KARDIYOJENIK ŞOK: TANıMLAR VE SıNıFLANDıRMALAR

DR. TOLGA SINAN GÜVENÇ

DR. SIYAMI ERSEK GKDC EAH

KARDIYOLOJI KLINIĞI

ÇıKAR ÇATıŞMASı BEYANı

- BU SUNUM ILE ILGILI HERHANGI BIR ÇıKAR ÇATıŞMASı BEYANıM YOKTUR.

‘ŞOK’

- O2’NIN YETERSIZ TAŞıNMASı...
- O2 IHTIYACıNıN AŞıRı DERECEDE ARTMASı...
- O2’NIN HÜCRE DÜZEYINDE KULLANıLMASıNDAKI BOZUKLUK...

... SONUCUNDA MEYDANA GELEN, HÜCRESEL VE DOKU DÜZEYINDE HIPOKSI ILE KARAKTERIZE KLINIK SENDROM[1,2].

1 BARBER AE, SHIRES GT. CELL DAMAGE AFTER SHOCK.NEW HORIZ. 1996 MAY;4(2):161-7.

2 ANGUS DC, VAN DER POLL T. SEVERE SEPSIS AND SEPTIC SHOCK. N ENGL J MED. 2013 AUG 29;369(9):840-51. DOI: 10.1056/NEJMRA1208623.

‘KARDIYOJENIK’ ‘ŞOK’

- İNTRAKARDIYAK SEBEPLER SONUCU KALBIN POMPA FONKSIYONUNDA BOZULMA SONUCU OLUŞAN,
- O2’NIN HÜCRE DÜZEYINDE KULLANıLMASıNDAKI BOZUKLUK…

… SONUCUNDA MEYDANA GELEN, HÜCRESEL VE DOKU DÜZEYINDE HIPOKSI ILE KARAKTERIZE KLINIK SENDROM.

! TANıM BU OLMAKLA BERABER, BAZı OBSTRÜKTIF VEYA PRELOAD AZALMASı SONUCU GELIŞEN ŞOK SEBEPLERI DE GENELLIKLE KARDIYOJENIK ŞOK SıNıFLAMASı IÇINE DAHIL EDILIR.

