POINT OF CARE TECHNOLÓGIA

SÜRGŐSSÉGI ALAPELVEK ÚJRATÖLTVE

BERÉNYI TAMÁS – KANIZSAI PÉTER

Semmelweis Egyetem AITK Sürgősség

* Budapest *

„POINT OF CARE DIAGNOSTIC”

NAGYJÁBÓL **849 000 000** TALÁLAT

(0,39 MÁSODPERC)

TODAY'S PoCT DEFINITION

BÁRHOL, BÁRMIKOR, BÁRMELYIK BETEGHEZ RENDELTEN VÉGEZHETŐ, DÖNTÉST TÁMOGATÓ VIZSGÁLAT

Health Data Management, December 2003

Az Egészségügyi Minisztérium szakmai irányelve
A betegágy melletti (Point-of-Care, POC) laboratóriumi diagnosztika
fekvőbeteg intézmények sürgős és intenzív terápiás betegellátásában való alkalmazására

Készítette: *Az Orvosi laboratóriumi vizsgálatok Szakmai Kollégiuma*
és az Aneszteziológiai és Intenzív Terápiás Szakmai Kollégium

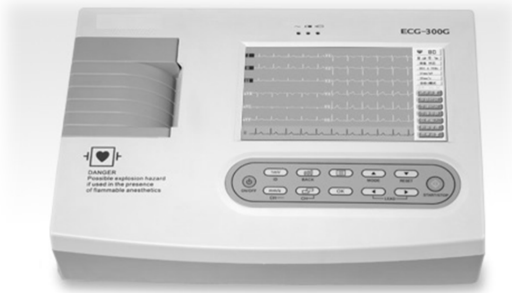
I.5. Az irányelv érvényessége és frissítési stratégiája

Felelős kiadója: Egészségügyi Minisztérium

A szakmai irányelv érvényessége: 2008. december 31.


Joycare


GE Healthcare
Vscan


ECG-300G
DANGER
Possible explosion hazard
if used in the presence
of flammable anesthetics


Eject
clearblue


hCG
Single Test Card


OMRON


Roche OMNI S

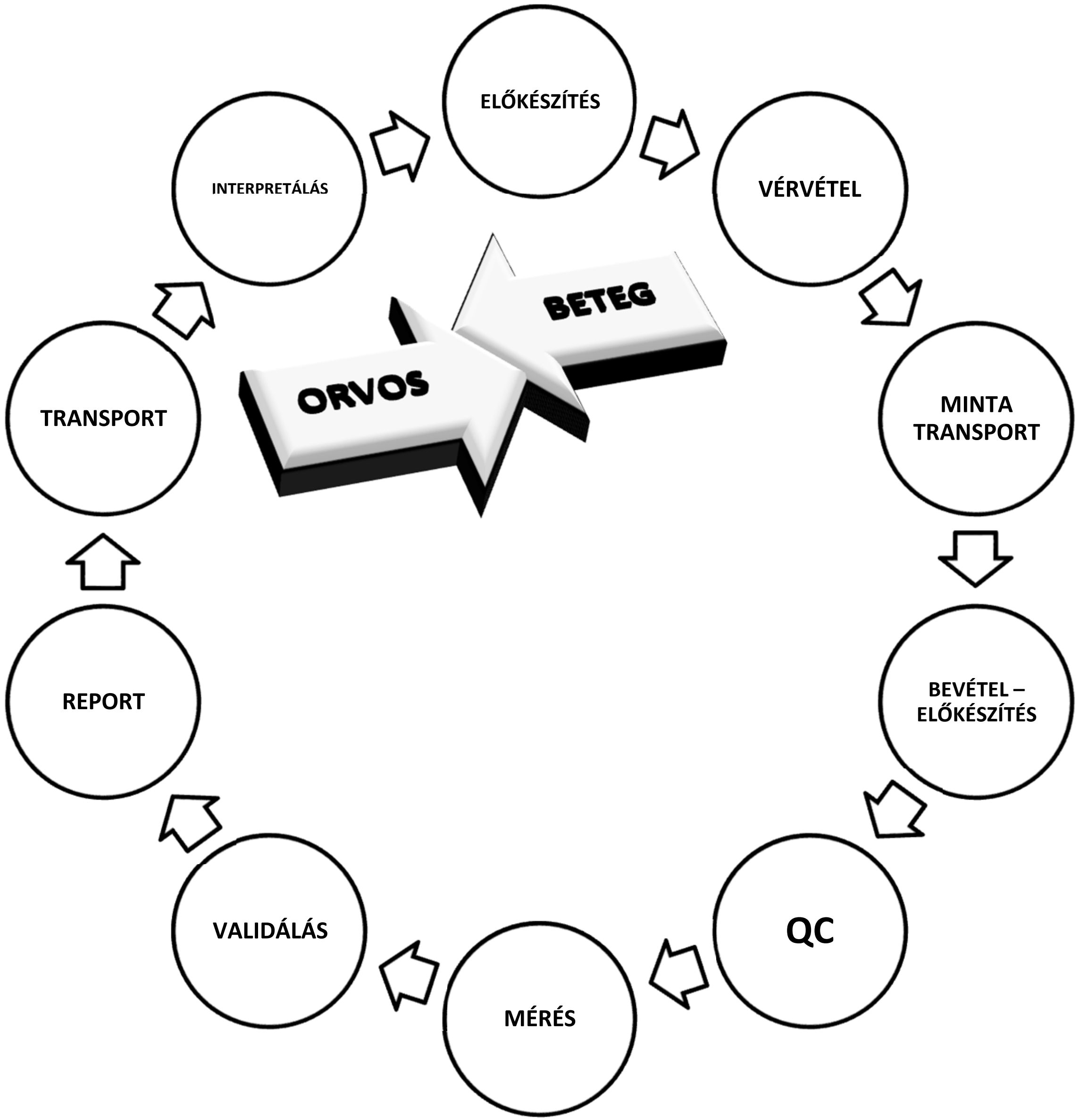

EXIT

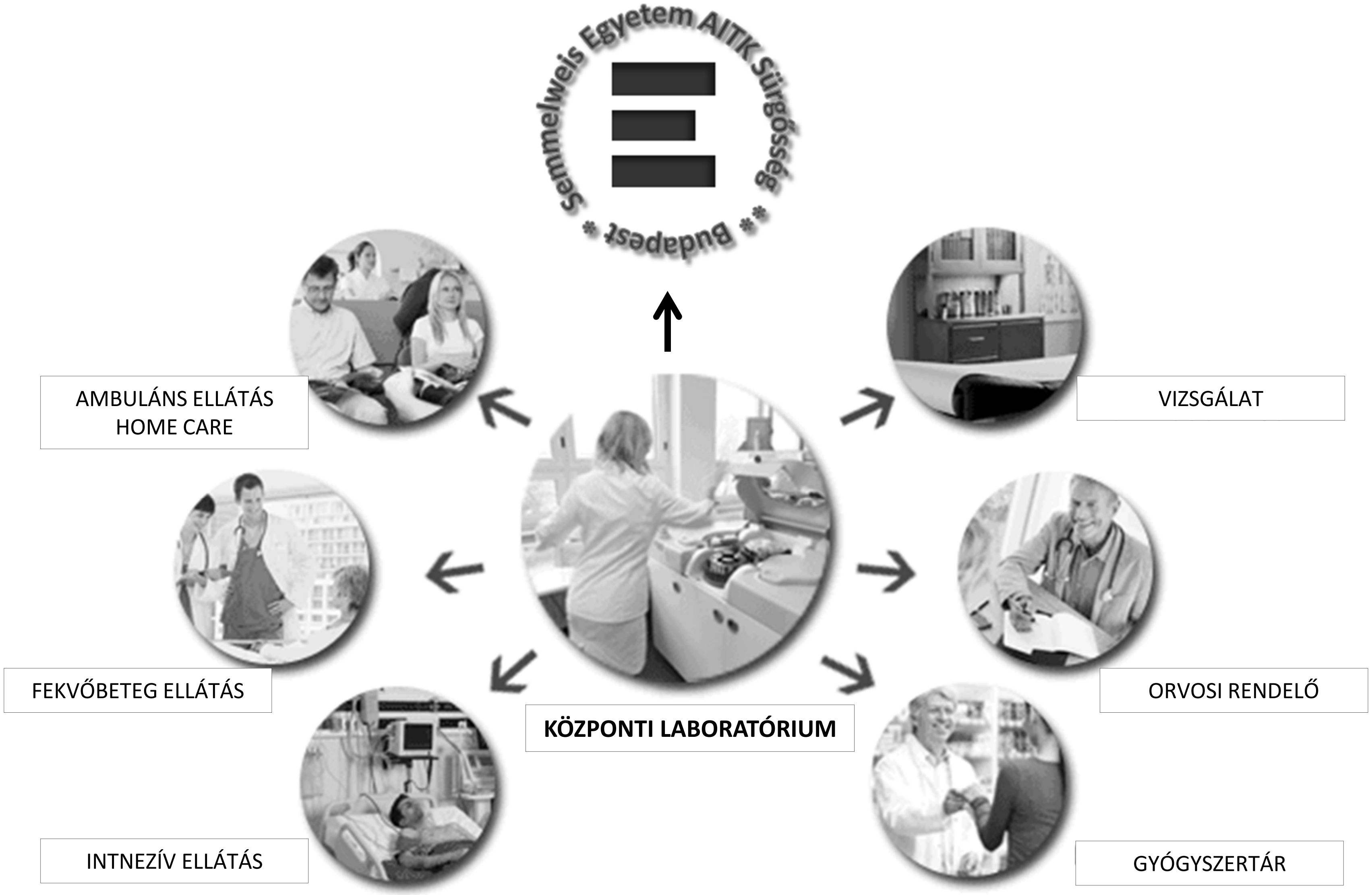
POINT OF CARE TESTING

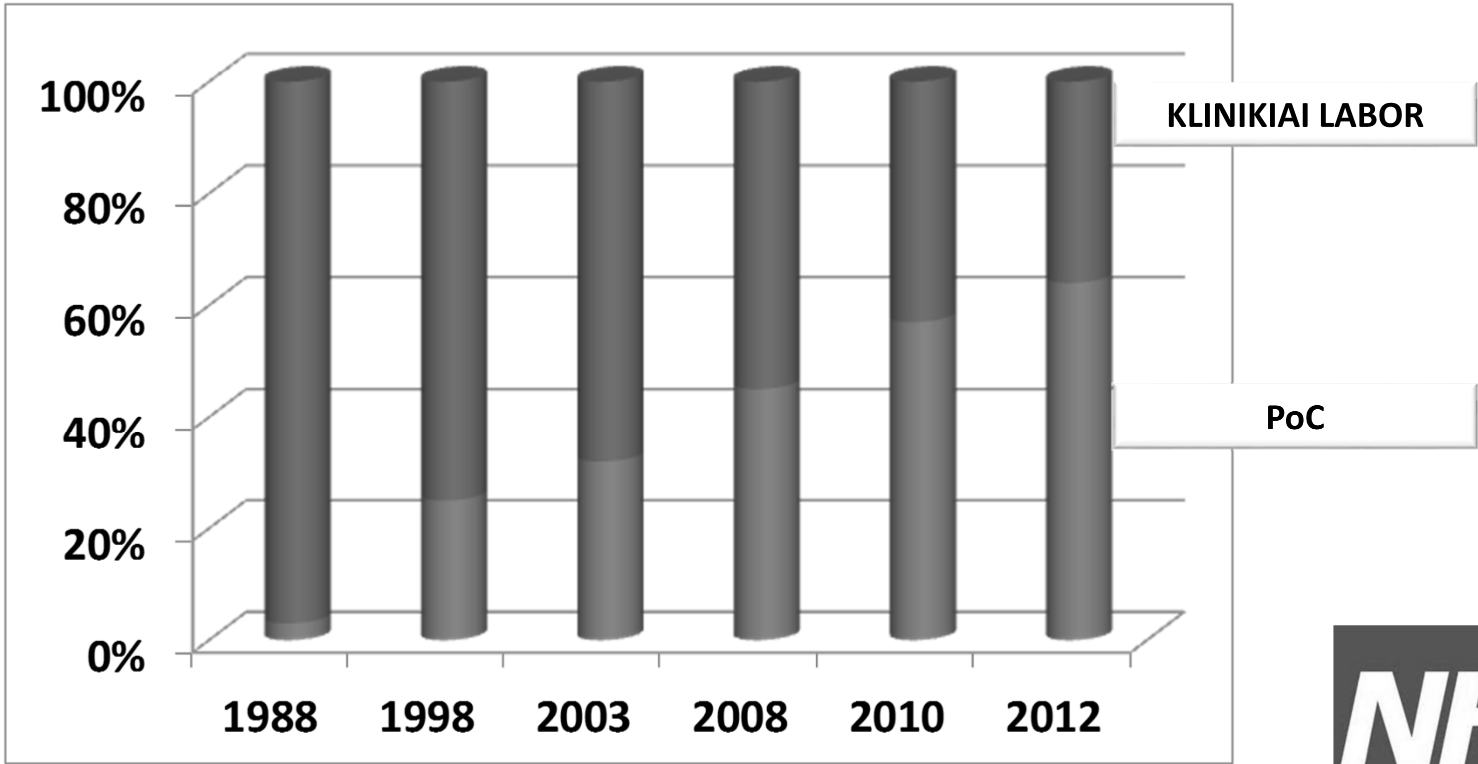
- ☞NEM SZOKÁSOS VIZSGÁLATI KÖRNYEZETBEN
- ☞A BETEGELLÁTÁS KÖZELÉBEN, ANNAK RÉSZEKÉNT
- ☞NEM DIAGNOSZTIKAI VÉGZETTSÉGŰ SZEMÉLYZET ÁLTAL VÉGZETT
- ☞MINŐSÉGÜGYI VIZSGÁLAT

