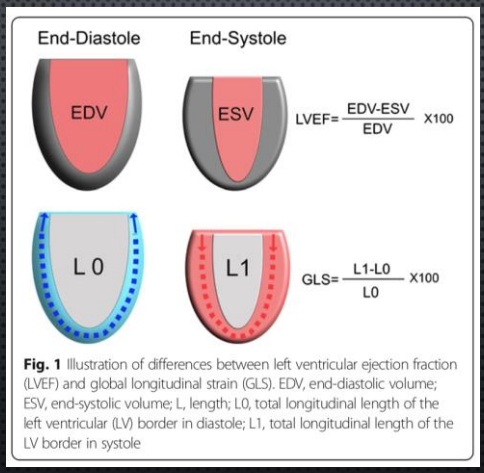

Fig. 1 Illustration of differences between left ventricular ejection fraction (LVEF) and global longitudinal strain (GLS). EDV, end-diastolic volume; ESV, end-systolic volume; L, length; L0, total longitudinal length of the left ventricular (LV) border in diastole; L1, total longitudinal length of the LV border in systole

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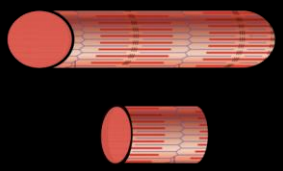
Myocardial Strain



L_0 = 10cm

L_1 = 8cm

$$\text{Strain } (\varepsilon) = \frac{L_1 - L_0}{L_0} = \frac{8-10}{10} = -20\%$$

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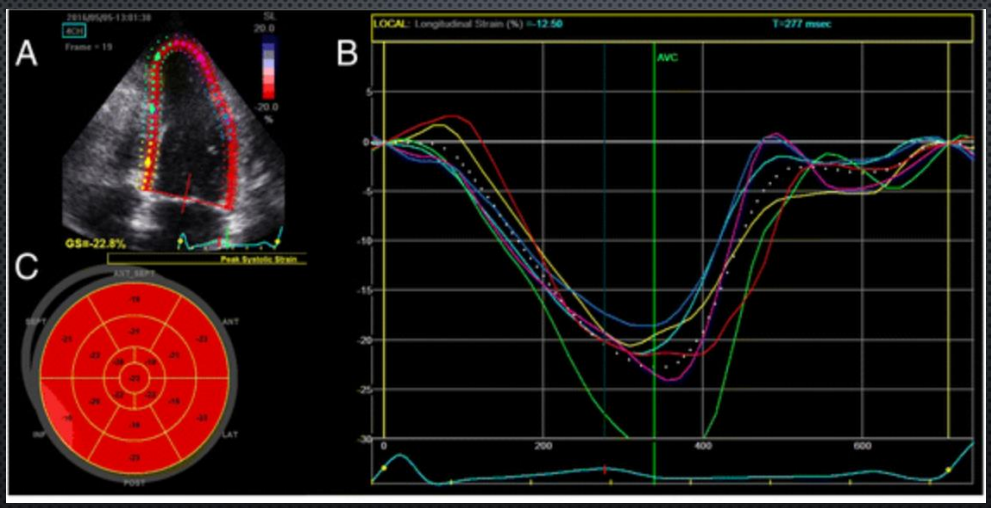
WHAT IS GLS?



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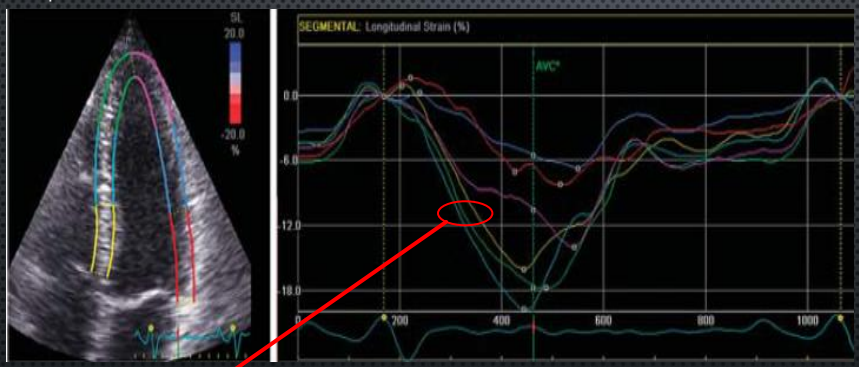
WHAT IS GLS?

