GÖĞÜS KALP DAMAR ANESTEZİ VE YOĞUN BAKIM DERNEĞİ

27. Ulusal Kongresi

24 - 25 Eylül 2021 Wyndham Grand İzmir Özdilek

Non-kardiyak cerrahi sonrası miyokard hasarı

Elvin Kesimci

NON-KARDİYAK CERRAHİ SONRASI MİYOKARD HASARI

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Başkent Üniversitesi Tıp Fakültesi

Anesteziyoloji Anabilim Dalı

25.9.2021

BAŞKENT ÜNİVERSİTESİ

Dünyada

> 300 milyon cerrahi işlem/yıl

> 200 milyon majör non-kardiyak cerrahi işlem/yıl

Puelacher C. Eur J Anaesthesiol 2021;38:600-608.
Szczeklik W. Curr Opin Anesthesiol 2021;34:381-386.

Anesteziye bağlı intraoperatif mortalite çok nadirdir

ABD'de

° cerrahi geçiren hastalarda non-kardiyak cerrahiden sonraki bir ay içinde mortalite ~ %1

Ruetzler K. Anesth Analg 2020;131:173-186.

Postoperatif ilk 30 gündeki mortalitenin en sık sebebi

miyokard iskemisi ve majör kanama

- ✓ Postoperatif ölümler global mortalitenin % 7.7’ si
- ✓ Dünyada mortalitenin 3. sebebi

Botto F. Anesthesiology. 2014;120:564-578.
Devereaux PJ. JAMA 2012; 307:2295-2304.
Devereaux PJ. Ann Intern Med 2011; 154:523-528.

postoperatif mortalite % 1 ↑ % 9.5

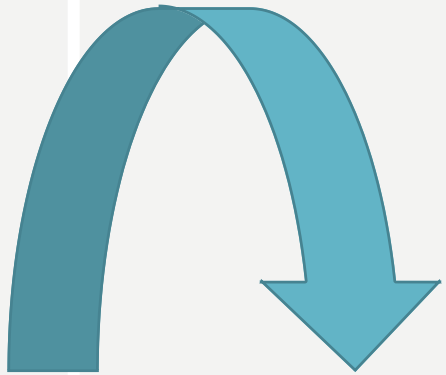
MINS varlığında

konjestif kalp yetmezliği, inme ve ölümcül olmayan MI riski % 2.4 ↑ % 18.5

Botto F. Anesthesiology. 2014;120:564-578.
Devereaux PJ. JAMA 2012; 307:2295-2304.
Devereaux PJ. Ann Intern Med 2011; 154:523-528.

Non-kardiyak cerrahi

- ✓ hemodinamik bozulma
- ✓ hiperkoagülasyon
- ✓ inflammasyon
- ✓ sempatik stimülasyon
- ✓ kanama



iskemik yaralanmalar

- ✓ Major non-kardiyak cerrahi sonrasında kardiyak komplikasyonlar sıktır (%5)

Devereaux PJ. N Engl J Med 2015; 373:2258-2269.

- miyosit nekrozu
- volüm ve basınç yükü
- inflamasyon

Akut kardiyak olayların patofizyolojisi altındaki mekanizmalar

De Hert S. Anesth Analg 2020;131:187-195.

KARDİYAK

Perioperatif Faktörler

- inflammasyon
- hiperkoagülabilite
- sempatik sistem aktivasyonu
- hemodinamik değişiklikler

hassas plak rüptürü

Tip I miyokard iskemisi

Postoperatif Faktörler

- hiper-veya hipotansiyon
- taşikardi
- anemi

- öncesinde stenozu olan hastada oksijen sunum-ihtiyaç uyumsuzluğu

NON-KARDİYAK

Non-kardiyak komplikasyonlar

- pulmoner emboli
- sepsis
- böbrek hasarı
- solunum yetmezliği

- öncesinde stenozu olmasa bile kalbin üzerindeki artmış ihtiyaç

Tip II miyokard iskemisi

Postoperatif miyokard hasarı

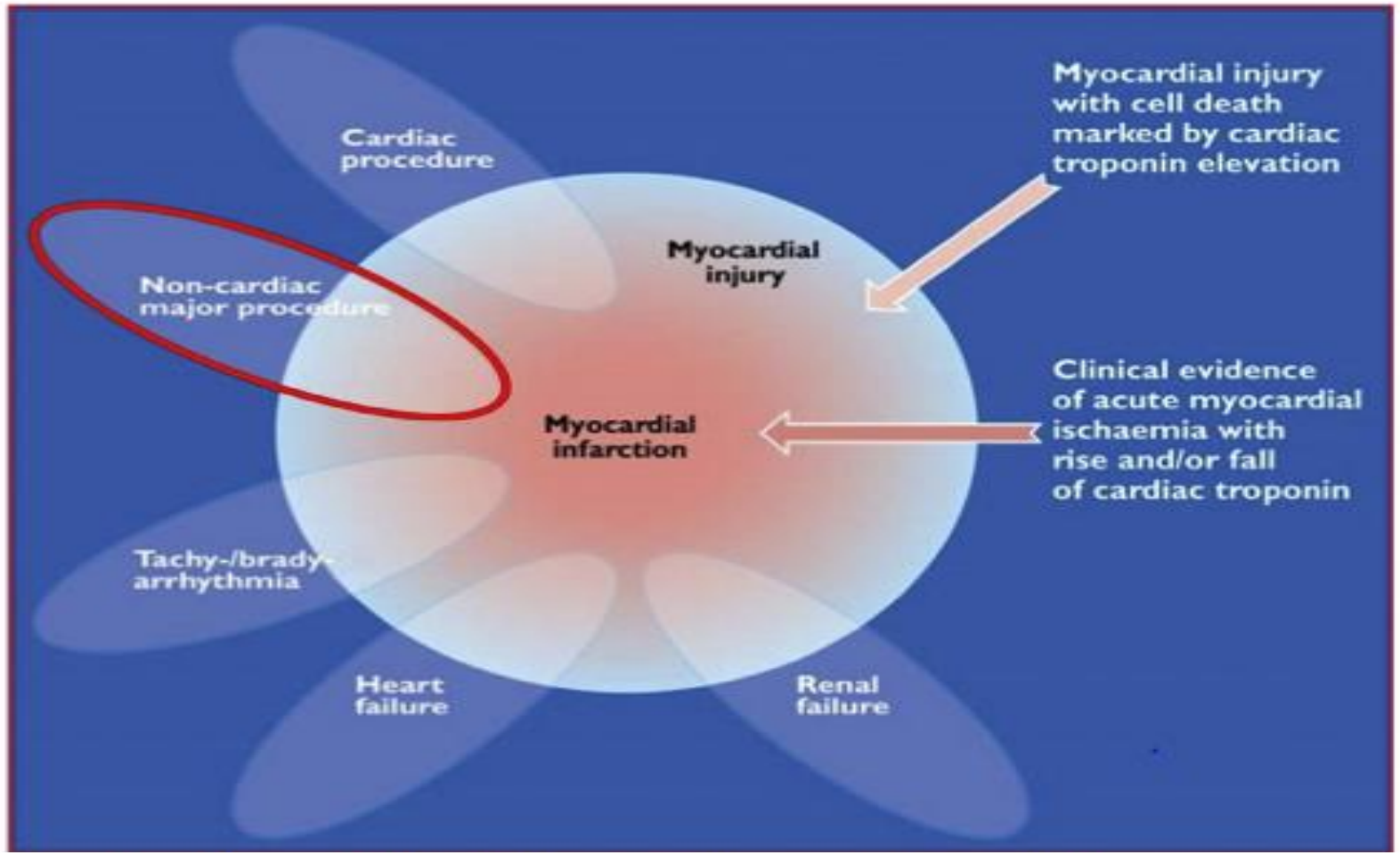
Ooi DS. Clin Chem. 2000;46:338–344.

MİYOKARD HASARI

MİYOKARD İNFARKTÜSÜ

NON-KARDİYAK CERRAHİ SONRASI MİYOKARD HASARI

Miyokard infarktüsü tüm miyokard hasarlarının alt kümesidir



Aboyans V. *Eur J Vasc Endovasc Surg 2018;56:161-162.*

Miyokard iskemisi: kan damarlarındaki obstrüksiyona bağlı olarak kalp dokusuna kan akımı ve oksijen sunumunda kısıtlılık

Sıklıkla miyokard nekrozu veya infarktüsü ile sonuçlanır

Crossman DC. Heart 2004;90:576-580.

MINS (NON-KARDİYAK CERRAHİ SONRASI MİYOKARD HASARI)

nonkardiyak cerrahi sonrası 30 gün içinde ortaya çıkan iskemiye bağlı miyokard hasarı

- ✓ iskemi kaynaklı
- ✓ MI gibi FM ve EKG'de tipik miyokard iskemi bulguları vermeyen (göğüs ağrısı, anjina pektoris, veya dispne)
- ✓ artmış postoperatif troponin seviyesi ile karakterize

klinik bir durum !

Devereaux PJ. JAMA 2017;317:1642-1651.

MINS kriterleri

- ✓ En az 20 ng/l high sensitivity cTn T konsantrasyonu beraberinde en az 5 ng/l lik mutlak değişiklik, veya
- ✓ Mutlak postoperatif hs-cTn T değerinin en az 65 ng/l olması

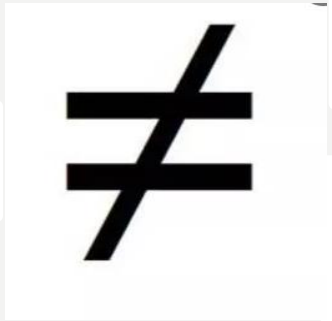
Puelacher C. Eur J Anaesthesiol 2021;38:600-608.

Pek çok araştırmada

- ✓ hastaların miyokard hasarı yaşadığı ama bunun MI için diagnostik kriterleri tam olarak karşılamadığı
- ✓ bu olayların uygun zamanda fark edilip zamanında müdahale edilmediği için kötü prognozlu olduğu kanıtlanmıştır

Ali ZA. Eur J Vasc Endovasc Surg 2008;35:413-419.

MINS



- ✓ MINS troponitis değil
- ✓ Non iskemik sebeplerle oluşan perioperatif miyokard hasarı (sepsis, hızlı AF, pulmoner emboli, renal yetmezlik)

Szczeklik W. Curr Opin Anesthesiol 2021;34:381-386.

MINS’ın % 93’ü

MI’ların % 68’i

Troponin tarama yapmadan tipik olarak tanınmaz

Devereaux PJ. JAMA 2017;317:1642-1651.
Levy M. Anesthesiology 2011;114:796-806.
BeattieWS. Can J Anaesth 2012;59:1013-1022.

Miyokard hasarı tanısı; * sistemik dolaşımda kardiyomiyosit (‘e özel) proteinlerin tayinine dayanır

Çok sayıda biyobelirteç

- Aspartat transaminaz ilk kullanılan biyobelirteç 1960’larda MI tanımında (WHO’nun belirlediği)

Thygesen K. Circulation 2018;136:e618-e651.

Çok sayıda biyobelirteç

- > 2000 yıl troponinler (Tn I veya T) kreatin kinaz-MB (CK-MB)'nin yerini aldı
- 2018'de de 'Dördüncü Evrensel MI Tanımında' yüksek spesifisite ve sensitiviteleri nedeni ile troponinler

Thygesen K. Circulation 2018;136:e618-e651.