- ☞RÖVIDEBB IDŐ (TURN-AROUND-TIME)
  - ☞ *TAT = MINTAVÉTEL/VIZSGÁLAT-EREDMÉNY*
- ☞ GAZDASÁGOSSÁG
- ☞ POCT ÉS DIAGNOSZTIKA
  - ☞ HELY – FUNKCIÓ
  - ☞ SZEMÉLYZET
  - ☞ KONTROLL
  - ☞ DOKUMENTÁLÁS
  - ☞ KARBANTARTÁS
  - ☞ MINŐSÉGBIZTOSÍTÁS


TURN AROUND TIME
< 10’


ELŐKÉSZÍTÉS
VÉRVÉTEL
MINTA TRANSPORT
BEVÉTEL – ELŐKÉSZÍTÉS
QC
MÉRÉS
VALIDÁLÁS
REPORT
TRANSPORT
INTERPRETÁLÁS
ORVOS
BETEG


Semmelweis Egyetem AITK Sürgősség
** Budapest **
AMBULÁNS ELLÁTÁS
HOME CARE
VIZSGÁLAT
FEKVŐBETEG ELLÁTÁS
ORVOSI RENDELŐ
KÖZPONTI LABORATÓRIUM
INTNEZÍV ELLÁTÁS
GYÓGYSZERTÁR


100%
80%
60%
40%
20%
0%
1988
1998
2003
2008
2010
2012
KLINIKIAI LABOR
PoC

NHS


integrated
care
patients
patient
integration
co-ordination
sharing
seamless
support
housing
working
health
together
listening
joined
holistic
staff
outcomes
Specialised
people
social
long-term
collaboration
team
budgets
embracing
joined-up
preferences
conditions
pathways
improving
experience
communicated
ideas
collaborating
belief
user
person
delay
clear
trained
uncomplicated
professionals
patient-centric
co-ordinating
service
complex
input
agencies
meaningful
accessible
ownership
respect
sector
knowledge
help

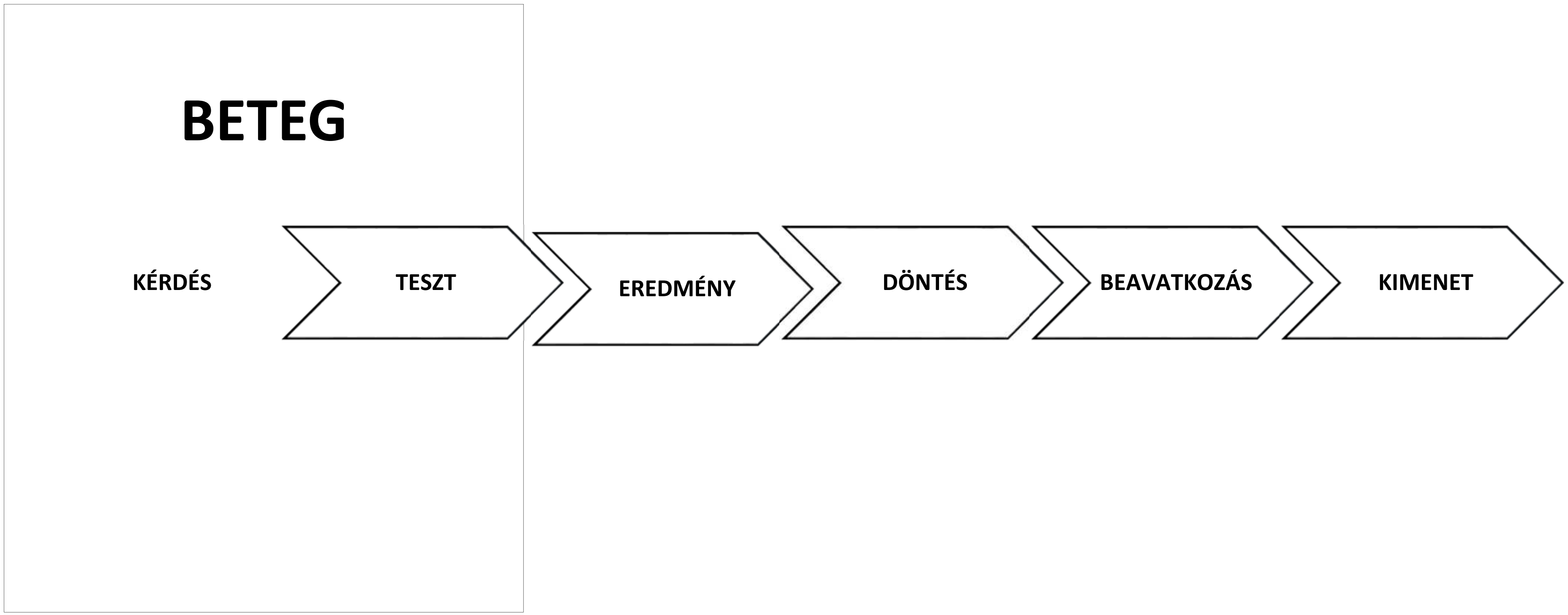
☞ RÖVID TAT
☞ GYORSABB DIAGNÓZIS
☞ PORTABILITÁS
☞ BETEGHEZ RENDELHETŐ
☞ HATÉKONYABB
☞ AZONNALI ANALÍZIS (MINTA MINŐSÉG)
☞ EGYSZERŰ / KÉNYELMES

☞ HATÉKONYABB KEZELÉS ÉS MENEDZSMENT
☞ HATÉKONY ÉSZLELÉS (KRITIKUS ÉRTÉKEK)
☞ KÖZPONTI DIAGNOSZTIKA TERHELÉSÉT CSÖKKENTI

PoCT TOP 10

1. HELYI DIAGNOSZTIKAI EGYSÉGEKKEL VALÓ KAPCSOLAT
2. SZERVEZETI EGYSÉG – MENEDZSMENT
3. SZABÁLYOZOTT TEVÉKENYSÉG
4. KÉPZÉS
5. ELŐÍRÁS SZERINTI HASZNÁLAT
6. STANDARDIZÁLÁS
7. MINŐSÉGBIZTOSÍTÁS
8. EREDMÉNYEK – KONTROLL
9. ADATTÁROLÁS – ARCHIVÁLÁS
10. KARBANTARTÁS

A PoC TESZT CSAK ADAT AZ ELLÁTÓN KERESZTÜL LESZ BELŐLE EREDMÉNY!


BETEG
KÉRDÉS
TESZT
EREDMÉNY
DÖNTÉS
BEAVATKOZÁS
KIMENET


IDŐ DEPENDENS
INTEGRATÍV
ALLOKATÍV


E
F P
T O Z
L P E D

BIOMARKER

SZŰRÉS – ALLOKÁCIÓ – RIZIKÓ - DÖNTÉS

DIAGNÓZIS - DÖNTÉS

RIZIKÓ STRATIFICATIO - DÖNTÉS

MONITORING – DÖNTÉS

VÉGPONT – DÖNTÉS

Clin Pharmacol Ott 2001 69:89-95,
Crit Care Med 2009; 37 (7) :2290-8

MIT VÁRUNK A BIOMARKEREKTŐL?

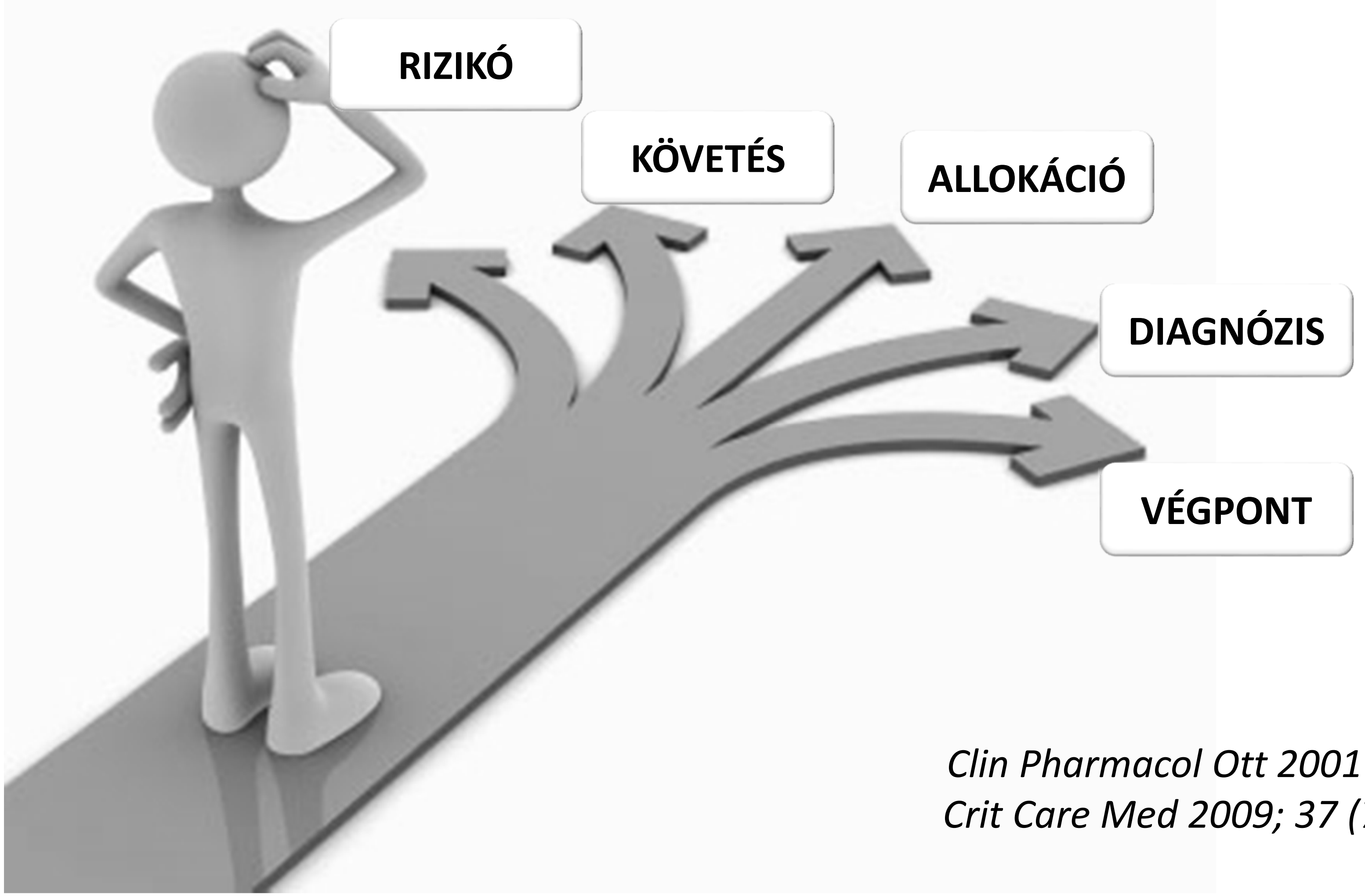
HELYES DIAGNÓZIS FELÁLLÍTÁSA

RIZIKÓBECSLÉS

MONITORING

Ray P et al:Statistical Evaluation of a Biomarker Anesthesiology 2010; 112:1023– 40

BIOMARKER



Clin Pharmacol Ott 2001 69:89-95,
Crit Care Med 2009; 37 (7) :2290-8

CARDIOMARKER

1. A MYOCARDIUMBAN MAGAS, EGYÉB SZÖVETBEN, COMPARTEMENTBEN ALACSONY KONCENTRÁCIÓBAN VAN JELEN
2. A MYOCARDIUM KÁROSODÁSAKOR GYORSAN ÉS ÉRTÉKELHETŐ KONCENTRÁCIÓBAN ÁRAMLIK KI A VÉRBE
3. A KÓRÁLLAPOT FOLYAMATÁBAN VÉGIG ÉRTÉKELHETŐ
4. RÖVID TAT

