Σηπτικό σοκ

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Παρουσίαση περιστατικού

- Άνδρας 64 ετών επισκέπτεται ιδιωτικό Θεραπευτήριο τον Ιανουάριο λόγω κακουχίας, ρίγους και κοιλιακού άλγους αιφνίδιας έναρξης προ ωρών
- Ατομικό αναμνηστικό : αρτηριακή υπέρταση, δυσλιπιδαιμία, σπληνεκτομή προ έτους λόγω ατυχήματος
- Φαρμακευτική αγωγή: περινδοπρίλη, ατορβαστατίνη
- Πρώην καπνιστής (40 PY), διακοπή προ 2ετίας. Αλλεργίες δεν αναφέρονται.
- Από τον έλεγχο: ζωτικά σημεία κ.φ. ECG, Ro Θώρακος χωρίς παθολογικά ευρήματα. WBC 11.35G/l, Hb 14.1g/dl PLT 249G/l, CRP 0.6mg/dl
- Εξέρχεται σπίτι με οδηγίες για οσελταμιβίρη και παρακεταμόλη.

Παρουσίαση περιστατικού (συνέχεια)

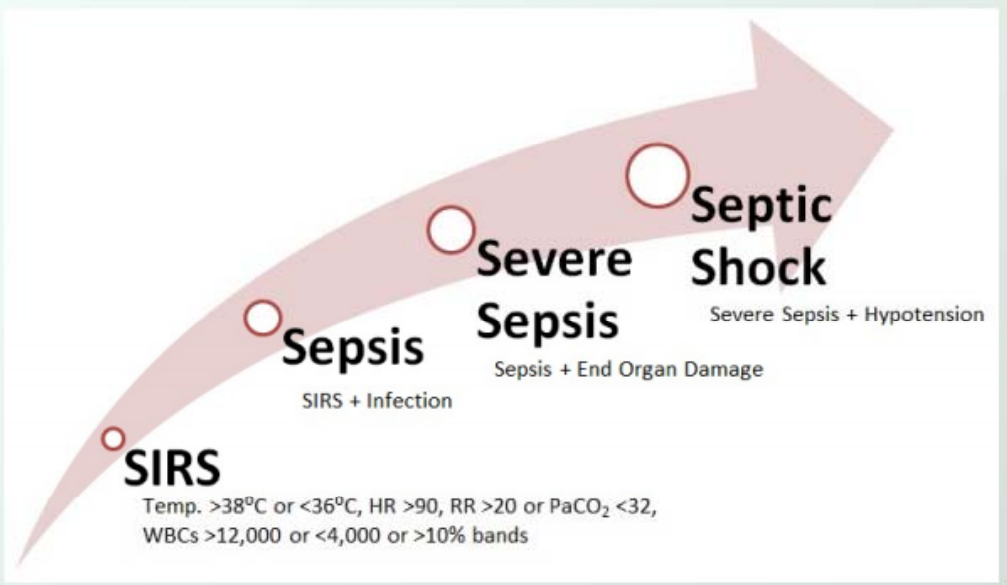
- 8 ώρες μετά προσέρχεται στα ΤΕΠ λόγω επιδείνωσης της γενικής του κατάστασης
- Ο ασθενής είναι συγχυτικός με κρύα άκρα και περιφερική κυάνωση. ΑΠ 90/50mmHg, Σφ 110/min, SO2 90% στον αέρα, RR 24/min
- Κλινική εξέταση: μείωση αναπνευστικού ψιθυρίσματος ΔΕ, S1S2 βύθιοι, ταχείς, ρυθμικοί, κοιλία μαλακή, ευπίεστη
- Τίθεται οξυγονοθεραπεία με MV, ζητείται εργαστηριακός έλεγχος και ενυδάτωση με N/S

Με βάση τον καινούριο ορισμό της σήψης ο ασθενής είναι σηπτικός?

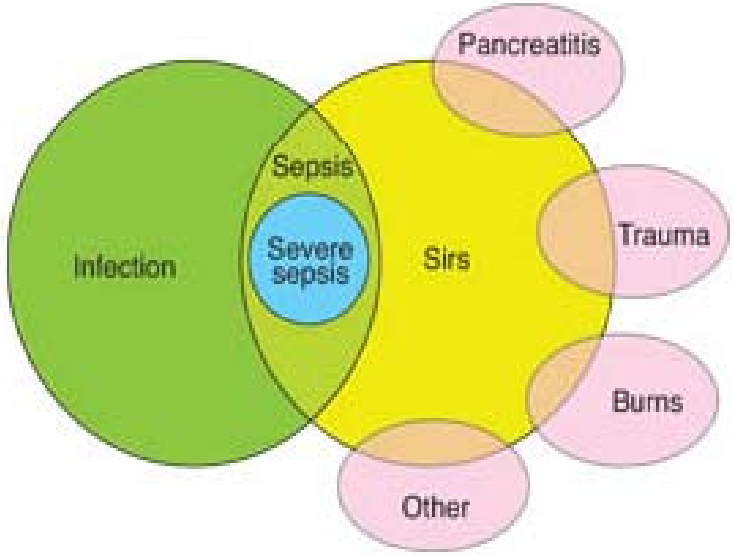
Surviving Sepsis Campaign

π.X.

ACCP/SCCM Consencus Conference 1991 (Sepsis-1)



Sepsis is the host's inflammatory response to



Incidence and Prognostic Value of the Systemic Inflammatory Response Syndrome and Organ Dysfunctions in Ward Patients

Matthew M. Churpek[1], Frank J. Zadravecz[1], Christopher Winslow[2], Michael D. Howell[1], and Dana P. Edelson[1]

Am J Respir Crit Care Med. 2015

Nearly half of all patients hospitalized on the wards met SIRS criteria at least once during their stay.

Systemic Inflammatory Response Syndrome Criteria in Defining Severe Sepsis

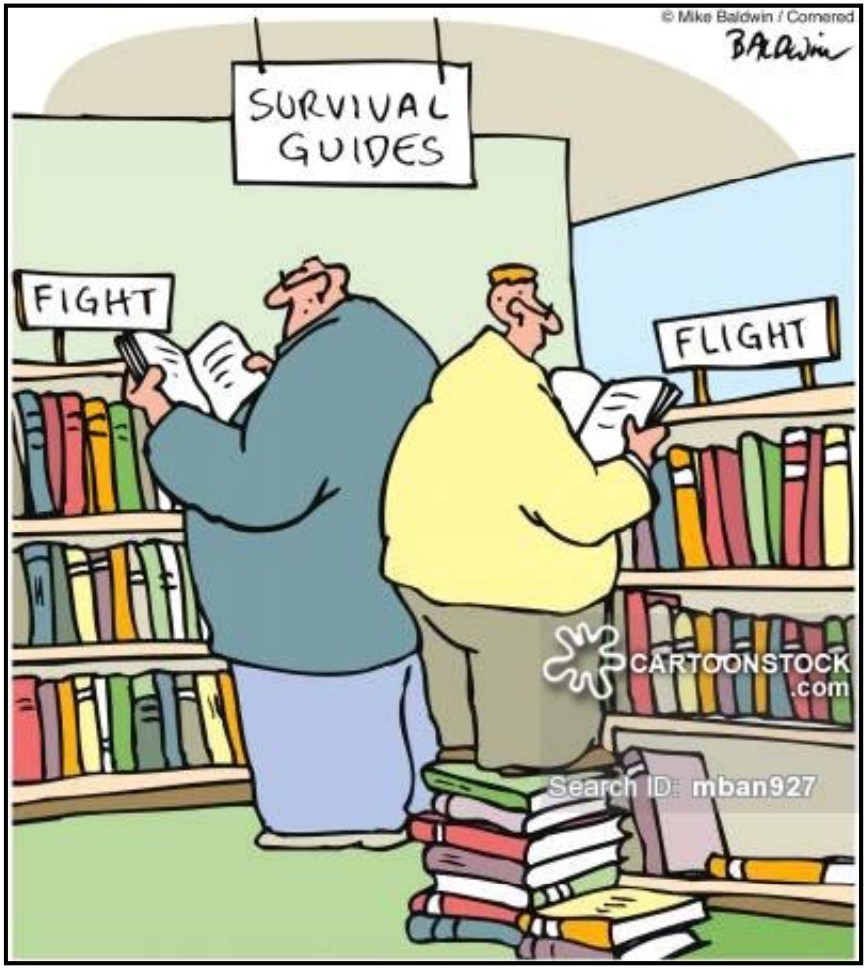
Kirsi-Maija Kaukonen, M.D., Ph.D., Michael Bailey, Ph.D., David Pilcher, F.C.I.C.M., D. Jamie Cooper, M.D., Ph.D., and Rinaldo Bellomo, M.D., Ph.D.

N Engl J Med. 2015

- SIRS-criteria rule missed one patient in eight with severe sepsis
- No transition point in mortality with "2 or more SIRS criteria"!

Surviving Sepsis Campaign

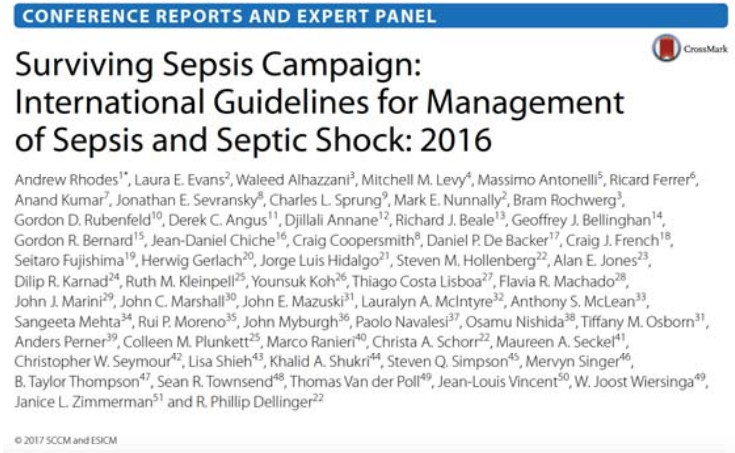
μ.X.



The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3)

Sepsis: life-threatening organ dysfunction caused by a dysregulated host response to infection

Septic shock: subset of sepsis in which particularly profound circulatory, cellular, and metabolic abnormalities are associated with a greater risk of mortality than with sepsis alone.


