Neden ? Ne zaman ? Hangi Sıvı? Ne kadar ?

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İç Hastalıkları Anabiim Dalı

YEDİTEPE ÜNİVERSİTESİ
HASTANESİ

TÜRK İÇ HASTALIKLARI UZMANLIK DERNEĞİ 1995

TDCY

ESICM
EUROPEAN SOCIETY OF INTENSIVE CARE MEDICINE

KEPAN
KLİNİK ENTERAL PARENTERAL NÜTRİSYON DERNEĞİ

Hastaya Sıvı Verilmesi

- **Neden ?**
- **Ne zaman ?**
- **Hangi Sıvı?**
- **Ne kadar ?**
- **Fazla sıvının zararları**

NEDEN?

1900 lu yılların başlarında sıvı tedavisi; strep sepsis, post partum hemoraji , diabetik koma da kullanılıyordu

Hipotansiyon
Taşikardi
Bulantı kusma
Karın ağrısı
Akut böbrek hasarı
Elektrolit imbalansı
Açlık

Son 100 yılda sıvı tedavisi benign kabül edilmiş

Refleks olarak sıvı tedavisi başlanmış

Doku perfüzyon bozukluğu ile seyreden hipovolemi, preload düşüklüğü durumlarında verilmeli

Foex BA. How the cholera epidemic of 1831 resulted in a new technique for fluid resuscitation. Emerg Med J. 2003;20:316-318.

Tablo 1- Şok Tiplerinde Hemodinamik Paternler

Parametre	Hipovolemik şok	Kardiyojenik şok	Vazodilatatör şok
CVP veya PAWB	Düşük	Yüksek	Düşük
Kardiyak Autput	Düşük	Düşük	Yüksek veya normal
Sistemik vasküler direnç	Yüksek	Yüksek	Düşük

PAWB: Pulmoner arter giriş basıncı

NEDEN?

Şoku sadece ‘hipotansiyon’ değil ,
‘hipoperfüzyon ve yetersiz doku oksijenlenmesi’
olarak tanımlamak daha kullanışlı

Kan basıncı henüz normal olmasına rağmen ScvO2 düşüklüğü ve kanda laktat yüksekliği olabilmektedir 1.

Tersi de olabilmeketedir, laktat yüksekliği olmadan persistan hipotansiyon olabilmektedir2.

Doku perfüzyonunun düzeltilmesi ve
organ disfonksiyonunun önlenmesi için intravasküler volüm durumu ve
kardiyak hemodinamikler çok önemlidir.

1. Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Peterson E, Tomlanovich M, Early Goal-Directed Therapy Collaborative G: **Early goal-directed therapy in the treatment of severe sepsis and septic shock**. *New Engl JMed* 2001, **345**(19):1368-1377.
2. Hernandez G, Castro R, Romero C, de la Hoz C, Angulo D, Aranguiz I, Larrondo J, Bujes A, Bruhn A: **Persistent sepsis-induced hypotension without hyperlactatemia: is it really septic shock?** *J Crit Care* 2011, **26**(4):435 e439-414.

NEDEN?

Oksijen transportu ve doku perfüzyonu yeterliliği En iyi Santral venöz oksijen saturasyonu (ScvO2) Serum laktat klerensi ile değerlendirilir 1

1. Jones AE, Shapiro NI, Trzeciak S, Arnold RC, Claremont HA, Kline JA: **Lactate clearance vs central venous oxygen saturation as goals of early sepsis therapy: a randomized clinical trial**. *JAMA* 2010, **303**(8):739-746.

NEDEN?

Resussitasyon sıvıları

İntravasküler volüm defisitini, ya da akut hipovolemiyi düzeltmek için kullanılan sıvıdır. Son 3 dekat kolloid kristalloid tartışması yapılmıştır . Son klinik çalışmalarda kolloidlerin sınırlı rolü gösterildi , daha yakın zamanda dengeli solüsyonların normal saline üstünlüğü gösterildi

Maintenans sıvıları

Günlük bazal su ve elektrolit ihtiyaçlarının karşılanması

Replasman sıvıları

Drenler , stoma, fistüller, ateş, poliüri, açık yara gibi durumlarda, oral alım ile kompanse edilemeyen sıvının intravenöz olarak verilmesidir

NEDEN?

Fluid Balance.

Günlük sıvı balansı, birgünde total alınan ve total çıkarılan sıvılar arasındaki farktır. Kümülatif sıvı balansı belli bir periyodda total alınan ve total çıkarılan sıvılar arasındaki farktır

Fluid overload.

Sıvı akümülasyon yüzdesi, kümülatif sıvı volümü (L) / Vücut ağırlığı x 100

Sıvı akümülasyonu %10 üzerinde ise : sıvı yüklenmesi

Fluid bolus.

Hipotansif ve hipovolemik şokta , 4 ml /kg , 10-15 dk da (hızlı) sıvı infüzyonudur.

Fluid challenge.

100-200 ml sıvının 5-10 dk da verilmesi , doku perfüzyonunun iyileştirilmesi için hemodinamik parametrelerin tekrar değerlendirilmesi

CVP de 2-5 kuralı , PCWP de 3-7 kuralı , her bolustan sonra uygulanır.

NEDEN?

Table 1. The 2–5 rule using dynamic changes in CVP (ΔCVP) to guide a fluid challenge

1. Measure baseline CVP (mm Hg):

- CVP < 8: give 4 mL kg^{-1} bolus over 10 minutes
- CVP 8—12: give 2 mL kg^{-1} bolus over 10 minutes
- CVP > 12: give 1 mL kg^{-1} bolus over 10 minutes

2. Re-assess increase in CVP at the end of the bolus (i.e. after 10 minutes from start at point 1)

- ΔCVP > 5: STOP fluid challenge
- ΔCVP < 2: restart with point 1
- ΔCVP 2—5: wait for another 10 minutes and move to point 3

3. Re-assess increase in CVP after another 10 minutes (i.e. after 20 minutes from start at point 1)

- ΔCVP > 2: STOP fluid challenge
- ΔCVP < 2: restart with 1

4. Repeat until CVP of 14 mm Hg or rule broken

NE ZAMAN ?

van Haren *Critical Care* 2017, **21**(Suppl 3):313
DOI 10.1186/s13054-017-1909-5

Critical Care

REVIEW Open Access

Personalised fluid resuscitation in the ICU: still a fluid concept?

Frank van Haren[1,2,3]

Personalised fluid resuscitation requires careful attention to the mnemonic CIT TAIT: context, indication, targets, timing, amount of fluid, infusion strategy, and type of fluid.

C	CONTEX	
I	INDİCATİON	ENDİKASYON
T	TARGETS	HEDEFLER
T	TİMİNG	ZAMANLAMA
A	AMOUNT OF FLUİD	SIVI MİKTARI
I	İNFUSİON STRATEGY	İNFÜZYON STRATEJİSİ
T	TYPE OF FLUİD	SIVI ÇEŞİDİ

NE ZAMAN ?

Septik şok , aynı miktarda sıvı
Erken hedefe yönelik tedavi kolu (EGDT), 6 saat
Standart tedavi kolu , 72 saat

EGDT de sonuçlar daha iyi , Miktar kadar zaman da önemli 1

1. Rivers E, Nguyen B, Havstad S, et al. Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med. 2001;345:1368-1377

NE ZAMAN ?