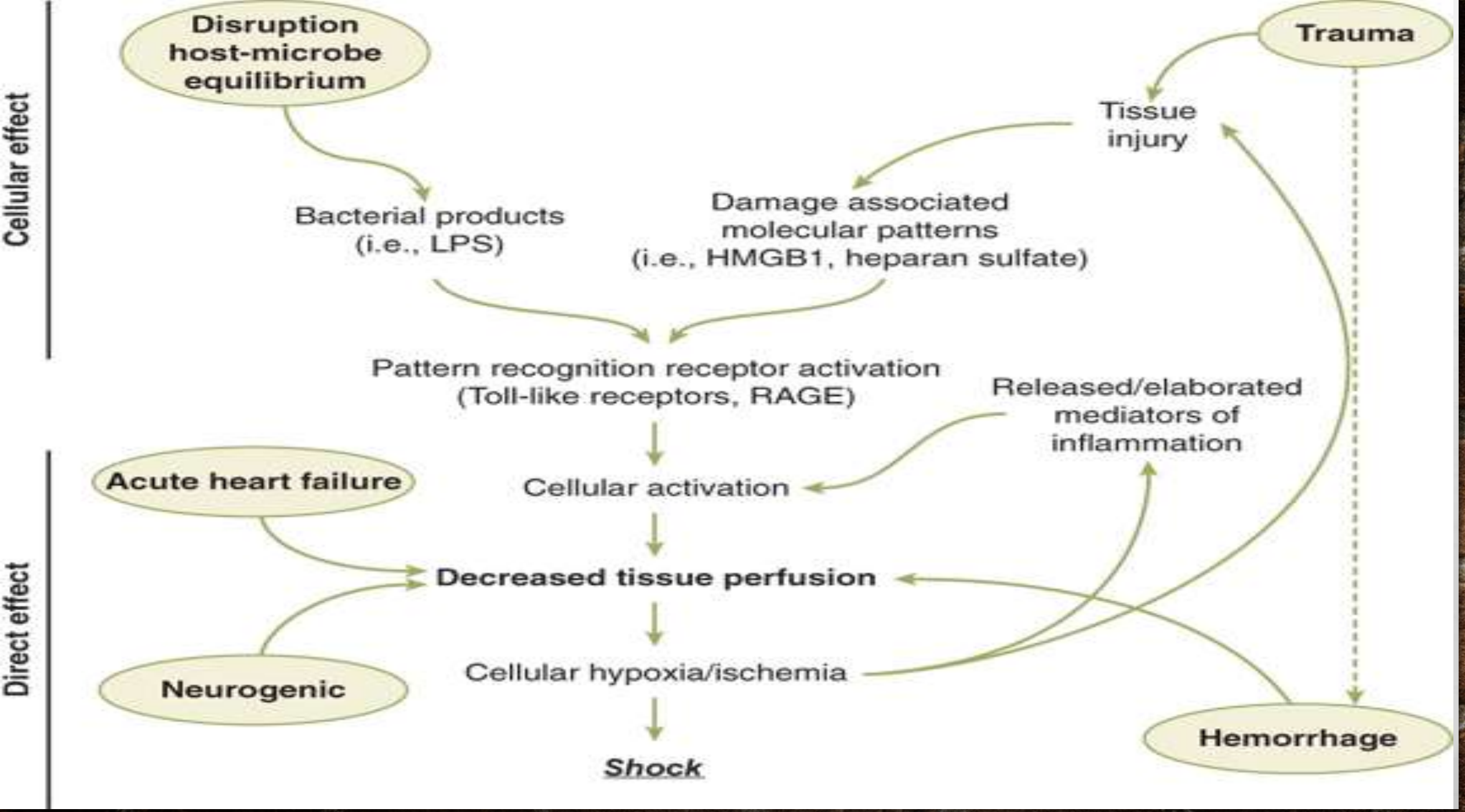
PATOFIZYOLOJI

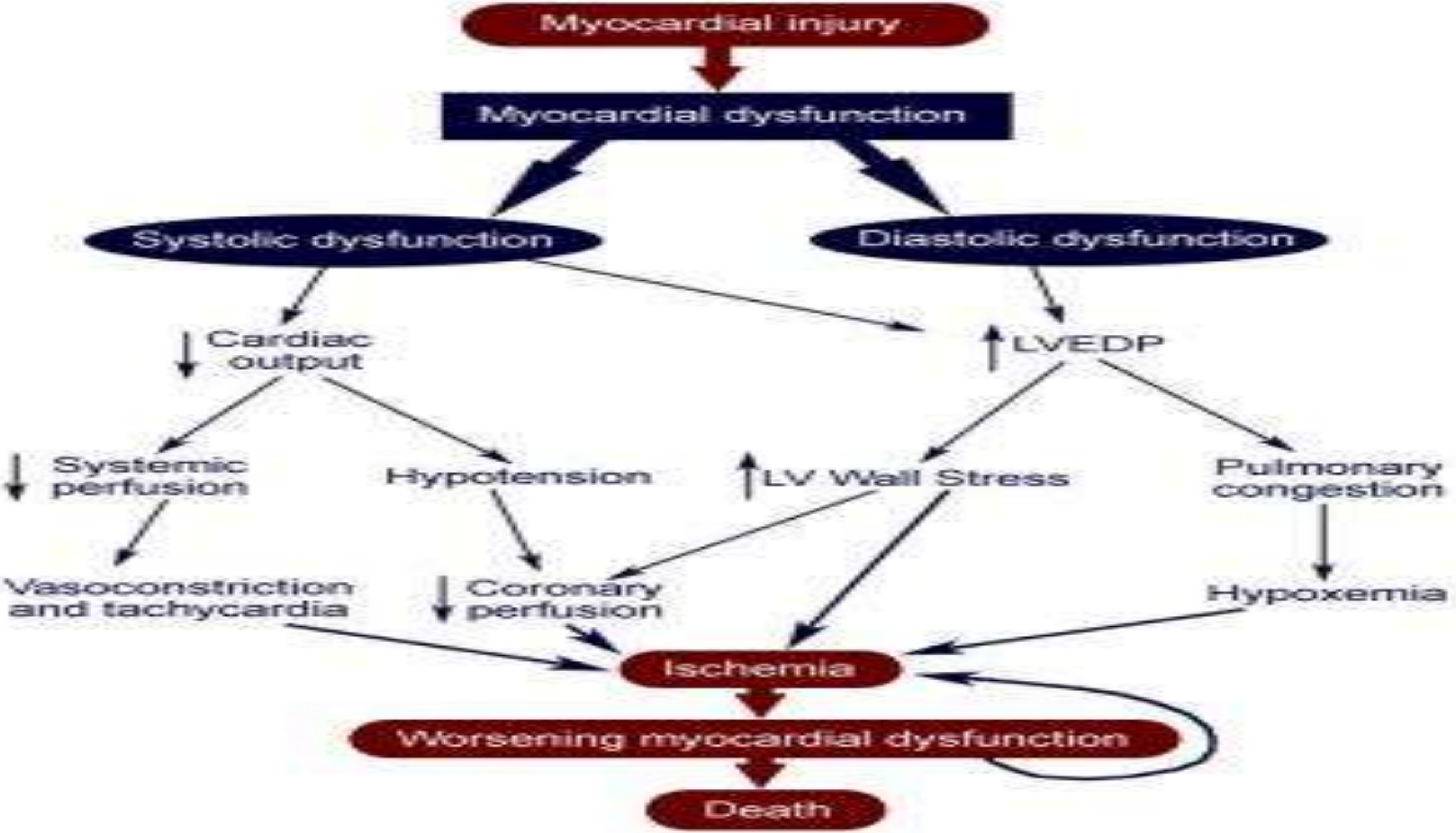
- HÜCRESEL HIPOKSI
- HIPOKSIYE SEKONDER IYON KANALLARıNDA DISFONKSIYON, HÜCRE ÖDEMI, HÜCRESEL ENERJI ÜRETIMINDE BOZULMA, MEMBRAN DISRUPSIYONU VE INTRASELÜLER IÇERIĞIN EKSTRASELÜLER ALANA GEÇIŞI, HÜCRE IÇI ASIDOZ
- SISTEMIK ASIDOZ, ENDOTEL DISFONKSIYONU, INFLAMASYON, KOMPANSATUAR MEKANIZMALARıN AKTIVASYONU
- DOLAŞıMDA DAHA FAZLA BOZULMA, DAHA FAZLA HÜCRESEL HIPOKSI...

.... BUNA EK OLARAK KARDIYOJENIK ŞOKTA, ŞOKA SEKONDER OLARAK KORONER PERFÜZYONDA BOZULMA ILE DAHA FAZLA MIYOKARDIYAL ISKEMI VE DEPRESYON GÖRÜLÜR[1,2].

1) HOLLENBERG SM, KAVINSKY CJ, PARRILLO JE. CARDIOGENIC SHOCK. ANN INTERN MED 1999; 131:47.

2) CALIFF RM, BENGTSON JR. CARDIOGENIC SHOCK. N ENGL J MED 1994; 330:1724.


Cellular effect
Direct effect
Disruption host-microbe equilibrium
Trauma
Tissue injury
Bacterial products (i.e., LPS)
Damage associated molecular patterns (i.e., HMGB1, heparan sulfate)
Pattern recognition receptor activation (Toll-like receptors, RAGE)
Released/elaborated mediators of inflammation
Acute heart failure
Cellular activation
Decreased tissue perfusion
Neurogenic
Cellular hypoxia/ischemia
Hemorrhage
Shock


Myocardial injury
Myocardial dysfunction
Systolic dysfunction
Diastolic dysfunction
Cardiac output
LVEDP
Systemic perfusion
Hypotension
LV Wall Stress
Pulmonary congestion
Vasoconstriction and tachycardia
Coronary perfusion
Hypoxemia
Ischemia
Worsening myocardial dysfunction
Death