CONFERENCE REPORTS AND EXPERT PANEL

Surviving Sepsis Campaign: International Guidelines for Management of Sepsis and Septic Shock: 2016

Andrew Rhodes[1*], Laura E. Evans[2], Waleed Alhazzani[3], Mitchell M. Levy[4], Massimo Antonelli[5], Ricard Ferrer[6], Anand Kumar[7], Jonathan E. Sevransky[8], Charles L. Sprung[9], Mark E. Nunnally[2], Bram Rochwerg[3], Gordon D. Rubenfeld[10], Derek C. Angus[11], Djillali Annane[12], Richard J. Beale[13], Geoffrey J. Bellinghan[14], Gordon R. Bernard[15], Jean-Daniel Chiche[16], Craig Coopersmith[8], Daniel P. De Backer[17], Craig J. French[18], Seitaro Fujishima[19], Herwig Gerlach[20], Jorge Luis Hidalgo[21], Steven M. Hollenberg[22], Alan E. Jones[23], Dilip R. Karnad[24], Ruth M. Kleinpell[25], Younsuk Koh[26], Thiago Costa Lisboa[27], Flavia R. Machado[28], John J. Marini[29], John C. Marshall[30], John E. Mazuski[31], Lauralyn A. McIntyre[32], Anthony S. McLean[33], Sangeeta Mehta[34], Rui P. Moreno[35], John Myburgh[36], Paolo Navalesi[37], Osamu Nishida[38], Tiffany M. Osborn[31], Anders Perner[39], Colleen M. Plunkett[25], Marco Ranieri[40], Christa A. Schorr[22], Maureen A. Seckel[41], Christopher W. Seymour[42], Lisa Shieh[43], Khalid A. Shukri[44], Steven Q. Simpson[45], Mervyn Singer[46], B. Taylor Thompson[47], Sean R. Townsend[48], Thomas Van der Poll[49], Jean-Louis Vincent[50], W. Joost Wiersinga[49], Janice L. Zimmerman[51] and R. Phillip Dellinger[22]

JAMA. 2016

- Organ dysfunction can be represented by an increase in the Sequential (sepsis-related) Organ Failure Assessment (SOFA) score of 2 points or more

- Septic shock should be defined as a subset of sepsis and should be clinically identified by a vasopressor requirement to maintain a mean arterial pressure of 65 mm Hg or greater AND serum lactate level greater than 2 mmol/L (>18 mg/dL) in the absence of hypovolemia.

Sequential [Sepsis-related] Organ Failure Assessment (SOFA) score. Adapted from Sepsis-3.

PaO_2/FiO_2 (mmHg)	SOFA score
<400	1
<300	2
<200 and mechanically ventilated	3
<100 and mechanically ventilated	4
Glasgow coma scale	
13–14	1
10–12	2
6–9	3
<6	4
Mean arterial pressure OR administration of vasopressors required	SOFA score
MAP <70 mm/Hg	1
dop ≤5 or dob (any dose)	2
dop >5 OR epi ≤0.1 OR nor ≤0.1	3
dop >15 OR epi >0.1 OR nor >0.1	4
Bilirubin (mg/dl) [μmol/L]	
1.2–1.9 [>20–32]	1
2.0–5.9 [33–101]	2
6.0–11.9 [102–204]	3
>12.0 [>204]	4
Platelets × 10^3/μl	
<150	1
<100	2
<50	3
<20	4
Creatinine (mg/dl) [μmol/L] (or urine output)	
1.2–1.9 [110–170]	1
2.0–3.4 [171–298, 305]	2
3.5–4.9 [300–440] (or <500 ml/d)	3
>5.0 [>440] (or <200 ml/d)	4

ALTERED MENTAL STATUS

FAST RESPIRATORY RATE

LOW BLOOD PRESSURE

qSOFA

Why vasopressors AND lactate?

- Risk-adjusted hospital mortality was significantly higher in patients with fluid-resistant hypotension requiring vasopressors and hyperlactatemia.

- Lactate level is a sensitive, albeit nonspecific, stand-alone indicator of cellular or metabolic stress rather than "shock."
- Identification of septic shock as a distinct entity is of epidemiologic rather than clinical importance.
- Although hyperlactatemia and hypotension are clinically concerning as separate entities, and although the proposed criteria differ from those of other recent consensus statements, clinical management should not be affected.

Distribution and Mortality in Septic Shock Cohorts from Surviving Sepsis Campaign Database

	Hypotension after fluids	*Vasopressors*	*Lactate >2 mmol/L*	*Prevalence, Surviving Sepsis Campaign Database (n = 18,840 patients)*	*Hospital mortality*
Group 1[a]	Yes	Yes	Yes	8,520 (45.2%)	42.3%
Group 2[b]	Yes	Yes	No	3,985 (21.2%)	30.1%
Group 3	Yes	No	Yes	223 (1.2%)	28.7%
Group 4	No	No	Yes	3,266 (17.3%)	25.7%
Group 5	Never (pre)	No	Yes	2,696 (14.3%)	29.7%
Group 6	Yes	No	No	150 (0.8%)	18.7%

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016

Ερώτηση 1

Ποια είναι η πρωταρχική παθοφυσιολογική ανωμαλία στο σηπτικό σοκ;

1. Απώλεια αγγειοκινητικου τόνο με αποτέλεσμα την μείωση του προφόρτιου και την αύξηση του μεταφόρτιου της αριστερής κοιλίας, χαμηλή μέση αρτηριακή πίεση και αυξημένη ιστική άρδευση

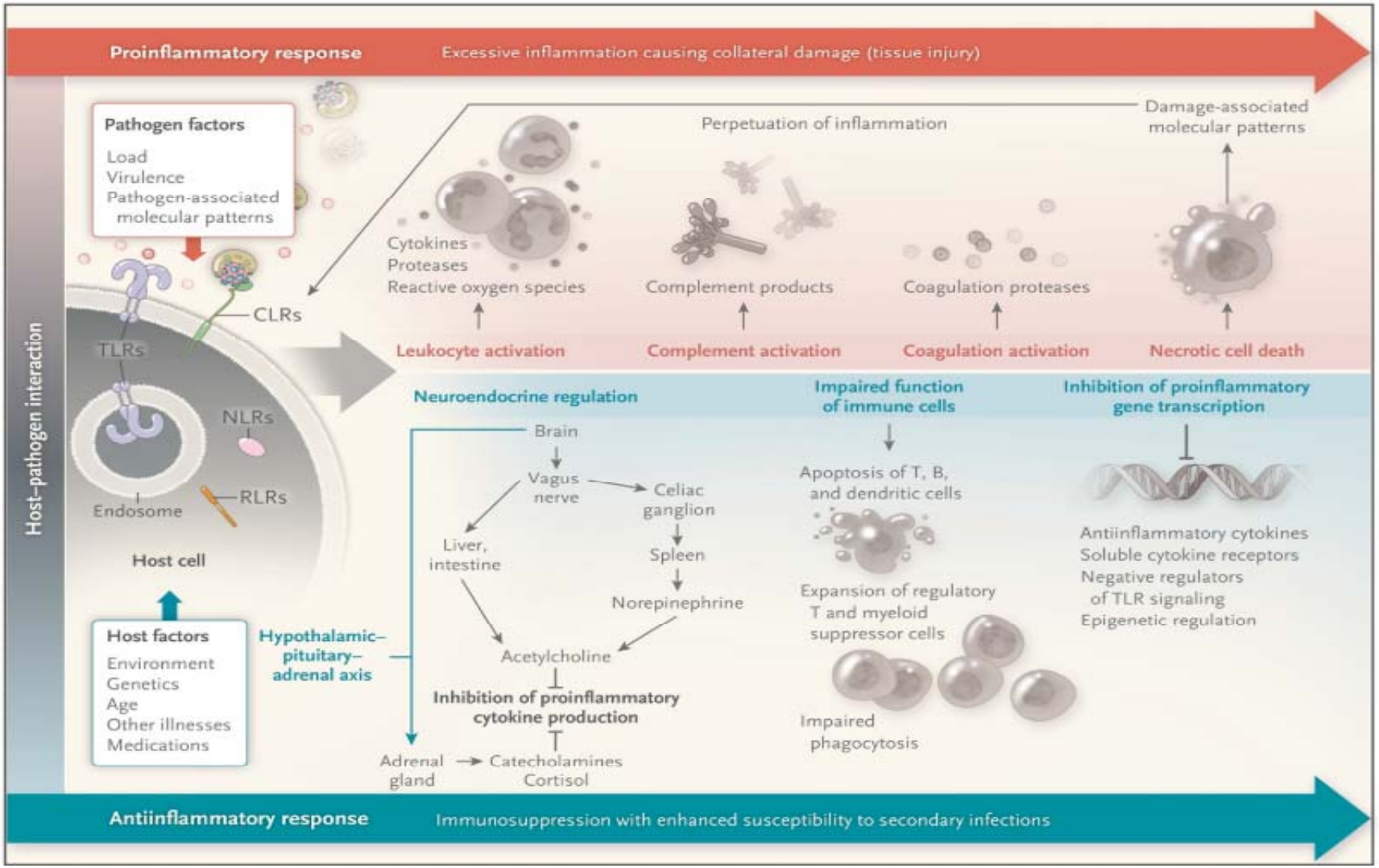
2. Απώλεια αγγειοκινητικου τόνο με αποτέλεσμα την αύξηση του προφόρτιου και του μεταφόρτιου της αριστερής κοιλίας, χαμηλή μέση αρτηριακή πίεση και φτωχή ιστική άρδευση

3. Απώλεια αγγειοκινητικου τόνο με αποτέλεσμα την μείωση του προφόρτιου και του μεταφόρτιου της αριστερής κοιλίας, χαμηλή μέση αρτηριακή πίεση και φτωχή ιστική άρδευση

4. Απώλεια αγγειοκινητικου τόνο με αποτέλεσμα την αύξηση του προφόρτιου και του μεταφόρτιου της αριστερής κοιλίας, χαμηλή μέση αρτηριακή πίεση και αυξημένη ιστική άρδευση