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Early Goal-Directed Therapy in the Treatment of Severe Sepsis and Septic Shock

Emanuel Rivers, M.D., M.P.H., Bryant Nguyen, M.D., Suzanne Havstad, M.A., Julie Ressler, B.S., Alexandria Muzzin, B.S., Bernhard Knoblich, M.D., Edward Peterson, Ph.D., and Michael Tomlanovich, M.D., for the Early Goal-Directed Therapy Collaborative Group*

N Engl J Med 2001; 345:1368-1377 | November 8, 2001 | DOI: 10.1056/NEJMoa010307

TABLE 4. TREATMENTS ADMINISTERED.*

TREATMENT	HOURS AFTER THE START OF THERAPY		
	0–6	7–72	0–72
Total fluids (ml)			
Standard therapy	3499±2438	10,602±6,216	13,358±7,729
EGDT	4981±2984	8,625±5,162	13,443±6,390
P value	<0.001	0.01	0.73
Red-cell transfusion (%)			
Standard therapy	18.5	32.8	44.5
EGDT	64.1	11.1	68.4
P value	<0.001	<0.001	<0.001
Any vasopressor (%)†			
Standard therapy	30.3	42.9	51.3
EGDT	27.4	29.1	36.8
P value	0.62	0.03	0.02
Inotropic agent (dobutamine) (%)			
Standard therapy	0.8	8.4	9.2
EGDT	13.7	14.5	15.4
P value	<0.001	0.14	0.15
Mechanical ventilation (%)			
Standard therapy	53.8	16.8	70.6
EGDT	53.0	2.6	55.6
P value	[illegible]	<0.[illegible]	[illegible]
Pulmonary-artery catheterization (%)‡			
Standard therapy	3.4	28.6	31.9
EGDT	0	18.0	18.0
P value	0.12	0.04	0.01

TABLE 3. KAPLAN–MEIER ESTIMATES OF MORTALITY AND CAUSES OF IN-HOSPITAL DEATH.*

VARIABLE	STANDARD THERAPY (N=133)	EARLY GOAL-DIRECTED THERAPY (N=130)	RELATIVE RISK (95% CI)	P VALUE
	no. (%)			
In-hospital mortality†				
All patients	59 (46.5)	38 (30.5)	0.58 (0.38–0.87)	0.009
Patients with severe sepsis	19 (30.0)	9 (14.9)	0.46 (0.21–1.03)	0.06
Patients with septic shock	40 (56.8)	29 (42.3)	0.60 (0.36–0.98)	0.04
Patients with sepsis syndrome	44 (45.4)	35 (35.1)	0.66 (0.42–1.04)	0.07
28-Day mortality†	61 (49.2)	40 (33.3)	0.58 (0.39–0.87)	0.01
60-Day mortality†	70 (56.9)	50 (44.3)	0.67 (0.46–0.96)	0.03
Causes of in-hospital death‡				
Sudden cardiovascular collapse	25/119 (21.0)	12/117 (10.3)	—	0.02
Multiorgan failure	26/119 (21.8)	19/117 (16.2)	—	0.27

Sıvı replasmanı erken yapılmalı

[illegible]

‡The denominators indicate the numbers of patients in each group who completed the initial six-hour study period.

Sıvılar

Kolloidler / kristalloidler

Kolloidler; moleküler ağırlıkları büyük, kapiller membrandan daha az geçer, vasküler kompartmanda kalma eğilimindeler, onkotik basıncı oluştururlar , intravasküler volüm resussitasyonu Human albümin & HES, dextran, gelatinler

Kristalloidler: daha ucuz, molekül ağırlığı düşük, memebran geçişi yüksek, interstisyel ödem riski daha fazla , daha büyük volümlerde uygulanması gerekir

HANGİ SIVI ?

Fluid as a Drug: Balancing Resuscitation and Fluid Overload in the Intensive Care Setting

Matthew D. McGuire and Michael Heung

Advances in Chronic Kidney Disease, Vol 23, No 3 (May), 2016: pp 152-159

CLINICAL SUMMARY

- There is no clear evidence to support colloids as first-line resuscitation fluids.
  - Randomized clinical trials, such as Saline versus Albumin Fluid Evaluation and Albumin Italian Outcome Sepsis, have found that resuscitation with albumin is associated with no difference in mortality or acute kidney injury (AKI) compared with crystalloids.
  - Starches, regardless of their molecular weight or substitution ratio, increase the risk of kidney injury when used as a resuscitation fluid.
- Crystalloids should remain the resuscitation fluid of choice.
  - Observational studies have suggested that use of chloride-rich crystalloid solutions are associated with worse outcomes (increased AKI or higher mortality) compared to balanced solutions.
  - The recently published randomized controlled 0.9% Saline vs Plasma-Lyte 148 for Intensive Care Unit Fluid Therapy study found no difference in outcomes when comparing balanced vs chloride-rich solutions for resuscitation, but the predominantly surgical patient population required low volumes of fluid, and these results may not extend to intensive care unit patients who require larger volumes of resuscitative fluids.
- After initial resuscitation, positive fluid balances are associated with higher mortality.
  - Fluid overload, most commonly defined by either positive fluid balance or weight gain exceeding 10% of intensive care unit admission weight, has been variously associated with longer lengths of stay, higher mortalities, and decreased rates of recovery from AKI.

s ubiquitous throughout medicine and is often considered a benign procedure. Yet, there is now
ial harms of fluid overload after initial resuscitation. In recent years, there has also been an
various resuscitation fluids with respect to both benefits and risks. Studies have examined
starches, against the clinical standard of crystalloids. In addition, evidence has emerged to
different between resuscitation with chloride-rich vs balanced crystalloid solutions. In this
iterature regarding choice of intravenous fluids for resuscitation in the intensive care setting
iated with fluid overload in critically ill patients.

ls, Balanced solutions, Resuscitation, Fluid overload

*Kolloidin daha üstün olduğunu gösteren çalışma yoktur
SAFE, AIOS , albümin & kristalloid , mortalite , AKI fark yok
HES, AKI riski var
*Kristalloid birinci seçenek
Klor zengin kristalloid, moratalite ve AKI daha fazla & dengeli solüsyon
(yüksek volüm sıvı resussitasyonları)
*Başlangıç sıvı resussitasyonundan sonra pozitif sıvı balansı mortalite artışı
Sıvı yüklenmesi (Pozitif sıvı dengesi, %10 ağırlık artışı) yatış, AKI, mortalite artışı ile ilişkili

HANGİ SIVI ?

Kristalloidlerden dengeli olanlar mı? Normal salin mi (klor zengin)

Meta analiz , Mortalite üzerine anlamlı fark yok

Klor zengin solüsyonlarda AKI riski anlamlı olarak fazla

(RR 1.64, 95% CI 1.27-2.13).

Krajewski ML, Raghunathan K, Paluszkiewicz SM, et al. Meta-analysis of high- versus low-chloride content in perioperative and critical care fluid resuscitation. Br J Surg. 2015;102:24-36.

*Normal anyon açıklı (hiperkloremik)Metabolik asidoz

*Artmış anyon açıklı Metabolik asidoz

Normal anyon açıklı (hiperkloremik)Metabolik asidoz

- Gastrointestinal sistemden HCO3 kaybı: Diyare
- Enterokütanöz fistül (pankreatit), idrarın üriner diversiyonu (ileal loop mesane), mesane drenajı olan pankreas tx
- Böbrekten HCO3 kaybı:
  - proksimal RTA (tip II),
  - karbonik anhidraz inh
- Böbreğin H sekresyonunda yetersizlik:
  - Distal RTA (tipI),
  - Hiperkalemik RTA (tip IV),
  - Böbrek yetmezliği
- Asid infüzyonu, amonyum klorid, hiperalimentasyon
- Diğer: normal salinle volüm genişletilmesi

Kristaloid sıvıların kompozisyonu

Sıvılar	Tonisite (mOsm/L)	Na^+ (mEq/L)	Cl^+ (mEq/L)	K^+ (mEq/L)	Ca^+ (mEq/L)	Mg^+ (mEq/L)	Glukoz (g/L)	Laktat (mEq/L)	HCO_3 (mEq/L)	Asetat (mEq/L)	Glukonat (mEq/L)
%5 Dekstroz (D_5)	Hipo (253)						50				
Normal Salin %09	İzo (308)	154	154								
D_5 ¼NS	İzo (355)	38.5	38.5				50				
D_5 ½NS	Hiper (432)	77	77				50				
D_5 NS	Hiper (586)	154	154				50				
LaktatlıRinger (LR)	İzo (273)	130	109	4	3			28			
D_5LR	Hiper (525)	130	109	4	3		50	28			
½ NaCl	Hipo (154)	77	77								
%3 Salin	Hiper (1026)	513	513								
İsolyte	İzo (294)	140	98	5		3				27	23

Sıvı Monitorizasyonu

- **OAB (ortalama arter basıncı)**
- **Kapiller dolma zamanı**
- **Ciltte renk değişikliği**
- **İdrar çıkışı**
- **Santral venöz basınç (CVP)**
- **Santral venöz oksijen konsantrasyonu (Scvo2)**
- **Laktat klerensi**
- **Pasif bacak kaldırma**
- **EKO,**
- **VCI kompressibilite**
- **PİCCO (Stroke volüm variation, pulse pressure variation)**