KARDIYOJENIK ŞOK ETIYOLOJISI

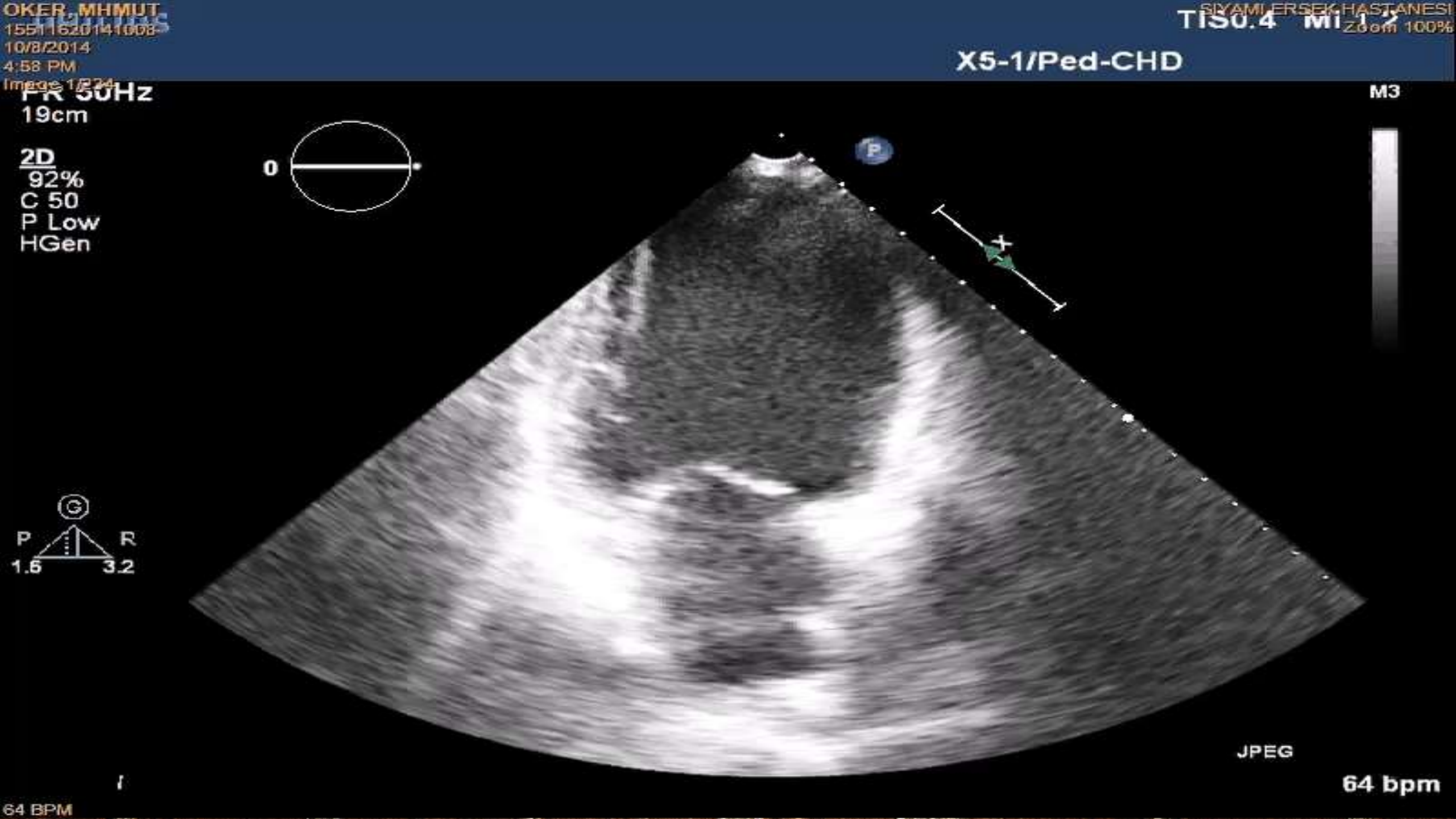
- KARDIYOMYOPATIK: MIYOKARD KONTRAKTILITESINDE, HERHANGI BIR NEDENDEN DOLAYı AZALMAYA IKINCIL GELIŞEN ŞOK – MYOKARD INFARKTı.
- ARITMIK: VENTRIKÜLER YA DA SUPRAVENTRIKÜLER ARITMILERE BAĞLı OLARAK KARDIYAK KONTRAKTILITEDE AZALMA VE BUNA BAĞLı GELIŞEN POMPA DISFONKSIYONU – VENTRIKÜLER TAŞIKARDILER.
- MEKANIK: INTRAKARDIYAK YAPıLARDA MEYDANA GELEN BOZULMAYA BAĞLı OLARAK GELIŞEN KARDIYOJENIK ŞOK – AKUT MITRAL YETERSIZLIĞI.

Cardiogenic	**Cardiomyopathic**	▪ Myocardial infarction (involving >40% of the left ventricle or with extensive ischemia) ▪ Severe right ventricle infarction ▪ Acute exacerbation of severe heart failure from dilated cardiomyopathy ▪ Stunned myocardium from prolonged ischemia (eg, cardiac arrest, hypotension, cardiopulmonary bypass) ▪ Advanced septic shock ▪ Myocarditis ▪ Myocardial contusion ▪ Drug-induced (eg, beta blockers)
	Arrhythmogenic	▪ Tachyarrhythmia – Atrial tachycardias (fibrillation, flutter, reentrant tachycardia), ventricular tachycardia and fibrillation ▪ Bradyarrhythmia – Complete heart block, Mobitz type II second degree heart block
	Mechanical	▪ Severe valvular insufficiency, acute valvular rupture (papillary or chordae tendineae rupture, valvular abscess), critical valvular stenosis, acute or severe ventricular septal wall defect, ruptured ventricular wall aneurysm, atrial myxoma

NONKARDIYAK KARDIYOJENIK ŞOK ETIYOLOJISI

- OBSTRÜKTIF ŞOK: SOL VEYA SAĞ VENTRIKÜL ÇıKıŞ YOLUNDAKI EXTRAKARDIYAK YAPıLARDA MEYDANA GELEN ANI DIRENÇ ARTıŞıNA SEKONDER OLARAK KARDIYAK OUTPUTTAKI ANI DÜŞÜŞ – AKUT PULMONER EMBOLI.
- MEKANIK ŞOK: SOL VEYA SAĞ VENTRIKÜLÜN DOLUŞUNUN MEKANIK SEBEPLERE BAĞLı OLARAK BOZULMASıNA IKINCIL GELIŞEN ŞOK – PERIKARDIYAL TAMPONAD