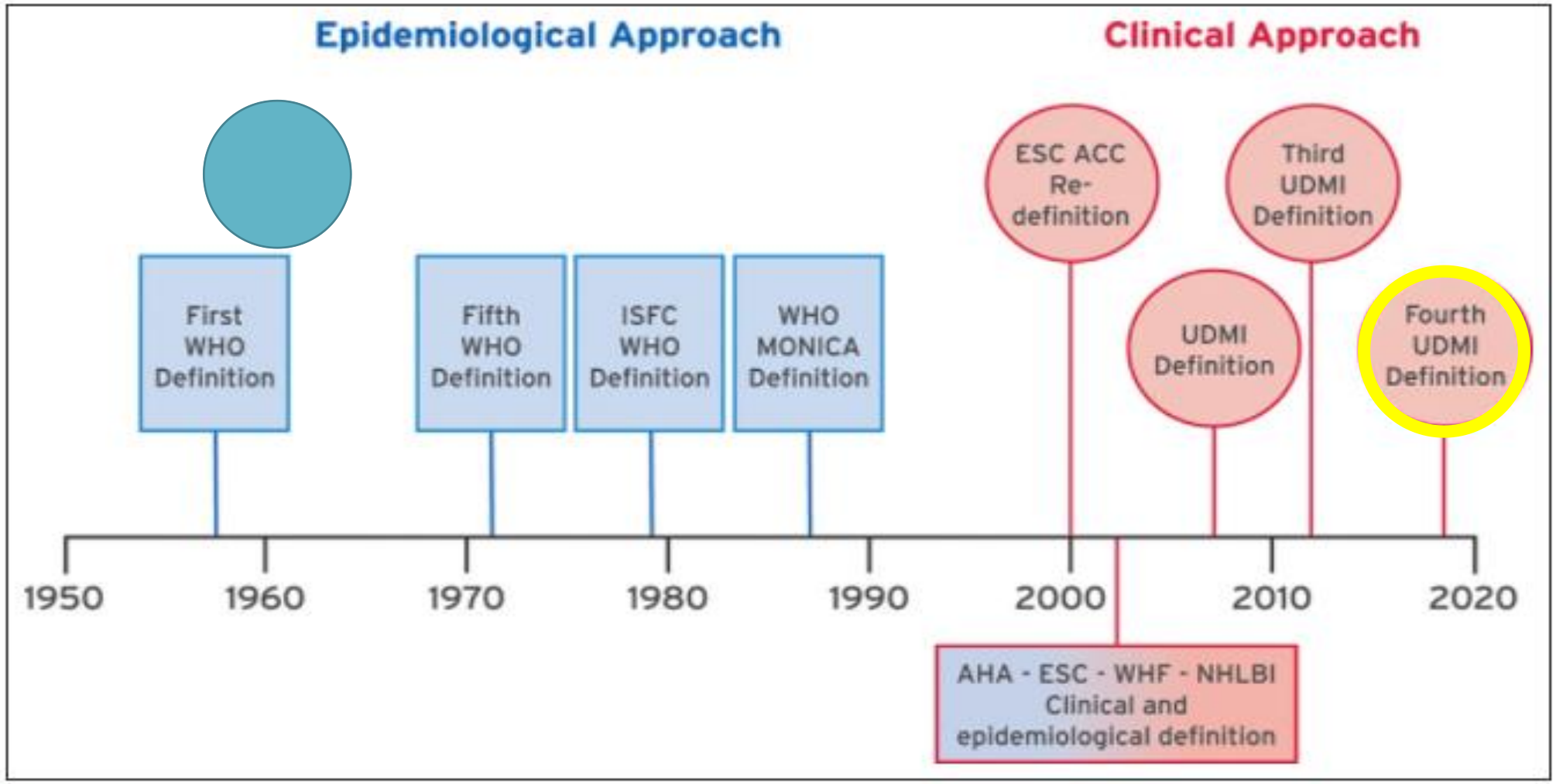


Figure 1. History of documents on the definition of myocardial infarction. ACC indicates American College of Cardiology; AHA, American Heart Association; ESC, European Society of Cardiology; ISFC, International Society and Federation of Cardiology; MONICA, MONItoring of trends and determinants in CArdiovascular disease; NHLBI, National Heart, Lung, and Blood Institute; UDMI, Universal Definition of Myocardial Infarction; WHF, World Heart Federation; WHO, World Health Organization.

Circulation
Volume 138, Issue 20, 13 November 2018; Pages e618-e651
https://doi.org/10.1161/CIR.0000000000000617

ESC/ACC/AHA/WHF EXPERT CONSENSUS DOCUMENT

Fourth Universal Definition of Myocardial Infarction (2018)

Kristian Thygesen, Joseph S. Alpert, Allan S. Jaffe, Bernard R. Chaitman, Jeroen J. Bax, David A. Morrow, Harvey D. White, and The Executive Group on behalf of the Joint European Society of Cardiology (ESC)/American College of Cardiology (ACC)/American Heart Association (AHA)/World Heart Federation (WHF) Task Force for the Universal Definition of Myocardial Infarction

Postoperatif MI için miyokard hasarı + (en az) bir tanesi;

- Akut miyokard iskemi belirtileri
- Yeni iskemik EKG değişiklikleri
- Patolojik Q dalgaları gelişmesi
- Görüntülemede →
  - yeni duvar hareket bozukluğu veya
  - anjiyo ya da otopsi ile koroner trombüs

Miyokard İnfarktüs Tipleri

Tip I: koroner arterin aterotrombotik tıkanıklığına bağlı olarak miyokarda kan akışının azalması (rüptür veya erozyon)

Tip II: O_2 ihtiyacı ve sunumu arasındaki dengesizlik (koroner spazm, koroner emboli, aritmi, anemi veya hipotansiyon)

Tedavi: volüm düzenlenmesi, kan basıncı yönetimi, kan transfüzyonu, kalp atım hızı kontrolü, ve solunumsal destek

Tip III: ani kardiyak olay ve ölüm

serum biyobelirteçleri sonradan yükselir

Thygesen K. Eur Heart J 2018; 39: 3757–3765.

Miyokard İnfarktüs Tipleri

Tip IV: Kardiyak biyobelirteçlerin 5 kat artması (bazal değerleri N ise)

Bazal değerleri artmış olanlarda > % 20 değişiklik

Tip V: Kardiyak biyobelirteçlerin 10 kat artması (bazal değerleri N ise)

Bazal değerleri artmış olanlarda > % 20 değişiklik

Thygesen K. Eur Heart J 2018; 39: 3757–3765.

Akut MI tip 1,2, ve 3

- ✓ akut miyokard iskeminin klinik belirtileri ve troponin konsantrasyonlarında artış ile görülen akut miyokard hasarı (en az 1 değeri 99th persentili geçecek şekilde)

Akut MI tip 4 ve 5

- ✓ normal bazal kardiyak biyobelirteçleri olan hastada bazal değerin > % 20 değişiklik vardır

Circulation
Volume 138, Issue 20, 13 November 2018; Pages e618-e651
https://doi.org/10.1161/CIR.0000000000000617

ESC/ACC/AHA/WHF EXPERT CONSENSUS DOCUMENT

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MI için Klinik Kriter

Akut miyokard iskemisi kanıtı ile aynı zeminde anormal kardiyak biyobelirteçlerle tespit edilen akut miyokard hasarı

MINS için

Bu metodolojik yaklaşım perioperatif uygulamada uygun yaklaşım değil

- ✓ postoperatif analjezik alan hastalar göğüs ağrısını hissedemez
- ✓ patognomik EKG değişiklikleri nadirdir
- ✓ ekokardiyografi tarama amaçlı rutin kullanılmaz
- ✓ anjiyografide postoperatif MI'ların dörtte biri trombotik lezyonları gösterir

Lange RA. Cath Cardiovasc Interv 2012;80:777-778.

Simpson GC *et al. SA Orthop J* 2018;17(2)
DOI 10.17159/2309-8309/2018/v17n2a1

South African Orthopaedic Journal
http://journal.saoa.org.za

Perioperatif Miyokard Hasarı Sık, Sessiz ve Ölümcül

GENERAL ORTHOPAEDICS

The silent killer: myocardial injury after non-cardiac surgery (MINS)

Simpson GC[1], Marais LC[2], Rodseth RN[3], Outcomes Research Consortium[4]

- MINS hastalarının
  - % 93'nde semptom yoktur

MINS ile ilgili farkındalığın artması, sonuçları iyileştirecek ve perioperatif morbidite ve mortaliteyi azaltacaktır

MINS patogenezi tam olarak bilinmese de…

altta yatan koroner arter lezyonu üzerine eklenen O_2 sunum-ihtiyaç arasındaki dengesizlik, uyumsuzluk vakaların çoğunluğundan sorumludur

Sessler DI. Br J Anaesth 2019,122,563-574.
Turan A. Anesth Analg 2020,131,822-829.
Chew E. Eur j anaesthesiol 2019,36,884-886.
Abbott TEF. Anesth Analg 2018,126,1936-1945.

MINS patogenez…

Major non-kardiyak cerrahi sırasında karşılaşılan sıvı shiftleri, hipotansiyon, ağrı, anemi, taşikardi, hipoksemi, ve katekolamin deşarjı

MINS'ı başlatabilir

Sessler DI. Br J Anaesth 2019,122,563-574.
Turan A. Anesth Analg 2020,131,822-829.
Chew E. Eur j anaesthesiol 2019,36,884-886.
Abbott TEF. Anesth Analg 2018,126,1936-1945.

Circulation

Circulation. 2018;137:1221–1232.

ORIGINAL RESEARCH ARTICLE

Perioperative Myocardial Injury After Noncardiac Surgery

Incidence, Mortality, and Characterization

Editorial, see p 1233

Christian Puelacher, MD, PhD

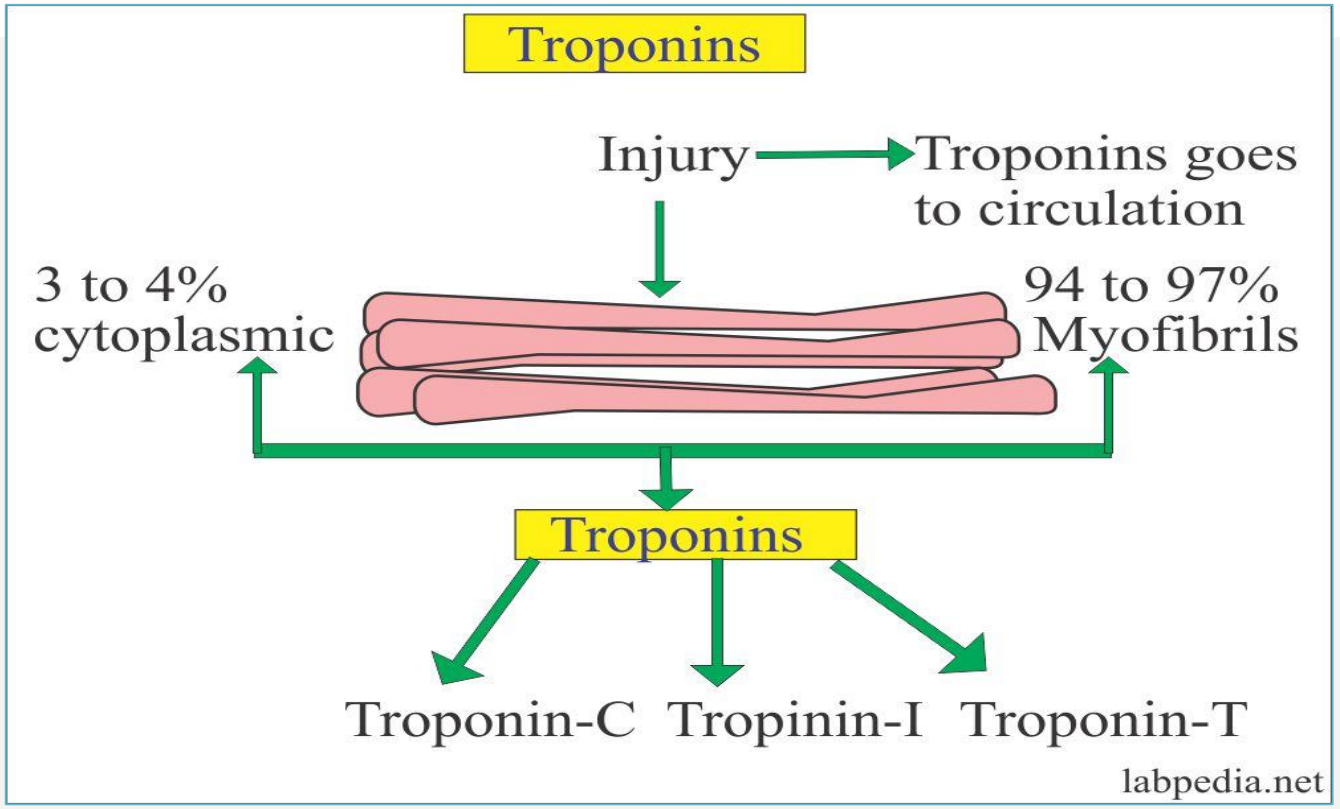
- ✓ Sunum-ihtiyaç uygunsuzluğu
- ✓ Daha ender klinikte Tip I MI'a benzeyen intrakoroner trombus oluşumu ile görülen plak rüptürü

Trombotik olay perioperatif strese bağlı yolaklar, inflammasyon ve hiperkoagülabilite ile artar

Ackland GL. Br J Anesth 2019;122:180-187.