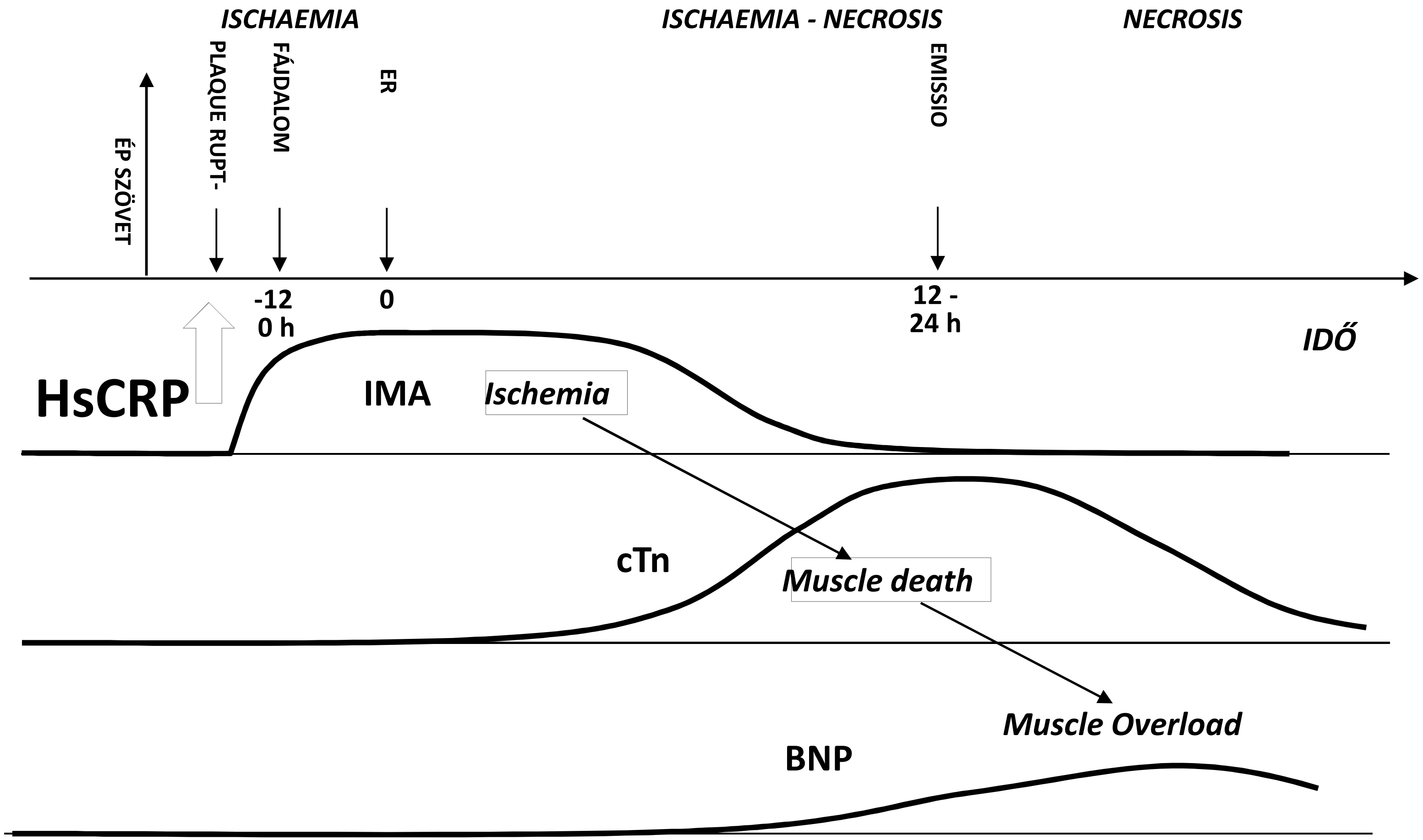
CARDIAC MARKER

- MYOCARDIALIS KÁROSODÁS KIMUTATÁSÁRA
- KÁROSODÁS MÉRTÉKÉNEK MEGÍTÉLÉSÉRE
- KÓRFOLYAMAT MONITORIZÁLÁSÁRA (REINF.)
- REPERFUSISO EFFECTUS IGAZOLÁSÁRA

CARDIALIS MARKEREK

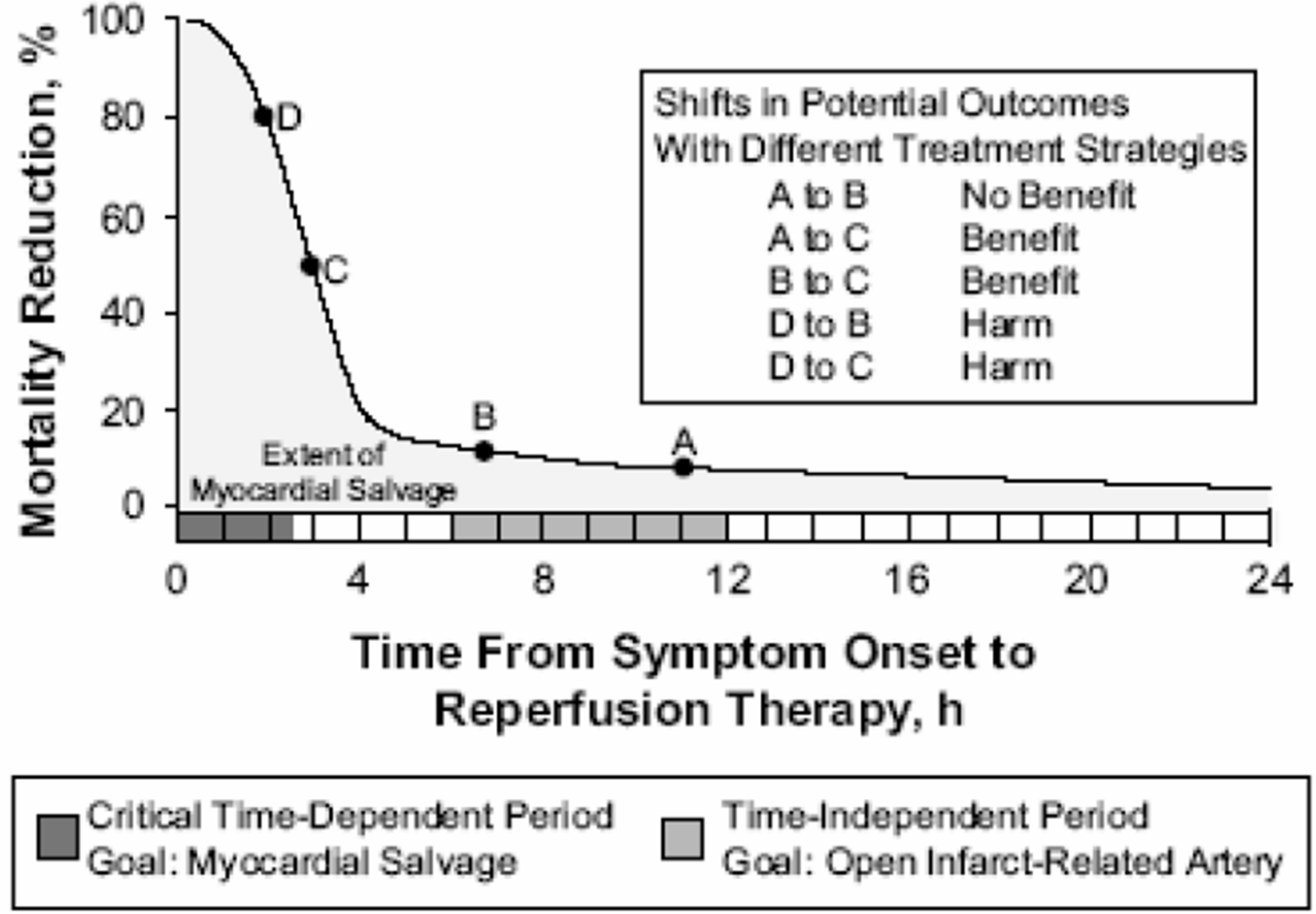
- ☞ **CK-MB**
- ☞ **TROPONIN (I/T)**
- ☞ **BNP**
- ☞ **MYOGLOBIN**
- ☞ **hsCRP**
- ☞ **D-DIMER**

ACS FOLYAMATA – IDŐ - MARKER



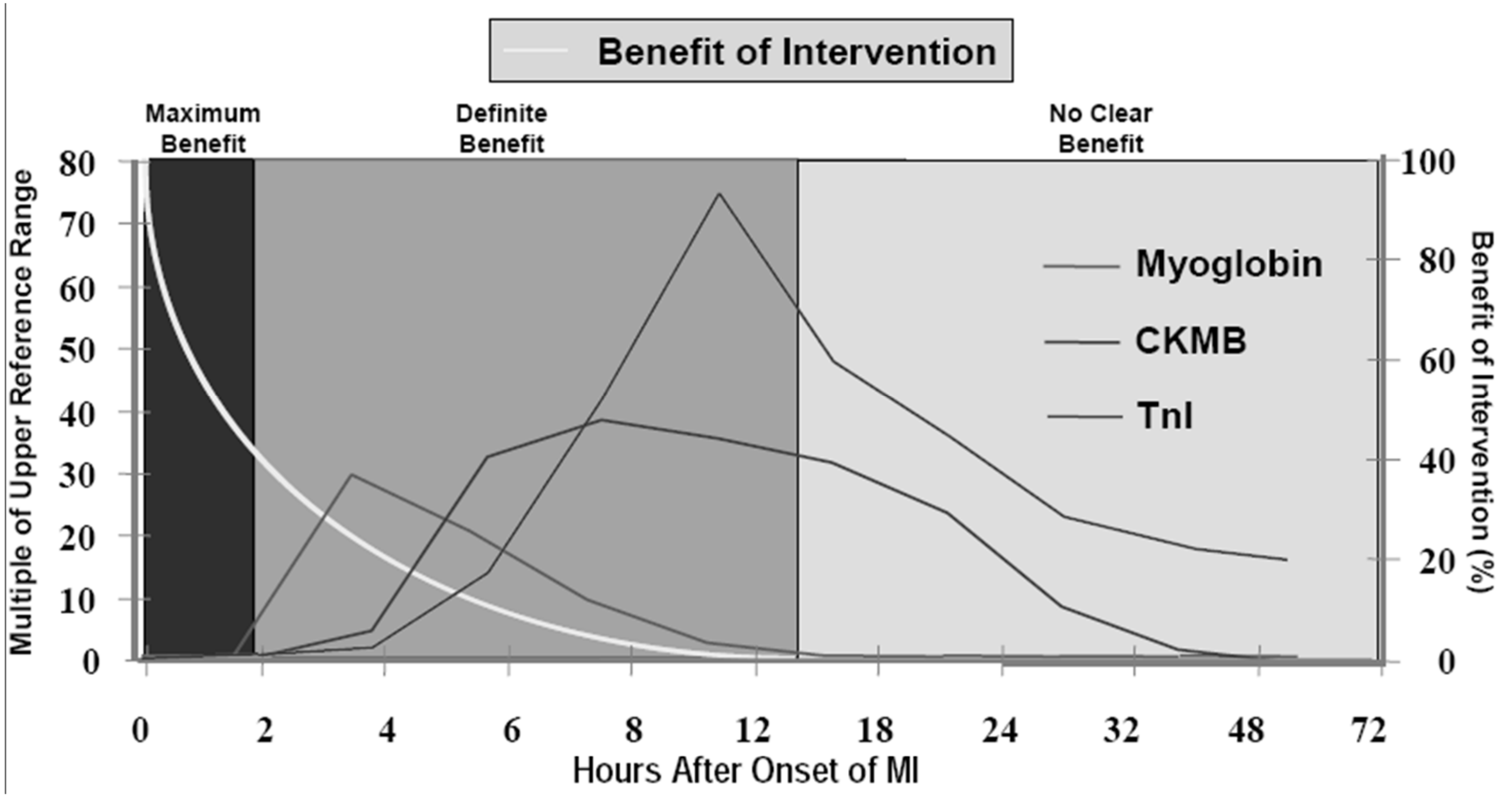
JAMA®
The Journal of the American Medical Association