Obstructive	**Pulmonary vascular**	▪ Hemodynamically significant pulmonary embolus, severe pulmonary hypertension, severe or acute obstruction of the pulmonic or tricuspid valve, venous air embolus
	Mechanical	▪ Tension pneumothorax or hemothorax (eg, trauma, iatrogenic), pericardial tamponade, constrictive pericarditis, restrictive cardiomyopathy, severe dynamic hyperinflation (eg, elevated intrinsic PEEP), left or right ventricular outflow tract obstruction, abdominal compartment syndrome, aorto-caval compression (eg, positioning, surgical retraction)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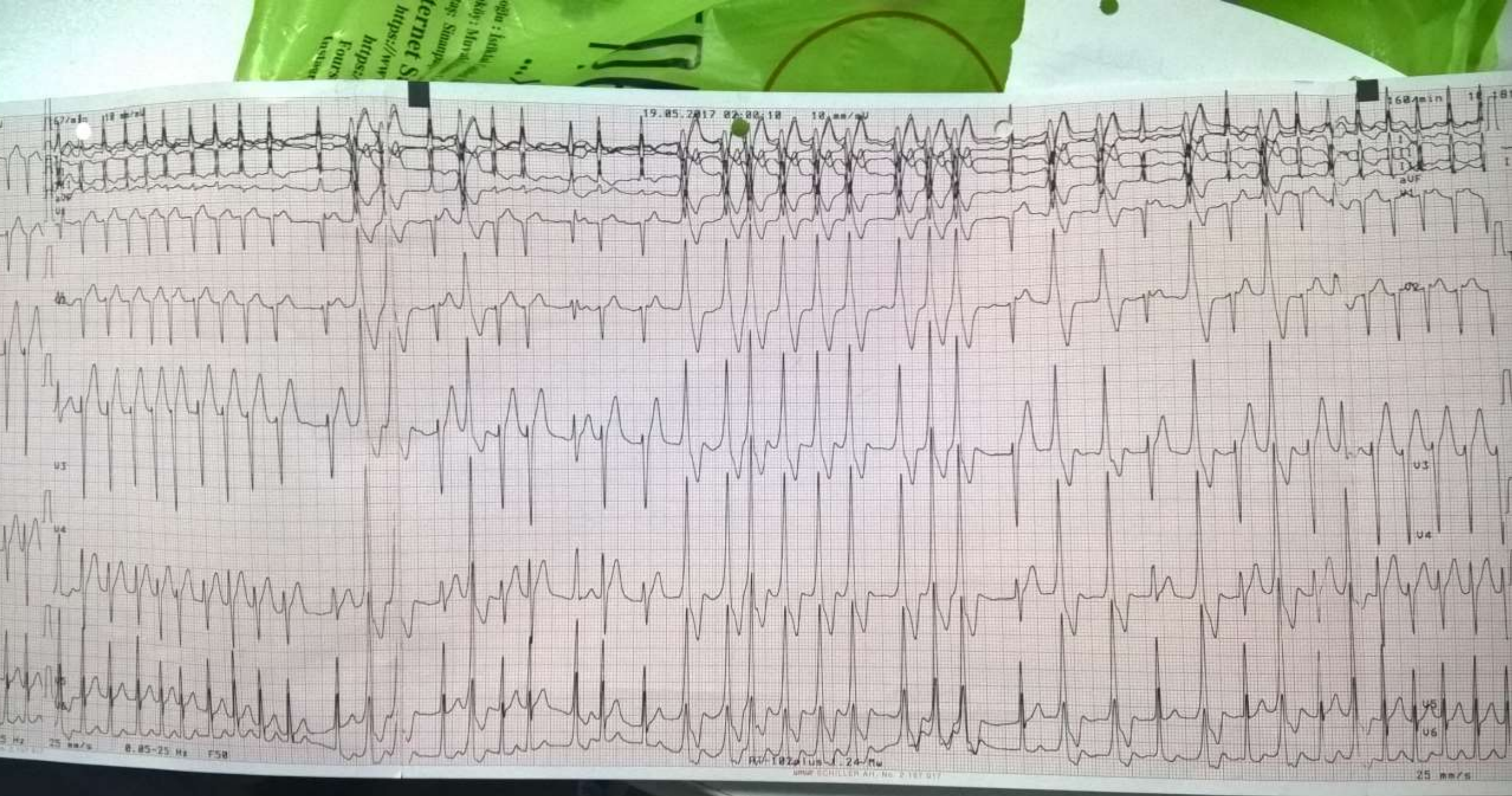
64 BPM