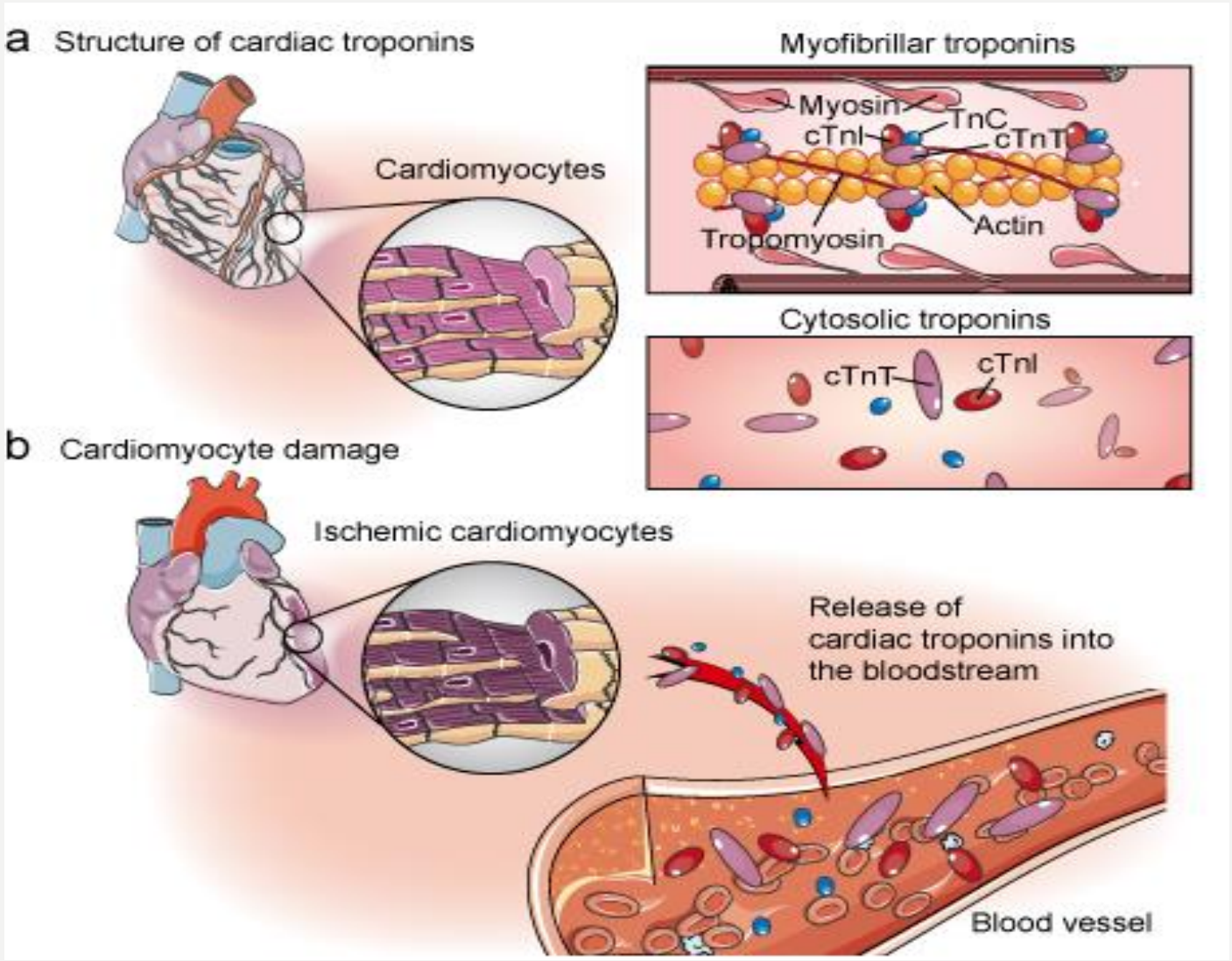
Troponinler

- iskelet ve kardiyak miyositlerdeki kontraktil filamentin iç kısmıdır
- miyosin ve aktin filamentleri arasındaki etkileşimi kolaylaştırırlar
- 3 subgrubu vardır: C, T ve I



De Hert. Anesth Analg 2020;131:187-195.

kardiyak troponin T ve troponin I (cTnT ve cTnI)



aktin ve miyozin arasındaki Ca^{+2} aracılı etkileşimi kontrol eden kardiyak düzenleyici proteinlerdir

Sharma S. J Clin Pathol 2004t; 57: 1025–1026.

TnT Tn'i aktin filamentine bağlar

TnC kalsiyum bağlayan kısımdır

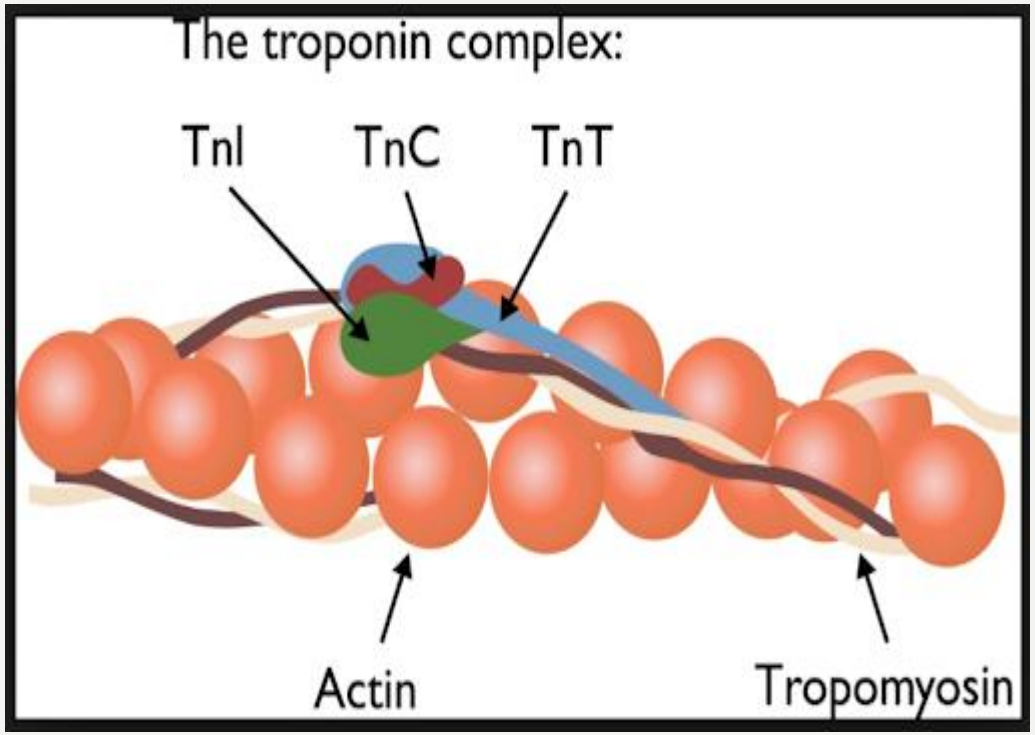
TnI miyosin ve aktinin etkileşimini inhibe eder

Testler, kardiyak izoformları için yapılmıştır

Kardiyak TnI ve TnT testleri kalbe spesifik

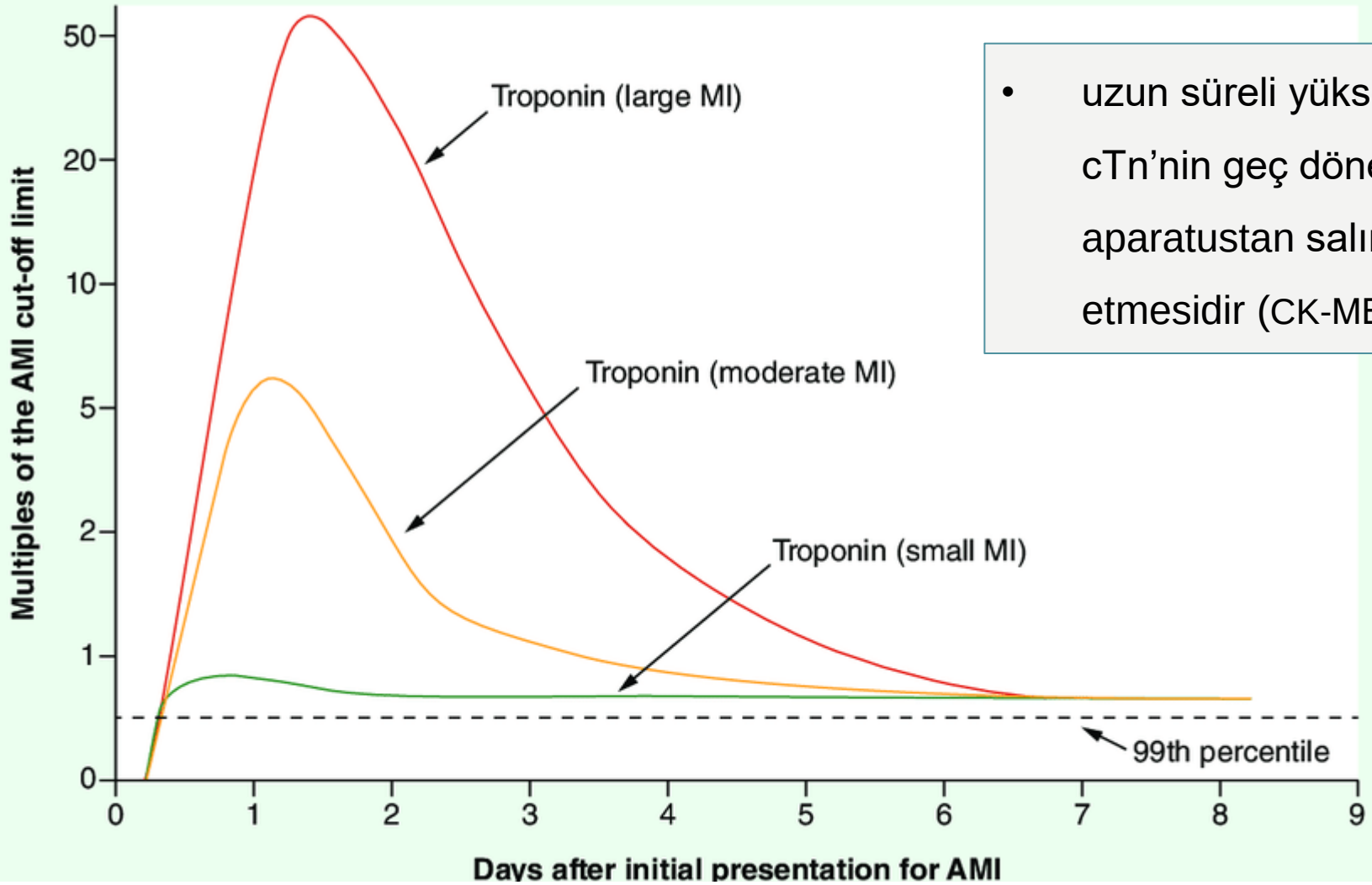
De Hert. Anesth Analg 2020;131:187-195.

✓ cTn-T ve I farklı genlerce kodlandıkları için iskelet kasındaki troponinlerden tamamıyla farklıdır



Serum cTnT ve cTnI ölçümü, kalp kası hasarının tanımlanmasında kalp kası enzim ölçümlerine duyarlılık ve özgüllük açısından üstündür

Sharma S. J Clin Pathol 2004; 57: 1025–1026.



- uzun süreli yüksekliğinin nedeni, cTn'nin geç dönemde kontraktil aparatustan salınımının devam etmesidir (CK-MB'den farklı olarak)

- ✓ Normalde kardiyak spesifik troponin kanda tespit edilmez veya zor tespit edilir
- ✓ Ama kardiyomiyosit nekrozundan 3-4 saat sonra kanda troponin açığa çıkar
- ✓ Kan konsantrasyonları 10-14 gün boyunca yüksek seyreder

Braunwald E. Circulation 2002;106:1893-1900.

- kardiyak troponin-T(cTn-T) için tek bir ölçüm kiti bulunur
- kardiyak troponin- I (cTn-I) için birden fazla ölçüm kiti vardır

ölçümler arasında standardizasyon Ø

Farklı ölçüm kitlerine ait cTn-I sınır (cut-off) değerleri değişmektedir.

Böbrek yetmezliği, sepsis, yanık troponin seviyelerini yükselten non-kardiyak etiyolojik sebeplerdir

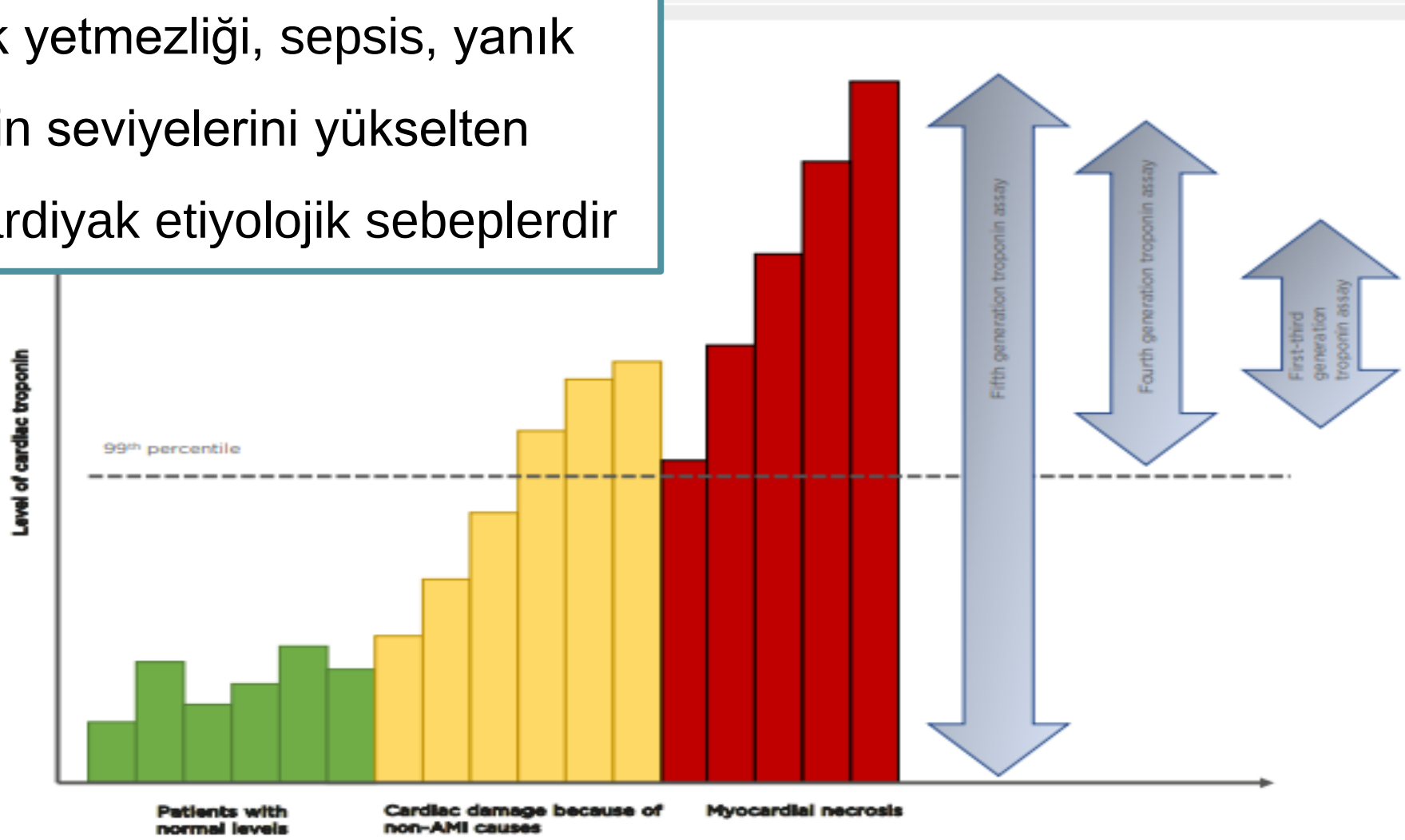


Figure 1: Improving sensitivities of troponin assays.