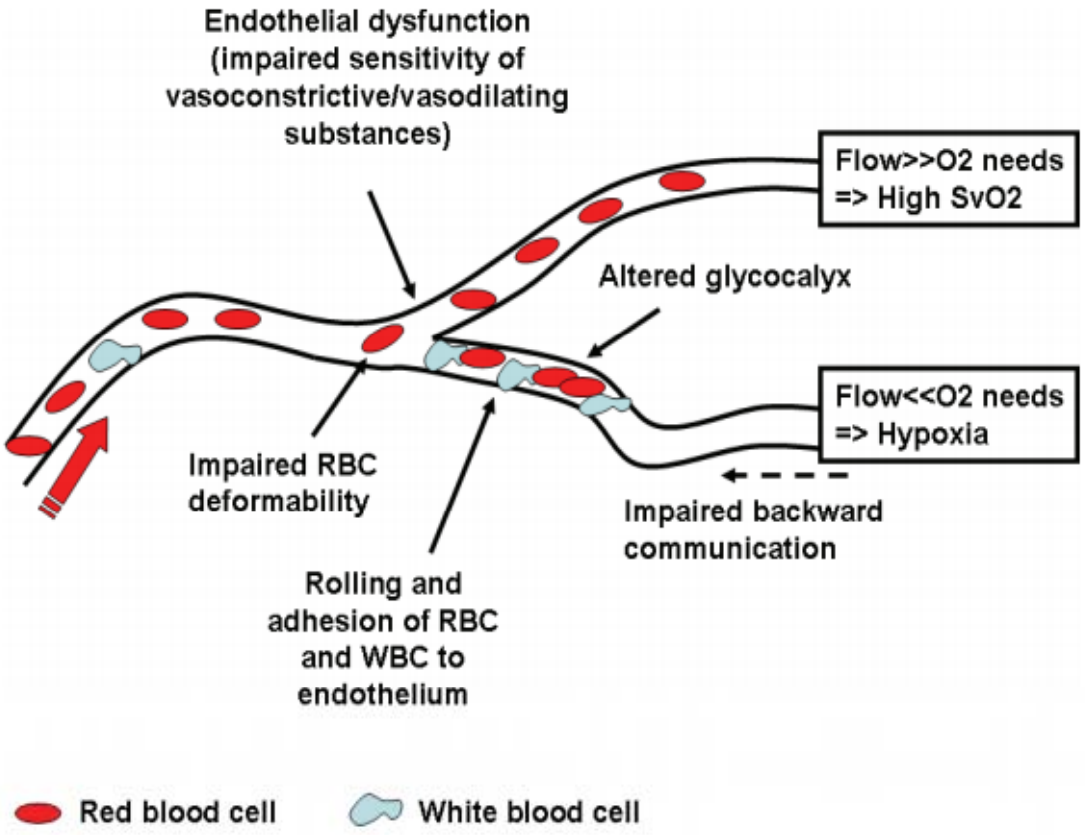
5. Όλα είναι λάθος

Sepsis pathogenesis



Severe sepsis and septic shock. NEJM. 2013

Microcirculation in sepsis

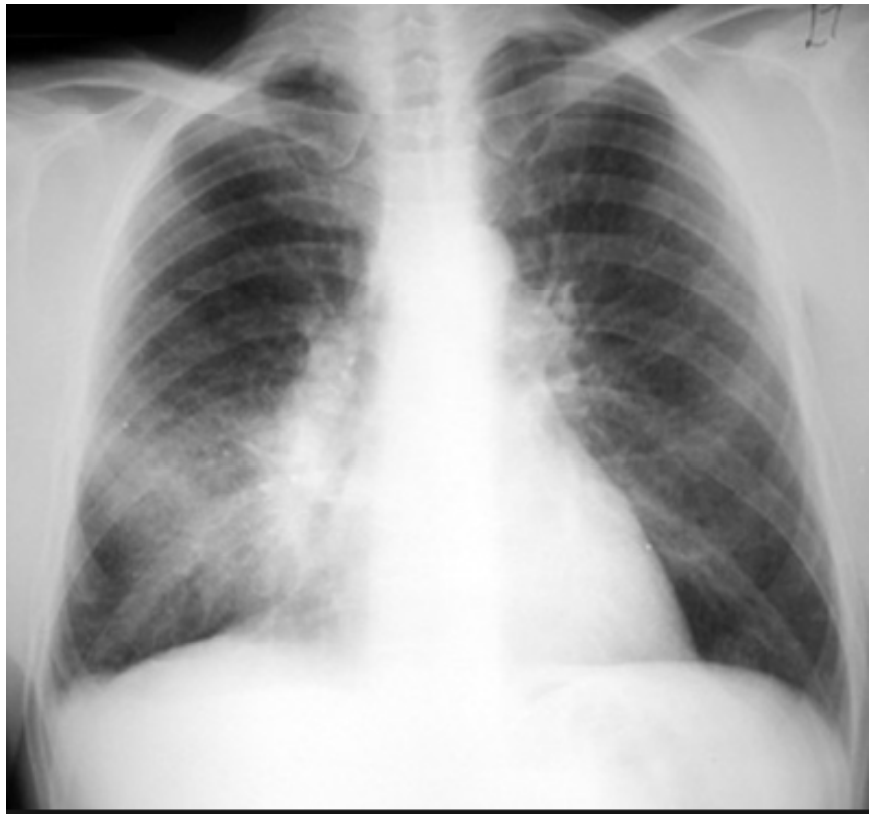


Microcirculatory alterations: potential mechanisms and implications for therapy. Ann Intensive care. 2011

Παρουσίαση περιστατικού (συνέχεια)

Παρακλινικός έλεγχος

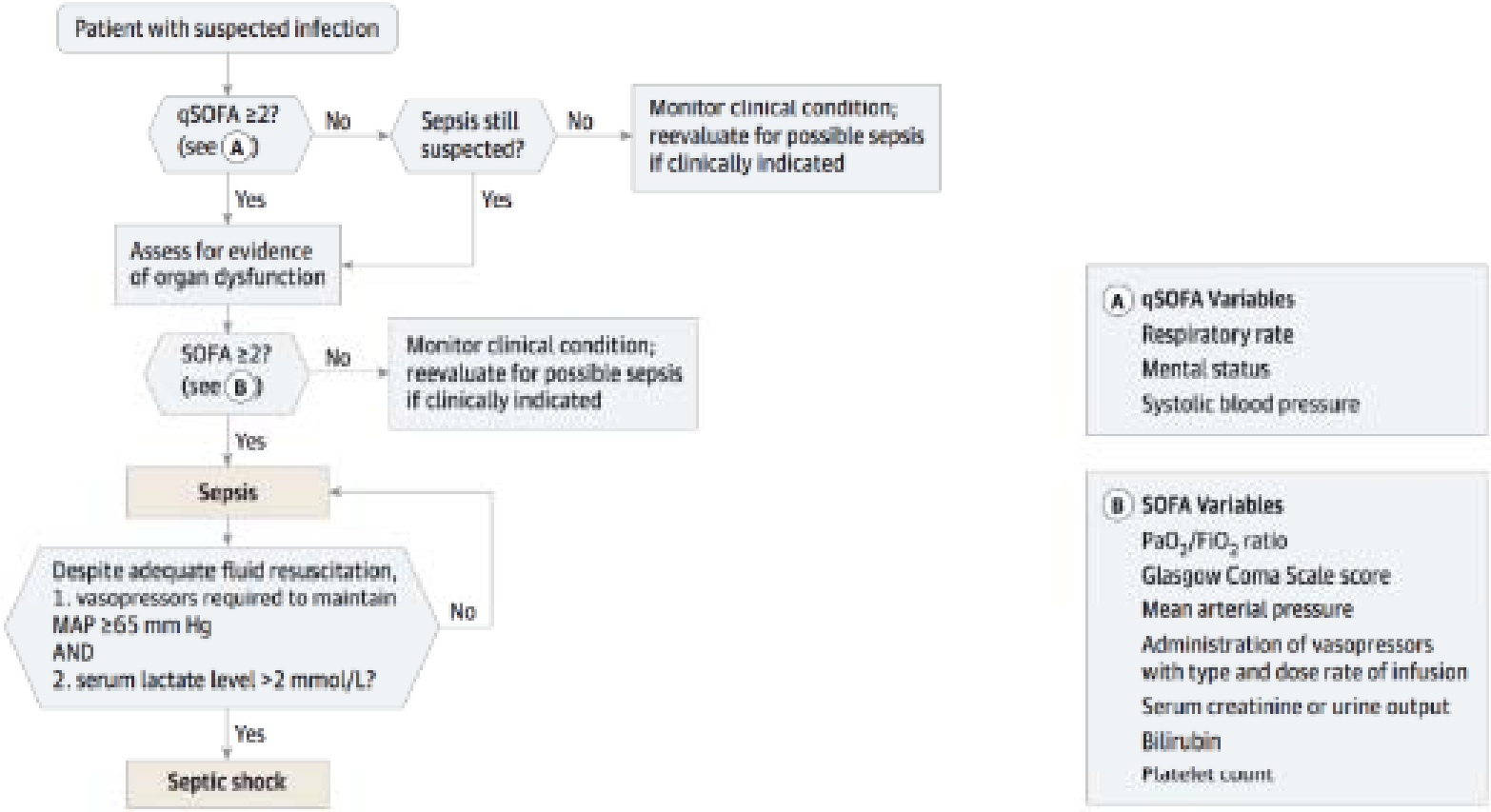
- WBC 18.8G/l, Hb 14.5g/dl, PLT 128G/l
- creat 1.8mg/dl, bil 0.6, ηπατική βιοχημεία κ.φ, CRP 5.5mg/dl
- Ετοιμάζεται εισαγωγή στην Παθολογική Κλινική ως λοίμωξη αναπνευστικού



Κατά την παραμονή στα ΤΕΠ:

- Χορήγηση υγρών (N/S 0.9% 1l)
- Λήψη κ/α αίματος
- Έναρξη αντιβιοτικής αγωγής με πιπερακιλλίνη/ταζομπακτάμη και μοξιφλοξασίνη
- ABG : Arterial blood gases: pH 7.21, pCO2 25 mmHg, PaO2 58 mmHg, HCO3 16 mmol/l), lactate 5 mmol/L
- Ο ασθενής παραμένει αιμοδυναμικά ασταθής. Βύθιος.
- Έναρξη έγχυσης νορεπινεφρίνης 0.5μg/kg/min
- Ο ασθενής διασωληνώτεται λόγω αιμοδυναμικής αστάθειας και χαμηλού επιπέδου συνείδησης
- ΑΠ 100/75mmHg

Figure. Operationalization of Clinical Criteria Identifying Patients With Sepsis and Septic Shock



The baseline Sequential [Sepsis-related] Organ Failure Assessment (SOFA) score should be assumed to be zero unless the patient is known to have preexisting (acute or chronic) organ dysfunction before the onset of infection. qSOFA indicates quick SOFA; MAP, mean arterial pressure.

The Third International Consensus Definitions for Sepsis and Septic Shock (Sepsis-3). JAMA. 2016

qSOFA：3
SOFA score: 6

Ερώτηση 2

Θεωρείτε πως σωστά ξεκίνησαν στον ασθενή αγγειοσυσπαστικά ενώ δεν έχει ολοκληρωθεί η χορήγηση των υγρών ανάνηψης?

1. Ναι
2. Όχι

The Surviving Sepsis Campaign Bundle: 2018 Update

Mitchell M. Levy, MD, MCCM[1]; Laura E. Evans, MD, MSc, FCCM[2]; Andrew Rhodes, MBBS, FRCA, FRCP, FFICM, MD (res)[3]

Intensive Care Med. 2018

The hour-1 bundle

The most important change in the revision of the SSC bundles is that the 3-h and 6-h bundles have been combined into a single "hour-1 bundle" with the explicit intention of beginning resuscitation and management immediately. *We believe this reflects the clinical reality at the bedside of these seriously ill patients with sepsis and septic shock—that clinicians begin treatment immediately, especially in patients with hypotension, rather than waiting or extending resuscitation measures over a longer period.* More than 1 hour may be required for resuscitation to be completed, but initiation of resuscitation and treatment, such as obtaining blood for measuring lactate and blood cultures, administration of fluids and antibiotics, and in the case of life-threatening hypotension, initiation of vasopressor therapy, are all begun immediately. *We believe the new bundle is an accurate reflection of actual clinical*

Bundle Element	Grade of Recommendation and Level of Evidence
Measure lactate level. Re-measure if initial lactate is >2 mmol/L	Weak recommendation, low quality of evidence
Obtain blood cultures prior to administration of antibiotics	Best practice statement
Administer broad-spectrum antibiotics	Strong recommendation, moderate quality of evidence
Rapidly administer 30mL/kg crystalloid for hypotension or lactate ≥ 4 mmol/L	Strong recommendation, low quality of evidence
Apply vasopressors if patient is hypotensive during or after fluid resuscitation to maintain mean arterial pressure ≥ 65 mm Hg	Strong recommendation, moderate quality of evidence

Surviving sepsis campaign Bundle: 2018 Update

Surviving Sepsis Campaign

- We recommend that in the resuscitation from sepsis-induced hypoperfusion, at least 30ml/kg of intravenous crystalloid fluid be given within the first 3 hours.

(Strong recommendation; low quality of evidence)

- We recommend that following initial fluid resuscitation, additional fluids be guided by frequent reassessment of hemodynamic status.

(Best Practice Statement)

IV Fluids : Is there any safety limit needed (how much is too much)?