TIME IS MUSCLE



JAMA 2005;293:973-986

IDŐ FAKTOR – INTERVENCIÓS HATÉKOYNSÁG



TROPONIN EMELKEDÉSSEL JÁRÓ KÓRFOLYAMATOK

Table 2. Elevations of Troponin in the Absence of Overt Ischemic Heart Disease

Trauma (including contusion, ablation, pacing, implantable cardioverterdefibrillator firings including atrial defibrillators, cardioversion, endomyocardial biopsy, cardiac surgery, after interventional closure of atrial septal defects)
Congestive heart failure—acute and chronic
Aortic valve disease and hypertrophic obstructive cardiomyopathy with significant left ventricular hypertrophy
Hypertension
Hypotension, often with arrhythmias
Postoperative noncardiac surgery patients who seem to do well
Renal failure
Critically ill patients, especially with diabetes, respiratory failure
Drug toxicity, e.g., adriamycin, 5-fluorouracil, herceptin, snake venoms
Hypothyroidism
Apical ballooning syndrome
Coronary vasospasm
Inflammatory diseases, e.g., myocarditis, e.g., Parvovirus B19, Kawasakidisease, sarcoid, smallpox vaccination, or myocardial extension of bacterial endocarditis
Post-percutaneous coronary intervention patients who seem to have no complications
Pulmonary embolism, severe pulmonary hypertension
Sepsis
Burns, especially if total body surface area is >30%
Infiltrative diseases including amyloidosis, hemachromatosis, sarcoidosis, and scleroderma
Acute neurological disease, including cerebrovascular subarachnoid bleeds
Rhabdomyolysis with cardiac injury
Transplant vasculopathy
Vital exhaustion

Jaffe AS, MD, Babuin L, Apple FS. Biomarkers in Acute Cardiac Disease The Present and the Future. *J Am Coll Cardiol* 2006;48:1–11

SPECIFICITÁS

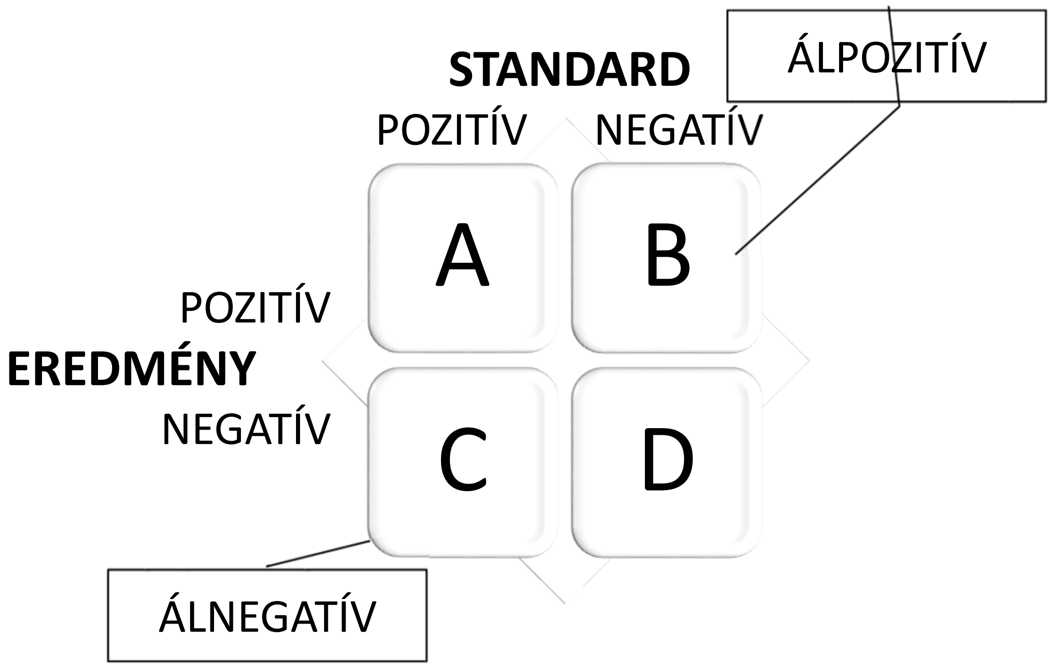
… annak a valószínűsége, hogy a kórfolyamat hiánya, negatív eredménnyel jár.

= d/b+d

SZENZITIVITÁS

… annak a valószínűsége, hogy a kórfolyamat fennállta, pozitív eredménnyel jár.

= c/a+c



POZITÍV PREDIKTÍV ÉRTÉK

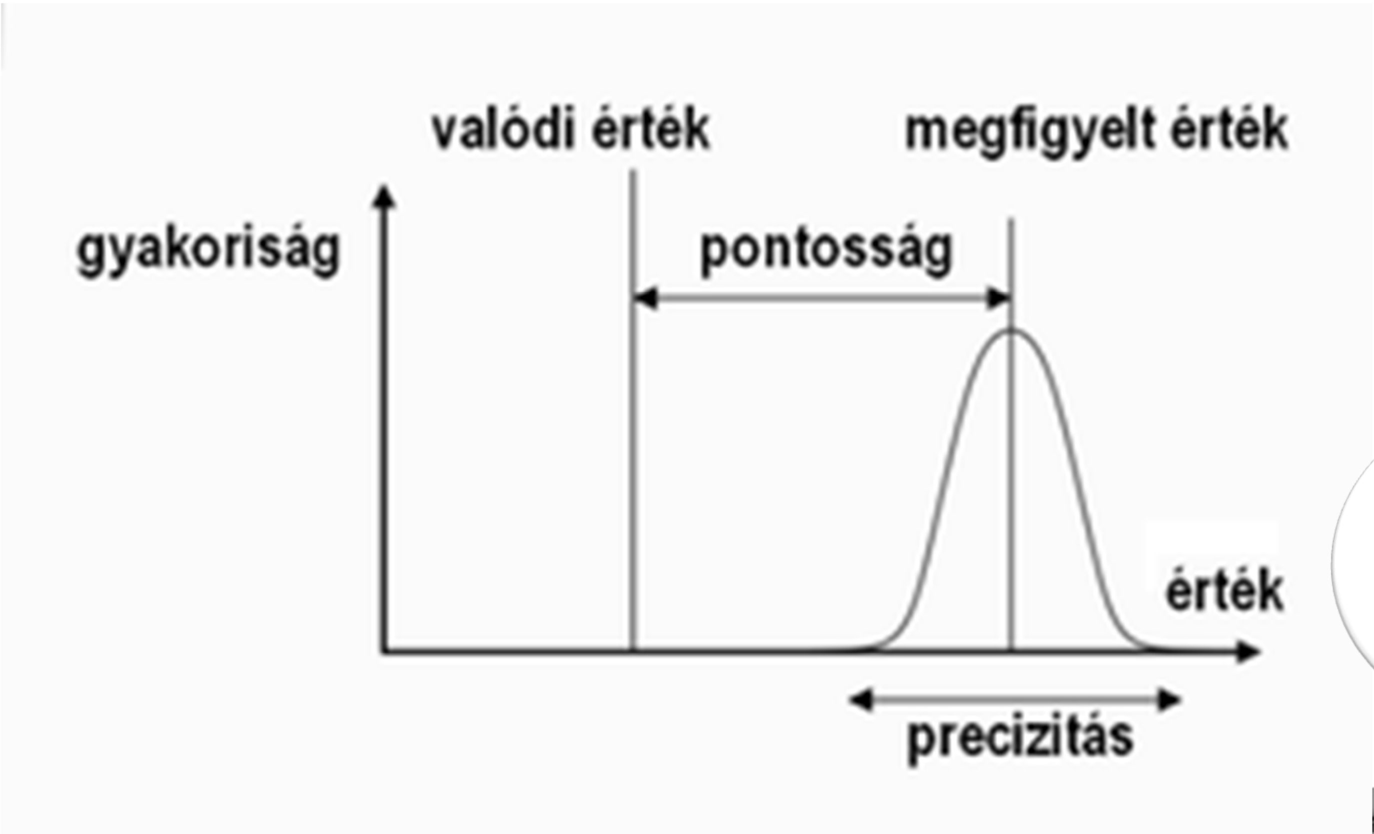
… az adott vizsgálat pozitivitása biztosan az adott betegség fennálltát igazolja.

= a/a+b

NEGATÍV PREDIKTÍV ÉRTÉK

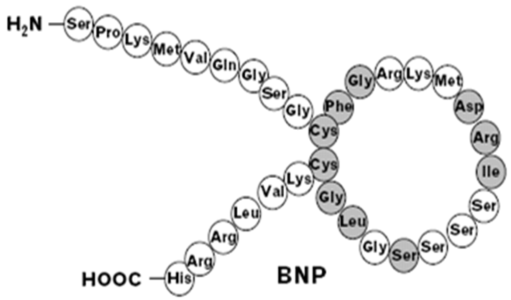
… az adott vizsgálat negativitása biztosan kizárja az adott betegség fennálltát.

= d/c+d

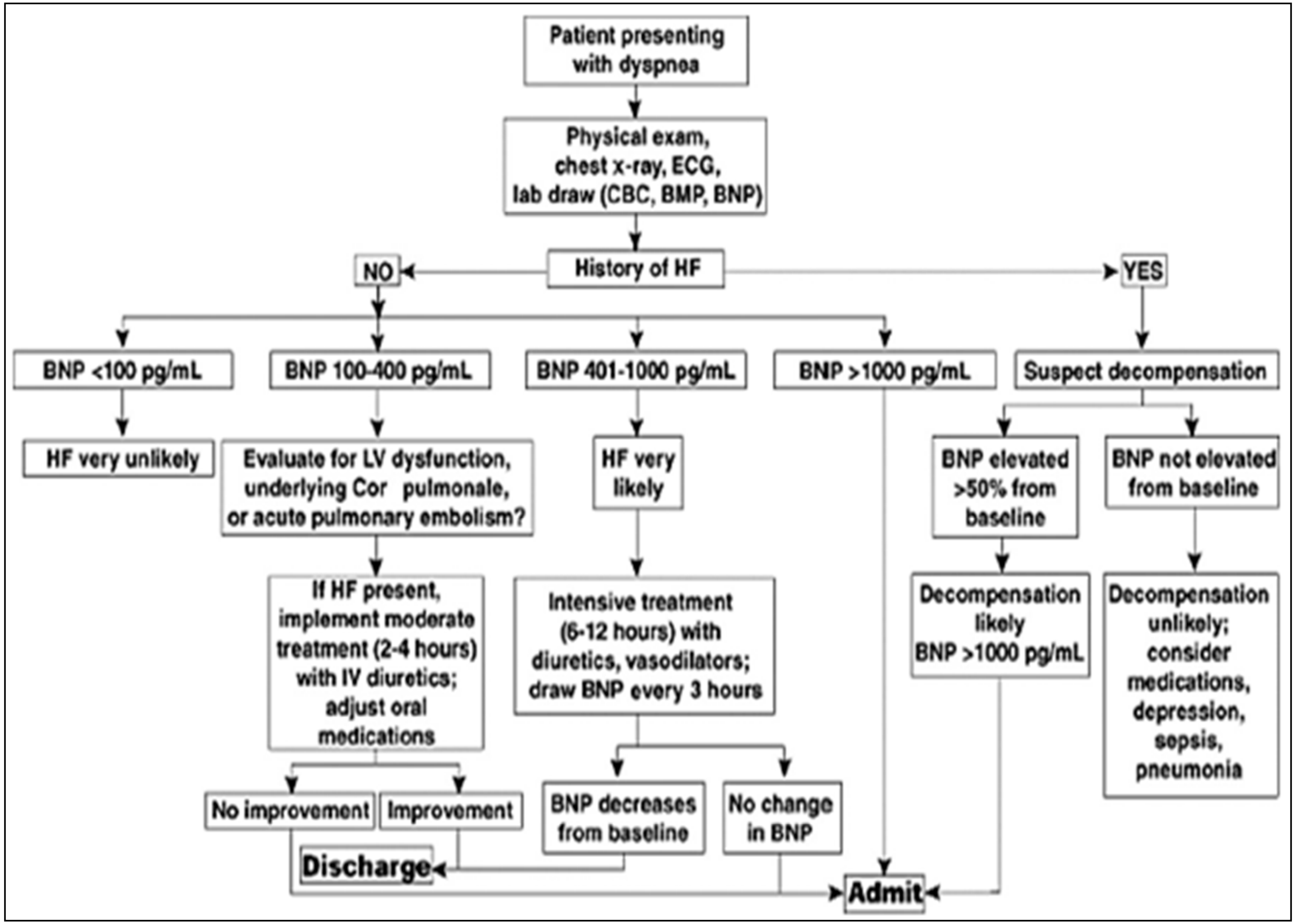

valódi érték
megfigyelt érték
gyakoriság
pontosság
érték
precizitás

> 99%

BIOMARKER – SZÍVELÉGTELENSÉG



- Unrecognized HF
- Prior HF
- LVH
- Valvular heart disease
- Atrial fibrillation
- Advancing age
- Myocarditis
- ACS
- Pulmonary hypertension
- Congenital heart disease
- Anemia
- Pulmonary embolism
- Cardiac surgery
- Sleep apnea
- Critical illness
- Sepsis
- Burns
- Renal failure
- Toxic-metabolic insults



Patient presenting with dyspnea
Physical exam, chest x-ray, ECG, lab draw (CBC, BMP, BNP)
History of HF
NO
YES
BNP <100 pg/mL
BNP 100-400 pg/mL
BNP 401-1000 pg/mL
BNP >1000 pg/mL
Suspect decompensation
HF very unlikely
Evaluate for LV dysfunction, underlying Cor pulmonale, or acute pulmonary embolism?
HF very likely
BNP elevated >50% from baseline
BNP not elevated from baseline
If HF present, implement moderate treatment (2-4 hours) with IV diuretics; adjust oral medications
Intensive treatment (6-12 hours) with diuretics, vasodilators; draw BNP every 3 hours
Decompensation likely BNP >1000 pg/mL
Decompensation unlikely; consider medications, depression, sepsis, pneumonia
No improvement
Improvement
BNP decreases from baseline
No change in BNP
Discharge
Admit

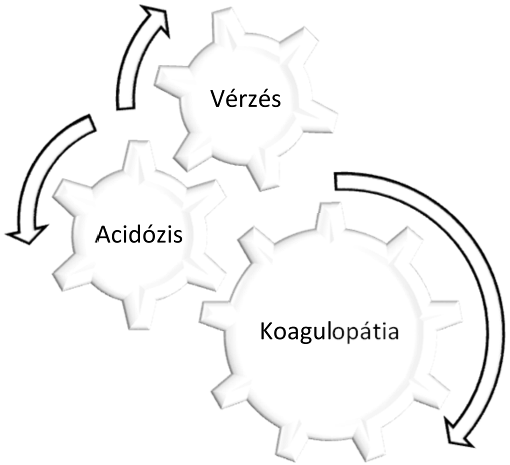
BIOMARKER – TRAUMA (1.)