Apical 4-Chamber View



Septal segments

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WHAT IS GLS?

Apical 4-Chamber View

Lateral segments

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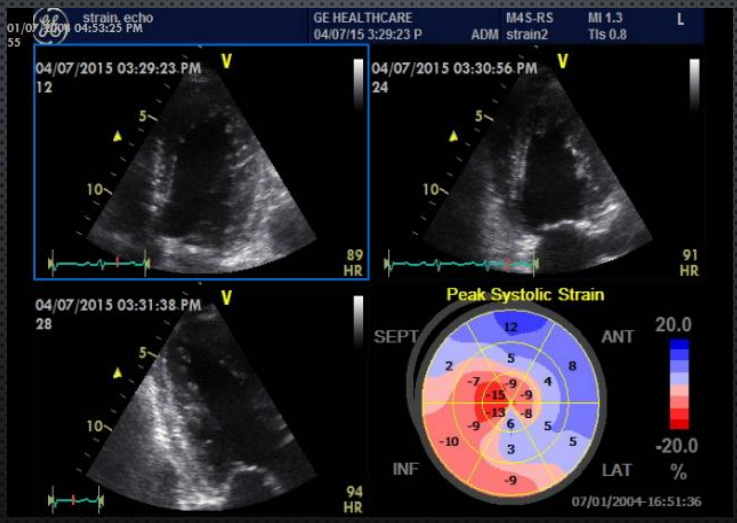
WHAT IS GLS?



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LIMITATIONS OF GLS

- IMAGE QUALITY
  - "SPECKLES"
  - OPERATOR SKILL
- HIGH FRAME RATES
  - DEPTH
  - SECTOR WIDTH
- GOOD ECG TRACING
- HIGH HEART RATES (UNRELIABLE > 120 BPM)
- "NEGATIVE" CONNOTATION
- VENDOR DIFFERENCES
- LOAD DEPENDENCY

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SCM LITERATURE: GLS

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Table 2 Characteristics of included observational studies

Author, year population (number)	Echocardiography timing	GLS software and TTE views used	Data reported	SAPS SOFA APACHE	MV	Mortality	Longest follow up
Boissier, 2017 78 ICU patients with septic shock	TTE within 24 h of ICU admission	Philips' Qlab 8.1 (Philips®) Ap: 4ch, 2ch	GLS and LVEF	60.1 ± 20.5 11.7 ± 3.4 -	84.6%	43.6%	Hospital
Chang, 2015 111 ICU patients with septic shock	TTE within 24 h of ICU admission	EchoPAC v. BT09 (GE®) Ap: 4ch, 2ch, 3ch	GLS and LVEF	- - 21 ± 8	65.8%	35.1%	Hospital
De Geer, 2014 50 ICU patients with septic shock	TTE within 24 h of ICU admission	EchoPac v. 112 (GE®) Ap: 4ch, 2ch, 3ch	GLS and LVEF	- 11 (9–12) -	84%	34%	90-day
Innocenti, 2016 56 ED patients with septic shock	TTE within 24 h if ICU admission	Philips' Qlab 8.1 (Philips®) Ap: 4ch, 2ch	GLS and LVEF	- 6.3 ± 2.8 -	–	27.2%	28-day
Landesberg, 2014 106 ICU patients with severe sepsis or septic shock	TTE on ICU admission day or as soon as possible	Philips' Qlab 8.1 (Philips®) Ap: 4ch, 2ch	GLS and LVEF	- - 21.61 ± 6.8	100%	39%	Hospital
Lanspa, 2017 298 ICU patients with severe sepsis or septic shock	TTE within 24 h of ICU admission	Image-Arena platform (TomTec®) Ap: 4ch	GLS	- 9 (6–12) 25 (18–23)	–	23%	28-day
Orde, 2014 60 adult patients with severe sepsis or septic shock	TTE within 24 h of meeting severe sepsis criteria	Syngo Velocity Vector Imaging (Siemens®) Ap: 4ch, 2ch, 3ch	GLS and LVEF	- 11 ± 4 -	65%	48%	180-day
Shahul, 2015* 35 ICU patients with sepsis and septic shock	TTE on admission and at 24 h post	cardiac perf. Analysis v1.1 (TomTec®) Ap: 4ch	GLS and LVEF	- 6 (2.1–9) 21.7 ± 6.2	69%	23.3%	30-day