Fluid volume, fluid balance and patient outcome in severe sepsis and septic shock: A systematic review

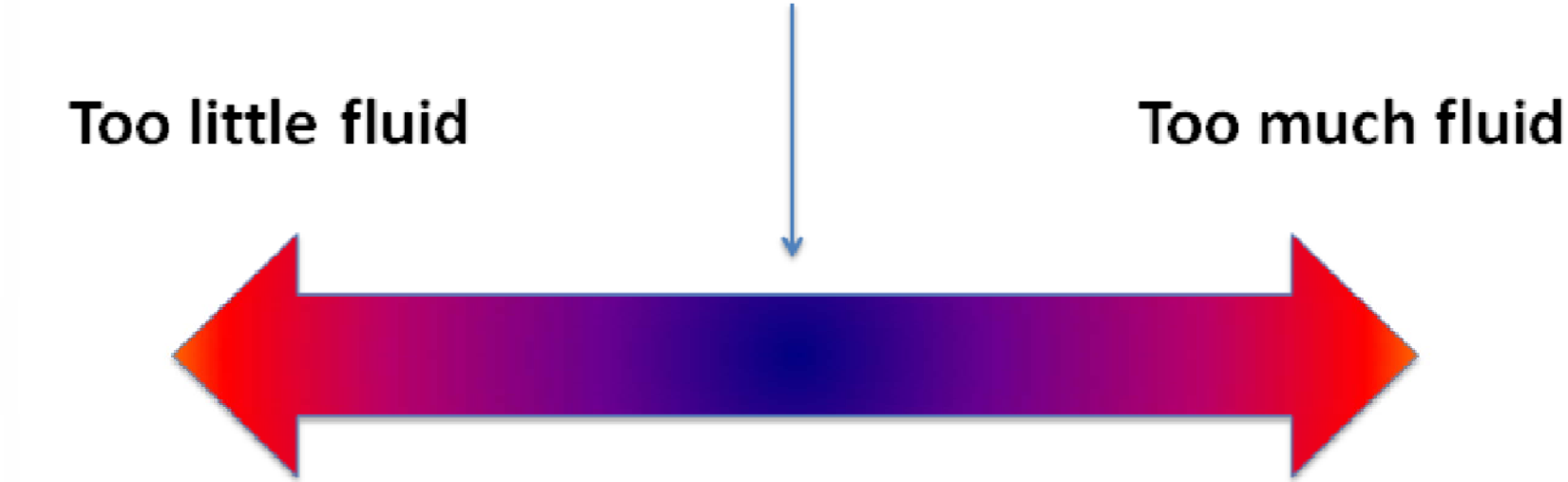
J Crit Care.2018

- Patients with a high fluid balance have a 70% increased risk of mortality. Survivors of severe sepsis and/or septic shock received higher fluid volume in the first three hours. However, fluid volume administered in the first 24 h was higher for non-survivors. Low volume resuscitation in the first 24 h had a significant mortality reduction.

- Heterogeneity regarding the setting, timing, and fluid dosing strategy used.

IV Fluids

Finding the middle for each patient requires combining many variables: physical exam, passive leg raise, IVC ultrasound, lactate, $ScvO_2$, urine output, trial and error, etc.



- More organ injury
- Higher mortality
- Vasopressor-induced digital necrosis

- Worse edema
- Impaired oxygen diffusion
- Worse ARDS

Is there an optimal MAP for patients with septic shock?

- Multicenter, open-label trial. 776 patients with septic shock
- 2 groups : MAP target of either 80 to 85 mm Hg (high-target group) or 65 to 70 mm Hg (low-target group)
- No significant difference in 28-day mortality. No significant between-group differences in the overall rates of organ dysfunction or death at 90 days.
- In patients with a history of chronic arterial hypertension, targeting a mean arterial pressure of 80 to 85 mm Hg reduced both the incidence of a doubling of the blood creatinine level and the rate of renal-replacement therapy.
- No significant between-group difference in the overall rate of serious adverse events, but patients in the high-target group had significantly more episodes of atrial fibrillation.

Ερώτηση 3

Αν το γαλακτικό είναι αυξημένο (> 2mmol/L) πόσο συχνά πρέπει να το μετράμε?

1. Κάθε 1 ώρα
2. Κάθε 2−4 ώρες
3. Κάθε 8-12 ώρες
4. Δεν χρειαζεται να ξαναμετρηθεί

Measure lactate level

- We suggest guiding resuscitation to normalize lactate in patients with elevated lactate levels as a marker of tissue hypoperfusion.

  (Weak recommendation; low quality of evidence)

- While serum lactate is not a direct measure of tissue perfusion, it can serve as a surrogate, as increases may represent tissue hypoxia, accelerated aerobic glycolysis driven by excess beta-adrenergic stimulation, or other causes associated with worse outcomes. Randomized controlled trials have demonstrated a significant reduction in mortality with lactate- guided resuscitation

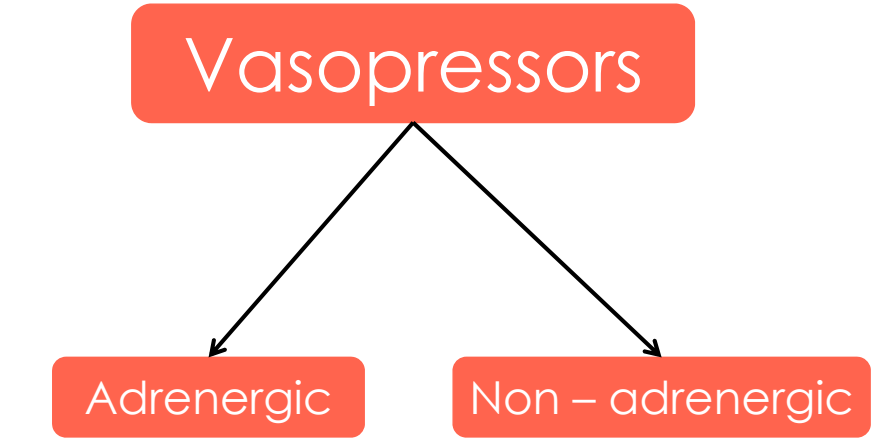
Τι έγινε όμως τελικά με τον άρρωστο?

3 ώρες μετά μεταφέρεται στην ΜΕΘ

- Ο ασθενής παραμένει αιμοδυναμικά ασταθής, ανουρικός με δικτυωτή πελίωση

  Ag Str. Pneumoniae ούρων Θετικό

- ΤοποΘέτηση ΚΦΓ
- CVP 2mmHg, ScvO2 52%. ABG: pH 7.2, pCO2 39mmHg, PaO2 75 mmHg, HCO3 14 mmol/l), lactate 10 mmol/L
- Χορήγηση 2l R/L
- Νορεπινεφρίνη σε δόση 1μg/kg/h
- Έναρξη CRRT



- Norepinephrine
- Epinephrine (adrenaline)
- Dopamine
- Phenylephrine

- Vasopressin
- Angiotensin*

Inotropes

Dobutamine

No study to date has demonstrated a statistically significant survival benefit of one vasopressor over another. Therefore, the choice of vasopressor in septic shock is rather empiric

Vasoactive medication receptor activity and clinical effects

Drug	Receptor activity				Predominant clinical effects
	Alpha-1	Beta-1	Beta-2	Dopaminergic	
Phenylephrine	+++	0	0	0	SVR ↑ ↑, CO ↔/↑
Norepinephrine	+++	++	0	0	SVR ↑ ↑, CO ↔/↑
Epinephrine	+++	+++	++	0	CO ↑ ↑, SVR ↓ (low dose) SVR/↑ (higher dose)
Dopamine (mcg/kg/min)*					
0.5 to 2.	0	+	0	++	CO
5. to 10.	+	++	0	++	CO ↑, SVR ↑
10. to 20.	++	++	0	++	SVR ↑ ↑
Dobutamine	0/+	+++	++	0	CO ↑, SVR ↓

Vasopressors for the Treatment of Septic Shock: Systematic Review and Meta-Analysis

PLoS One. 2015

Norepinephrine versus Dopamine

- Absolute reduction of 11% in 28-days all-cause mortality with norepinephrine compared with dopamine corresponding to a number needed to treat of 9
- Dopamine resulted in more than twice the risk for major adverse effects including a twofold increase in the risk for cardiac arrhythmias.
- No mortality benefit with norepinephrine over epinephrine, phenylephrine and vasopressin / terlipressin or between the other comparisons, although a trend towards reduced mortality with norepinephrine was seen in all comparisons.

Epinephrine

- Epinephrine has potent inoconstriction and vasoconstriction effect via a- and β- adrenergic stimulation
- Increases mean arterial pressure by increasing both the cardiac index and peripheral vascular tone.
- Side effects: tachyarrhythmias, ischemia, hypoglycemia, increase in lactate concentrations (stimulation of β2-adrenergic receptors)
- As compared to the norepinephrine plus dobutamine regimen, epinephrine has no significant effect on mortality reduction
- For some patients with compromised cardiac function, epinephrine can be a useful alternative medication.

Norepinephrine plus dobutamine versus epinephrine alone for management of septic shock: a randomised trial. Lancet. 2007

Phenylephrine

- Pure α-adrenergic agonist
- Vasoconstriction with minimal cardiac inotropy or chronotropy
- Good option when tachyarrhythmias limit therapy with other vasopressors
- Fewer attentions in medical literature.
- Drug of choice in anesthesia-induced hypotension

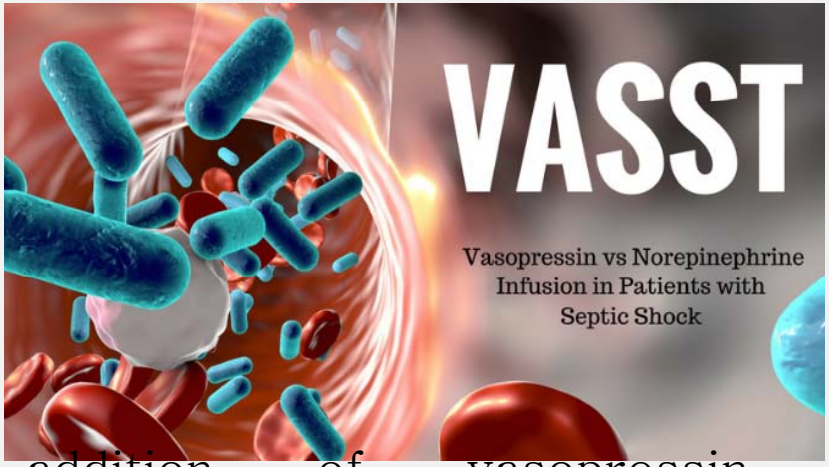
The Clinical Implications of Isolated Alpha1 Adrenergic Stimulation. Anesth Analg. 2011

Dobutamine

- Inotropic effect via stimulation of $\beta_1$receptors, with a variable effect on blood pressure. Increases cardiac output by increasing both contractility and heart rate, to a different extent in different patients.
- Dobutamine is also the first-line inotropic agent for patients with septic shock and low cardiac output in the presence of adequate filling pressures.

Vasopressin

Vasoconstriction via V1 vascular smooth muscle receptors.
Relative vasopressin deficiency in septic shock
Catecholamine-sparing effect



- addition of vasopressin to norepinephrine vs norepinephrine alone (0.01U-0.03U/min)
- No significant difference in mortality at 28/90 days, days alive and free of organ dysfunction, need for renal replacement therapy, need for corticosteroids, length of stay (ICU and hospital), adverse incidents.