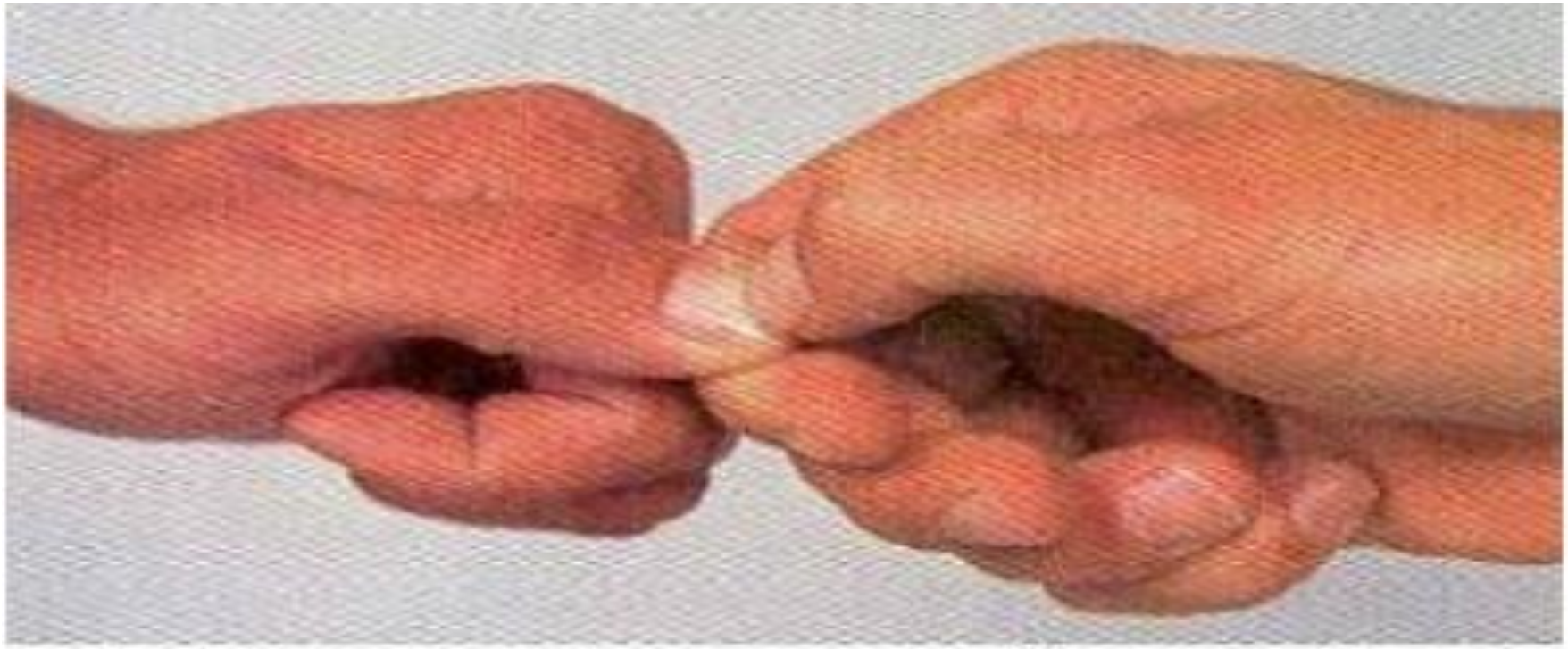
$$OAB: \frac{SKB + 2DK}{3}$$

Dellinger RP, 2016 Surviving Sepsis Guidelines. Critical Care Medicine 2017;45(3):381-385.

Statik ve dinamik hemodinamik parametreler	
Statik parametreler	Santral venöz basınç Pulmoner arter okluzyon basıncı Vena cava inferior çapı VCI kollapsibilitesi End diastolik volüm
Dinamik parametreler	Nabız basıncı değişimi (PPV) Stroke volüm değişimi (SVV) Pletysismografik değişkenlik indeksi
Modifiye Sıvı Challenge	Pasif bacak kaldırma Mini sıvı bolus (100-200 ml)

NE KADAR?

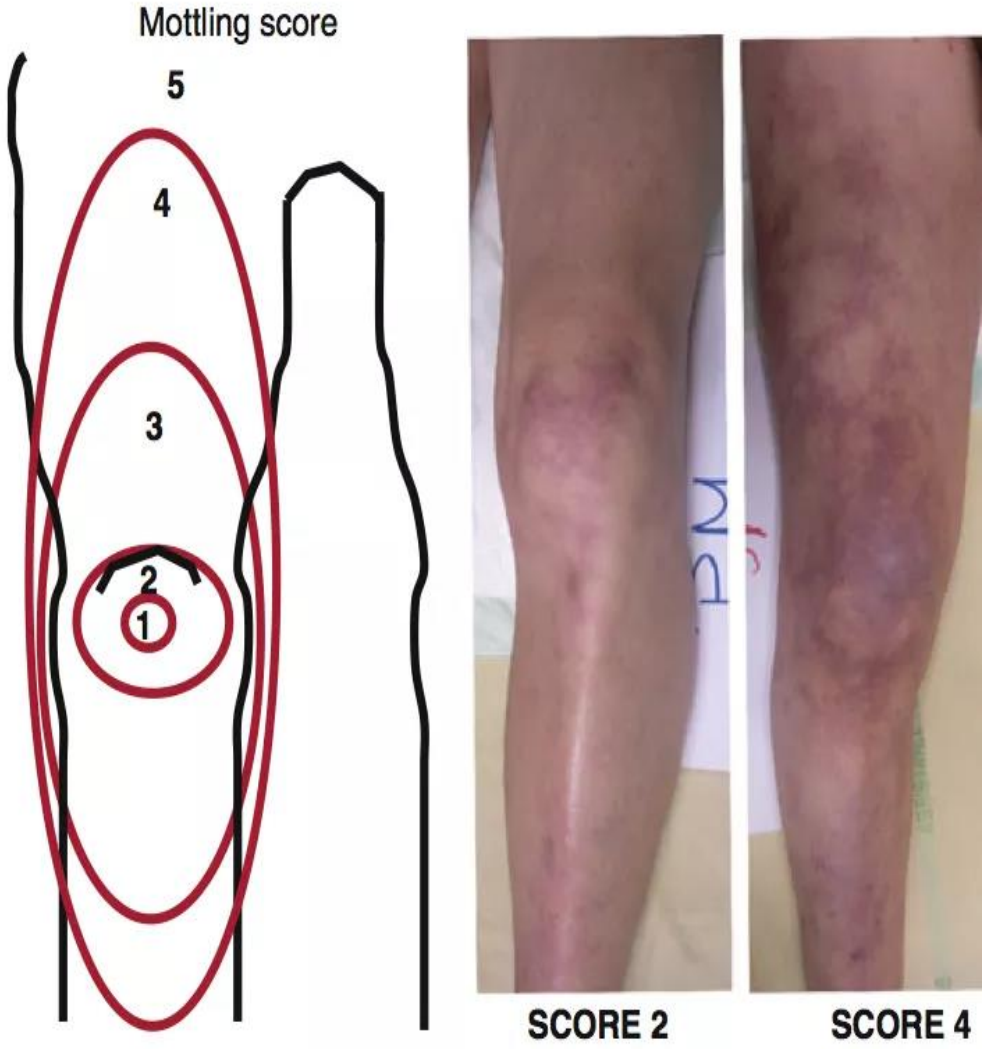
MONİTORİZASYON



KAPİLLER GERİ DOLUMU DEĞERLENDİRME

Dolum Zamanı hastanın yeterli volümü olup olmadığının en iyi göstergelerinden biridir. Hastanın eli kalbinin hizasında tutularak tırnak yatağından bakılır. Normali 2-4 saniyedir. (tırnağa basıldıktan sonra tekrar kanlanması için geçen süre)

Cilt Renk Değişiklikleri



ŞOK VE DOKU HİPOPERFÜZYONUNUN KLİNİK BELİRTİLERİ VE SEMPTOMLARI	
Santral sinir sistemi	Mental durum değişikliği (sersemlik, güçsüzlük, letarji , koma)
Dolaşım	
Kalp	Taşikardi, disritmiler, hipotansiyon, yeni üfürüm
Sistemik	Hipotansiyon, juguler venöz basıncın artması veya azalması
Solunum	Takipne, dispne
Böbrek	Oligüri (< 0.5 ml /kg/saat)
Cilt	Mottle , soğuk, sıcak , livedo retikülaris

NE KADAR?