19.05.2017 02:00:10
10 mm/mV
aVF
V2
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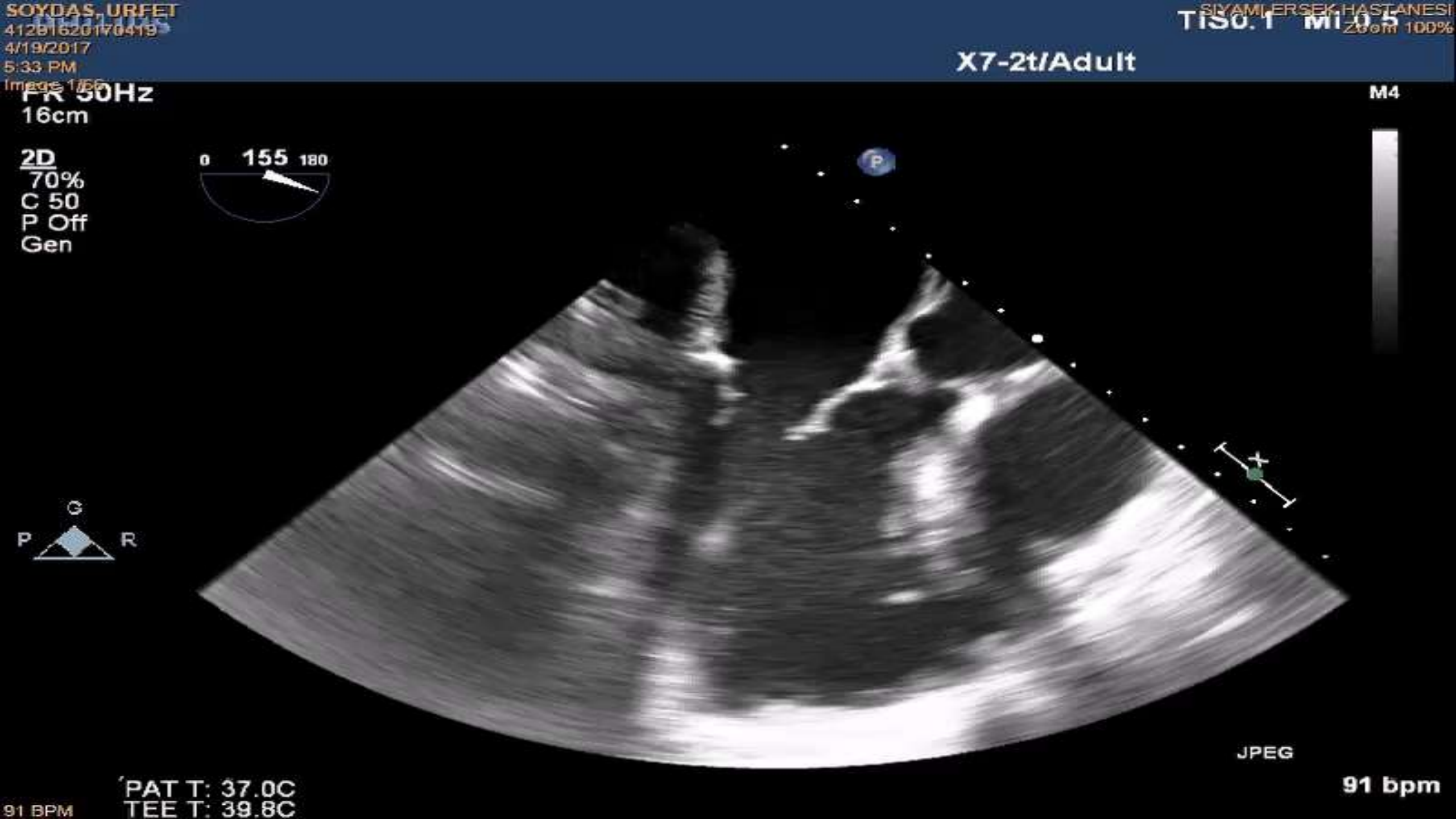
V5
V6
0.05-25 Hz
F50
25 mm/s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SIYAMI ERSEK HASTANESI
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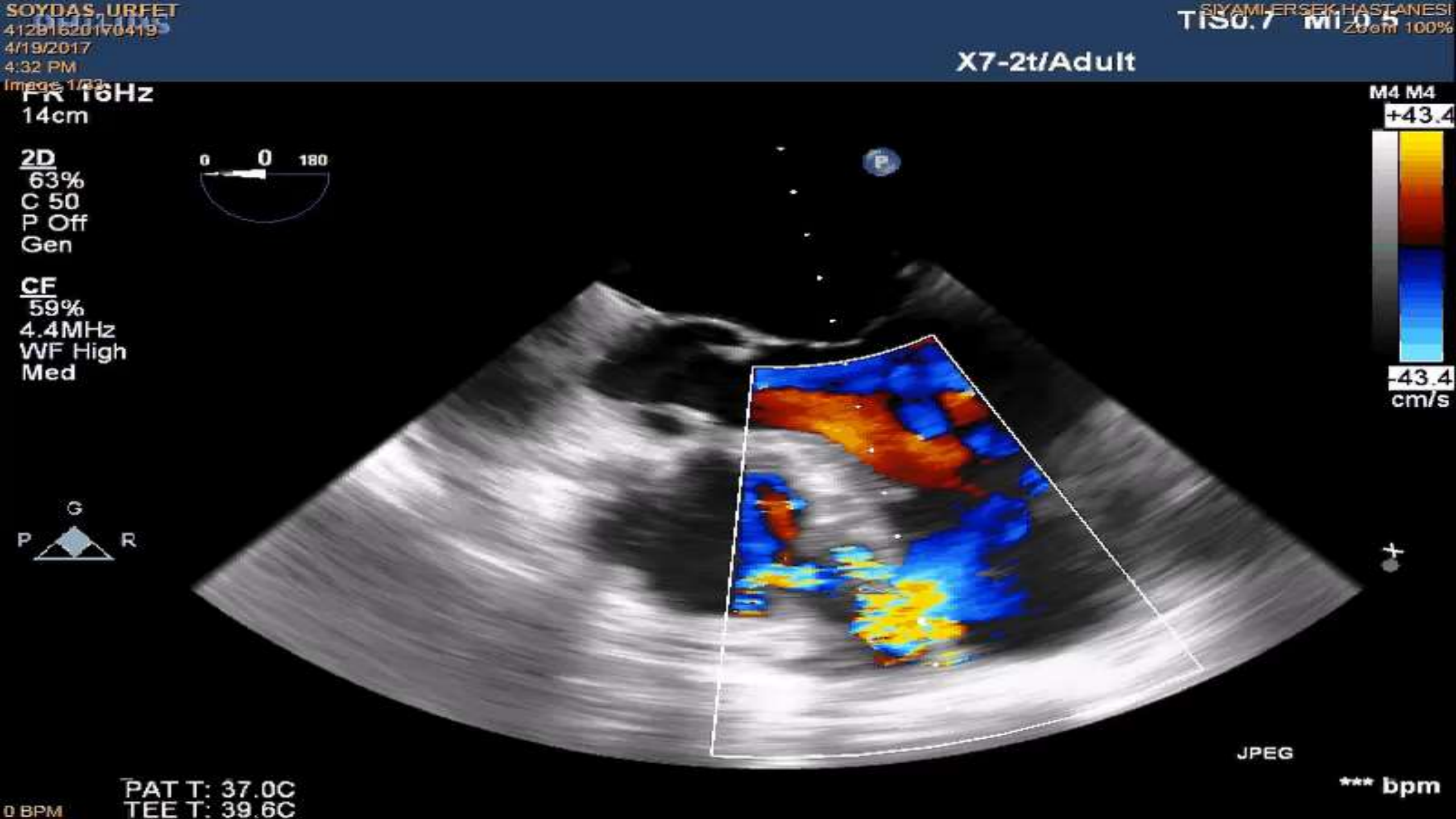
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SIYAMI ERSEK HASTANESI
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C 50
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CF
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-43.4
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P
R
JPEG
PAT T: 37.0C
TEE T: 39.6C
0 BPM
*** bpm

KARDIYOJENIK ŞOK

$$\Delta P = CO \times SVR$$

$$CO = SV \times HR$$

KARDIYOJENIK ŞOK

- TEMEL ÖZELLIKLER:

  - KARDIYAK OUTPUTTA/İNDEXTE AZALMA: CI>2.5 L/DK/M2 ISE ŞOK OLUŞMAZ. KARDIYOJENIK ŞOK OLABILMESI IÇIN CI<2.2 L/DK/M2 OLMALı.