N Engl J Med. 2008

Effect of Early Vasopressin vs Norepinephrine on Kidney Failure in Patients With Septic Shock The VANISH Randomized Clinical Trial

- 4 groups. Study drug 1: Vasopressin (titrated up to 0.06 U/min) vs Norepinephrine. Study drug 2: hydrocortisone vs placebo
- Early use of vasopressin to treat septic shock did not increase the number of kidney failure-free days compared with norepinephrine.
- Mortality rates were similar between all groups and there was no interaction on outcome between vasopressin and corticosteroids.

JAMA. 2016

Angiotensin II for the Treatment of Vasodilatory Shock

NEJM. 2017

- Multinational, double-blind, randomized, controlled trial. 344 enrolled patients
- Patients with vasodilatory shock who were receiving more than 0.2 µg of norepinephrine per kilogram of body weight per minute or the equivalent dose of another vasopressor to receive infusions of either angiotensin II or placebo.
- Angiotensin II administered intravenously increased blood pressure and allowed catecholamine dose reductions in patients with vasodilatory shock who were receiving high-dose vasopressors.
- Adverse effects : increased thrombotic events, thrombocytopenia, and infection risk

Ερώτηση 4

Ο ασθενής εμφανίζει ανθιστάμενο σοκ. Τι από τα παρακάτω θα χορηγούσατε;

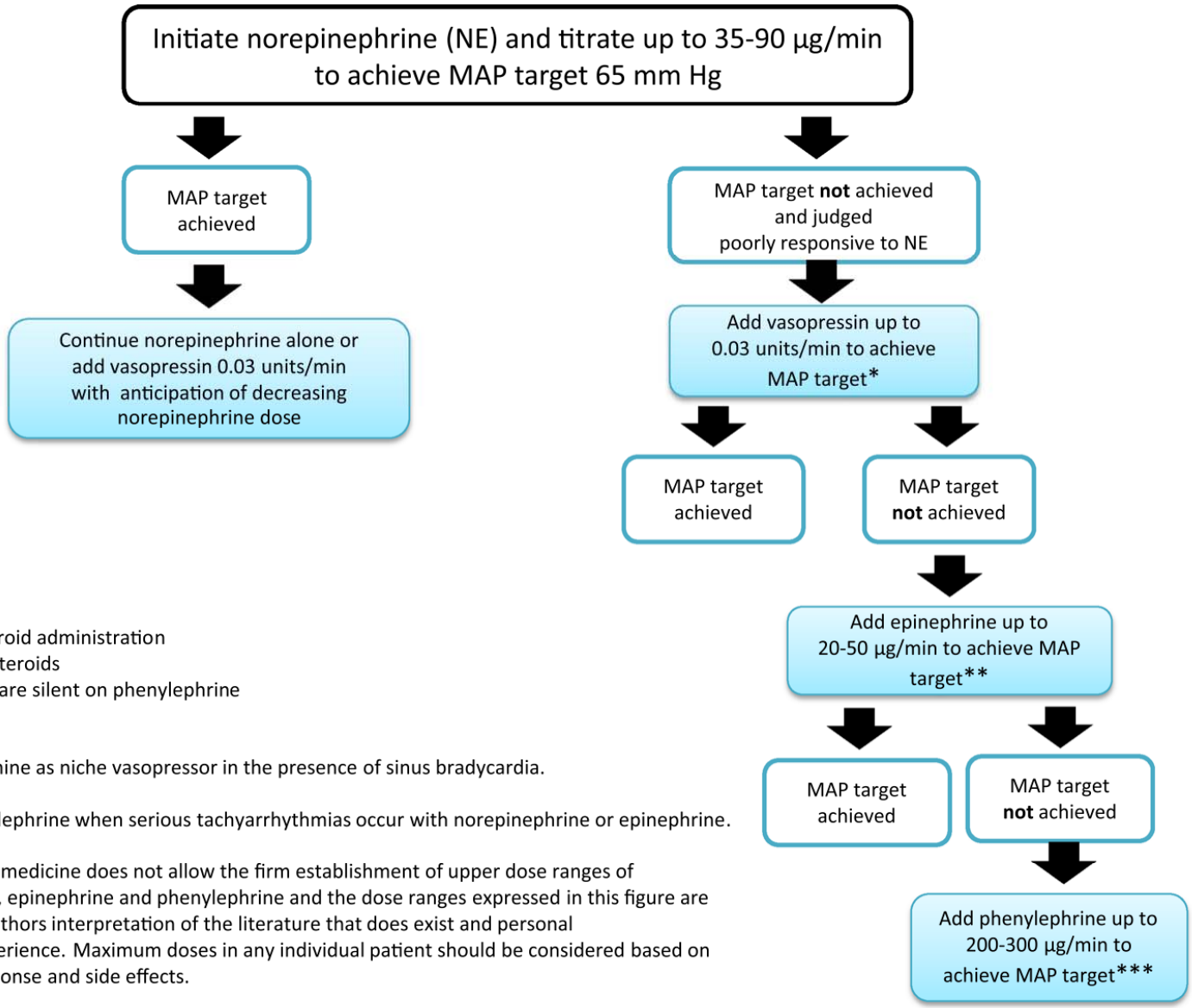
1. Υδροκορτιζόνη
2. Υδροκορτιζόνη και φθοριουδροκορτιζόνη
3. Βιταμίνη C
4. Υδροξυκοβαλαμίνη
5. Όλα τα παραπάνω είναι σωστά
6. Όλα τα παραπάνω είναι λάθος

Refractory shock

- Norepinephrine-equivalent doses of 0.5 mcg/kg/min or 1 mcg/kg/min have been proposed as thresholds to define high-dose vasopressor therapy and refractory shock.
- A reasonable definition of refractory shock would be an inadequate response to high-dose vasopressor therapy (defined as ≥0.5 mcg/kg/min norepinephrine-equivalent dose).

Management of Refractory Vasodilatory Shock. Chest 2018

Vasopressor Use for Adult Septic Shock (with guidance for steroid administration)

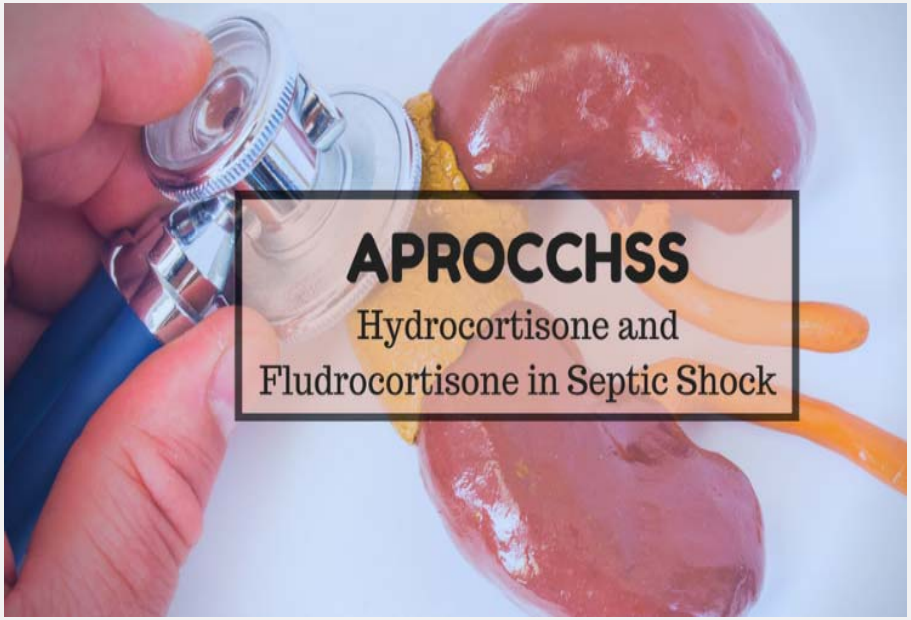


* Consider IV steroid administration
** Administer IV steroids
*** SSC guidelines are silent on phenylephrine

Notes:

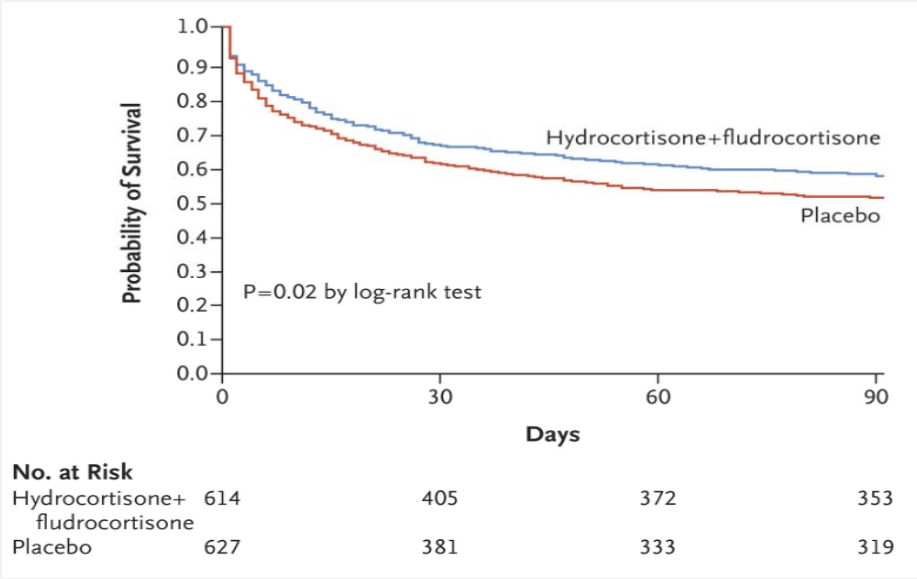
- Consider dopamine as niche vasopressor in the presence of sinus bradycardia.
- Consider phenylephrine when serious tachyarrhythmias occur with norepinephrine or epinephrine.
- Evidence based medicine does not allow the firm establishment of upper dose ranges of norepinephrine, epinephrine and phenylephrine and the dose ranges expressed in this figure are based on the authors interpretation of the literature that does exist and personal preference/experience. Maximum doses in any individual patient should be considered based on physiologic response and side effects.

A Users' Guide to the 2016 Surviving Sepsis Guidelines. Crit Care Med. 2017



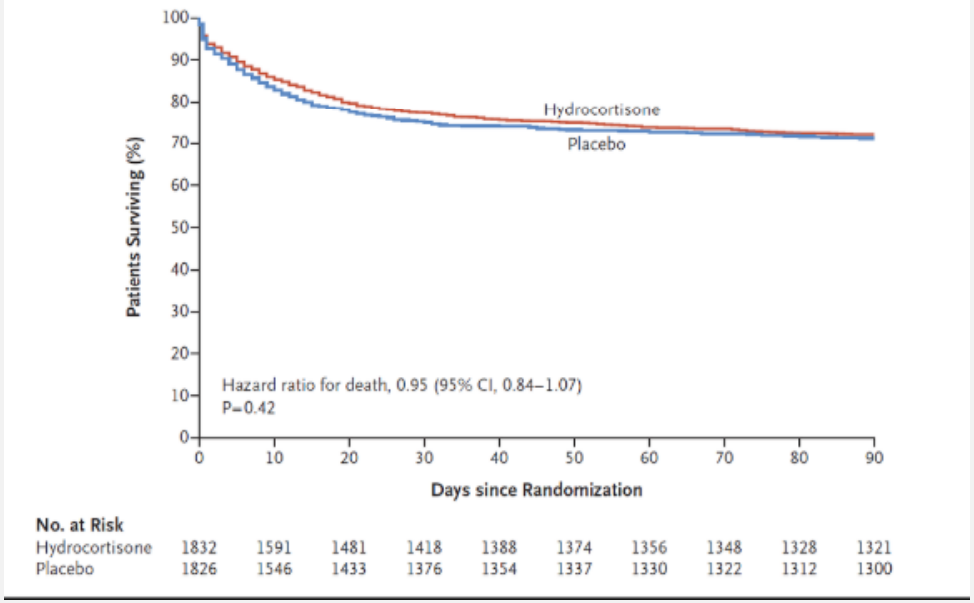
NEJM. 2018

Hydrocortisone 50mg x4 + fludrocortisone 50 mgx1 for 7 days. 1241 patients



NEJM. 2018

Hydrocortisone (at a dose of 200 mg per day) vs placebo for 7 days. 3800 patients



Corticosteroids in sepsis: The end of the road?