SCM LITERATURE: GLS

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SCM LITERATURE: GLS

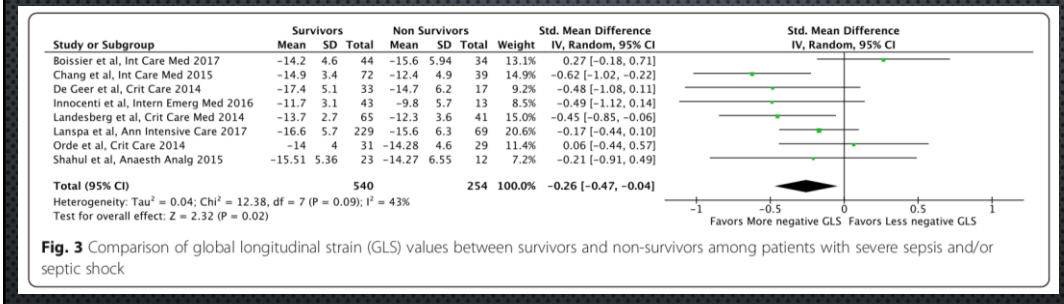
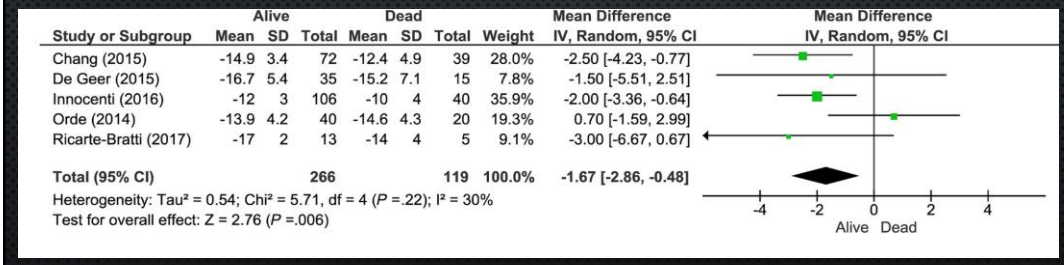


Fig. 3 Comparison of global longitudinal strain (GLS) values between survivors and non-survivors among patients with severe sepsis and/or septic shock



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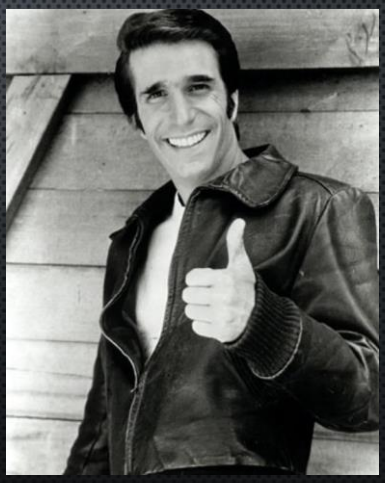
SCM LITERATURE: WHAT'S OUT THERE

- LEFT VENTRICULAR EF*
- LV DIASTOLIC FUNCTION*
- GLOBAL LONGITUDINAL STRAIN (GLS)*



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SCM LITERATURE: LIMITATIONS

- TIMING OF ECHOCARDIOGRAPHY
  - DONE IN ICU
  - 24-72 HOURS AFTER ADMISSION!
- CARDIAC FUNCTION ASSESSED AT SINGLE TIME POINT
- PRE-MORBID CARDIAC FUNCTION UNKNOWN
- MORTALITY IS MULTIFACTORIAL
- HETEROGENEITY OF TREATMENT EFFECT

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SCM: WHAT'S NEXT?

- More data needed
  - Early in treatment course (0-24 hours)

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SCM: UNANSWERED QUESTIONS

- WHAT IS SEPTIC CARDIOMYOPATHY?
- IS FUNCTION AT PRESENTATION PROGNOSTICALLY USEFUL?
- CAN TRAJECTORY OF CARDIAC FUNCTION INFORM PROGNOSIS OR GUIDE TREATMENT DECISIONS?