Santral Venöz Basınç

Sıvı yönetiminde eski bir yöntemdir

Halen kullanılır, santral venöz katetere ihtiyaç vardır.1

Sıvı boluslarına yanıtı değerlendirmede yetersizdir2

23 Araştırma , metaanaliz, infeksiyon ve mekanik komplikasyon riski var

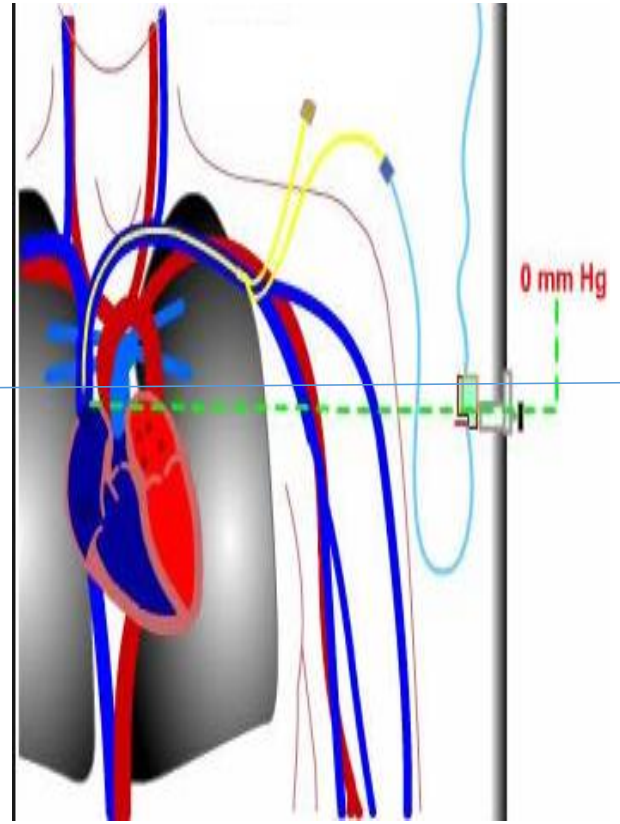
1. Dellinger RP, Levy MM, Rhodes A, et al. Surviving sepsis campaign: international guidelines for management of severe sepsis and septic shock: 2012. Crit Care Med 2013;41:580-637.
2. Osman D, Ridel C, Ray P, et al. Cardiac filling pressures are not appropriate to predict hemodynamic response to volume challenge. Crit Care Med 2007;35:64-8.

NE KADAR?

Yanlış

Doğru

CVP seti



NE KADAR?

- **Laktat klerensi : ilk laktat düzeyi – sonraki laktat düzeyi x 100**
  ilk laktat düzeyi

Laktat klerensinde %10 artma , mortalitede %11 azalmaya yol açar

Crit Care Med. 2004 Aug;32(8):1637-42.

Early lactate clearance is associated with improved outcome in severe sepsis and septic shock.

Nguyen HB[1], Rivers EP, Knoblich BP, Jacobsen G, Muzzin A, Ressler JA, Tomlanovich MC.

Author information

Abstract

OBJECTIVE: Serial lactate concentrations can be used to examine disease severity in the intensive care unit. This study examines the clinical utility of the lactate clearance before intensive care unit admission (during the most proximal period of disease presentation) as an indicator of outcome in severe sepsis and septic shock. We hypothesize that a high lactate clearance in 6 hrs is associated with decreased mortality rate.

DESIGN: Prospective observational study.

SETTING: An urban emergency department and intensive care unit over a 1-yr period.

PATIENTS: A convenience cohort of patients with severe sepsis or septic shock.

INTERVENTIONS: Therapy was initiated in the emergency department and continued in the intensive care unit, including central venous and arterial catheterization, antibiotics, fluid resuscitation, mechanical ventilation, vasopressors, and inotropes when appropriate.

MEASUREMENTS AND MAIN RESULTS: Vital signs, laboratory values, and Acute Physiology and Chronic Health Evaluation (APACHE) II score were obtained at hour 0 (emergency department presentation), hour 6, and over the first 72 hrs of hospitalization. Therapy given in the emergency department and intensive care unit was recorded. Lactate clearance was defined as the percent decrease in lactate from emergency department presentation to hour 6. Logistic regression analysis was performed to determine independent variables associated with mortality. One hundred and eleven patients were enrolled with mean age 64.9 +/- 16.7 yrs, emergency department length of stay 6.3 +/- 3.2 hrs, and overall in-hospital mortality rate 42.3%. Baseline APACHE II score was 20.2 +/- 6.8 and lactate 6.9 +/- 4.6 mmol/L. Survivors compared with nonsurvivors had a lactate clearance of 38.1 +/- 34.6 vs. 12.0 +/- 51.6%, respectively (p =.005). Multivariate logistic regression analysis of statistically significant univariate variables showed lactate clearance to have a significant inverse relationship with mortality (p =.04). There was an approximately 11% decrease likelihood of mortality for each 10% increase in lactate clearance. Patients with a lactate clearance> or =10%, relative to patients with a lactate clearance <10%, had a greater decrease in APACHE II score over the 72-hr study period and a lower 60-day mortality rate (p =.007).

NE KADAR?

J Intensive Care. 2017; 5: 36.

Passive leg raising test with minimally invasive monitoring: the way forward for guiding septic shock resuscitation?

Patrick M. Honore and Herbert D. Spapen

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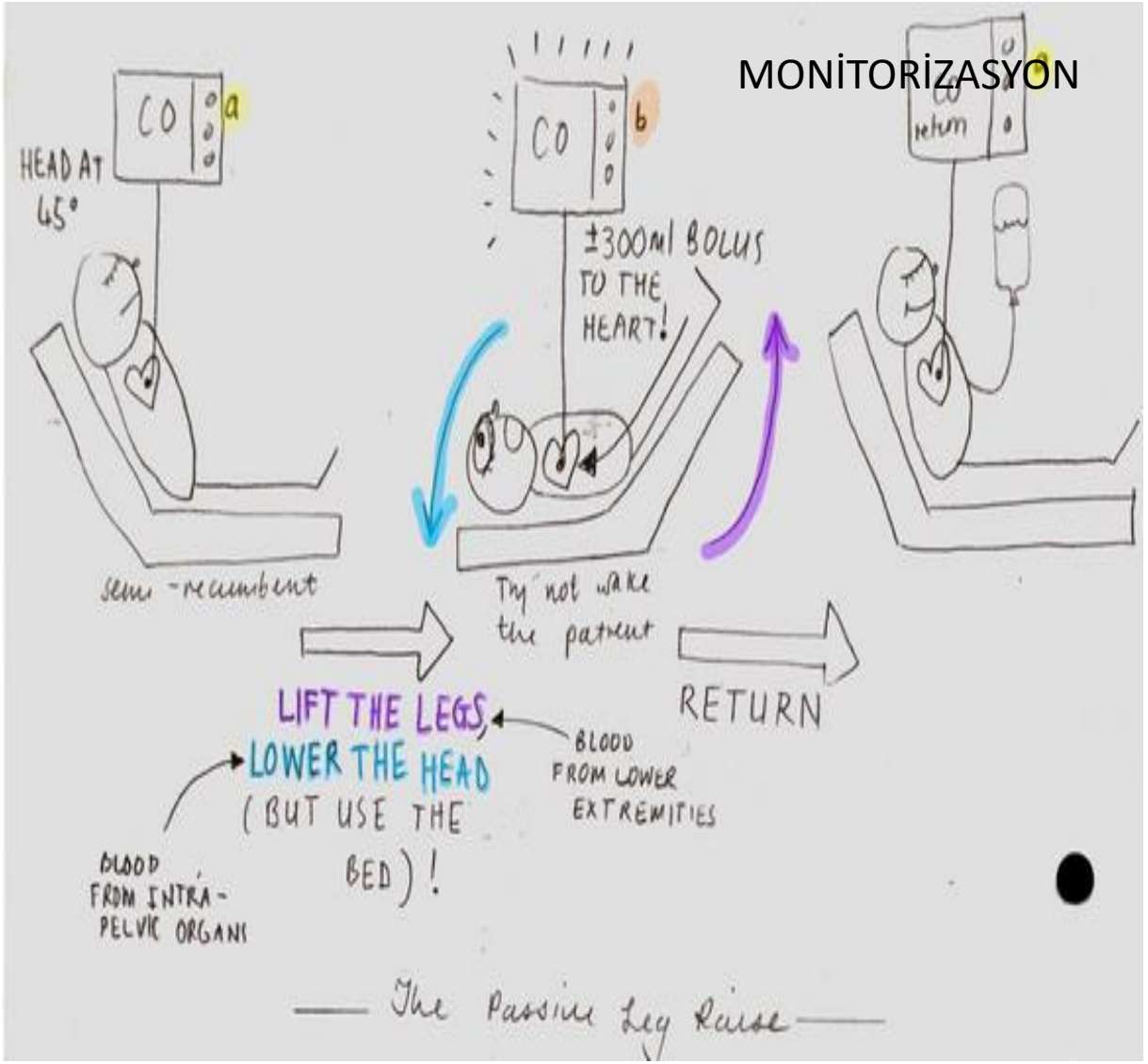
MONİTORİZASYON



Pasif bacak kaldırma , kardiyak autput ölçen metod ile kombine edildiğinde kolay kullanışlı, hızlı, doğru , yatak başı değerlendirme olanağı sağlar.

oto-bolus

Kardiyak aoutput artıyorsa sıvı vermeye devam etmek gerekir

Gereksiz sıvı infüzyonlarının önüne geçer

Spontan soluyan ve disritmisi olan hastalarda da kullanılabilir.