  - DOLUM BASıNÇLARıNDA ARTıŞ.

  - MIYOKARDIYAL O2 IHTIYACıNDA ARTıŞ VE SUNUMUNDA AZALMA SONUCU OKSIJEN TÜKETIMININ AZALMASı.

  - SISTEMIK VASKÜLER DIRENÇTE ARTıŞ: METABOLIK SEBEPLER, E/NE VE ATII (VE ILAÇLAR!!!) ARTıŞıNA BAĞLı SVR ARTAR. ANCAK AKUT MYOKARD HASARı SEYRINDE ORTAYA ÇıKAN INFLAMATUAR MEDIATÖRLER PERIFERIK VAZODILATASYONA BAĞLı SVR'YI AZALTABILIR[1].

1 HOCHMAN JS. CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION: EXPANDING THE PARADIGM. CIRCULATION. 2003 JUN 24;107(24):2998-3002.

MAJÖR BULGULAR

- HIPOTANSIYON: ŞOK TANıSı IÇIN ŞART DEĞIL! ANCAK KARDIYOJENIK ŞOKTA HEMEN HER ZAMAN BULUNUR.
- TAŞIKARDI: BETA BLOKER KULLANANLARDA OLMAYABILIR.
- TAŞIPNE
- OLIGÜRI
- MENTAL DURUMDA BOZULMA
- SOĞUK-SOLUK-NEMLI DERI
- METABOLIK ASIDOZ VE LAKTATEMI (LA>2.0 MG/DL).

+ KONJESYON BULGULARı: AKCIĞER RALLERI, JUGULER VENÖZ DISTANSIYON – 1/3 HASTADA YOK[1]!

+ S3 GALO RITMI

1 MENON V1, WHITE H, LEJEMTEL T, WEBB JG, SLEEPER LA, HOCHMAN JS. THE CLINICAL PROFILE OF PATIENTS WITH SUSPECTED CARDIOGENIC SHOCK DUE TO PREDOMINANT LEFT VENTRICULAR FAILURE: A REPORT FROM THE SHOCK TRIAL REGISTRY. SHOULD WE EMERGENTLY REVASCULARIZE OCCLUDED CORONARIES IN CARDIOGENIC SHOCK? J AM COLL CARDIOL. 2000 SEP;36(3 SUPPL A):1071-6.

ÖNERILEN TANıMLAMALAR

Table 12.2 Definitions of the terms used in Section 12 on acute heart failure

Term	Definition
Symptoms/signs of congestion (left-sided)	Orthopnoea, paroxysmal nocturnal dyspnoea, pulmonary rales (bilateral), peripheral oedema (bilateral).
Symptoms/signs of congestion (right-sided)	Jugular venous dilatation, peripheral oedema (bilateral), congested hepatomegaly, hepatojugular reflux, ascites, symptoms of gut congestion.
Symptoms/signs of hypoperfusion	Clinical: cold sweated extremities, oliguria, mental confusion, dizziness, narrow pulse pressure. Laboratory measures: metabolic acidosis, elevated serum lactate, elevated serum creatinine. Hypoperfusion is not synonymous with hypotension, but often hypoperfusion is accompanied by hypotension.
Hypotension	Systolic BP <90 mmHg
Bradycardia	Heart rate <40 bpm
Tachycardia	Heart rate >120 bpm
Abnormal respiratory effort	Respiratory rate >25 breaths/min with use of accessory muscles for breathing, or respiratory rate <8 breaths/min despite dyspnoea.
Low O_2 saturation	O_2 saturation (SaO_2) <90% in pulse oximetry Normal SaO_2 neither excludes hypoxaemia (low PaO_2) nor tissue hypoxia.
Hypoxaemia	O_2 partial pressure (PaO_2) in arterial blood <80 mmHg (<10,67 kPa) (blood gas analysis).
Hypoxaemic respiratory failure (type I)	PaO_2 <60 mmHg (<8 kPa)
Hypercapnia	CO_2 partial pressure ($PaCO_2$) in arterial blood >45 mmHg (>6 kPa) (blood gas analysis).
Hypercapnic respiratory failure (type II)	$PaCO_2$ >50 mmHg (>6,65 kPa).
Acidosis	pH <7.35
Elevated blood lactate	>2 mmol/L
Oliguria	Urine output <0.5 mL/kg/h

BP = blood pressure; bpm = beats per minute; $PaCO_2$ = partial pressure of carbon dioxide in arterial blood; PaO_2 = partial pressure of oxygen in arterial blood; SaO_2 = oxygen saturation.

HIPOTANSIYON NEDIR?

- MUTLAK HIPOTANSIYON: SBP<90 MMHG VEYA MAP<65 MMHG.
- RÖLATIF HIPOTANSIYON: SBP'DE 40 MMHG VEYA DAHA FAZLA PERSISTAN AZALMA.
- ORTOSTATIK HIPOTANSIYON: AYAĞA KALKıNCA SBP'DE 20 MMHG, DBP'DE 10 MMHG AZALMA.

AKUT MYOKARD ENFARKTı SEYRINDE KARDIYOJENIK ŞOK

* KARDIYOJENIK ŞOKUN EN SıK SEBEBIDIR.

- 79% - SOL VENTRIKÜL YETERSIZLIĞI[1]: ŞOK OLABILMESI IÇIN LV TOTAL KITLESININ EN AZ 40%'ı INFARKTE OLMALı.
- 7% - MITRAL YETERSIZLIĞI
- 4% - VENTRIKÜLER SEPTAL RÜPTÜR
- 2% - RV INFARKTı
- 1.4% - TAMPONAD
- 7% - DIĞER.