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LONGITUDINAL CARDIAC FUNCTION AND ILLNESS SEVERITY

Table 3: Mixed-Effects Model Results Showing The Relationship Between Emergency Department Sequential Organ Failure Assessment Score and Marginal Mean of Echocardiography Parameters

	ED SOFA Score			
	Parameter Estimate	95% CI	Percent Residual Variation Not Explained by Within-Subject Effects	Number of Observations Included (%)
Echocardiography Measure				
LVEF (%)	-1.80	-3.18 to -0.42	78.2	185 (84)
TAPSE (mm)	-0.76	-1.2 to -0.28	49.1	103 (47)
GLS (%)	0.53	0.02 to 1.03	84.3	89 (41)
Lateral e'(cm)	-0.04	-0.06 to -0.01	33.3	147 (67)
Septal e' (cm)	-0.04	-0.06 to -0.02	61.4	149 (68)
Average e' (cm)	-0.04	-0.06 to -0.02	60.2	139 (63)
Lateral E/e'	0.87	0.26 to 1.47	77.5	126 (58)
Septal E/e'	1.29	0.54 to 2.04	75.0	126 (58)
Average E/e'	1.07	0.40 to 1.75	78.3	126 (58)

Table legend: Each row represents a single linear mixed-effects model with the outcome being repeated measures (at 0, 3 and 24 hours) of the cardiac function parameters listed in column 1 and the parameter estimate for ED SOFA score (covariate parameter estimates not shown). *LVEF= left ventricular ejection fraction; TAPSE= tricuspid annular plan systolic excursion; GLS= global longitudinal strain; e' = mitral annular velocity; E/e= trans-mitral inflow velocity/mitral annular velocity ratio.*

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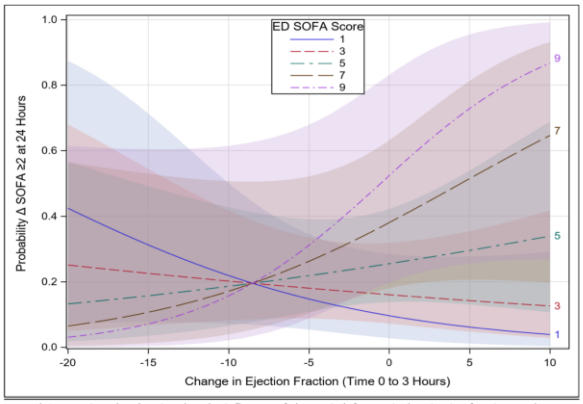
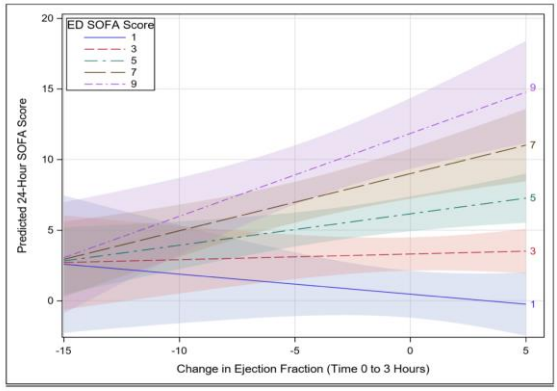
EARLY CHANGE IN LVEF AND 24-HOUR ILLNESS SEVERITY

Figure 1



Legend: Interaction plot showing that the influence of change in left-ventricular ejection fraction on the probability of meeting the outcome of ΔSOFA ≥ 2 at 24 hours varies by baseline (ED) SOFA Score, adjusted for age, troponin-I, average change in E/e' from 0-3hrs, and history of heart failure= "yes"; bands represent 95% CIs.

Figure 2



Legend: Interaction plot for showing that the influence of change in left-ventricular ejection fraction on predicted SOFA Score at 24-hours varies by baseline (ED) SOFA Score, adjusted for age, troponin-I, Average change in E/e' from 0-3hrs, and history of heart failure= "yes"; bands represent 95% CIs.

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SCM: HIBERNATION VS KAROSHI?