Cavallaro F, Sandroni C, Marano C, et al. Diagnostic accuracy of passive leg raising for prediction of fluid responsiveness in adults: systematic review and meta-analysis of clinical studies. Intensive Care Med 2010;36:1475-83.
Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. Crit Care Med 2006;34:1402-7.
Boulain T, Achard JM, Teboul JL, Richard C, Perrotin D, Ginies G. Changes in BP induced by passive leg raising predict response to fluid loading in critically ill patients. Chest 2002;121:1245-52.

NE KADAR?

Journal of Critical Care 30 (2015) 217.e1–217.e5

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journal homepage: www.jccjournal.org

The reliability and validity of passive leg raise and fluid bolus to assess fluid responsiveness in spontaneously breathing emergency department patients☆,☆☆

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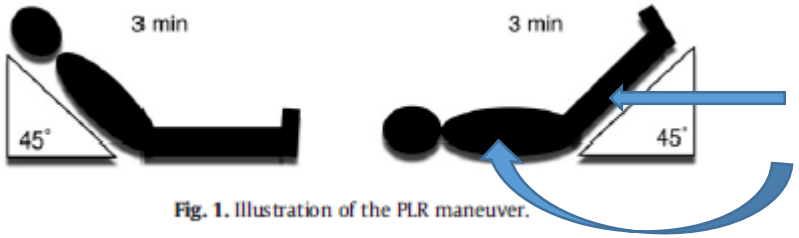


Fig. 1. Illustration of the PLR maneuver.

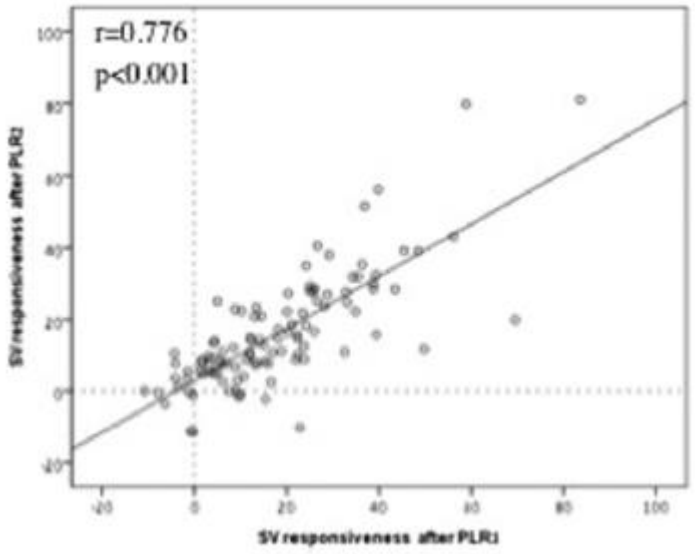


Fig. 3. Correlation between SV responsiveness to PLR1 and PLR2.

A B S T R A C T

Purpose: We investigated the reproducibility of passive leg raise (PLR) and fluid bolus (BOLUS) using the Non-Invasive Cardiac Output Monitor (NICOM; Cheetah Medical, Tel Aviv, Israel) for assessment of fluid responsiveness (FR) in spontaneously breathing emergency department (ED) patients.

Methods: Prospective, observational study of a convenience sample of adult ED patients receiving intravenous fluid bolus. We assessed stroke volume (SV) using NICOM and obtained results from PLR, where the head of the bed was changed from semirecumbent to supine while the patients' legs raised to 45° for 3 minutes. *Fluid bolus* was defined as 5 mL/kg normal saline infusion. Maximal increase in SV was recorded. *Fluid responsiveness* was defined as an increase of SV greater than 10% from baseline. We obtained 4 consecutive responses for each patient; PLR1, PLR2, BOLUS1 separated each by 10 minutes, and BOLUS2 initiated immediately after the end of BOLUS1. We calculated κ statistics, correlation coefficients, and odds ratios with 95% confidence interval and Bland-Altman plots.

Results: We enrolled 109 patients enrolled in this study. The 2 PLRs were significantly correlated ($r = 0.78$, $P < .001$) with $\kappa = 0.46$ for FR ($P < .001$). The 2 BOLUSES less strongly correlated ($r = 0.14$, $P = .001$) and $\kappa = 0.06$ for FR ($P < .001$). Patients who were responsive to PLR1 had 9.5 (3.6-25) odds of being FR for PLR2, whereas those responsive to BOLUS1 had a 1.8 (0.76-4.3) increased odds of FR for BOLUS2.

Conclusion: In conclusion, we have found PLR as measured by the NICOM to be a promising tool for the evaluation of SV responsiveness. It was feasible for use in the ED, and the data suggest that the PLR technique may be more reproducible than the fluid bolus technique for assessing volume responsiveness.

Bacak kompresyon cihazı
Abdominal kompartman sendromu varsa uygulanmaz

NE KADAR?

Sıvıya Yanıtın Trans Torasik Ekokardiyografi (TTE) ile değerlendirilmesi

SVP , PİCCO (PPV,SVV) invaziv kan basıncı monitorizasyonu damar girişimi gerektirir.

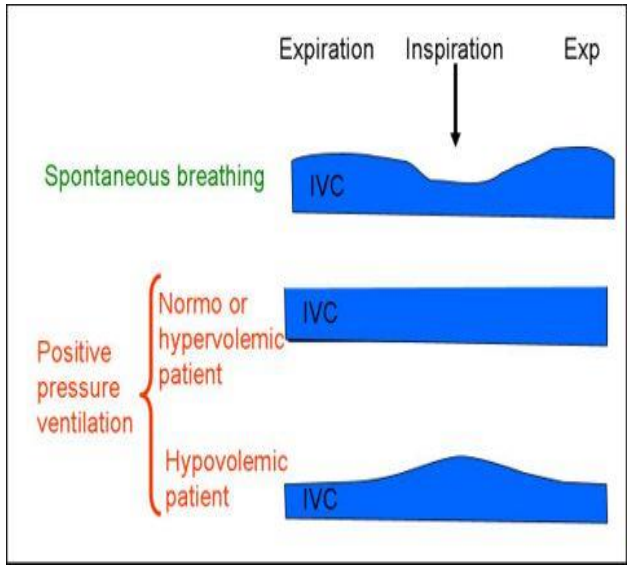
Kanama infeksiyon pnömotoraks tromboz riski vardır

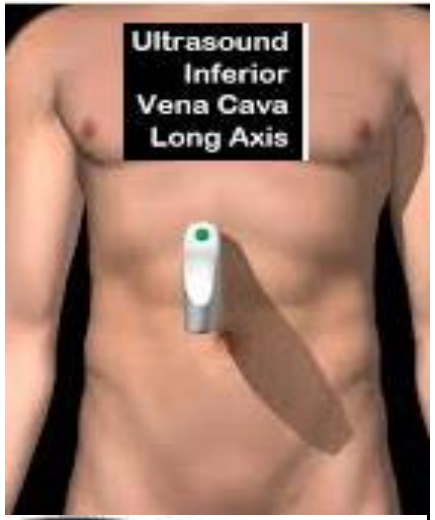
TTE non- invaziv , yatak başı değerlendirme

Vena cava inferior genişliğinin solunum ile değişim oranına göre değerlendirme yapılır.

Kircher BJ, Himelman RB, Schiller NB: **Noninvasive estimation of right atrial pressure from the inspiratory collapse of the inferior vena cava**. *Am J Cardiol* 1990, **66**(4):493-496.