There is a background, normal, turnover of cardiac troponin (green bars). With the onset of AMI there is a rise in cardiac troponin that represents either ischemia-induced release of cytosolic troponin or micro-necrosis (orange bars). Between 2 and 6 hours there is a steep increase in cardiac troponin representing myocardial necrosis (red bars).

Benedict McD. EMJ Cardiol. 2020;8:97-106.

Bu hastalarda rutin biyobelirteç taraması yapılmalı mı?

Kardiyak biyobelirteçler?

GFR artar,

Na reabsorpsiyonu azalır

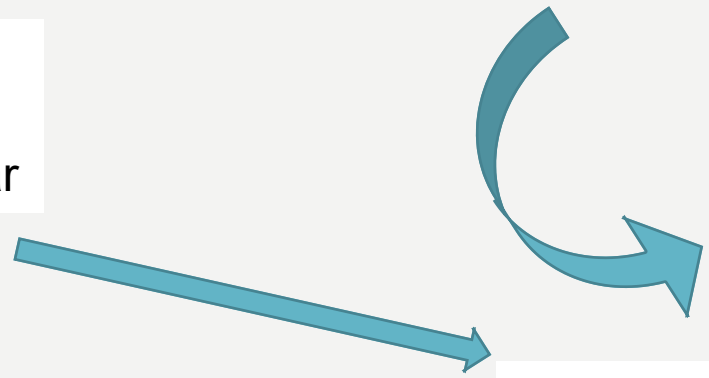
Sonuç

natriürez ve diürez

B-tip natriüretik peptidler (BNP'ler)

sol atriyum miyokardiyal gerilme

iskemi
inflamasyon
nöroendokrin uyarılar



sistemik dolaşıma katılırlar

karşı salınır

Struthers A. Eur Heart J.2007;28:1678-1682.
Clerico A. Am J Physiol Heart Circ Physiol. 2011;301:H12-H20.

Pro-BNP → aktif BNP ve inaktif NTpro-BNP

preop BNP, N terminal fragment BNP

+

postop BNP ve NT-proBNP değerleri

→ ölüm ve non fatal MI

(tahmininde faydalı)

(non-kardiyak cerrahi sonrası 30. ve 180. günde)

BNP testlerinde %50 ye varan varyasyonlar

dikkat !

Struthers A. Eur Heart J.2007;28:1678-1682.
Clerico A. Am J Physiol Heart Circ Physiol. 2011;301:H12-H20.

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doi:10.1016/S0735-1097(03)01069-6

Association of Cardiac Troponin, CK-MB, and Postoperative Myocardial Ischemia With Long-Term Survival After Major Vascular Surgery

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Jerusalem, Israel

OBJECTIVES The aim of this study was to determine the long-term prognosis with postoperative markers of myocardial ischemia and infarction.

BACKGROUND Cardiac troponins (cTn) are superior to creatine kinase-MB fraction (CK-MB) in detecting perioperative myocardial infarction (PMI). However, their threshold levels signifying PMI and their long-term prognostic value are not yet determined.

METHODS A cohort of 447 consecutive patients who underwent 501 major vascular procedures was prospectively studied. Perioperative continuous 12-lead electrocardiogram monitoring, cardiac troponin-I (cTn-I) and/or cardiac troponin-T (cTn-T), and CK-MB levels on the first three postoperative days, and long-term survival were determined. The association of different cutoff levels of CK-MB, troponin, and ischemia duration with long-term survival was investigated.

RESULTS Between 14 (2.9%) and 107 (23.9%) of the patients sustained PMI, depending on the biochemical criteria used. Elevated postoperative CK-MB, cTn, and prolonged (>30 min) ischemia, at all cutoff levels examined, predicted long-term mortality independent of the preoperative predictors: patient's age, type of vascular surgery, previous myocardial infarction, and renal failure (Cox multivariate analysis). Both CK-MB >10% and cTn-I >1.5 ng/ml and/or cTn-T >0.1 ng/ml independently predicted a 3.75-fold and 2.06-fold increase in long-term mortality ($p = 0.006$ and 0.012, respectively). Similarly, both CK-MB >5% and cTn-I >0.6 ng/ml and/or cTn-T >0.03 ng/ml independently predicted a 2.15-fold and

CONCLUSIONS Postoperative CK-MB and troponin, even at low cutoff levels, are independent and complementary predictors of long-term mortality after major vascular surgery. (J Am Coll Cardiol 2003;42:1547–54) © 2003 by the American College of Cardiology Foundation

ESA Kılavuzları

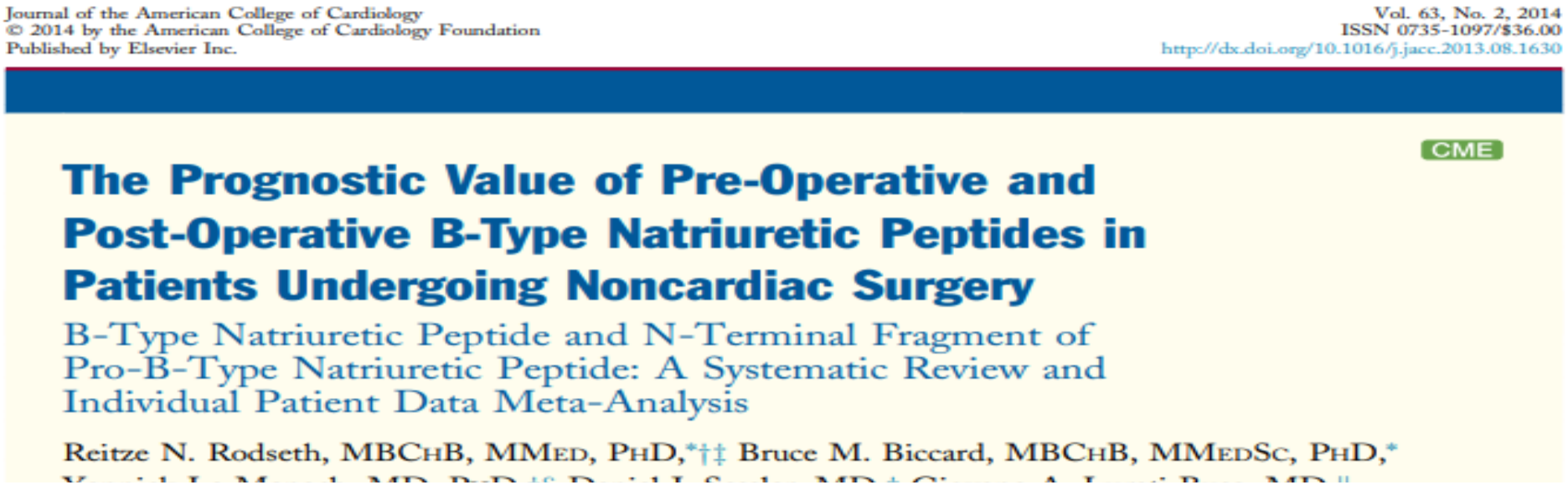
- ✓ preoperatif natriüretik peptidleri
  - ° yüksek riskli hastalarda → kanıt 2C

    (major genel ve ortopedik cerrahi)

  - ° orta ve yüksek riskli hastalarda → kanıt 1C

    (vasküler veya majör torasik cerrahi)

De Hert S. Eur J Anaesthesiol 2018;35:407-465.



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http://dx.doi.org/10.1016/j.jacc.2013.08.1630

CME

The Prognostic Value of Pre-Operative and Post-Operative B-Type Natriuretic Peptides in Patients Undergoing Noncardiac Surgery

B-Type Natriuretic Peptide and N-Terminal Fragment of Pro-B-Type Natriuretic Peptide: A Systematic Review and Individual Patient Data Meta-Analysis

Reitze N. Rodseth, MBChB, MMed, PhD,*†‡ Bruce M. Biccard, MBChB, MMedSc, PhD,*

Preop risk belirlemede araç

Preop bazal değer-postop değer: miyokard iskemi monitorizasyonu

rutin troponin ölçümü

° cost-effectif

° MINS veya postoperatif MI belirlemede güvenilir başka yol yok

"troponin yüksekliği saptandığında" EKG'si çekilen

MINS hastalarının %35'nde EKG'de iskemik bulgular (+)

Daha yüksek oranlarda hastalarda iskemi olduğu düşünülmekte

ama

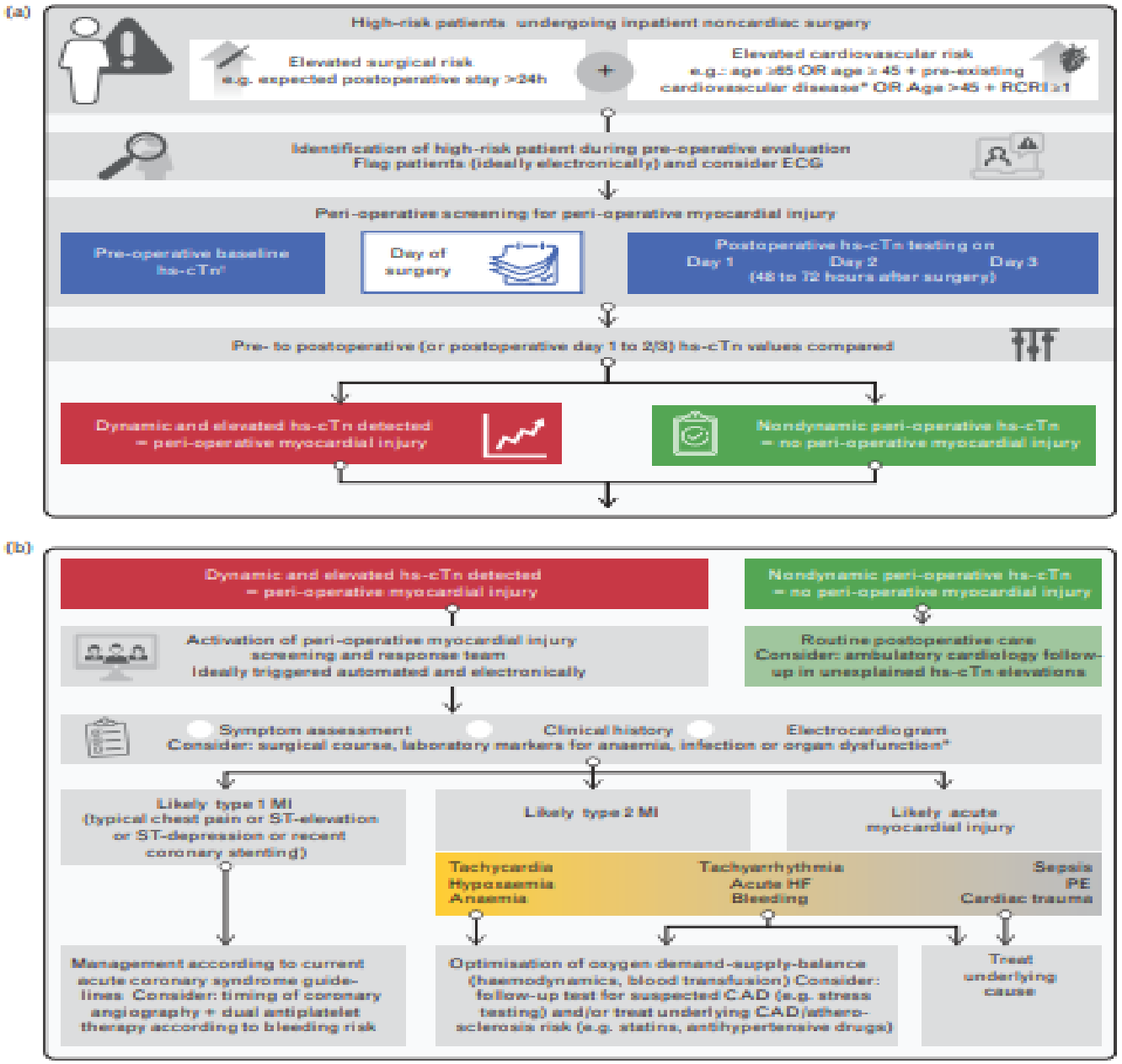
anlaşılamamaktadır

çünkü

- EKG çektirecek kadar iskemik semptom yaşamayabilirler
- Çoğu EKG, troponin yükseldikten sonra çekilir
- Troponin yüksekliği iskemik olay başladıktan saatler sonra gelişir

Devereaux PJ. JAMA 2012;307:2295-2304.