- HIBERNATION DURING STRESS → PROTECTIVE?
  - RECOGNIZED PHENOMENA IN ISCHEMIA
  - REVERSIBILITY OF SCM IN SOME STUDIES
  - LACK OF MYONECROSIS IN SOME STUDIES

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SCM: WHAT ARE THE PATHOMECHANISMS?

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SCM: PATHOMECHANISMS

- Sepsis is a balance between pro & anti-inflammatory mediators
- Pro: TNF, cytokines, ROS, lipids, hormones, genetics
- Anti: Specialized pro-resolving mediators (SPMs)
  - maresins, resolvins, protectins
  - Omega-3s

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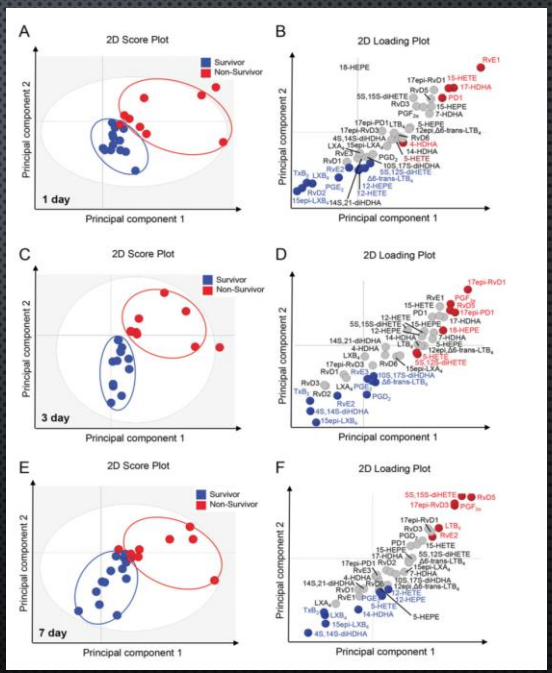
SCM: LIPIDOMICS

Alive vs Dead



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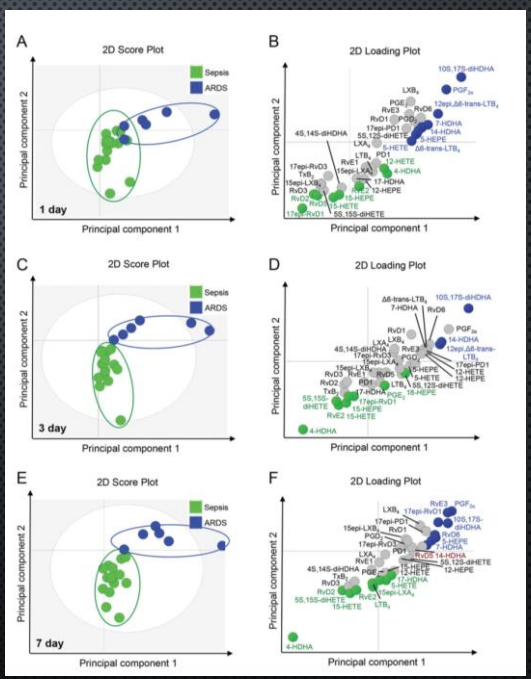
SCM: LIPIDOMICS

Sepsis vs ARDS



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BIOMARKERS IN SCM: NEXT STEPS

- IDENTIFY CANDIDATE MOLECULES
- *IN VITRO* & ANIMAL MODELS
  - DIAGNOSTICS
  - THERAPEUTICS
- BACK TO HUMANS

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SCM: BIOMARKER DEVELOPMENT

Cardiac function and Biomarkers?

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SCM: WHAT DO WE WANT?

- Diagnostics
- Prognostication
- Therapeutics
  - Echo-guided resuscitation
  - Medications/interventions

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SCM: CONCLUSIONS

- SCM remains a poorly defined and understood entity
- Crisitunity!
- ED is the ideal location to study SCM
- Longitudinal use of imaging + biomarkers at POC

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THE END


"WHILE DOING THE RESEARCH, KEEP IN MIND THERE ARE ONLY TWO KINDS OF FACTS... THOSE THAT SUPPORT MY POSITION... AND INCONCLUSIVE."

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