VOLÜM CEVABININ DEĞERLENDİRİLMESİNDE VCI

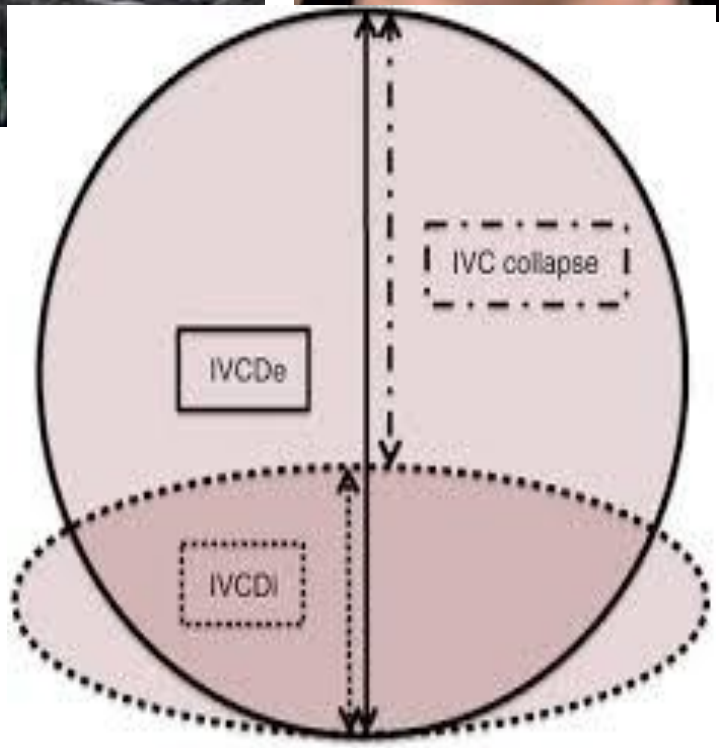




Correlations Between IVC Size and CVP

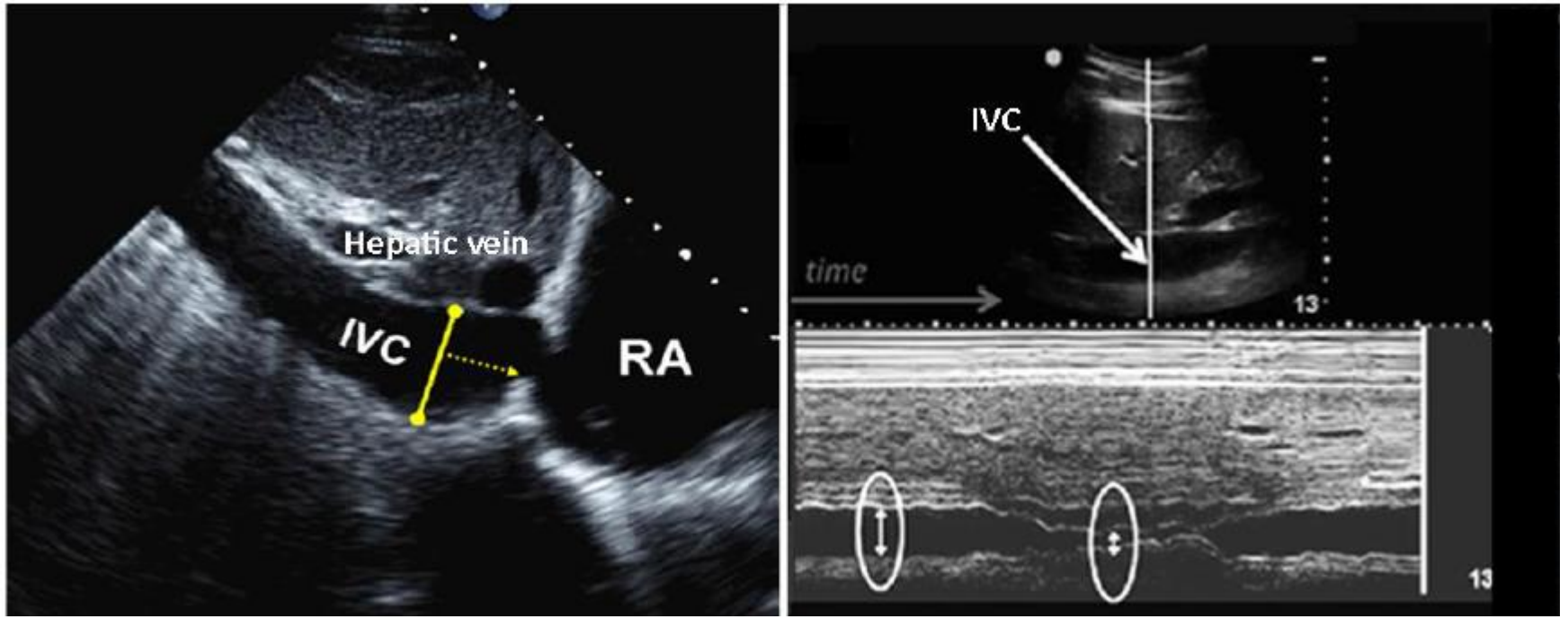
Inferior vena cava size (cm)	Respiratory change	Central venous pressure (cm H_2O)
<1.5	Total collapse	0-5
1.5-2.5	>50% collapse	6-10
1.5-2.5	<50% collapse	11-15
>2.5	<50% collapse	16-20
>2.5	No change	>20

ELSEVIER GLOBAL MEDICAL NEWS



IVC-CI = (IVCDe – IVCDi)/IVCDe

NE KADAR?



Collapsibility index or caval index is calculated as:
[(maximal diameter–minimal diameter)÷maximal diameter]×100%. (Based on Kent et al.[63])

Kent A, Bahner DP, Boulger CT, Eiferman DS, Adkins EJ, Evans DC, Springer AN, Balakrishnan JM, Valiyaveedan S, Galwankar SC *et al*:
Sonographic evaluation of intravascular volume status in the surgical intensive care unit: a prospective comparison of subclavian vein and inferior vena cava collapsibility index. *J Surg Res* 2013, **184**(1):561-566

NE KADAR?

CVP ile VCI çap değişikleri arasında anlamlı korelasyon vardır

Preload reservi hakkında bilgi verir.

Ölçümler hepatik ven girişinin distalinden yapılır

Respiratuar siklusta ölçümler M Mode ile yapılır.

Prekker ME, Scott NL, Hart D, Sprenkle MD, Leatherman JW. Point-of-care ultrasound to estimate central venous pressure: a comparison of three techniques. Crit Care Med 2013;41:833-41.
Nagdev AD, Merchant RC, Tirado-Gonzalez A, Sisson CA, Murphy MC. Emergency department bedside ultrasonographic measurement of the caval index for noninvasive determination of low central venous pressure. Ann Emerg Med 2010; 55:290-5.

NE KADAR?