- Between 1976 and 2017, 22 randomised placebo-controlled trials with conflicting results.
- 2 recent meta-analyses in 2018 with conflicting results
- The available evidence suggests that in patients with septic shock, treatment with hydrocortisone reduces vasopressor dependency, time to extubation, and ICU length of stay.
- Hydrocortisone appears to reduce mortality in the sickest subgroup of patients with septic shock.

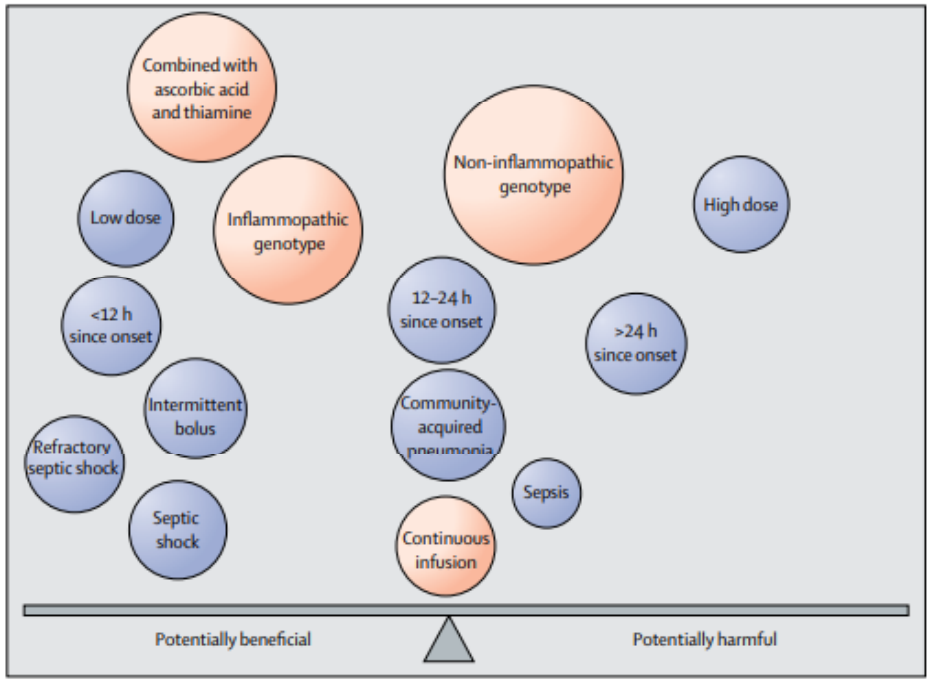
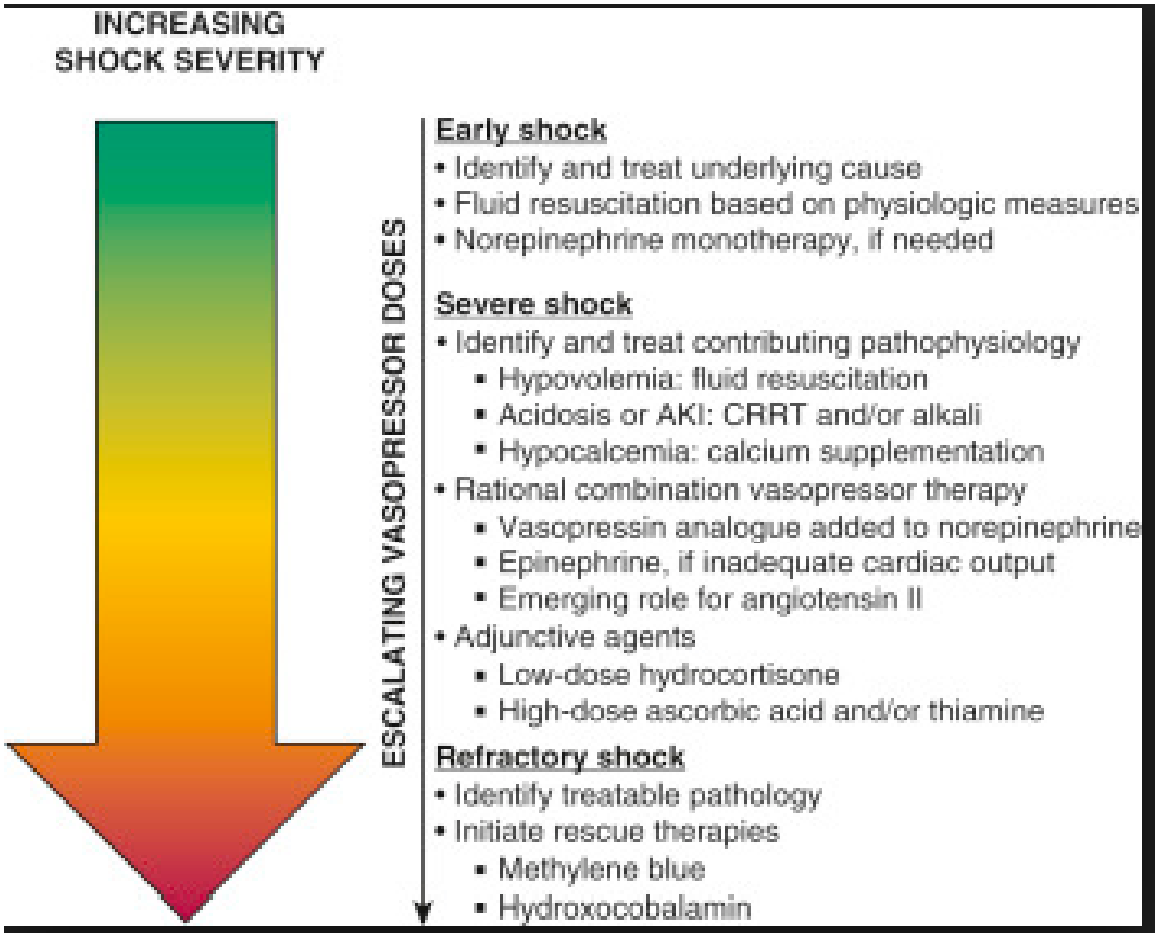


Figure: **Balance of the benefits and potential harms associated with the use of glucocorticoids in patients with sepsis and septic shock**
The orange bubbles are those with lower certainty of evidence. The blue bubbles are those with higher certainty of evidence.

The role of glucocorticoids as adjunctive treatment for sepsis in the modern era. Lancet Respir Med 2018



Management of refractory vasodilatory shock. Chest 2018

Hydrocortisone, Vitamin C, and Thiamine for the Treatment of Severe Sepsis and Septic Shock

Chest. 2017

Small, before-after study. 47 patients. Hospital mortality was 8.5% in the treatment group compared with 40.4% in the control group ($P < .001$).

6 ώρες μετά ...

- Επιδεινούμενη γαλακτική οξέωση (15mmol/l), CVP 15mmHg, ScvO2 48%.
- Προσθήκη βαζοπρεσίνης

7 ώρες μετά ...

- Άσφυγμη ηλεκτρική δραστηριότητα. Επαναφορά αυτόματης κυκλοφορίας μετά από 1 κύκλο CPR.
- Αύξηση νορεπινεφρίνης 3μg/kg/h
- 1l R/L
- Προσθήκη επινεφρίνης

Ποιά είναι κατά την γνώμη σας η αιτία της άσφυγμης ηλεκτρικής δραστηριότητας;

- Έμφραγμα μυοκαρδίου
- Πνευμονική εμβολή
- Απόφραξη στο χώρο εξόδου της αριστερής κοιλίας
- Καρδιακός επιπωματισμός
- Μη αναστρέψιμο σηπτικό σοκ

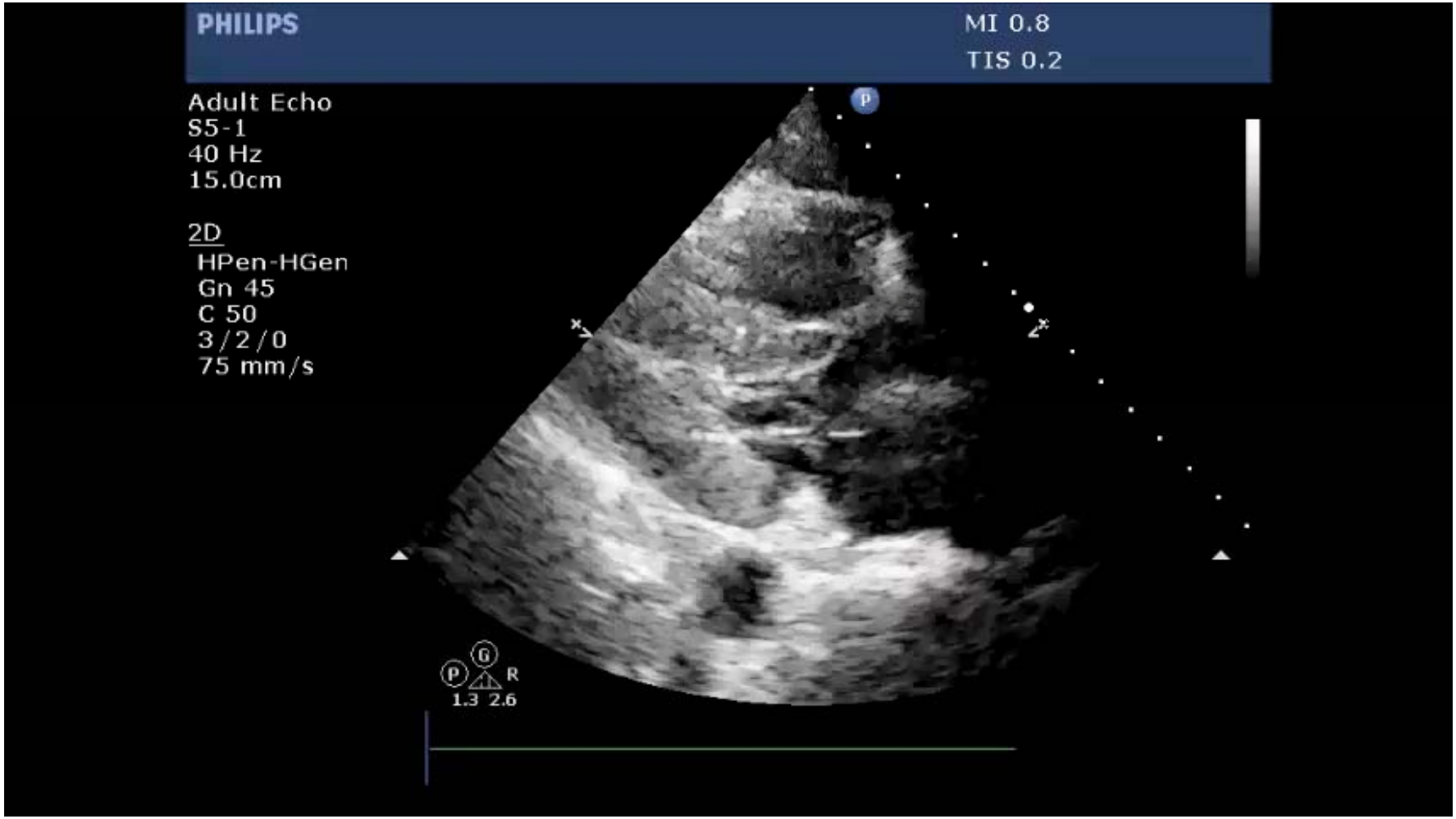
Ερώτηση 5

Ποιά εξέταση Θα ζητούσατε αυτή την στιγμή?