Fig. 1 Example of a potential peri-operative cTn screening and care programme



Puelacher C. Eur J Anaesthesiol 2021;38:600-608.

Bu hastalarda rutin Troponin taraması yapılmalı mı?

- ✓ 2017 Canadian Cardiology Society (CCS) kılavuzuna göre;

orta düzeyde kardiyovasküler riske sahip hastalarda

2-3 gün süre ile günlük troponin ölçümü yapılmalıdır

Duceppe E. Can J Cardiol 2017;33:17-32.

Bu yaklaşım ile MINS hastalarının < %95 belirlenir

Sessler DJ.Anesth Analg 2016;123:359-360.

Bu hastalarda rutin Troponin taraması yapılmalı mı?

- ✓ 4. Evrensel MI Tanımına göre;

yüksek riskli cerrahi hastalarda postoperatif troponin izlenmesi önerilmektedir

Thygesen K.Circulation 2018;138:e618-e651.

Bu hastalarda rutin Troponin taraması yapılmalı mı?

- 45-64 yaş, en az 1 kardiyak risk faktörü olan non-kardiyak cerrahi hastalarında ve
- ≥ 65 y tüm cerrahi hastalarda

Troponin ölçümü faydalı, basit ve pratik yaklaşımdır

Ruetzler K. Anesth Analg 2020;131:173-186.

2018 ESA Kılavuzu:

✓ Non-kardiyak cerrahi geçirecek KAH hastasında risk belirlemede preop Tn ölçümü : 2C (zayıf tavsiye, düşük kanıt kalitesi)

De Hert. Eur J Anaesthesiol 2018;35:407-465.

2018 ESA Kılavuzu:

✓ Akut/kronik miyokard hasarı arasında ayrım yapmak için preop Tn ölçümü :2B (zayıf tavsiye, orta kalite)

Non-kardiyak cerrahide

eğer postop Tn monitorizasyonu planlanıyorsa preop Tn ölçümü tavsiye edilir

Fleisher LA. J Am Coll Cardiol 2014;64:e77-137.
Duceppe E. Can J Cardiol 2017;33:17-32.

Table 3. Overview of Recommendations on Preoperative Biomarker Measurement by Different Professional Societies

Reference	Society	Preoperative Natriuretic Peptide Measurement Recommended	Grade
Fleisher et al[28] (2014)	American Heart Association and American College of Cardiologist	None	...
Kristensen et al[29] (2014)	European Societies of Anesthesiology and of Cardiology	If ≤4 METS or RCRI >1+ major vascular surgery or RCRI >2	IIb recommendation ("may be considered"), level B (large nonrandomized studies)
Duceppe et al[32] (2017)	Canadian Cardiology Society	In-patient noncardiac surgery in patients ≥65 y or ≥45 y and Hx of cardiovascular disease or RCRI >1	Strong recommendation; moderate-quality evidence
De Hert et al[31] (2018)	European Society of Anesthesiology	High-risk patients and vascular or thoracic surgery	Strong recommendation; low-quality evidence
		High-risk patients and major general or orthopedic surgery	Weak recommendation; low-quality evidence
Fleisher et al[28] (2014)	American Heart Association and American College of Cardiologist	...	...
Kristensen et al[29] (2014)	European Societies of Anesthesiology and of Cardiology	...	...
Duceppe et al[32] (2017)	Canadian Cardiology Society	...	...
De Hert et al[31] (2018)	European Society of Anesthesiology	As risk prediction tool before major surgery if Hx of CAD	Weak recommendation; low-quality evidence
		As baseline in case of routine postoperative troponin measurement	Weak recommendation; moderate quality evidence

Abbreviations: CAD, coronary artery disease; Hx, history; METS, metabolic equivalent; RCRI, Revised Cardiac Risk Index.

De Hert S. Anesth Analg. 2020;131:187-195.

MINS,

- ✓ miyokard iskemisi ve MI'a kıyasla patofizyolojide farklılık gösterir.

bu nedenle bilinen tedaviler MINS'a kolayca uygulanmaz

EJA

OPEN

Eur J Anaesthesiol 2021; **38**:600–608

REVIEW ARTICLE

Expert consensus on peri-operative myocardial injury screening in noncardiac surgery

A literature review

Christian Puelacher, Bernardo Bollen Pinto, Nicholas L. Mills, Emmanuelle Duceppe, Ekaterine Popova, Andreas Duma, Peter Nagele, Torbjørn Omland, Angelika Hammerer-Lercher and Giovanna Lurati Buse

Peri-operative myocardial injury, detected by dynamic and elevated cardiac troponin (cTn) concentrations, is a common complication of noncardiac surgery that is strongly associated with 30-day mortality. Although active screening for peri-operative myocardial injury has been suggested in recent guidelines, clinical implementation remains tentative due to a lack of examples on how to tackle such an interdisciplinary project at a local level. Moreover, consensus on which assay and cTn cut-off values should be used has not yet been reached, and guidance on whom to screen is lacking. In this article, we aim to summarise local examples of successfully implemented cTn screening practices and review the current literature in order to provide information and suggestions for patient selection, organisation of a screening programme, caveats and a potential management pathway.

Published online 1 March 2021

Table 2 Summary of studies of myocardial injury associated with noncardiac surgery

Study	Patient population[a]	Patients (n)	Definition of myocardial injury	Incidence of event (%)	Cut-off	Timing of cTn measurements	Key data
hs-cTnT assay (Roche Diagnostics)							
VISION 2017[10]	Aged ≥45 years staying at least one night in-hospital after surgery	21 842	MINS	17.9	Absolute postoperative hs-cTnT of >20 ng l^{-1} combined with an absolute change of ≥5 ng l^{-1}, excluding extra-cardiac events	Pre-operative: 1 to 28 days before surgery (available in 40.4% of patients) Postoperative: 6 to 12 h postop and on days 1 to 3	Crude 30-day mortality: 4.1% (HR = 3.10; 95% CI: 2.39 to 4.02)
BASEL-PMI study 2018[6] (see also Lism *et al.*[17] and Mol *et al.*[18])	High-risk patients (aged ≥65 years of age, OR ≥45 years with history of CAD, peripheral artery disease or stroke)	2018	Peri-operative myocardial injury (PMI) Cardiac PMI Extra-cardiac PMI	15.8 13.4 2.2	Absolute increase of ≥14 ng l^{-1} hs-cTnT above pre-operative value within 7 days postsurgery	Pre-operative: Within 30 days before surgery Postoperative: Days 1 and 2	Crude 30-day mortality: 9.8% (HR = 2.73; 95% CI: 1.54 to 4.84) Crude 1-year mortality: 22.5% (HR = 1.58; 95% CI 1.16 to 2.15)
Gillmann *et al.* 2014[30]	Aged ≥18 years undergoing elective open aortic, peripheral vascular or carotid surgery	455	MACE	9.0	Absolute change of ≥6 ng l^{-1} hs-cTnT	Pre-operative: N/A Postoperative: 24 h postop	Increased risk of MACE independently associated with history of renal failure (OR = 3.4; 95% CI 1.4 to 8.1; *P* = 0.006) and heart failure (OR = 2.8; 95% CI 1.3 to 5.7; *P* = 0.008)
Fourth-generation Elecsys cTnT assay (Roche Diagnostics)							
VISION 2014[46] (see also VISION 2012[9])	Aged ≥45 years staying at least one night in-hospital after surgery	15 065	MINS	8.0	Peak conventional cTnT ≥0.04 µg l^{-1} postoperatively with an ischaemic feature	Pre-operative: N/A Postoperative: 6 to 12 h postop and on days 1 to 3	MINS was an independent predictor of 30-day mortality (HR = 3.87; 95% CI 2.98 to 5.08)
Dimension RxL cTnI assay (Siemens Healthcare)							
Nagele P *et al.* 2011[19]	Patients undergoing head and neck cancer surgery	378	Peri-operative myocardial injury	15.1	Peak conventional cTnI ≥0.07 µg l^{-1} postoperatively	Pre-operative: N/A Postoperative: Immediately after surgery, 8 h and 16 h after surgery	8-fold increase in mortality 60 days postop (OR = 8.01; 95% CI 2.03 to 31.56)
AccuTnI assay (Beckman Coulter)							
van Waes *et al.*[12]	Intermediate- to high-risk patients aged ≥60 years	1627	Postoperative myocardial injury	19.4	Peak conventional cTnI >0.06 µg l^{-1} postoperatively	Pre-operative: N/A Postoperative: Days 1 to 3	Crude 30-day mortality: 8.6%; 95% CI 6.0 to 12.2%
Architect$_{STAT}$ TnI assay (Abbott Diagnostics)							
Chong *et al.*[15]	Aged ≥60 years	102	Postoperative myocardial injury	52.9	Peak conventional cTnI >0.03 µg l^{-1} postoperatively	Pre-operative: Before surgery (unspecified) Postoperative: Days 1 to 3	Crude 1-year mortality: 37.0%
Architect$_{STAT}$ hs-TnI assay (Abbott Diagnostics)							
Gillies *et al.*[16]	Aged ≥65 years or aged 50 to 64 years with one or more of the following: nonelective surgery, acute or chronic renal impairment, diabetes mellitus or presence of a risk factor for cardiac or respiratory disease.	288	Peri-operative myocardial injury	Intervention group: 46.0 Usual care: 48.0	Peak hs-cTnI ≥26 ng l^{-1} postoperatively	Pre-operative: Induction of anaesthesia Postoperative: 24 and 72 h after surgery	Peak postoperative cTnI concentrations similar between intervention (haemodynamic therapy) and usual care groups
VISION (biobank cohort) Duceppe *et al.*[51]	Aged ≥45 years staying at least one night in-hospital after surgery	3953	MINS	6.1	Peak hs-cTnI ≥60 ng l^{-1} postoperatively	Pre-operative: N/A Postoperative: 6 to 12 h postop and on days 1 to 3	Overall incidence of MACE at 30 days was 1.7% (66/3947) Postoperative hs-cTnI ≥60 ng l^{-1} without clinical features of myocardial ischaemia was associated with six-fold increase in risk of subsequent major cardiac events at 30 days (adjusted HR 5.90; 95% CI 2.67 to 11.72)

Presented cut-offs were not validated in large external cohorts. CAD, coronary artery disease; CI, confidence interval; cTnI/cTnT, cardiac troponin I/T; EF, ejection fraction; HR, hazard ratio; hs, high-sensitivity; MACE, major adverse cardiac event; MINS, myocardial injury after noncardiac surgery; N/A, not applicable; PMI, peri-operative myocardial injury. [a]All patients were undergoing noncardiac surgery.

MINS tanısında zorluk nedir?

- ✓ MINS'lı hastaların sadece % 20'si 4. Evrensel MI Tanımı kriterlerine uyar,
- ✓ Ama MINS'ın sebep olduğu mortalite MI için olan kadar yüksektir

Levy M. Anesthesiology 2011;114:796-806.

VISION

(Vascular Events In-noncardiac Surgery patients cOhort evaluatioN)

- Geniş (15065 hasta)
- Uluslararası (8 ülke-12 merkez)
- Prospektif
- Cohort

Non-kardiyak cerrahi sonrası komplikasyonları değerlendirir

ORIGINAL CONTRIBUTION

Devereaux PJ. JAMA 2012; 307:2295–304.

Association Between Postoperative Troponin Levels and 30-Day Mortality Among Patients Undergoing Noncardiac Surgery

The Vascular Events In Noncardiac Surgery Patients Cohort Evaluation (VISION) Study Investigators

Context Of the 200 million adults worldwide who undergo noncardiac surgery each year, more than 1 million will die within 30 days.

Objective To determine the relationship between the peak fourth-generation troponin T (TnT) measurement in the first 3 days after noncardiac surgery and 30-day mortality.

Design, Setting, and Participants A prospective, international cohort study that enrolled patients from August 6, 2007, to January 11, 2011. Eligible patients were aged 45 years and older and required at least an overnight hospital admission after having noncardiac surgery.

Main Outcome Measures Patients' TnT levels were measured 6 to 12 hours after surgery and on days 1, 2, and 3 after surgery. We undertook Cox regression analysis in which the dependent variable was mortality until 30 days after surgery, and the independent variables included 24 preoperative variables. We repeated this analysis, adding the peak TnT measurement during the first 3 postoperative days as an independent variable and used a minimum *P* value approach to determine if there were TnT thresholds that independently altered patients' risk of death

WORLDWIDE, MORE THAN 200 million adults have major noncardiac surgery annually.[1,2] Despite benefits associated with surgery, major perioperative complications, including death, occur.[3] More than 1 million adults worldwide will die within 30 days of noncardiac surgery each year.[1,2]

Perioperative risk estimation identi-

non-kardiyak cerrahi sonrası ilk 3 günde ölçülen peak postoperatif TnT (4. jenerasyon) değerleri 30 günlük mortalite ile –anlamlı- ilişkilidir

JAMA | Original Investigation

Association of Postoperative High-Sensitivity Troponin Levels With Myocardial Injury and 30-Day Mortality Among Patients Undergoing Noncardiac Surgery

Writing Committee for the VISION Study Investigators

IMPORTANCE Little is known about the relationship between perioperative high-sensitivity troponin T (hsTnT) measurements and 30-day mortality and myocardial injury after noncardiac surgery (MINS).