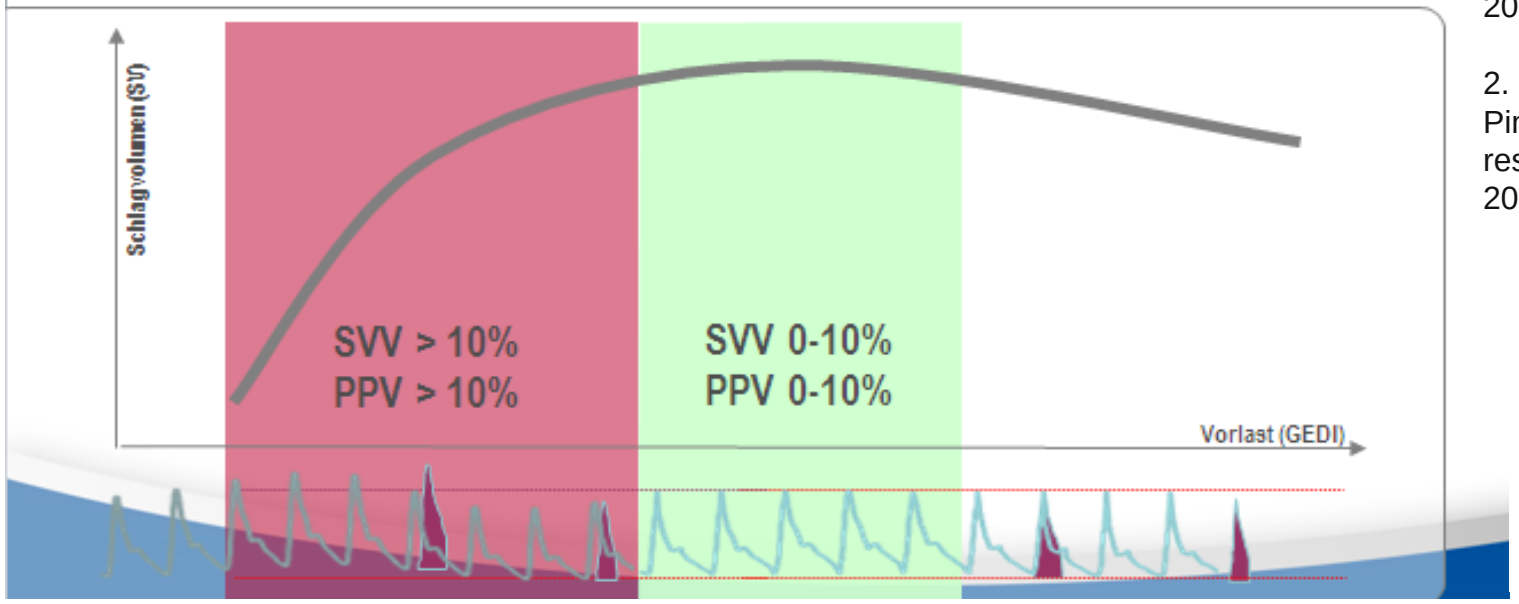
MONİTORİZASYON

SVV – Kan atım miktari değişimi

PPV – Nabız basinç değişimi

Düzenli kalp atışı olan (aritmisi olmayan), mekanik ventilasyonlu hastalarda SVV ve PPV'nin (>10%) 'dan büyük olması hacim yüklenmesinin kardiyak ejeksiyonda bir artışa yol açacağı anlamına gelir.

- SVV/PPV değeri >10% ise volüm yüklemesi faydalı olabilir.



Sıvı yanıtını değerlendirmede stroke volüm varyansı (SVV)
ve pulse pressure varyans (PPV) dinamik göstergeler kıymetlidir.1
Aritmi , spontan solunum varlığında endikasyon yok 1
Pasif bacak kaldırma , ekokardiyografi , yatak başı uygulanabilir alternatif izlem parametreleri 2

1 Michard F, Biais M. Rational fluid management: dissecting facts from fiction. Br J Anaesth. 2012;108(3):369–71.

2. Monnet X, Rienzo M, Osman D, Anguel N, Richard C, Pinsky MR, Teboul JL. Passive leg raising predicts fluid responsiveness in the critically ill. Crit Care Med. 2006;34(5):1402–7.

Table 2. Techniques and monitors for evaluating fluid responsiveness

Parameter	Monitor
Passive leg raise	Echocardiography, CardioQ (esophageal Doppler), NICOM, PiCCO, Vigileo FloTrac
Pulse pressure variation	LiDCO, Clearsight (Nexfin), PRAM
Stroke volume variation	LiDCO, PiCCO, Pulsioflex, PRAM, Vigileo FloTrac, VolumeView
Pleth variability index	Masimo Radical7

NE KADAR?

Sıvı yanıtının izlenmesinde metodların PPV NPV

Table 3. Positive and Negative Predictive Values of Dynamic Changes and Fluid Responsiveness

Source	No.	Setting	Breathing	Parameters Tested	Measure of response	Best Threshold value	PPV,%	NPV,%
Magder[32]	33	Mixed medical and surgical ICU	SB and MV	CVP dynamic changes	CI	≥1 mm Hg drop in CVP	84	93
Westphal[33]	30	Post-cardiac surgery	MV	VCCI	PPV>13%	VCCI>5%	94	97
Lapsa[34]	14	Septic shock	SB	VCCI	CI	VCCI≥15%	62	100
				SVV	CI	SVV≥17%	100	82
Michard[29]	40	Septic shock	MV	PPV	CI	PPV>13%	94	96
Nagdar[41]	73	Shock	SB, MV	Caval Index	CVP<8 mm Hg	Caval Index≥50%	87	96
Barbier[42]	20	Septic shock	MV	dIVC	CI≥15%	dIVC>18%	90	90
Feissel[43]	39	Septic shock	MV	dIVC	CI≥15%	dIVC>12%	93	92
Thiel[45]	89	Shock	SB, MV	dSV in response to PLR	SV	SV≥15%	91	85

SB, spontaneous breathing; MV, mechanical ventilation; CVP, central venous pressure; CI, cardiac index; VCCI, vena cava "pressure" collapsibility index; SVV, stroke volume variation; PPV, pulse pressure variation; caval index, expiratory IVC diameter – inspiratory IVC diameter ÷ expiratory IVC diameter on echocardiography multiplied by 100; dIVC, change in IVC diameter on echocardiography; dSV, change in stroke volume on echocardiography; PLR, passive leg raising fluid volume challenge

NE KADAR?

Servis odası veya yoğun bakım için volüm yanıtını değerlendirmede yöntem;

Non ınvaziv
Devamlı
Doğru
Ucuz
USG bazlı

FAZLA OLSA NE OLUR?

Yüksek volüm verilmesiyle
Endotelial hasar
Sıvı ekstravazasyonu
Doku ödemi
İnterstisiyel sıvı artışı
Akciğerde Ekstravasküler sıvı artışı
Progressif organ disfonksiyonu
ÖLÜM

1.Marik PE. Iatrogenic salt water drowning and the hazards of a high central venous pressure. Ann Intensive Care 2014;4:21.
2.Wang CH, Hsieh WH, Chou HC, et al. Liberal versus restricted fluid resuscitation strategies in trauma patients: a systematic review and meta-analysis of randomized controlled trials and observational studies. Crit Care Med 2014;42:954-61.

FAZLA OLSA NE OLUR?

VIA MEDICA

REVIEWS

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Fluid overload, de-resuscitation, and outcomes in critically ill or injured patients: a systematic review with suggestions for clinical practice

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[2]*Department of Internal Medicine, Pulmonary and Critical Care Medicine, Eastern Virginia Medical School, Norfolk, VA, USA*
[3]*Regional Trauma Services, Foothills Medical Centre, Calgary, Alberta, Canada*
[4]*Departments of Surgery, Critical Care Medicine, and Community Health Sciences,*

Yüksek Volumde sıvı verilmesi doku ödemi , klinik olarak sıvı yüklenmesi bulgularına yol açar.

Doku ödemi oksijen ve metabolit difüzyonunu bozar, doku yapısını bozar, kapiller kan akımı ve lenfatik drenaj bozulur, hücre - hücre bağlantıları bozulur.

Bu etkiler karaciğer ve böbrek gibi kapsüllü organlarda daha belirgindir, organ kan akımı bozulur .

Yüksek volüm resussitasyonunda Intra abdominal basınç artar, bu durum böbrek ve karaciğer perfüzyonunu daha da bozar