1. Αξονική τομογραφία Θώρακος με πρωτόκολλο πνευμονικής εμβολής
2. ΔιαΘωρακικό υπέρηχο καρδιάς
3. Ακτινογραφία Θώρακος
4. ΤοποΘέτηση καθετήρα swan ganz
5. Καρδιακά ένζυμα

Έγινε επειγόντως διαθωρακικό υπέρηχο καρδιάς για την αναζήτηση των πιθανών αιτίων για το ανθιστάμενο σοκ….

Υπέρηχο καρδιάς προ



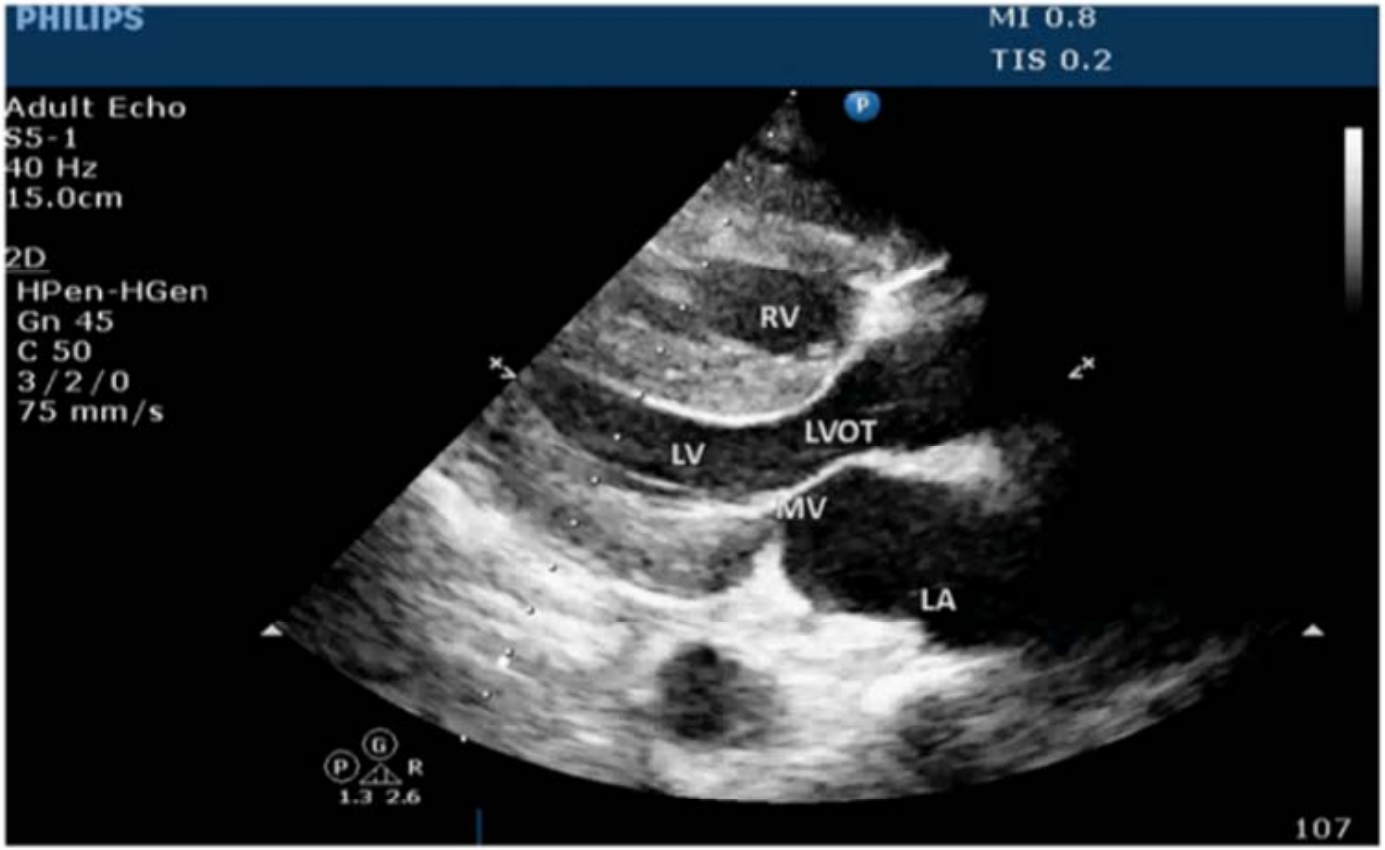


Figure 1. Parasternal long-axis view of the left atrium (LA), left ventricle (LV), mitral valve (MV), and left ventricle outflow tract (LVOT) during ventricular systole. BPM = beats per minute; RV = right ventricle.

Μικρή, υπερδυναμική αριστερή κοιλία με ανεπαρκή πλήρωση και συστολική πρόσθια κίνηση (SAM) της πρόσθιας μιτοειδικής γλωχίνας μέσα στον χώρο εξόδου της αριστερής κοιλίας.

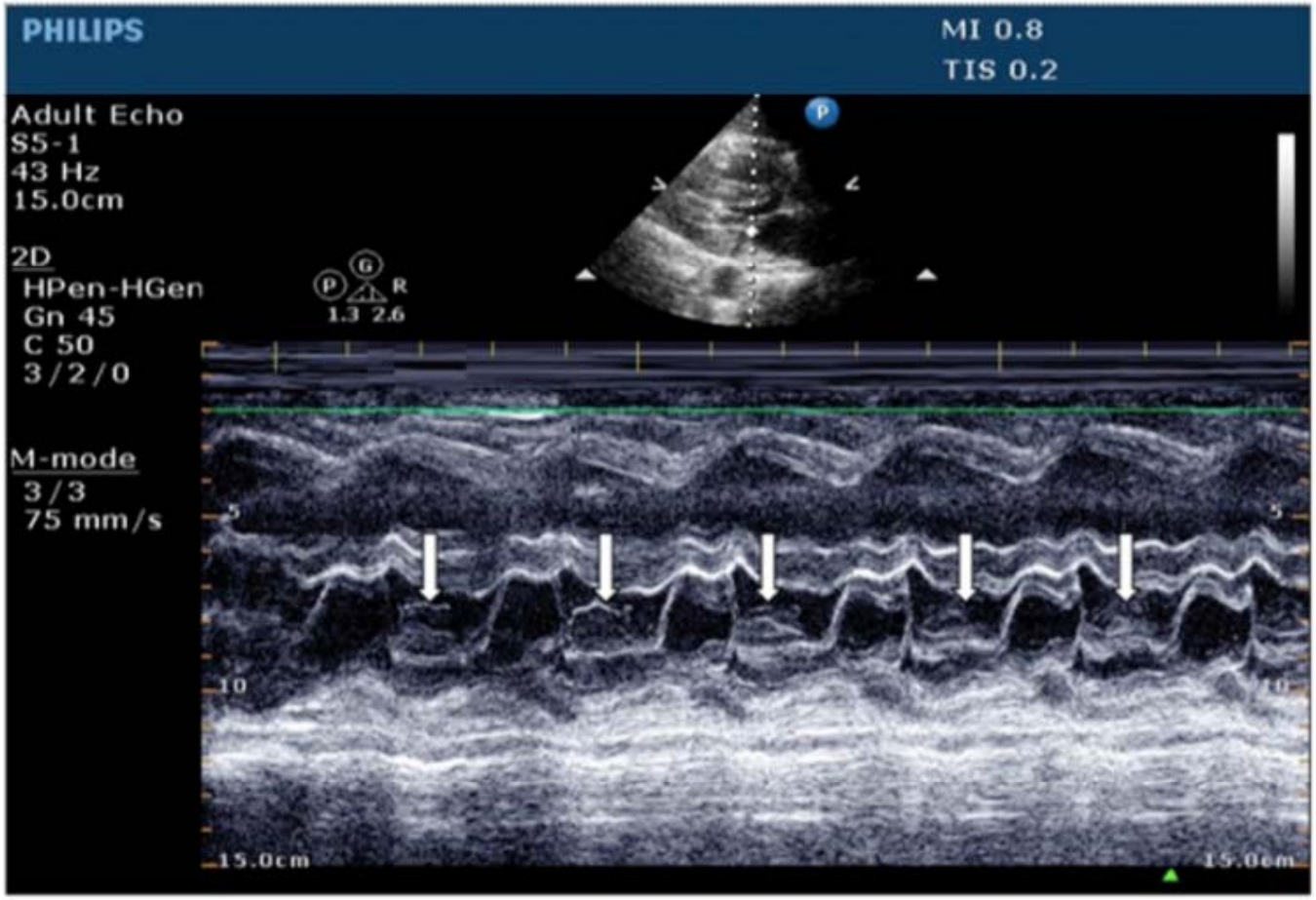


Figure 2. M-mode echocardiography. The *arrows* indicate systolic anterior motion of mitral leaflet during systole, which narrows the left ventricular outflow tract.

Η απόφραξη του χώρου εξόδου επιβεβαιώθηκε με την βοήθεια του M-n

Σηπτικό σοκ:

- απώλεια αγγειοκινητικού τόνου αγγείων
- μείωση προφορτίου και μεταφορτίου αριστερής κοιλίας
- χαμηλή ΜΑΠ και φτωχή ιστική οξυγόνωση

Απάντηση συμπαθητικού

- αύξηση καρδιακής συχνότητας και συσπαστικότητας μυοκαρδίου
- αύξηση καρδιακής παροχής
- βελτίωση ιστικής οξυγόνωσης

Εμφάνιση SAM, LVOT απόφραξης και καρδιαγγειακού collapse:

- μειωμένος τελοδιαστολικός όγκος αριστερής κοιλίας
- ταχυκαρδία
- υπερσυσπαστικότητα

Η χορήγηση 1L R/L στο CPR προφανώς ανέστρεψε την απόφραξη και βοήθησε στο να επανέλθει η κυκλοφορία….