1 HOCHMAN JS, BULLER CE, SLEEPER LA, BOLAND J, DZAVIK V, SANBORN TA, GODFREY E, WHITE HD, LIM J, LEJEMTEL T. CARDIOGENIC SHOCK COMPLICATING ACUTE MYOCARDIAL INFARCTION--ETIOLOGIES, MANAGEMENT AND OUTCOME: A REPORT FROM THE SHOCK TRIAL REGISTRY. SHOULD WE EMERGENTLY REVASCULARIZE OCCLUDED CORONARIES FOR CARDIOGENIC SHOCK? J AM COLL CARDIOL. 2000 SEP;36(3 SUPPL A):1063-70.

AKUT MYOKARD ENFARKTı SEYRINDE KARDIYOJENIK ŞOK

- İNSIDANSıNDA YıLLAR IÇERISINDE BIR AZALMA MEVCUT. DAHA ESKI ÇALıŞMALARDA SıKLıĞı 6-9% ARASıNDA BELIRTILIRKEN 2005'TE YAPıLAN BIR ÇALıŞMADA 5.7% OLARAK BULUNMUŞ[1].
- 97.5% ST ELEVASY6ONLU Mı SEYRINDE, 2.5% NON ST ELEVASYONLU Mı SEYRINDE GÖRÜLÜR.
- ST ELEVASYONLU Mı'DA ÇOĞU VAKA ILK 24 SAATTE ŞOK ILE PREZENTE OLURKEN NON-ST ELEVASYONLU Mı'DA PREZANTASYON HOSPITALIZASYONDAN SONRAKI 4-5 GÜNDE ZIRVE YAPAR[2].

1 AISSAOUI N, PUYMIRAT E, TABONE X, CHARBONNIER B, SCHIELE F, LEFÈVRE T, DURAND E, BLANCHARD D, SIMON T, CAMBOU JP, DANCHIN N. IMPROVED OUTCOME OF CARDIOGENIC SHOCK AT THE ACUTE STAGE OF MYOCARDIAL INFARCTION: A REPORT FROM THE USIK 1995, USIC 2000, AND FAST-MI FRENCH NATIONWIDE REGISTRIES. EUR HEART J. 2012 OCT;33(20):2535-43. DOI: 10.1093/EURHEARTJ/EHS264. EPUB 2012 AUG 26.

2 HOLMES DR JR, BERGER PB, HOCHMAN JS, GRANGER CB, THOMPSON TD, CALIFF RM, VAHANIAN A, BATES ER, TOPOL EJ. CARDIOGENIC SHOCK IN PATIENTS WITH ACUTE ISCHEMIC SYNDROMES WITH AND WITHOUT ST-SEGMENT ELEVATION. CIRCULATION. 1999 NOV 16;100(20):2067-73.

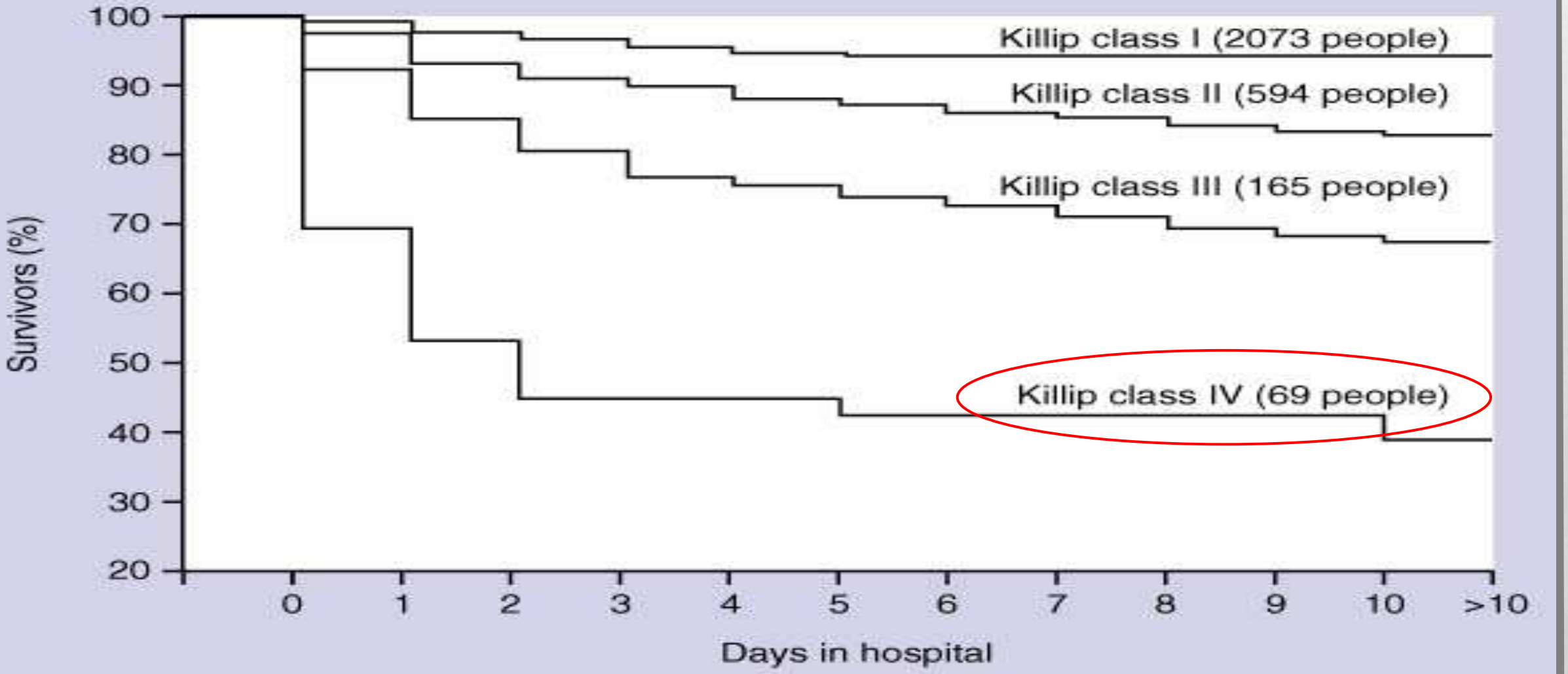
RISK FAKTÖRLERI

- İLERI YAŞ
- ANTERIOR Mı
- HIPERTANSIYON
- DIYABET
- ÇOKLU DAMAR HASTALıĞı
- DAHA ÖNCE GEÇIRILMIŞ MI
- SBP<120 MMHG
- KH>90 V/DAK
- BAŞVURU ANıNDA KALP YETERSIZLIĞI
- ST ELEVASYONLU Mı
- SOL DAL BLOĞU

TANI

- EKG: ISKEMI BULGULARı, TAŞIKARDI, ARITMI
- PA AKCIĞER GRAFISI: KONJESYON
- METABOLIK ASIDOZ
- HIPERLAKTATEMI
- ÜRE VE KREATININDE ARTıŞ
- EKOKARDIYOGRAFIDE AZALMıŞ SOL VENTRIKÜL SISTOLIK FONKSIYONU VEYA MEKANIK KOMPLIKASYON
- PULMONER KATETERIZASYONDA ARTMıŞ KAMA VE PULMONER ARTER BASıNCı.