OBJECTIVE To determine the association between perioperative hsTnT measurements and 30-day mortality and potential diagnostic criteria for MINS (ie, myocardial injury due to ischemia associated with 30-day mortality).

DESIGN, SETTING, AND PARTICIPANTS Prospective cohort study of patients aged 45 years or older who underwent inpatient noncardiac surgery and had a postoperative hsTnT measurement. Starting in October 2008, participants were recruited at 23 centers in 13 countries; follow-up finished in December 2013.

EXPOSURES Patients had hsTnT measurements 6 to 12 hours after surgery and daily for 3 days; 40.4% had a preoperative hsTnT measurement.

MAIN OUTCOMES AND MEASURES A modified Mazumdar approach (an iterative process) was used to determine if there were hsTnT thresholds associated with risk of death and had an adjusted hazard ratio (HR) of 3.0 or higher and a risk of 30-day mortality of 3% or higher. To determine potential diagnostic criteria for MINS, regression analyses ascertained if postoperative hsTnT elevations required an ischemic feature (eg, ischemic symptom or electrocardiography finding) to be associated with 30-day mortality.

Supplemental content

VISION "2. fazında" 21842 hasta, >45 y, non-kardiyak cerrahi

Fark ne? Postop high-sensitivity troponin (hsTnT)

Hastaların % 40.4'nde preop hsTnT

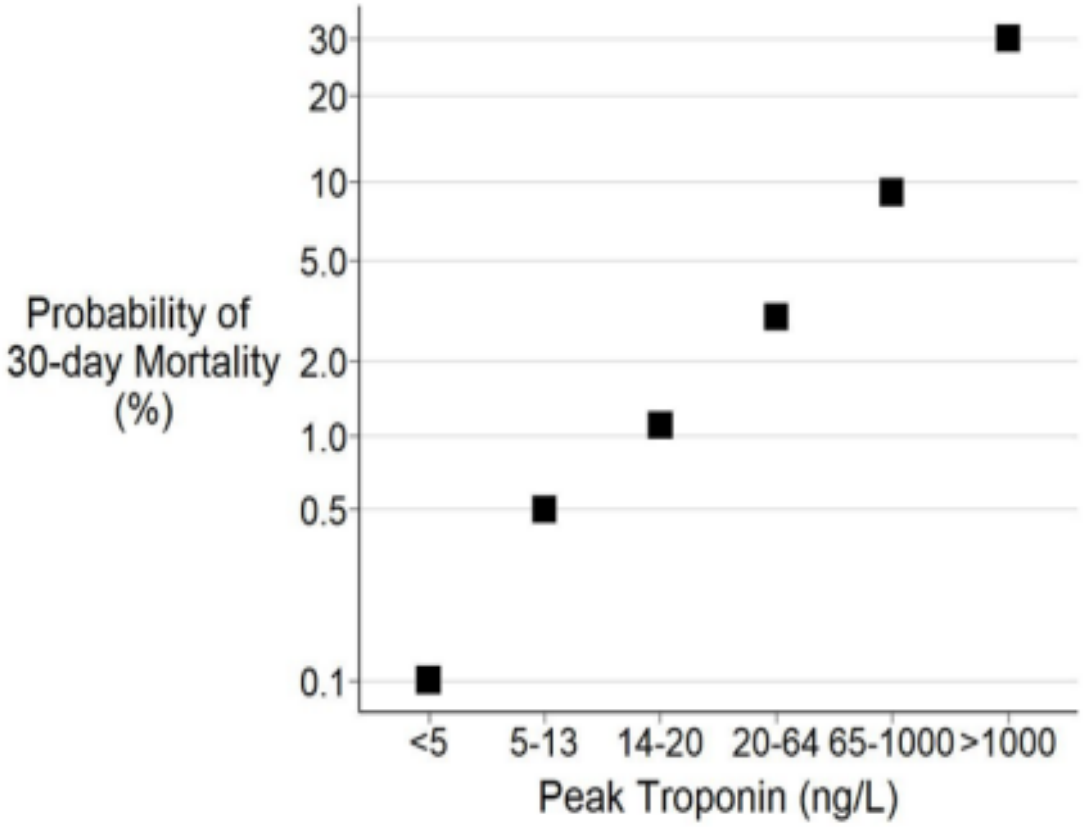


Figure 1. Thirty-day mortality as a function of postoperative peak high-sensitivity troponin T. Mortality increases markedly from 0.1% at a troponin T concentration <5 ng/L to 30% mortality when troponin T exceeds 1000 ng/L. Adapted with permission from the Writing Committee for the Vision Study Investigators, Devereaux PJ, Biccard BM, et al, "Association of Postoperative High-Sensitivity Troponin Levels With Myocardial Injury and 30-day Mortality Among Patients Undergoing Noncardiac Surgery." *JAMA*. 2017;317:1642–1651.[13]

VISION SONUÇ

Non-kardiyak cerrahiden sonraki ilk 3 gün boyunca hs TnT değerlerinin izlenmesi, preoperatif risk faktörleriyle sınırlı değerlendirmeye kıyasla 30 günlük mortalite riskini belirlemeyi önemli ölçüde iyileştirmektedir

Devereaux PJ. JAMA 2017;317:1642-1651.

sınırlı değerlendirme

Kardiak Risk Stratifikasyonu

Postop MINS rastlantı sonucu değildir:

- ✓ daha çok kardiyovasküler hastalığı olanlarda görülür
- ✓ cerrahinin tipi, süresi, büyüklüğü MINS'a katkıda bulunur ama asıl belirleyici;
- ✓ hastanın kendi riskidir!

Chamon GF. Anesthesiology 2018;128:109-116.

Kardiak Risk Stratifikasyonu

hastanın söylediği egzersiz toleransı ? kabaca dinçlik indeksi ?

"bu değerlendirme yetersizdir"

Klinikte kardiyak risk değerlendirme araçları (en sık 3 araç):

- ✓ Revize Kardiyak Risk İndeksi (RCRI)
- ✓ ACS NSQIP
- ✓ NSQIP MICA risk indeksi

3'ü de yetersiz !

MINS büyük oranda; koroner trombotik olayla tetiklenmez

O_2 ihtiyaç-sunum arasındaki dengesizlikten kaynaklanır

inatçı perioperatif hipotansiyon

ciddi anemi

taşiaritmi

Tedavi edilebilir

Hipotansiyon-taşikardi ve MINS ilişkisi

Ort Arter Basıncı (OAB) < 65 mm Hg ve

OAB'nin bazalinden % 30'luk relatif düşüş

~ miyokard hasarı

Van Waes JA. Anesthesiology 2016;124:35-44.

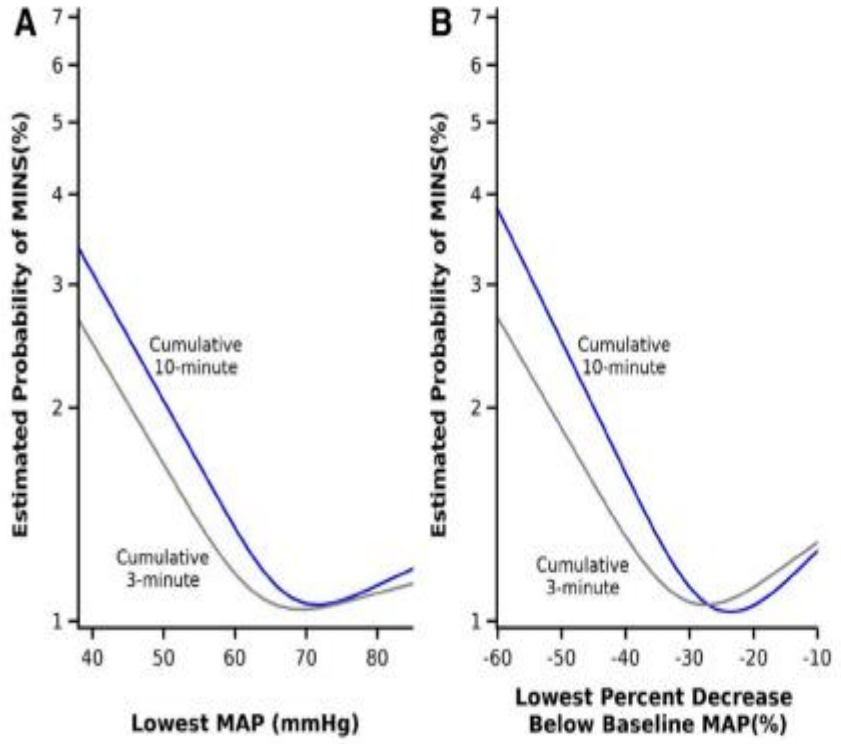


Figure 2. Lowest MAP thresholds for MINS. The left graph shows the relationship between the lowest cumulative absolute MAP maintained for 3 and 10 min and myocardial injury. The right graph shows the relationship between the lowest cumulative relative MAP maintained for 3 and 10 min and myocardial injury. Both graphs are multivariable logistic regression smoothed by restricted cubic spline with 3 degrees and knots at 10th, 50th, and 90th percentiles of given exposure variable. MAP indicates mean arterial pressure; MINS, myocardial injury after noncardiac surgery. Data were derived with permission from Salmasi V, Maheshwari K, Yang D, et al, "Relationship Between Intraoperative Hypotension, Defined by Either Reduction from Baseline or Absolute Thresholds, and Acute Kidney and Myocardial Injury After Noncardiac Surgery: A Retrospective Cohort Analysis." *Anesthesiology*. 2017;126:47–65. Available at: https://anesthesiology.pubs.asahq.org/article.aspx?articleid=2579833.[56]

Hipotansiyonun ciddiyeti ve süresi miyokard hasarı ve mortalite için anahtar belirleyicidir

Ruetzler K. Anesth Analg 2020;131:173-186.

Ama bu kontrol edilebilir bir faktör:

- tüm hipotansiyonun 1/3'ü indüksiyon sonrasında olur ve aslında önlenebilir
- sürekli invazif arter monitorizasyonu yapmak hipotansiyon epizotlarını önler

Maheshwari K. Anaesthesia 2018;73:1223-1228.

Taşikardi ile

miyokardın O_2 tüketimi artar

diastolik dolum zamanı azalır

Kronik taşikardi

non-operatif MI'a katkıda bulunabilir

MINS gelişimine katkıda bulunur

Ladha KS. Br J Anaesth 2018121722-729.

Postoperatif hipotansiyon ve taşikardi

MI ve MINS çoğunluğu postop ilk 48 saat içinde gelişiyor,

Vital bulgular postoperatif cerrahi kliniklerde nadiren ölçülür

(4- 8 saatte bir)

Bu nedenle hipotansiyon uzun süreli olabilir

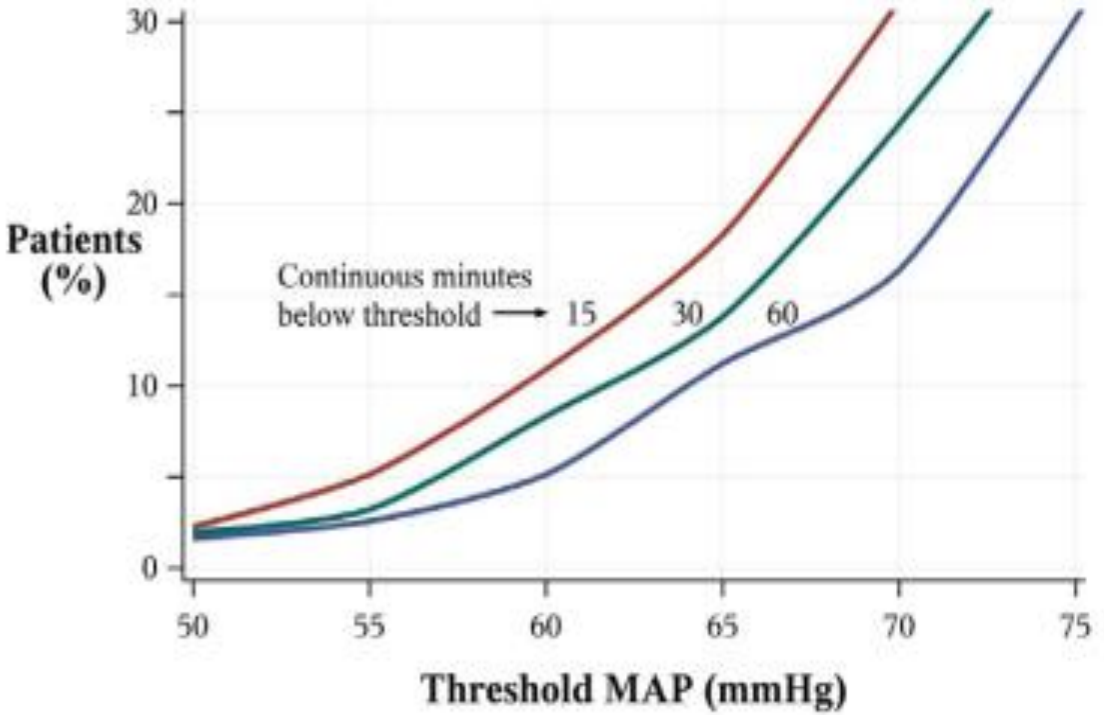
Postoperatif hipotansiyon ve taşikardi

- ✓ OAB <70 mmHg en az 30 dk olan hasta oranı % 24;
- ✓ OAB < 65 mmHg en az 15 dk olan hasta oranı % 14

Ama 4 saatlik takiplerde bu hastalardaki TA düşüklüğü % 70 oranında olduğu halde elektronik kayıtlarda hiç bahsedilmemiş

Turan A. Anesthesiology 2019;130:550-559.