REVIEW ARTICLE

J Trauma Acute Care Surg
Volume 74, Number 6

Practical application of point-of-care coagulation testing to guide treatment decisions in trauma

Herbert Schöchl, MD, Wolfgang Voelckel, MD, Alberto Grassetto, MD,
and **Christoph J. Schlimp, MD,** *Salzburg, Austria*



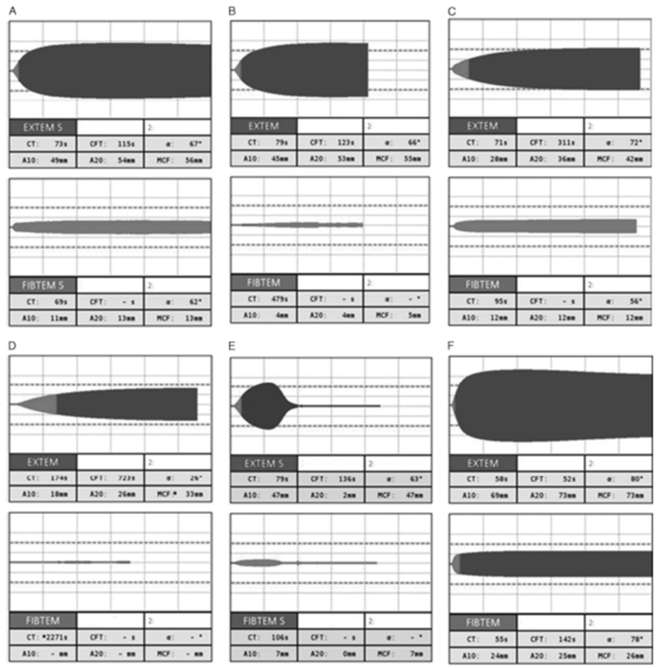
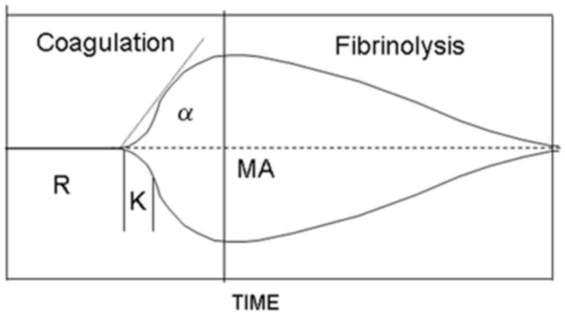
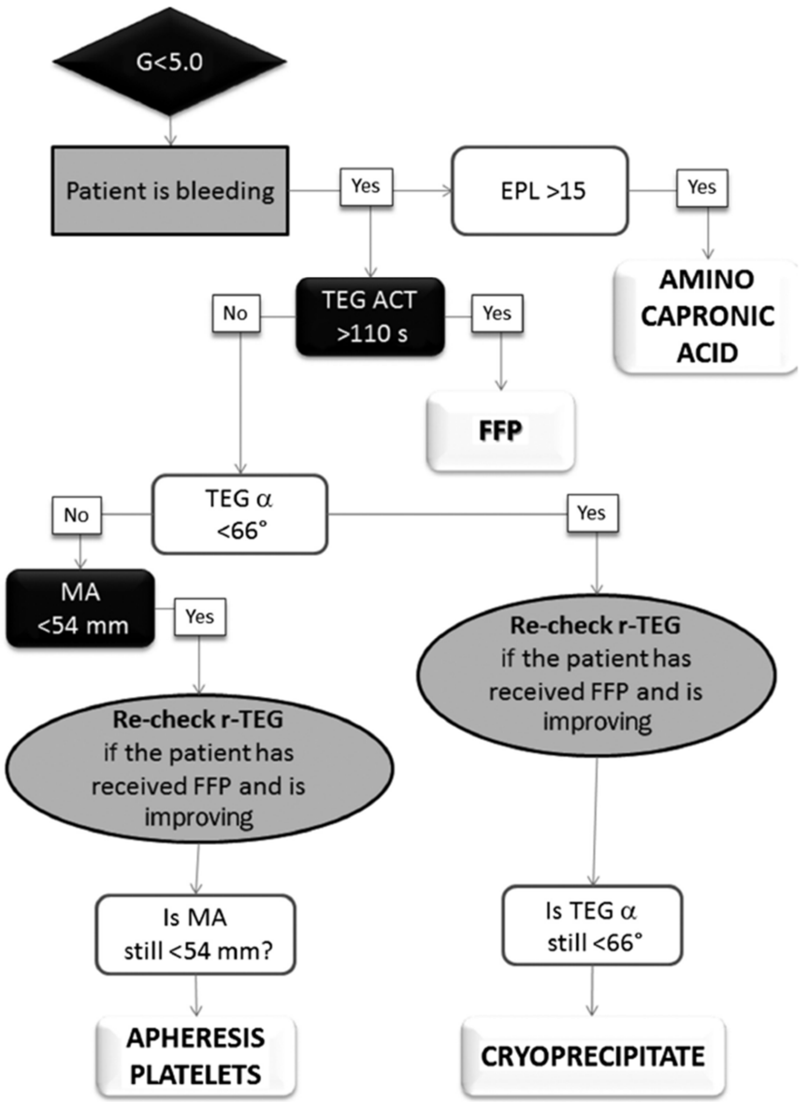
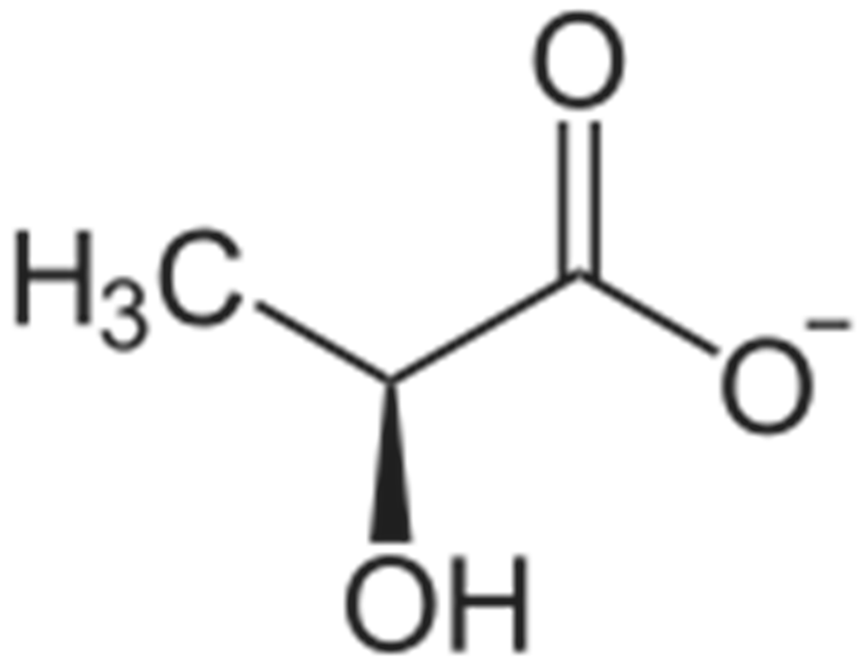


Figure 3. Thromboelastometry (EXTEM and FIBTEM) traces observed during normal, impaired, and hyperfibrinolytic hypercoagulable states.[19] *A*, Normal EXTEM and FIBTEM test result. *B*, Normal parameters in EXTEM but major reduction in FIBTEM MCF, suggesting fibrinogen deficit. *C*, Major prolongation of CFT and reduction in α angle and EXTEM MCF, normal FIBTEM MCF suggesting platelet deficit. *D*, Severe coagulopathy: delayed initiation of coagulation (prolonged CT), low CFT and MCF; no measurable FIBTEM. *E*, HF: complete breakdown of the clot in EXTEM and FIBTEM. *F*, Hypercoagulable state: short CT and CFT and high α angle and MCF in EXTEM, high MCF in FIBTEM. FIBTEM, extrinsically activated test plus cytochalasin D; EXTEM, extrinsically activated test.


Coagulation
Fibrinolysis
α
R
K
MA
TIME


G<5.0
Patient is bleeding
Yes
EPL >15
Yes
AMINO CAPRONIC ACID
TEG ACT >110 s
No
Yes
FFP
TEG α <66°
No
Yes
MA <54 mm
Yes
Re-check r-TEG if the patient has received FFP and is improving
Re-check r-TEG if the patient has received FFP and is improving
Is MA still <54 mm?
Is TEG α still <66°
APHERESIS PLATELETS
CRYOPRECIPITATE

BIOMARKER – TRAUMA (2.)



BIOMARKER – TRAUMA (3.)

Biomarkers of mild traumatic brain injury in cerebrospinal fluid and blood

Henrik Zetterberg, Douglas H. Smith and Kaj Blennow

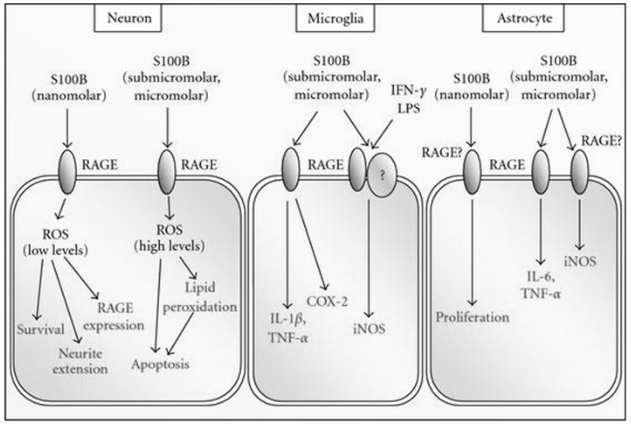


Table 1 | Potential fluid biomarkers of mild TBI

Marker	Process or structure affected	Research findings in mild TBI: Cerebrospinal fluid	Research findings in mild TBI: Blood
Cerebrospinal fluid:serum albumin ratio	Blood–brain barrier	Detection method not sensitive enough to changes in concentration[53,54]	NA
Interleukins and other acute-phase inflammatory response proteins	Neuroinflammation	NA	NA
Total tau protein	Axon	Levels peak 4–10 days after injury[54,71]	Elevated levels in hypoxic brain injury;[101,102] studies in mild TBI are lacking
Myelin basic protein	Axon	NA	Detection methods not sensitive in mild TBI[79]
Neurofilament light polypeptide	Axon	Levels peak 4–10 days after injury[54,71]	NA
Neurofilament heavy polypeptide	Axon	NA	NA
γ-Enolase	Neuron	Levels confounded by lysis of red blood cells in blood-contaminated cerebrospinal fluid[82]	Serum levels very sensitive to lysis of red blood cells in blood-contaminated cerebrospinal fluid[82]
S100-B	Astroglial cells	Levels are elevated, but a less sensitive marker than tau protein and neurofilaments[71]	Results may be confounded by release from extracerebral tissues[77]
Glial fibrillary acidic protein	Astroglial cells	Elevated levels, but a less sensitive marker than tau and neurofilament proteins[54,71]	Serum levels correlate with changes on brain imaging;[118] extracerebral protein has not been detected[74]
Secreted APP-α and APP-β	Axon	NA	NA
Amyloid-β_{40} and amyloid-β_{42}	Plaque pathology	No change in levels[57,74]	NA
Spectrin breakdown products	Neuron	NA	NA
Ubiquitin carboxyl-terminal hydrolase isoenzyme L1	Neuron	NA	One pilot study showed promising results[123]
Peripheral blood mononuclear small noncoding RNA molecules	Unknown	NA	One pilot study suggests altered expression of three RNA molecules in mild-TBI-exposed military personnel[125]

Abbreviations: APP, amyloid precursor protein; NA, not available; TBI, traumatic brain injury.