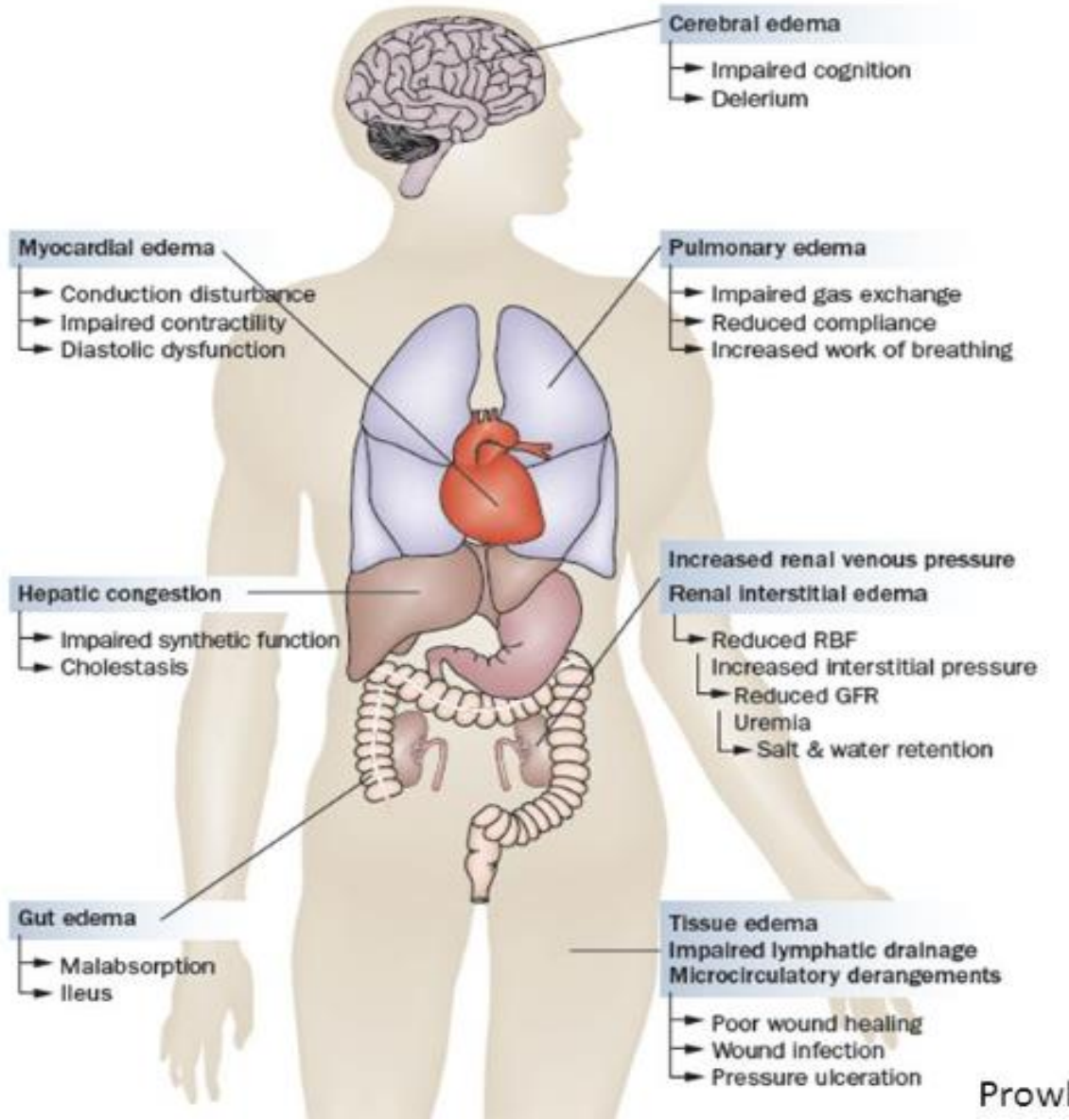
Marik PE: Iatrogenic salt water drowning and the hazards of a high central venous pressure. Ann Intensive Care 2014; 4: 21.
Kirkpatrick AW, Balogh Z, Ball CG et al.: The secondary abdominal compartment syndrome: iatrogenic or unavoidable? J Am Coll Surg 2006; 202: 668–679.

FAZLA OLSA NE OLUR?

Septik hastalarda sıvı yüklenmesinin
kötü sonuçlarına ilişkin veriler artmaktadır (1-4)

ARDS Network Fluids Treatment Trial
1000 hst, konservatif ve liberal sıvı stratejileri, 7 gün izlem
konservatif grupta
akciğer fonksiyonları daha iyi ,
mekanik ventilasyon daha kısa ,
non pulmoner organ yetmezliği daha az (5)

1. Sadaka F, Juarez M Fluid resuscitation in septic shock: the effect of increasing fluid balance on mortality. J Intensive Care Med. 2014;29(4):213–7.
2. Smith SH, Perner A. Higher vs. lower fluid volume for septic shock: clinical characteristics and outcome in unselected patients in a prospective, multicenter cohort. Crit Care. 2012;16(3):R76.
3. Boyd JH, Forbes J, Nakada TA, Walley KR, Russell JA. Fluid resuscitation in septic shock: a positive fluid balance and elevated central venous pressure are associated with increased mortality. Crit Care Med. 2011;39(2):259–65.
4. Samoni S, Vigo V, Resendiz LI, Impact of hyperhydration on the mortality risk in critically ill patients admitted in intensive care units: comparison between bioelectrical impedance vector analysis and cumulative fluid balance recording. Crit Care. 2016;20:95.
5. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network, Wiedemann HP, Wheeler AP, Bernard GR, Thompson BT, Hayden D, DeBoisblanc B, Connors Jr AF, Hite RD, et al. Comparison of two fluid-management strategies in acute lung injury. N Engl J Med. 2006;354(24):2564–75.



Fluid overload has many potentially deleterious effects

Prowle et al, Nat Rev Neph 2010

1. Prowle JR, Echeverri JE, Ligabo EV, et al. Fluid balance and acute kidney injury. Nat Rev Nephrol. 2010;6:107-115.
2. Heung M, Wolfgram DF, Kommareddi M, et al. Fluid overload at initiation of renal replacement therapy is associated with lack of renal recovery in patients with acute kidney injury. Nephrol Dial Transplant. 2012;27:956-961.
3. Bouchard J, Soroko SB, Chertow GM, et al. Fluid accumulation, survival and recovery of kidney function in critically ill patients with acute kidney injury. Kidney Int. 2009;76:422-427.

Öncelikle sıvı yanıtı olmayan hastalarda sıvı yüklenmesinin riskleri konusunda klinisyenleri eğitmek gerekir.

Hemodinaminin izlenerek konservatif sıvı verilmesi morbidite yi azaltır, sonlanmaları iyileştirir 1,2
Fazla Sıvı infüzyonunun immun sistem, endotel fonksiyonu , glycocalyx integrty üzerine olumsuz etkisi var .

Septik vasküler endotel hücre hasarında , vasküler luminal hücre membranlarında glycocalyx degradasyonu gösterilmiştir.3.

Özellikle hipervolemi ile sonuçlanan hızlı sıvı infüzyonlarında glcocalyx hasarı potansiyalize olmaktadır 4-6

1. Marik P, Bellomo R. A rational approach to fluid therapy in sepsis. Br J Anaesth. 2016;116(3):339–49.
2. Chen C, Kollef MH. Targeted fluid minimization following initial resuscitation in septic shock: a pilot study. Chest. 2015;148(6):1462–9.
3. Henrich M, Gruss M, Weigand MA. Sepsis-induced degradation of endothelial glycocalix. ScientificWorldJournal. 2010;10:917–23.
4. Berg S, Golster M, Lisander B. Albumin extravasation and tissue washout of hyaluronan after plasma volume expansion with crystalloid or hypooncotic colloid solutions. Acta Anaesthesiol Scand. 2002;46(2):166–72.
5. Berg S, Engman A, Hesselvik JF, Laurent TC. Crystalloid infusion increases plasma hyaluronan. Crit Care Med. 1994;22(10):1563–7.
6. Chappell D, Bruegger D, Potzel J, Jacob M, Brettner F, Vogeser M, Conzen P, Becker BF, Rehm M. Hypervolemia increases release of atrial natriuretic peptide and shedding of the endothelial glycocalyx. Crit Care. 2014;18(5):538.

CEEM Clinical and Experimental Emergency Medicine

Clin Exp Emerg Med 2014;1(2):67-77
http://dx.doi.org/10.15441/ceem.14.040

Assessing volume status and fluid responsiveness in the emergency department

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[2]Department of Emergency Medicine, Massachusetts General Hospital, Boston, MA, USA

Resuscitation with intravenous fluid can restore intravascular volume and improve stroke volume. However, in unstable patients, approximately 50% of fluid boluses fail to improve cardiac output as intended. Increasing evidence suggests that excess fluid may worsen patient outcomes. Clinical examination and vital signs are unreliable predictors of the response to a fluid challenge. We review the importance of fluid management in the critically ill, methods of evaluating volume status, and tools to predict fluid responsiveness.

Keywords Hemodynamics; Ultrasonography; Shock

Review Article

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04102, USA
E-mail: DMackenzie@mmc.org

Stabil olmayan hastalarda, bolus sıvı uygulamalarının % 50 sinde

kardiyak aoutput artışı olmaz

Septik şokta, Az sıvı resussitasyonu / sıvı vermeksizin supportif tedavi (eksperimental çalışma)

Bolus sıvı tedavisine alternatif olarak erken vasoaktif kullanımının araştırılması , prospektif randomize çalışma

Hipertanik sıvı kullanarak küçük volümlü sıvı resussitasyonu kullanılması

1. Hamzaoui O, Georger JF, Early administration of norepinephrine increases cardiac preload and cardiac output in septic patients with life-threatening hypotension. Crit Care. 2010;14(4):R142.
2. van Haren HMP. The use of hypertonic solutions in sepsis. Trends Anaesthesia Crit Care. 2013;3(1):37–41.

Bütün sıvı resusitasyonları zararlıdır anlamı çıkmaz

İntravenöz sıvı verilmesi ve izlenmesi ilaç gibi olmalıdır.

Potansiyel faydalar ve zararlar ile birliktedir.