Figure 3. Continuous hypotensive episodes of various durations under various thresholds. For each patient, the total time of the observed longest continuous hypotensive episode (ie, no gap) with MAP readings below various thresholds was computed. The percentage of patients with at least that many minutes below the threshold is plotted. For example, the green line shows that 24% of patients had a continuous episode of MAP <70 mm Hg lasting at least 30 min. MAP indicates mean arterial pressure. Data were derived with permission from Turan A, Chang C, Cohen B, et al. "Incidence, Severity, and Detection of Blood Pressure Perturbations After Abdominal Surgery: A Prospective Blinded Observational Study," *Anesthesiology*. 2019;130:550–559. Available at: https://anesthesiology.pubs.asahq.org/article.aspx?articleid=2723777.[70]



önceki yıllarda ° hasta yaşının ileri olmaması

° hastaların postop hastanede kalma süresinin ↗

uzun aralıklı vital bulgu takibi yapılmıştır

Çözüm; kontinü hemodinamik monitorizasyon

Association Between Mean Arterial Pressure and Acute Kidney Injury and a Composite of Myocardial Injury and Mortality in Postoperative Critically Ill Patients: A Retrospective Cohort Analysis

Ashish K. Khanna, MD, FCCP, FCCM[1,2]; Kamal Maheshwari, MD, MPH[3]; Guangmei Mao, MS[3,4]; Liu Liu, MS[3,4]; Silvia E. Perez-Protto, MD[3]; Praneeta Chodavarapu, MD[3]; Yehoshua N. Schacham, MD[3]; Daniel I. Sessler, MD[3]

Objectives: Hypotension thresholds that provoke renal injury, myocardial injury, and mortality in critical care patients remain unknown. We primarily sought to determine the relationship between hypotension and a composite of myocardial injury (troponin T ≥ 0.03 ng/mL without nonischemic cause) and death up to 7 postoperative days. Secondarily, we considered acute kidney injury (creatinine concentration ≥ 0.3 mg/dL or 1.5 times baseline).
Design: Retrospective cohort.
Setting: Surgical ICU at an academic medical center.
Patients: Two-thousand eight-hundred thirty-three postoperative patients admitted to the surgical ICU.
Interventions: A Cox proportional hazard survival model was used to assess the association between lowest mean arterial pressure on each intensive care day, considered as a time-varying covariate, and outcomes. In sensitivity analyses hypotension defined as pressures less than 80 mm Hg and 70 mm Hg were also considered.
Measurements and Main Results: There was a strong nonlinear (quadratic) association between the lowest mean arterial pressure and the primary outcome of myocardial injury after noncardiac surgery or mortality, with estimated risk increasing at lower pressures. The risk of myocardial injury after noncardiac surgery or mortality was an estimated 23% higher at the 25th percentile (78 mm Hg) of lowest mean arterial pressure compared with at the median of 87 mm Hg, with adjusted hazard ratio (95% CI) of 1.23 (1.12–1.355; $p < 0.001$). Overall results were generally similar in sensitivity analyses based on every hour of mean arterial pressure less than 80 mm Hg and any mean arterial pressure less than 70 mm Hg. Post hoc analyses showed that the relationship between ICU hypotension and outcomes depended on the amount of intraoperative hypotension. The risk of acute kidney injury increased over a range of minimum daily pressures from 110 mm Hg to 50 mm Hg, with an adjusted hazard ratio of 1.27 (95% CI, 1.18–1.37; $p < 0.001$).
Conclusions: Increasing amounts of hypotension (defined by lowest mean arterial pressures per day) were strongly associated with myocardial injury, mortality, and renal injury in postoperative critical care patients. (*Crit Care Med* 2019; 47:910–917)
Key Words: hypotension; intensive care; mean arterial pressure; outcomes; postoperative; surgical

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This work was performed by the Center for Critical Care and the Departments of General Anesthesiology & Outcomes Research at the Anesthesiology Institute, Cleveland Clinic, 9500 Euclid Avenue, G-58, Cleveland, OH.
Supplemental digital content is available for this article. Direct URL citations appear in the printed text and are provided in the HTML and PDF versions of this article on the journal's website (http://journals.lww.com/ccmjournal).
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Intensive Care Med (2018) 44:857–867
https://doi.org/10.1007/s00134-018-5218-5

ORIGINAL

The relationship between ICU hypotension and in-hospital mortality and morbidity in septic patients

Kamal Maheshwari[1,7*], Brian H. Nathanson[2], Sibyl H. Munson[3], Victor Khangulov[3], Mitali Stevens[4], Hussain Badani[3], Ashish K. Khanna[5] and Daniel I. Sessler[6]

Abstract

Purpose: Current guidelines recommend maintaining a mean arterial pressure (MAP) ≥ 65 mmHg in septic patients. However, the relationship between hypotension and major complications in septic patients remains unclear. We, therefore, evaluated associations of MAPs below various thresholds and in-hospital mortality, acute kidney injury (AKI), and myocardial injury.

Methods: We conducted a retrospective analysis using electronic health records from 110 US hospitals. We evaluated septic adults with intensive care unit (ICU) stays ≥ 24 h from 2010 to 2016. Patients were excluded with inadequate blood pressure recordings, poorly documented potential confounding factors, or renal or myocardial histories documented within 6 months of ICU admission. Hypotension exposure was defined by time-weighted average mean arterial pressure (TWA-MAP) and cumulative time below 55, 65, 75, and 85 mmHg thresholds. Multivariable logistic regressions determined the associations between hypotension exposure and in-hospital mortality, AKI, and myocardial injury.

Results: In total, 8,782 patients met study criteria. For every one unit increase in TWA-MAP < 65 mmHg, the odds of in-hospital mortality increased 11.4% (95% CI 7.8%, 15.1%, $p < 0.001$); the odds of AKI increased 7.0% (4.7, 9.5%, $p < 0.001$); and the odds of myocardial injury increased 4.5% (0.4, 8.7%, $p = 0.03$). For mortality and AKI, odds progressively increased as thresholds decreased from 85 to 55 mmHg.

Conclusions: Risks for mortality, AKI, and myocardial injury were apparent at 85 mmHg, and for mortality and AKI risk progressively worsened at lower thresholds. Maintaining MAP well above 65 mmHg may be prudent in septic ICU patients.

Keywords: Sepsis, Hypotension, Blood pressure monitoring, Mortality, Acute kidney injury, Myocardial injury

OAB için alt sınır?
" 65 mmHg da yetersiz "

✓ plak rüptürü çok baskın mekanizma olmasa bile; çoğu hastada KAH mevcut!

Bu hastalar KAH'ı önlemek için alınan tedbirlerden fayda görecektir.

Dabigatran :110 mg 16 ay boyunca / günde 2 kez:

- vasküler ölüm, arteriyal ve venöz trombozu önler,
- majör kanamayı arttırmaz

Devereaux PJ.Lancet 20183912325-2334.

Dabigatran’a alternatif?

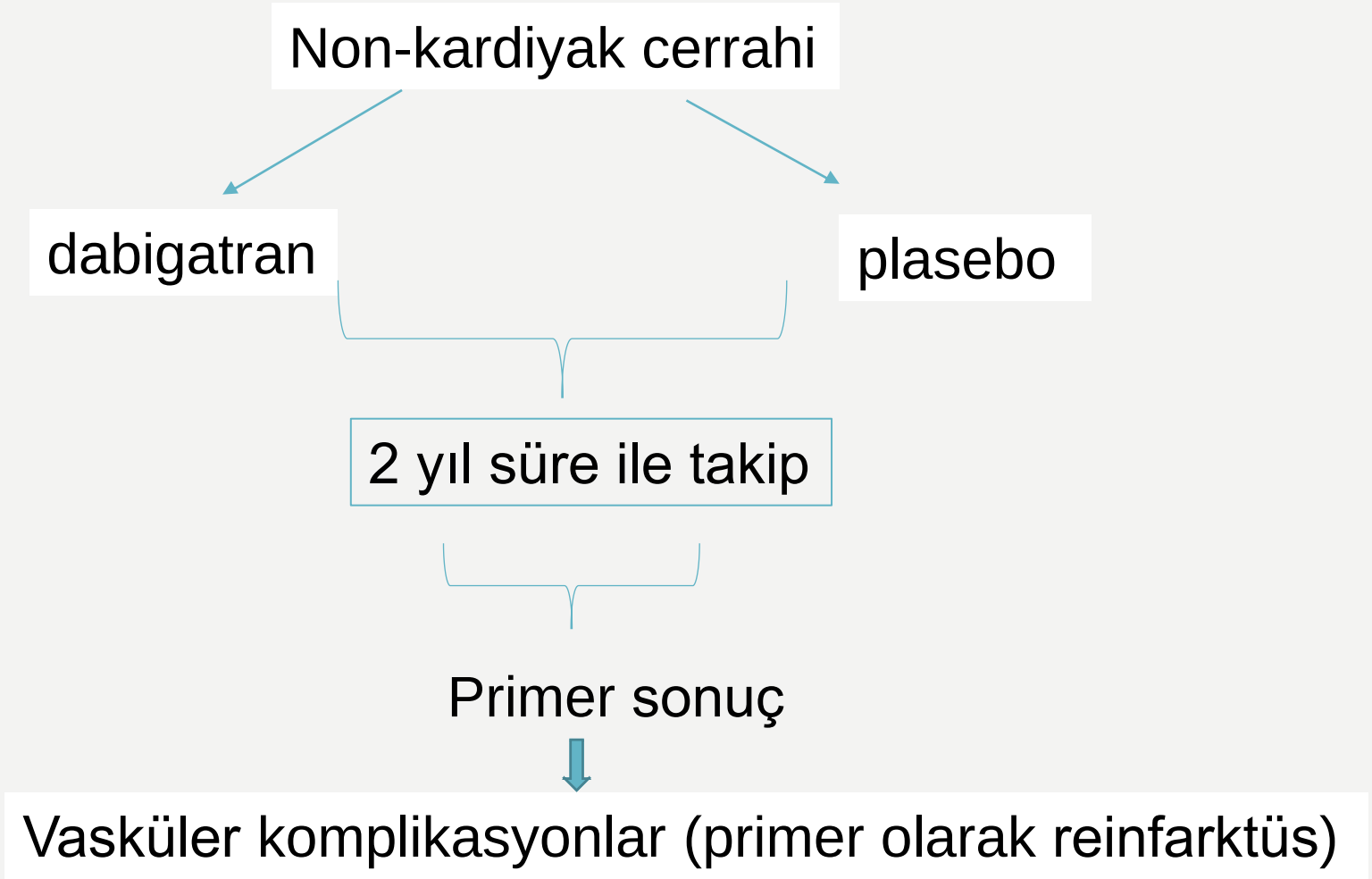
- ✓ Aspirin ve statin--- Kanada Kılavuzlarının önerisi

  Tedavi sonrası mortalitede azalma

Duceppe E. Can J Cardiol 2017;33:17-32.

MANAGE Trial

(Management of Myocardial Injury After Non-cardiac surgery)



Duceppe E. Can J Cardiol 2017;33:17-32.

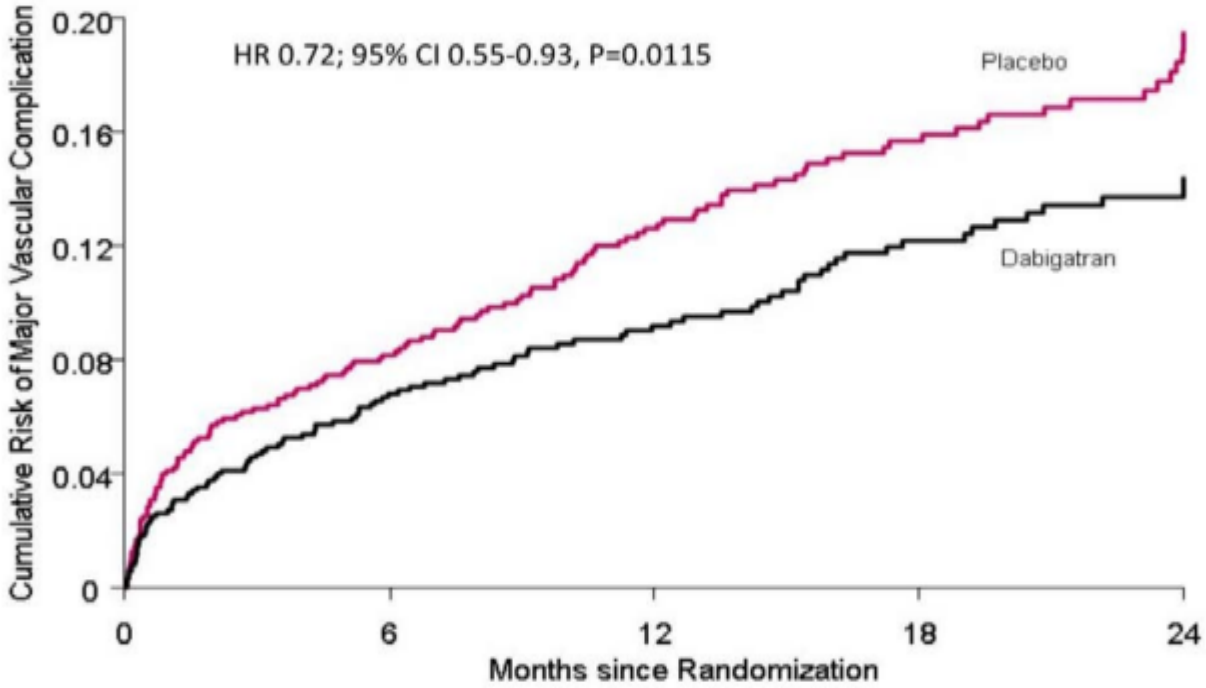


Figure 5. The MANAGE trial randomized 1754 patients who had MINS to dabigatran or placebo for up to 2 y. The primary outcome was a composite of vascular complications, primarily reinfarction. Dabigatran reduced the hazard ratio 28%, with a number-needed-to-treat of 24. Major bleeding was similar in each group. CI indicates confidence interval; HR, hazard ratio; MANAGE, Management of Myocardial Injury After Noncardiac Surgery trial; MINS, myocardial injury after noncardiac surgery. Adapted from *The Lancet*, 391, Devereaux PJ, Duceppe E, Guyatt G, et al, "Dabigatran in Patients with Myocardial Injury After Noncardiac Surgery (MANAGE): An International, Randomized, Placebo-Controlled Trial," 2325–2334, 2018, with permission from Elsevier.[95]

Nasıl önleyelim MINS'ı?