LIQUOR SZENZITÍVEBB
VÉRBŐL TECHNOLÓGIA ULTRASZENZITÍV
SPECIFICITÁS – SERIAL CT-VEL (!)

BIOMARKER - STROKE

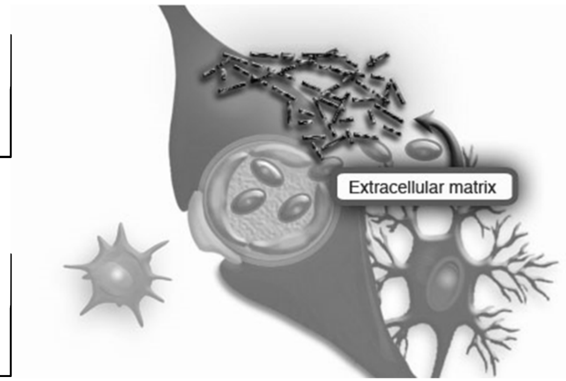
http://stroke-biomarkers.com/

RIZIKÓ (hsCRP)

DIAGNÓZIS (ARGININ, S100B, GFAB,ANFP)

ETIOLÓGIA (ANFP, SRAGE)

PROGNÓZIS



Available online at www.sciencedirect.com

SciVerse ScienceDirect

www.elsevier.com/locate/jprot

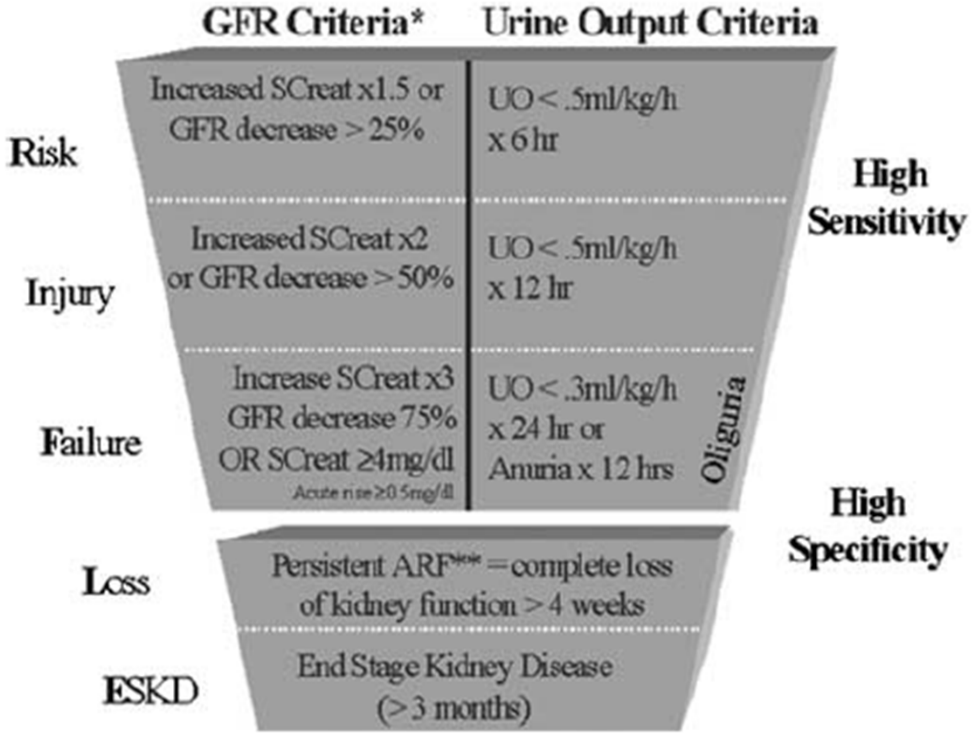
Differentiating ischemic from hemorrhagic stroke using plasma biomarkers: The S100B/RAGE pathway☆

7. Conclusions

A biochemical panel containing both S100B and sRAGE as a rapid blood test might help to distinguish between ischemic and hemorrhagic strokes, especially within the first hours after stroke onset. This approach would improve the management of both conditions in the pre-hospital setting.

BIOMARKER – ACUT RENAL FAILAURE

ß2 MICROGLOBULIN
ENGAL
IL-18
FABP



BIOMARKER – ACUT RENAL FAILAURE

Journal of the American College of Cardiology

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Vol. 59, No. 3, 2012
ISSN 0735-1097/$36.00
doi:10.1016/j.jacc.2011.10.854

Diagnostic and Prognostic Stratification in the Emergency Department Using Urinary Biomarkers of Nephron Damage

A Multicenter Prospective Cohort Study

Neutrophil Gelatinase-Associated Lipocalin

Cystatin C

Kidney Injury Molecule-1

NAG

Liver-Type Fatty Acid Binding Proteins

Interleukin-18

Conclusions

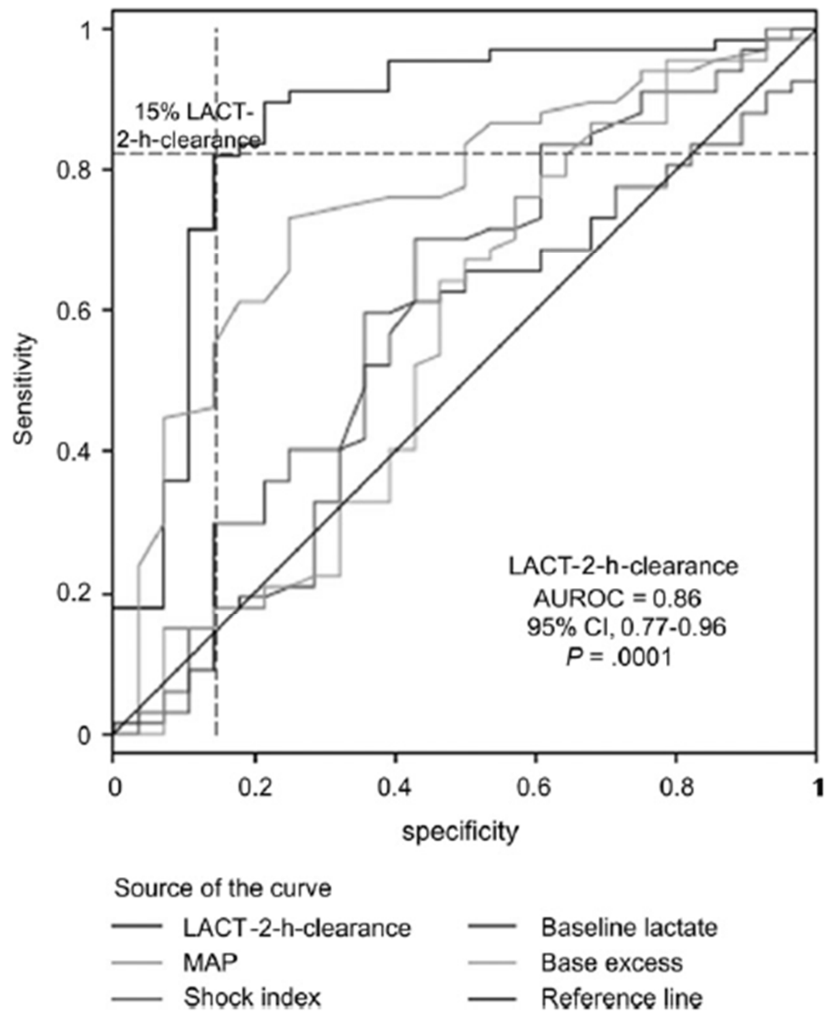
In summary, we characterized the performance of different urinary biomarkers obtained on patient entry to the hospital to diagnose iAKI (as defined by strict criteria) and to determine its severity and clinical sequelae. Our analysis prospectively validated the concept that the addition of urinary biomarkers and their interpretation together with sCr levels identified patients at risk who otherwise would have been missed during triage.

BIOMARKER - SEPSIS

PROCALCITONIN
- KINETIKA

PRESEPSIN

LAKTÁT
- CLEARANCE



Crit Care Res Pract 2010;

$$\text{Pyruvate} + NAD^+ + \text{CoA} \rightarrow \text{Acetyl CoA} + CO_2 + \text{NADH}$$

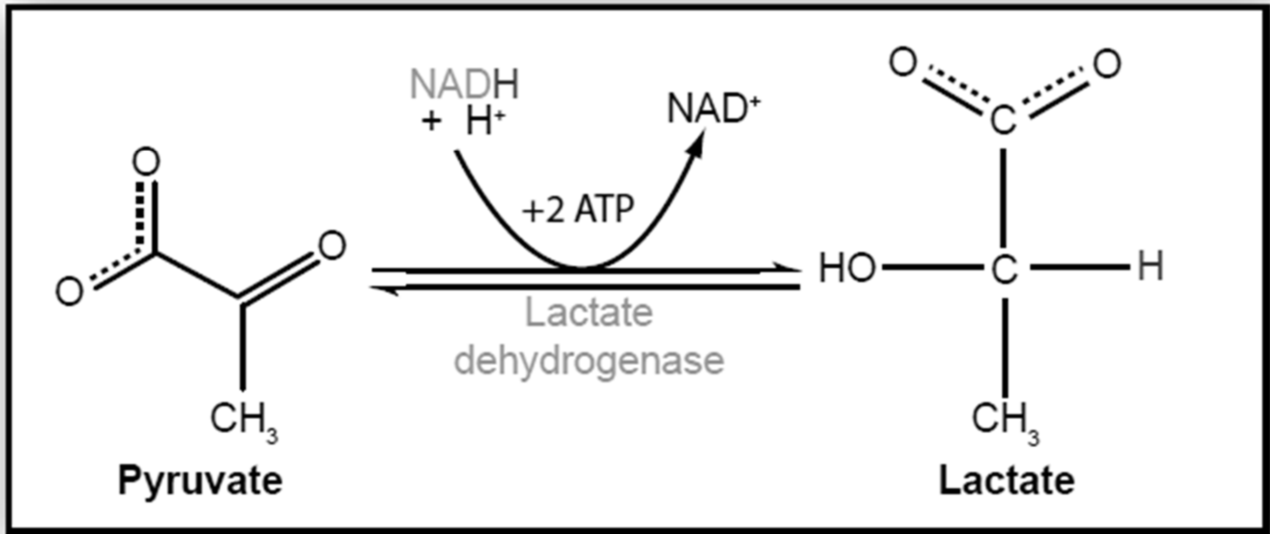


Table 1. Causes of Lactic Acidosis

Decreased oxygen delivery
Hypotension
Volume depletion
Blood loss
Cardiogenic shock
Septic shock
Severe anemia
Severe hypoxemia
Carbon monoxide poisoning
Increase oxygen demands
Exercise
Seizures
Shivering

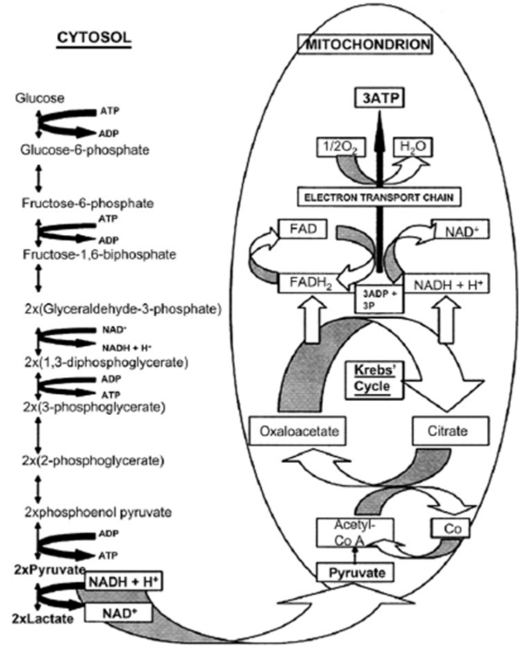
Cohen R, Woods H. Clinical and Biochemical Aspects of Lactic Acidosis. Blackwell Scientific Publications; 1976.

Table 2. Causes of Type B Lactic Acidosis

Inadequate oxygen utilization
Systemic inflammatory response syndrome
Diabetes mellitus
Malignancy
Total parenteral nutrition
Thiamine deficiency
Congenital lactic acidosis
Mitochondrial myopathies
HIV infection
Malaria
Drugs/toxins (see Table 3)
Other
D-lactic acidosis

Cohen R, Woods H. Clinical and Biochemical Aspects of Lactic Acidosis. Blackwell Scientific Publications; 1976.