PROGNOZ


Killip class I (2073 people)
Killip class II (594 people)
Killip class III (165 people)
Killip class IV (69 people)
Survivors (%)
100
90
80
70
60
50
40
30
20
0
1
2
3
4
5
6
7
8
9
10
>10
Days in hospital

MI'DA KARDIYOJENIK ŞOK: AYIRICI TANI

- 1-) AKUT MYOKARD ENFARKTı ILE BIRLIKTE BAŞKA ŞOK SEBEPLERI (ÖRN. SEPTIK ŞOK).
- 2-) AMI ILE KARıŞABILECEK PATOLOJILER:
  - TAKOTSUBO KARDIYOMYOPATISI
  - HIPERTROFIK KARDIYOMYOPATI VEYA AKUT MIYOKARDIT
  - AKUT PULMONER EMBOLI
  - AORT DISEKSIYONU SEYRINDE GELIŞEN MI

KARDIYOJENIK ŞOKUN SıNıFLAMASı VAR Mı?

- ETIYOLOJIK SıNıFLAMA HARIÇ YOKTUR.
- KARDIYOJENIK ŞOK, AKUT KALP YETERSIZLIĞININ EN CIDDI PREZANTASYONU OLARAK SıNıFLANıR.

Table II – Clinical and hemodynamic subgroups in acute myocardial infarction

Killip Subgroup	Clinical characteristics	Hospital mortality
I	No congestion signs	<6%
II	S3, basal rales	<17%
III	Acute pulmonary edema	38%
IV	Cardiogenic shock	81%
Forrester subgroup	**Hemodynamic characteristics**	**Hospital mortality**
I	PCP <18, IC >2.2	3%
II	PCP >18, IC >2.2	9%
III	PCP <18, IC <2.2	23%
IV	PCP >18, IC <2.2	51%

PCP- pulmonary capillary pressure; CI- cardiac index.

Table 13.2 INTERMACS (Interagency Registry for Mechanically Assisted Circulatory Support) stages for classifying patients with advanced heart failure

INTERMACS level	NYHA Class	Description	Device	1 y survival with LVAD therapy
1. Cardiogenic shock "Crash and burn"	IV	Haemodynamic instability in spite of increasing doses of catecholamines and/or mechanical circulatory support with critical hypoperfusion of target organs (severe cardiogenic shock).	ECLS, ECMO, percutaneous support devices	52.6±5.6%
2. Progressive decline despite inotropic support "Sliding on inotropes"	IV	Intravenous inotropic support with acceptable blood pressure but rapid deterioration of renal function, nutritional state, or signs of congestion.	ECLS, ECMO, LVAD	63.1±3.1%
3. Stable but inotrope dependent "Dependent stability"	IV	Haemodynamic stability with low or intermediate doses of inotropics, but necessary due to hypotension, worsening of symptoms, or progressive renal failure.	LVAD	78.4±2.5%
4. Resting symptoms "Frequent flyer"	IV ambulatory	Temporary cessation of inotropic treatment is possible, but patient presents with frequent symptom recurrences and typically with fluid overload.	LVAD	78.7±3.0%
5. Exertion intolerant "Housebound"	IV ambulatory	Complete cessation of physical activity, stable at rest, but frequently with moderate fluid retention and some level of renal dysfunction.	LVAD	93.0±3.9%[a]
6. Exertion limited "Walking wounded"	III	Minor limitation on physical activity and absence of congestion while at rest. Easily fatigued by light activity.	LVAD / Discuss LVAD as option	-
7. "Placeholder"	III	Patient in NYHA Class III with no current or recent unstable fluid balance.	Discuss LVAD as option	-

ECLS = extracorporeal life support; ECMO = extracorporeal membrane oxygenation; INTERMACS = Interagency Registry for Mechanically Assisted Circulatory Support; LVAD = left ventricular assist device; NYHA = New York Heart Association.

[a]Kaplan-Meier estimates with standard error of the mean for 1 year survival with LVAD therapy. Patients were censored at time of last contact, recovery or heart transplantation. Due to small numbers outcomes for INTERMACS levels 5, 6, 7 were combined[4,10].

TABLE 24-2 Simplified Classification and Common Clinical Characteristics of Patients with Acute Heart Failure

CLINICAL CLASSIFICATION	SYMPTOM ONSET	TRIGGERS	SIGNS AND SYMPTOMS	CLINICAL ASSESSMENT	COURSE
Decompensated heart failure	Usually gradual	Noncompliance, ischemia, infections	Peripheral edema, orthopnea, dyspnea on exertion	SBP: variable CXR: often clear despite elevated filling pressures	Variable; high rehospitalization rate
Acute hypertensive heart failure	Usually sudden	Hypertension, atrial arrhythmias, ACS	Dyspnea (often severe), tachypnea, tachycardia, rales common	SBP: high (>180/100 mm Hg) CXR: evidence of pulmonary edema Hypoxemia common	High acuity but often responds quickly to therapy with vasodilators, noninvasive ventilation; low postdischarge mortality
Cardiogenic shock	Variable	Progression of advanced HF or major myocardial insult (e.g., large-infarct AMI, acute myocarditis)	End-organ hypoperfusion; oliguria, confusion, cool extremities	SBP: Low or low-normal LV function usually severely depressed RV dysfunction common Laboratory evidence of end-organ dysfunction (renal, hepatic)	High inpatient mortality; poor prognosis except with readily reversible cause or mechanical support/ transplantation

AMI = acute myocardial infarction; CXR = chest x-ray [film/examination]; HF = heart failure.

- ***BENI DINLEDIĞINIZ İÇIN TEŞEKKÜR EDERIM...***