Yeterli yayın yok

3 büyük Trial var

Her 3 çalışmada da; MI primer sonuç

POISE 1: Perioperative Ischemic Evaluation Trial

POISE 2:

ENIGMA 2: Evaluation of Nitrous Oxide in the Gas Mixture of Anesthesia

Devereaux PJ. Lancet 2008;371:1839-1847.
Devereaux PJ. NEJM 2014;370:1494-1503.
Devereaux PJ. NEJM 2014;370:1504-1513.
Myles PS. Am Heart J 2009;157:488-494.e1.

ESC European Society of Cardiology

European Heart Journal (2020) **41**, 3083–3091
doi:10.1093/eurheartj/ehz301

CLINICAL REVIEW
Novel therapeutic concepts

Myocardial injury after non-cardiac surgery: diagnosis and management

P.J. Devereaux [1,2,3,4]* and Wojciech Szczeklik [5]

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Received 3 January 2019; revised 21 February 2019; editorial decision 27 March 2019; accepted 6 May 2019; online publish-ahead-of-print 16 May 2019

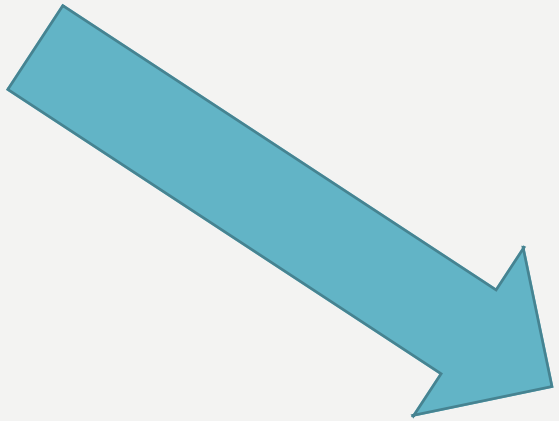
Myocardial injury after non-cardiac surgery (MINS) is due to myocardial ischaemia (i.e. supply-demand mismatch or thrombus) and is associated with an increased risk of mortality and major vascular complications at 30 days and up to 2 years after non-cardiac surgery. The diagnostic criteria for MINS includes an elevated post-operative troponin measurement judged as resulting from myocardial ischaemia (i.e. no evidence of a non-ischaemic aetiology), during or within 30 days after non-cardiac surgery, and without the requirement of an ischaemic feature (e.g. ischaemic symptom, ischaemic electrocardiography finding). For patients with MINS who are not at high risk of bleeding, physicians should consider initiating dabigatran 110 mg twice daily and low-dose aspirin. Physicians should also consider initiating statin therapy in patients with MINS. Most MINS patients should only be referred to cardiac catheterization if they demonstrate recurrent instability (e.g. cardiac ischaemia, heart failure). Patients ≥65 years of age or with known atherosclerotic disease should have troponin measurements on days 1, 2, and 3 after surgery while the patient is in hospital to avoid missing >90% of MINS and the opportunity to initiate secondary prophylactic measures and follow-up.

- ✓ Henüz bilinen güvenli profilaksi yok
- ✓ ß-blokerler belirgin düzeyde MI riskini düşürür ama inme ve mortalite riskini arttırır
- ✓ Nitroz Oksid'den kaçınmak kardiyovasküler olay riskini azaltmaz
- ✓ Klonidin ve aspirin riski azaltmaz, aksine komplikasyonları vardır

- ✓ ACE inhibitörleri

  Anjiyotensin reseptör blokerleri

  Statinler



sekonder vasküler komplikasyonları azaltır

Beattie WS. Am Coll Cardiol 2017;70:907-908.

Geniş çalışmalar:

- MINS olan hastaların % 8'i ve
- Perioperatif MI geçirenlerin % 21'i

Koroner anjiyografi

Puelacher C. Circulation 2018;137:1221-1232.
Smilowitz NR. Eur Heart J 2017382409-2417.

- Rekürren instabilitesi olan MINS'lı hastalarda (kardiyak iskemi, kalp yetmezliği)

Kardiyak kateterizasyon

Devereaux PJ. Eur Heart J 2020;41:3083-3091.

PLOS ONE

RESEARCH ARTICLE

Intraoperative blood loss may be associated with myocardial injury after non-cardiac surgery

Jungchan Park[1☯], Ji-hye Kwon[1☯], Seung-Hwa Lee[2*], Jong Hwan Lee[1], Jeong Jin Min[1], Jihoon Kim[2], Ah Ran Oh[1], Wonho Seo[1], Cheol Won Hyeon[2], Kwangmo Yang[3], Jin-ho Choi[2,4], Sang-Chol Lee[2], Kyunga Kim[5,6], Joonghyun Ahn[5], Hyeon-Cheol Gwon[2]

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Check for updates

intraoperatif hemoglobin düzeyi için hesaplanan eşik değeri 9.9 g / dL

- yeterli intraoperatif hemoglobin seviyesinin korunması

⮑ MINS'ın önlenmesinde yararlı olabilir

Published in final edited form as:
Lancet Diabetes Endocrinol. 2018 October ; 6(10): 790–797. doi:10.1016/S2213-8587(18)30205-5.

Pre-operative Glucose Levels, Myocardial Injury and Death after Non-cardiac Surgery (The GlucoVISION Cohort Study)

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[c]Centro Cochrane Iberoamericano, Instituto de Investigación Biomédica (IIB Sant Pau-CIBERESP), Barcelona, Spain

[d]Department of Clinical Epidemiology and Public Health, HSCSP CIBER de Salud Mental (CIBERSAM), Barcelona, Spain

[e]Universidad Peruana Cayetano Heredia, Lima, Peru

Abstract

Interpretation: Pre-operative glucose, particularly casual glucose predicts risk for post-operative cardiovascular outcomes, especially among patients without diabetes.

Sırasıyla 125mg/L ve 115 mg/L'lik rastgele ve açlık glikoz eşikleri, diyabeti olmayan kişilerde ve 143 mg/L'lik rastgele bir glikoz eşiği, diyabetli kişilerde MINS'ın en önemli belirleyicisidir.

Volatile Agents for Cardiac Protection in N
Controlled S

Alberto Zangrillo, MD,* Valentina Testa, MD,* Valeri
Giuseppina Casiraghi, MD,* Francesca Cavenago, MD,* M
Giovanni Landoni

Objective: **Volatile anesthetics reduce the risk of myocardial infarction and mortality in coronary artery surgery. Recently, the American College of Cardiology/American Heart Association Guidelines suggested the use of volatile anesthetic agents for the maintenance of general anesthesia during noncardiac surgery in patients at risk for perioperative myocardial ischemia, but no randomized experience to document the cardioprotective effects of these agents exists in this setting. Therefore, the authors performed a prospective, randomized, controlled trial to compare the effects of sevoflurane versus total intravenous anesthesia, in terms of postoperative cardiac troponin I release in patients undergoing noncardiac surgery.**

Design: **A randomized, controlled trial.**

Setting: **A teaching hospital.**

Participants: **Eighty-eight consecutive patients undergoing noncardiac surgery.**

Interventions: **Patients were allocated randomly to receive either volatile anesthetic (44 patients) as the main anesthetic agent or total intravenous anesthesia (TIVA) (44 patients).**

KEY
tota
surg
dam

Coronary Heart Disease

Randomized Comparison of Sevoflurane Versus Propofol to Reduce Perioperative Myocardial Ischemia in Patients Undergoing Noncardiac Surgery

Giovanna A.L. Lurati Buse, MD; Philippe Schumacher, MD; Esther Seeberger, CCRN; Wolfgang Studer, MD; Regina M. Schuman, MD; Jens Fassl, MD; Jorge Kasper, MD; Miodrag Filipovic, MD; Daniel Bolliger, MD; Manfred D. Seeberger, MD

Background—Volatile anesthetics provide myocardial preconditioning in coronary surgery patients. We hypothesized that sevoflurane compared with propofol reduces the incidence of myocardial ischemia in patients undergoing major noncardiac surgery.

Methods and Results—We enrolled 385 patients at cardiovascular risk in 3 centers. Patients were randomized to maintenance of anesthesia with sevoflurane or propofol. We recorded continuous ECG for 48 hours perioperatively, measured troponin T and N-terminal prohormone of brain natriuretic peptide (NT-proBNP) on postoperative days 1 and 2, and evaluated postoperative delirium by the Confusion Assessment Method. At 6 and 12 months, we contacted patients by telephone to assess major adverse cardiac events. The primary end point was a composite of myocardial ischemia detected by continuous ECG and/or troponin elevation. Additional end points were postoperative NT-proBNP concentrations, major adverse cardiac events, and delirium. Patients and outcome assessors were blinded. We tested dichotomous end points by χ^2 test and NT-proBNP by Mann–Whitney test on an intention-to-treat basis. Myocardial ischemia occurred in 75 patients (40.8%) in the sevoflurane and 81 (40.3%) in the propofol group (relative risk, 1.01; 95% confidence interval, 0.78–1.30). NT-proBNP release did not differ across allocation on postoperative day 1 or 2. Within 12 months, 14 patients (7.6%) suffered a major adverse cardiac event after sevoflurane and 17 (8.5%) after propofol (relative risk, 0.90; 95% confidence interval, 0.44–1.83). The incidence of delirium did not differ (11.4% versus 14.4%; $P=0.379$).

Conclusions—Compared with propofol, sevoflurane did not reduce the incidence of myocardial ischemia in high-risk patients undergoing major noncardiac surgery. The sevoflurane and propofol groups did not differ in postoperative NT-proBNP release, major adverse cardiac events at 1 year, or delirium.

Clinical Trial Registration—URL: http://www.clinicaltrials.gov. Unique identifier: NCT00286585.

(*Circulation*. 2012;126:2696-2704.)

Key Words: acute coronary syndrome ■ cardiovascular events ■ preconditioning ■ volatile anesthetics

Mantıklı strateji

Preop risk belirlemede serum NP

Preop prognoz için serum troponin konsantrasyonu

Postop ilk 2 gün serum troponin konsantrasyonu

→ bakılması

kimlerde

- ✓ cerrahi yapılacak, > 45 yaş, en az 1 kardiyovasküler risk faktörü (+) ve
- ✓ > 65 yaş tüm cerrahiye alınacak hastalarda

Tarama yapılmaması veya taramaya rağmen bir şey yapılmaması bu hastaların tedavi şansını da azaltır

MINS'lı hastaların
* %3'ü 30 gün içinde ölebilir
* artmış mortalite 1 yıl boyunca devam eder

Rodseth RN. J Am Coll Cardiol 2011;58:522-529.
Devereaux PJ. JAMA 2017;317:1642-1651.

ameliyat sonrası ideal yönetim

- ✓ analjezi optimizasyonu
- ✓ hedef Hb değeri: >9 g / dl
- ✓ hedef OAB >65 mmHg
- ✓ hedef KAH < 100 atım/dk
- ✓ hedef SaO_2 > %95
- ✓ postop 1. ve 2. günlerde EKG
  - T dalgasında inversiyon,
  - ST segment depresyonu, yükselmesi

Simpson GC. SA Orthop J 2018;17:12-15.

Troponin tarama ile

- ✓ hastalar miyokard hasarı yaşadıklarını ve gelecek kalp krizleri için riskli grup olduklarını
- ✓ aspirin başlanması için aday olduklarını
- ✓ statin ve/veya ACE inhibitör tedaviye başlanacağını
- ✓ HT kontrolünün daha ciddi olacağını
- ✓ egzersiz, diyet, sigarasız hayat: yeni bir düzen !!!

Sessler D. Anesth Analg 2016;123:359-360.

Eğer bir gün benim sözlerim bilim ile ters düşerse bilimi seçin.

Mustafa Kemal Atatürk

BİTTİ...

Teşekkürler...