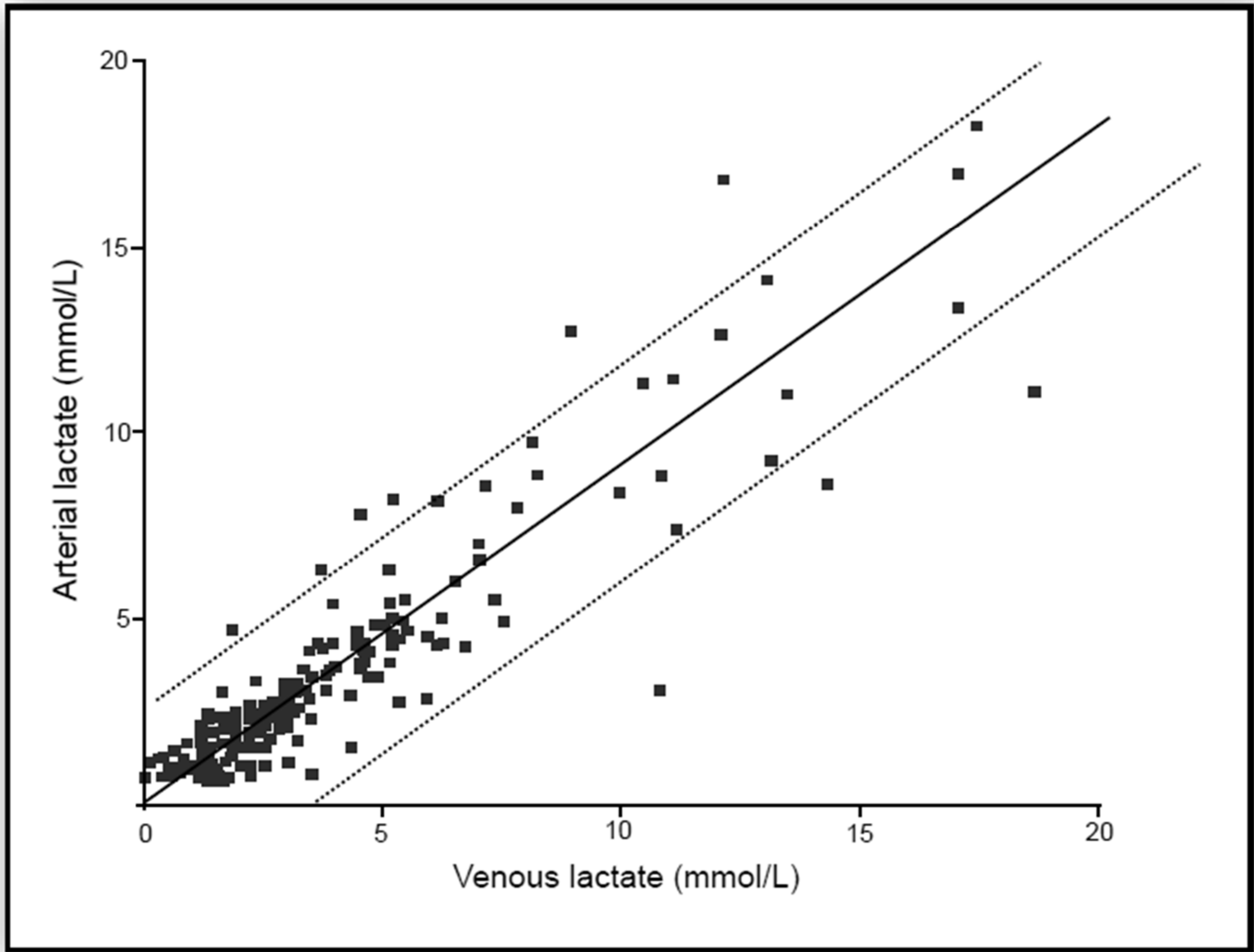
INADEQUAT OXYGEN KÍNÁLAT
VOLUMEN DEPLETIO / HYPOVOLAEMIA SZIGNIFIKÁNS VÉRVESZTÉS SÚLYOS ANAEMIA SÚLYOS HYPOXAEMIA CO – EXPOSITIO TRAUMA



ARÁNYTALAN OXYGEN IGÉNY
HYPERTHEMIA GÖRCS ÁLLAPOT REMEGÉS EXTRÉM IZOMTERHELÉS

INADEQUAT OXYGEN FELHASZNÁLÁS
SIRS DIABETES TPN THIAMIN HIÁNY HIV FERTŐZÉS DRUG (MERFORMIN, SALICYL, PROPOFOL, ISONIAZID, CYANID, ANTIRETROVIRALIS)


3.4
Lactate
BLOOD LACTATE TEST METER
Pro



Acta Med Austriaca 1976; 3:69-73.

JOURNAL OF EMERGENCY NURSING January 2011 VOLUME 37 • ISSUE 1

DETECTING AND TREATING SEPSIS IN THE EMERGENCY DEPARTMENT

Authors: Amy Michelle Vanzant, MSN, RN, CEN, and Marilee Schmelzer, PhD, RN, Fort Worth and Arlington, TX

SERUM LACTIC ACID LEVELS

Because the serum lactate level reflects cellular hypoxia, it seems logical to measure it to detect and track sepsis. The pathophysiology of sepsis suggests that lactate levels would rise early in the development of shock before organs start to fail.

RIVERS – STOP SEPSIS CAMPAIGN

MIKKELSEN (n=890 / LACTAT 2-3,9)

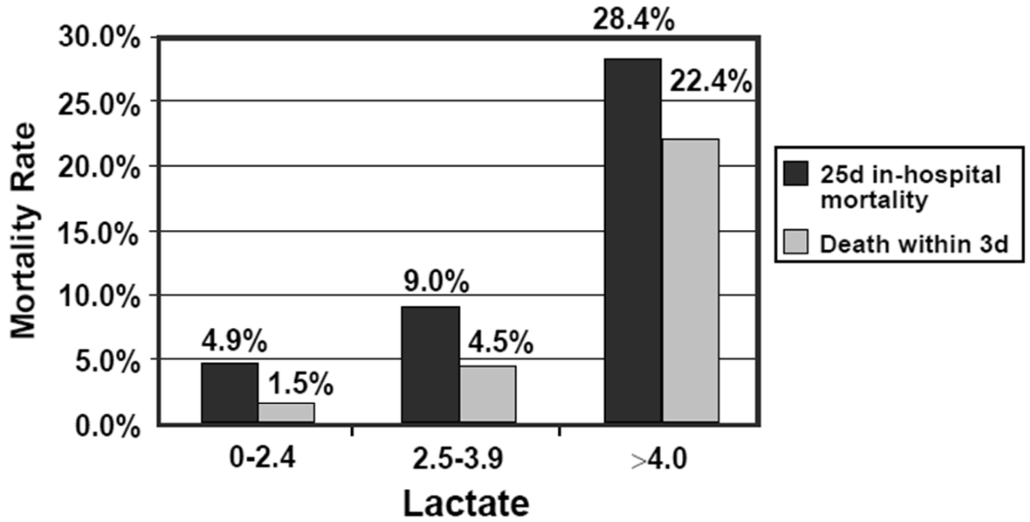
SHAPIRO (n=1.278 ⇨ 105 ✞)
0-2,5 :4,9% 2,6-4,0:28,4% > 4,0:36%

BAKKER – SERIAL

Serum Lactate as a Predictor of Mortality in Emergency Department Patients With Infection

Nathan I. Shapiro, MD, MPH

Conclusion: In this cohort of ED patients with signs and symptoms suggestive of infection, our results support serum venous lactate level as a promising risk-stratification tool. Multicenter validation, as well as comparison of the lactate level with clinical predictors, needs to be done before widespread implementation. [Ann Emerg Med. 2005;45:524-528.]



SHOCK, Vol. 32, No. 1, pp. 35–39, 2009

MULTICENTER STUDY OF EARLY LACTATE CLEARANCE AS A DETERMINANT OF SURVIVAL IN PATIENTS WITH PRESUMED SEPSIS

Ryan C. Arnold,* Nathan I. Shapiro,† Alan E. Jones,‡ Christa Schorr,§

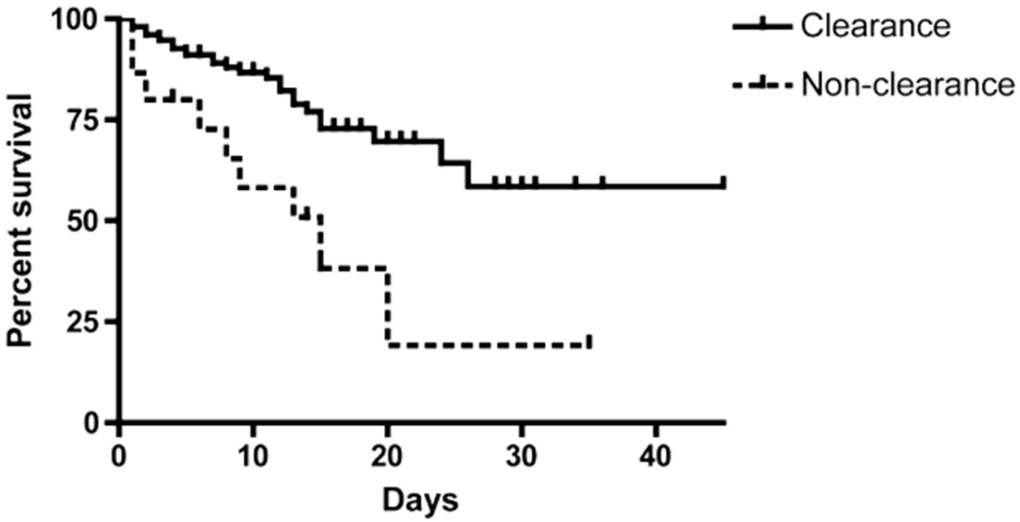


FIG. 2. **Kaplan-Meier survival curves.** This graph depicts survival curves over time for lactate clearance (lactate decrease by ≥10%) and lactate non-clearance (lactate decrease <10%) groups. The curves diverge significantly by log-rank test (P = 0.003).

Resuscitation (2007) 75, 229—234

ELSEVIER

RESUSCITATION

www.elsevier.com/locate/resuscitation

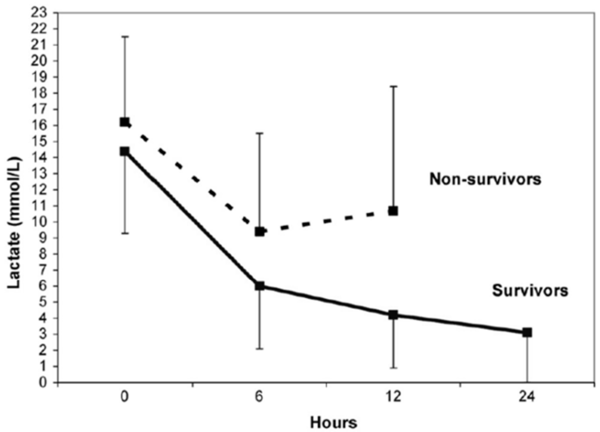
CLINICAL PAPER

Effective lactate clearance is associated with improved outcome in post-cardiac arrest patients

Michael W. Donnino*, Joseph Miller, Nikhil Goyal, Manisha Loomba,

Clearance

Conclusions: Early, effective lactate clearance is associated with decreased early and overall in-hospital mortality in post-cardiac arrest patients. These findings suggest that post-arrest tissue hypo-perfusion plays in an important role in early as well as overall mortality.



$$\text{Lactate Clearance} = \frac{(\text{Lactate}^{\text{ED Presentation}}) - (\text{Lactate}^{\text{Hour 6}})}{\text{Lactate}^{\text{ED Presentation}}} \times 100$$

Kruse *et al. Scandinavian Journal of Trauma, Resuscitation and Emergency Medicine* 2011, **19**:74
http://www.sjtrem.com/content/19/1/74

SCANDINAVIAN JOURNAL OF
trauma, resuscitation
& emergency medicine

REVIEW **Open Access**

Blood lactate as a predictor for in-hospital mortality in patients admitted acutely to hospital: a systematic review

Ole Kruse[1], Niels Grunnet[2] and Charlotte Barfod[3*]

THE COCHRANE
COLLABORATION®

Conclusions

Blood lactate monitoring is useful for risk assessment in patients admitted acutely to hospital, and especially the trend in serial lactate monitoring is valuable in predicting in-hospital mortality. All patients with a lactate at admission above 2.5 mM should be closely monitored for signs of deterioration, and patients with even lower lactate levels should be considered for serial lactate monitoring. Peripheral venous blood lactate is highly correlated with arterial blood lactate, and venous sampling should therefore be encouraged, as the risk and inconvenience for this procedure is minimal for the patient. The relevance of lactate guided therapy has to be supported by more studies.

http://ccforum.com/content/12/6/R160

Research

Open Access

The prognostic value of blood lactate levels relative to that of vital signs in the pre-hospital setting: a pilot study

Tim C Jansen[1], Jasper van Bommel[1], Paul G Mulder[2], Johannes H Rommes[3], Selma JM Schieveld[3] and Jan Bakker[1]