Αλλά το echo καρδίας ανέδειξε το πρόβλημα αφού παραμένουν:

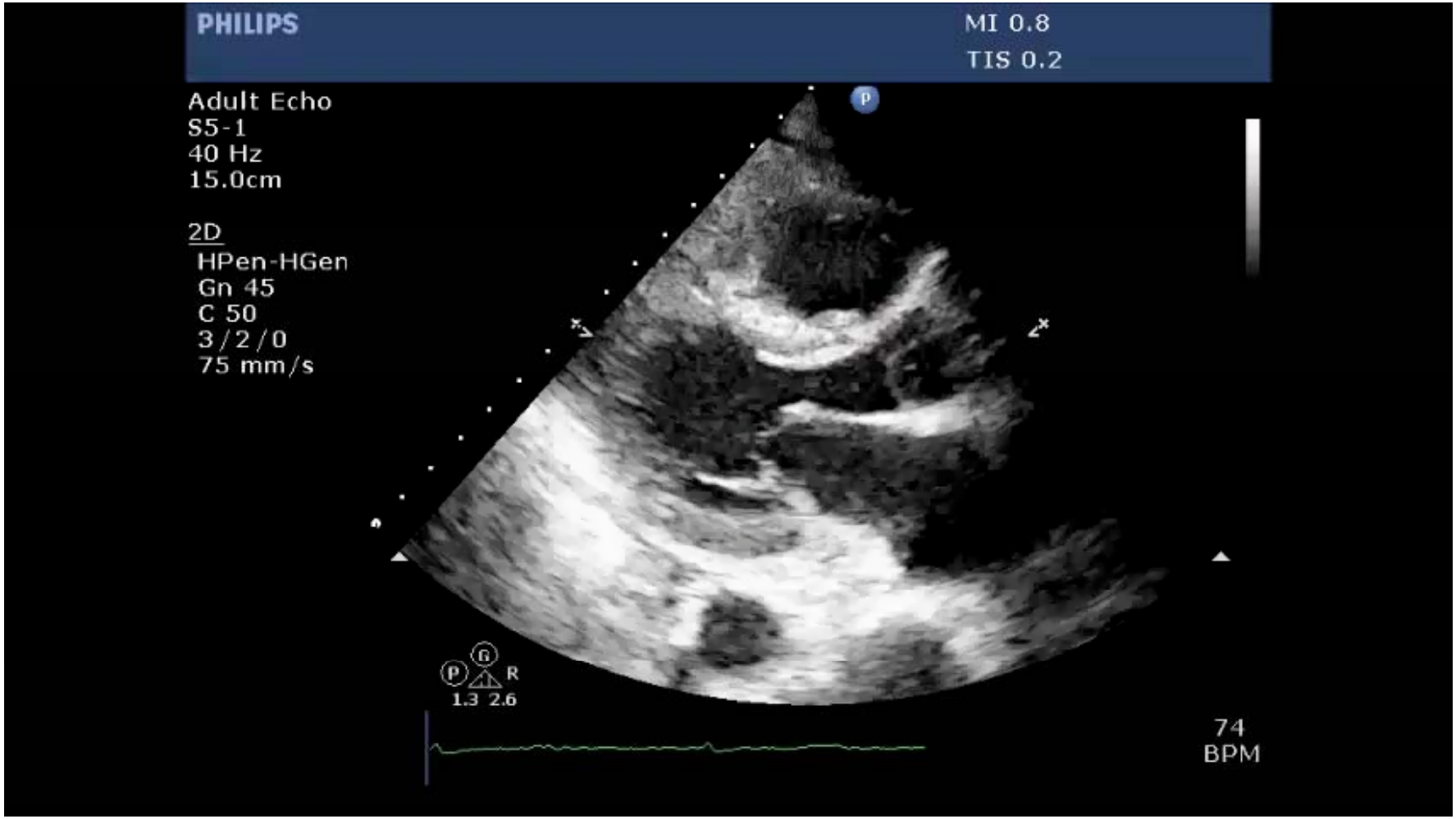
- Μειωμένος δραστικός ενδαγγειακός όγκος
- Ταχυκαρδία προκαλούμενη από κατεχολαμινεργικά φάρμακα
- Καθώς και καρδιακή υπερσυσπαστικότητα.

Θεραπευτική στρατηγική

- Χορήγηση επιπλέον υγρών (2 L Ringer lacate)
- Σταδιακή απόσυρση αγγειοσυσπαστικών
- Έναρξη χαμηλής δόσης β-blocker

Άμεση αιμοδυναμική βελτίωση

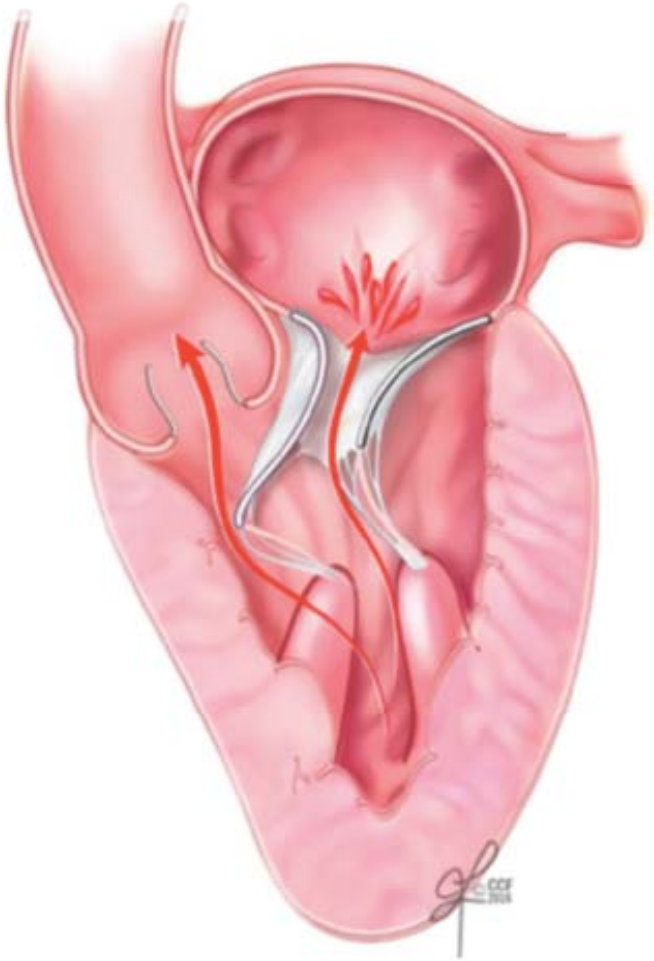
Υπέρηχος καρδιάς μετά



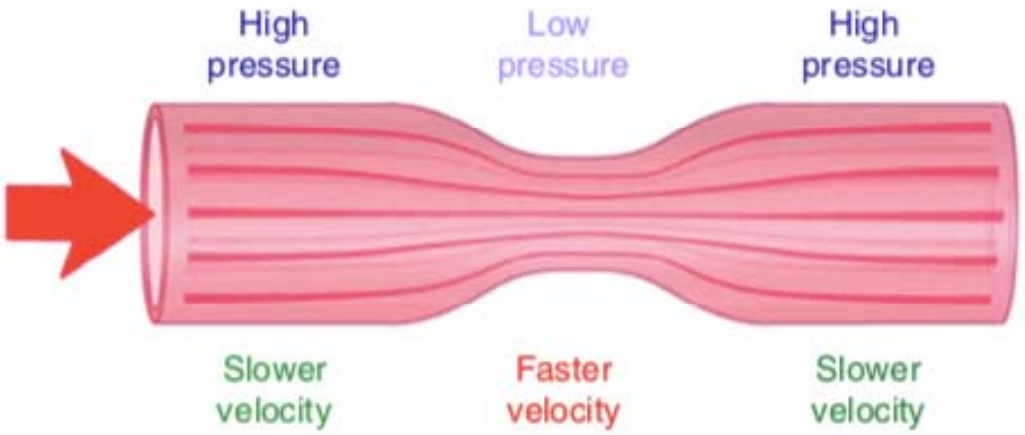
The Science behind the Solution

- SAM of mitral valve was first described in the 1960s. It is defined as the anterior excursion of one or both mitral valve leaflets into the LVOT during systole, which leads to narrowing of the LVOT

- The hemodynamic consequences of SAM are directly related to the duration and extent of contact between the mitral valve leaflet and the ventricular septum. Over the last decade, there has been growing recognition of the development of SAM in shock states, especially with the use of sympathomimetic medications.



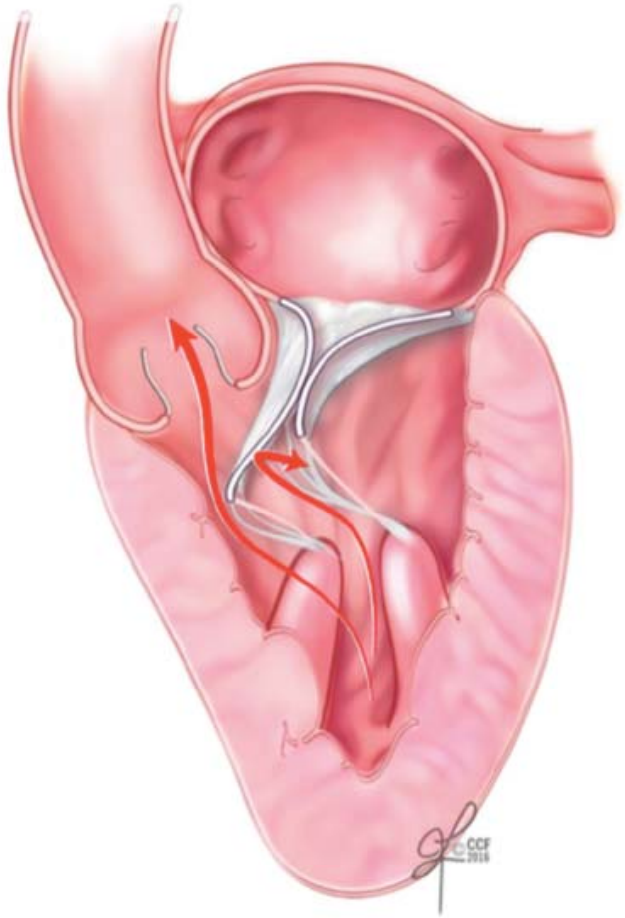
The Science behind the Solution



Systolic anterior motion of the mitral valve is believed to result, at least in part, from the Venturi effect, which describes the drop in pressure created when a liquid flows at high velocity through a narrowed orifice. When heart rate and contractility are increased, the high velocity through the LVOT is believed to pull the mitral valve leaflets toward the septum.

The Science behind the Solution

Alternatively, the drag effect hypothesizes that some patients are predisposed to the development of SAM because their mitral valve leaflets are positioned in the path of LVOT flow, which drags them anteriorly and superiorly toward the septum. Predisposing factors for SAM include any condition that reduces LV systolic cavity size or increases blood velocity through the LVOT.



Predisposing factors

Reduced LV cavity size during systole

- Decreased EDV
- Hypovolemia
- Tachycardia(reduced diastolic filling time)
- LV hypertrophy (diffuse or isolated to the septum)
- Increased contractility

Increased LV ejection velocity

- Increased contractility
- Reduced LV afterload

Effective therapy of SAM and LVOT obstruction is directed at reducing or correcting these predisposing factors.

Τελικά...

- Ο ασθενής σταθεροποιήθηκε αιμοδυναμικά.
- Ωστόσο παρέμεινε η νεφρική βλάβη για την οποία συνεχίστηκε η CRRT.
- Την 3η εβδομάδα νοσηλείας μπήκε σε πρόγραμμα αιμοκάθαρσης.
- Η νεφρική λειτουργία αποκαταστάθηκε σταδιακά μετά από 6 εβδομάδες.

«Όλα τα σηπτικά σοκ δεν είναι ίδια!»

Personalized medicine. One size does not fit all.

Ευχαριστώ!