[1]Department of Intensive Care, Erasmus MC University Medical Center, PO Box 2040, 3000 CA, Rotterdam, The Netherlands
[2]Department of Epidemiology & Biostatistics, Erasmus MC University Medical, PO Box 2040, 3000 CA, Rotterdam, The Netherlands
[3]Department of Intensive Care, Gelre Hospital, location Lukas, PO Box 9014, 7300 DS Apeldoorn, The Netherlands

Corresponding author: Jan Bakker, jan.bakker@erasmusmc.nl

Received: 29 Sep 2008 Revisions requested: 6 Nov 2008 Accepted: 17 Dec 2008 Published: 17 Dec 2008

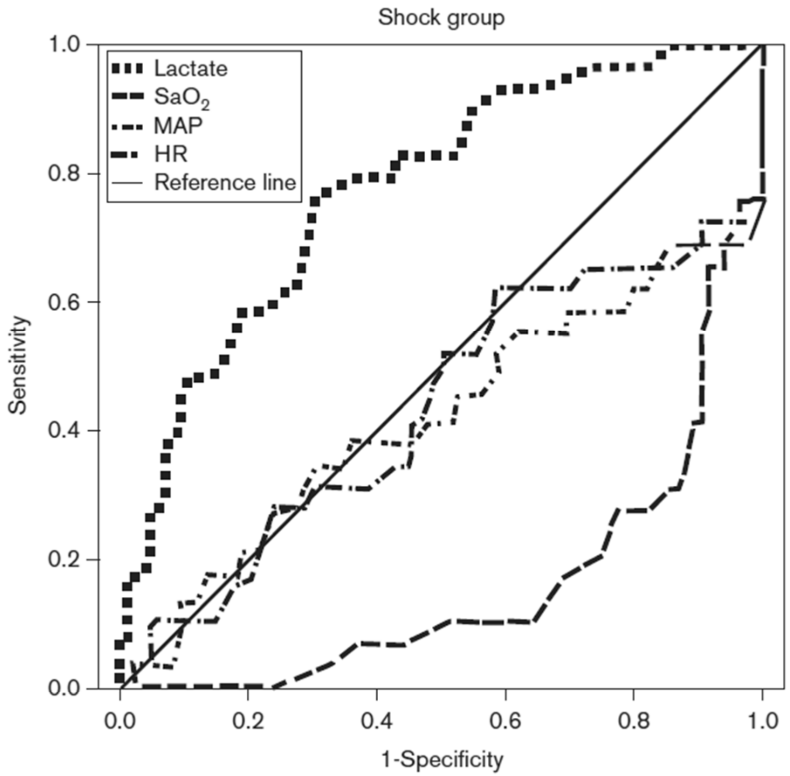
Key messages

- Pre-hospital blood lactate levels were associated with in-hospital mortality.
- A blood lactate level of 3.5 mmol/L was the best cut-off value in the pre-hospital phase to discriminate survivors from non-survivors.
- The prognostic value of pre-hospital blood lactate level was superior to that of heart rate and systolic blood pressure.
- The use of blood lactate measurement in EMS might have potential for triage decisions, earlier detection of occult shock and earlier start of goal-directed therapy.

European Journal of Emergency Medicine 2009, 16:318–322

Measurement of lactate in a prehospital setting is related to outcome

Paul A. van Beest[a,d,e], Peter Jan Mulder[c], Suparto Bambang Oetomo[c], Bert van den Broek[e], Michael A. Kuiper[d,f] and Peter E. Spronk[b,f]



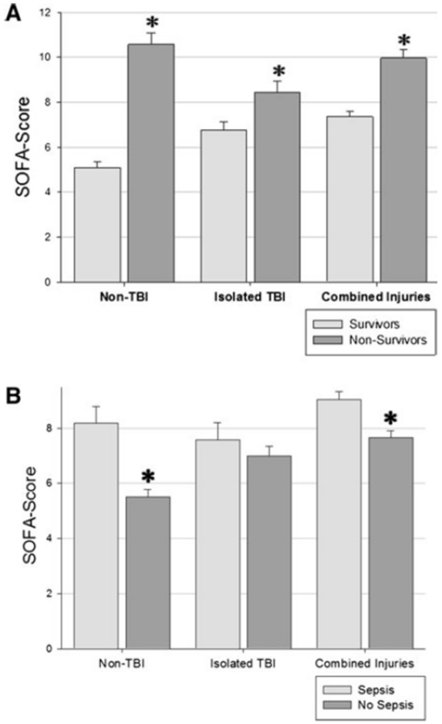
Published online: 27 July 2012

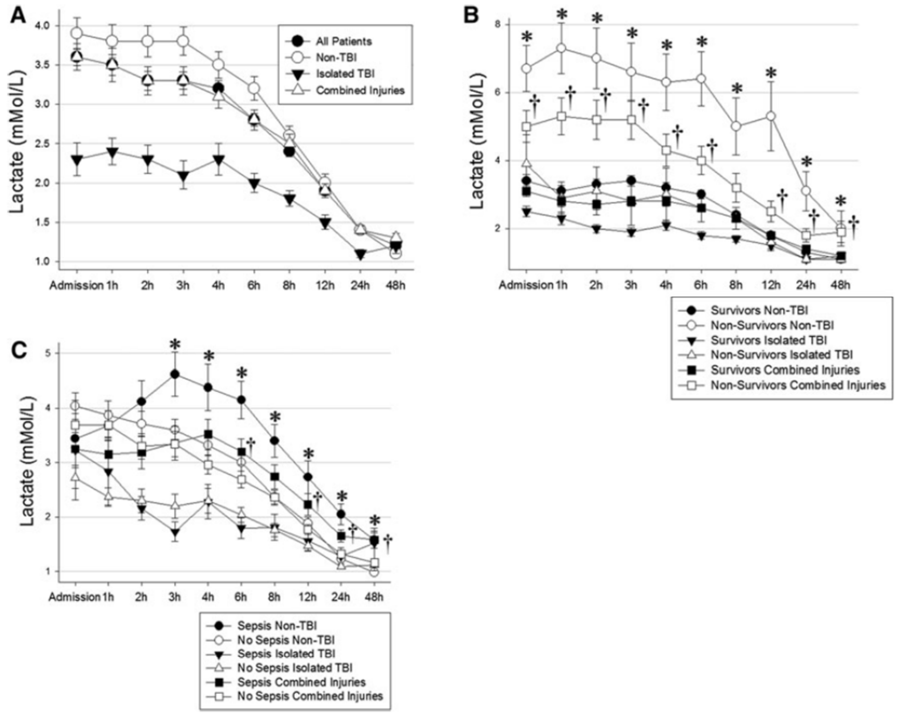
Eur J Trauma Emerg Surg
DOI 10.1007/s00068-012-0212-z

ORIGINAL ARTICLE

Serial lactate and admission SOFA scores in trauma: an analysis of predictive value in 724 patients with and without traumatic brain injury

C. Dübendorfer · A. T. Billeter ·
M. Keel · M. Turina





BURNS 33 (2007) 973–978

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/burns

Base deficit and lactate: Early predictors of morbidity and mortality in patients with burns

D. Andel [a], *L.-P. Kamolz* [b,*], *J. Roka* [b], *W. Schramm* [a], *M. Zimpfer* [a], *M. Frey* [b], *H. Andel* [a]

[a] *Department of Anesthesia and Intensive Care, Medical University Vienna, Vienna, Austria*
[b] *Division of Plastic and Reconstructive Surgery, Department of Surgery, Medical University Vienna, Waehringer Guertel 18-20, A-1090 Vienna, Austria*

Table 3 – Correlation coefficients between plasma lactate (PL), base deficit (BD) and total body surface area burned (TBSA)

	Correlation coefficient	Significance
PL-TBSA	0.45	NS
BD-TBSA	−0.48	NS

Table 4 – Details of 280 patients concerning additional inhalation injury

	Lactate	Base deficit		Mortality rate (%)
		Day 0	Day 1	
Inhal	4.0 ± 3.6	−5 ± 4.5	−2.4 ± 4.2	45
Non-Inhal	2.0 ± 1.9	−2.4 ± 3.9	−1.9 ± 4.1	25

Lactate in mmol/l and base deficit in mmol/l. Where applicable, data are presented as mean ± standard deviation.

Journal of Critical Care (2012) **xx**, xxx–xxx

ELSEVIER

Journal of Critical Care

Simplistic approach to prognosis after cardiopulmonary resuscitation—value of pH and lactate

Florian H. Seeger MD*, Moritz Toenne BSc, Ralf Lehmann MD, Joachim R. Ehrlich MD, FESC, FHRS, FAHA*

Medizinische Klinik III, Kardiologie, Universitätsklinikum Frankfurt am Main, Theodor-Stern-Kai 7, 60590 Frankfurt am Main, Germany

A: Univariate analysis overall cohort

	HR	95% CI	*P-value*
NSE after 72 h > 39 ng/ml	4.610	2.499 - 8.502	< .001
Lactate upon admission > 6.94 mmol/l (> 62.5 mg/dL)	2.772	1.953 - 3.936	< .001
pH upon admission < 7,21	2.706	1.912 - 3.830	< .001
Age ≥ 65 years	1.720	1.212 - 2.439	.002
Inital rhythm : non shockable	1.686	1.204 - 2.361	.002
Gender ♀	1.205	0.852 - 1.703	.292
Received bystander CPR	1.193	0.853 - 1.669	.303

B: Multivariate analysis overall cohort

	HR	95% CI	*P-value*
Inital rhythm: non shockable	1.078	0.749 - 1.550	.687
Age ≥ 65 years	1.854	1.297 - 2.651	.001
pH upon admission < 7,21	2.027	1.342 - 3.060	.001
Lactate upon admission > 6.94 mmol/l (> 62.5 mg/dL)	2.026	1.371 - 2.994	< .001

C: Multivariate analysis VF - shockable rhythm co

	HR	95% CI	*P-value*
Age ≥ 65 years	1.721	1.044 - 2.837	.033
Lactate upon admission > 6.94 mmol/l (> 62.5 mg/dL)	1.905	1.092 - 3.325	.023
pH upon admission < 7,21	3.129	1.795 - 5.452	< .001

Clinica Chimica Acta 413 (2012) 1512–1515

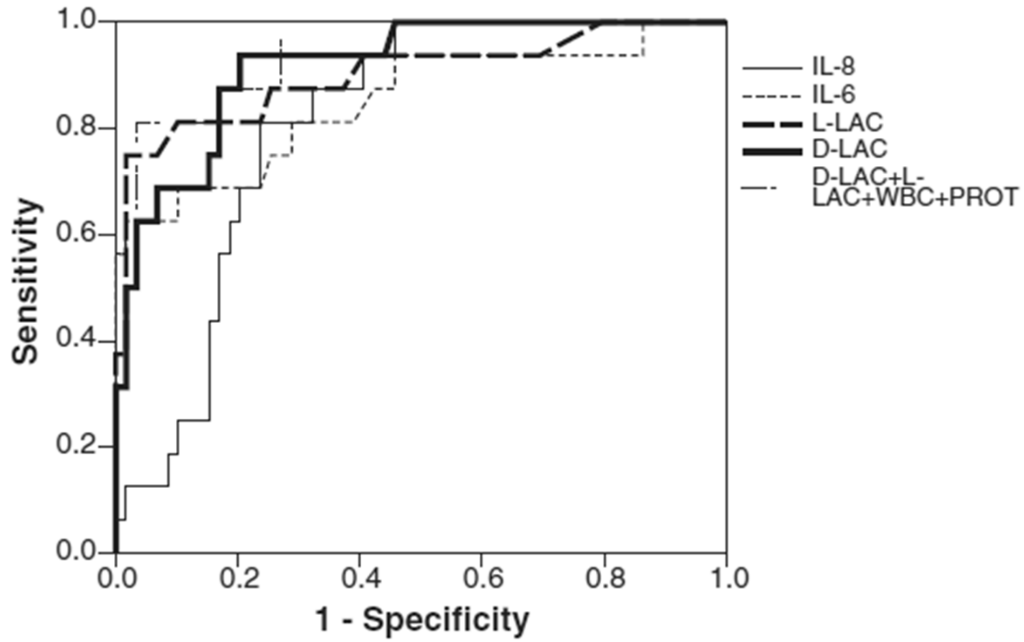
Contents lists available at SciVerse ScienceDirect

Clinica Chimica Acta

journal homepage: www.elsevier.com/locate/clinchim

The clinical diagnostic significance of cerebrospinal fluid D-lactate for bacterial meningitis

Zengqiang Chen [a,1], Yumin Wang [a,1], Aibing Zeng [a], Lijiang Chen [a], Ruihao Wu [a], Bicheng Chen [a],



PoCT (VBG)

TRIAGE – BIOMARKER

RIZIKÓ – BIOMARKER

DIAGNOSZTIKUS – BIOMARKER

MONITOR BIOMARKER

Lactate: Biomarker and Potential Therapeutic Target

Okorie Nduka Okorie, MD[a,b,*], Phil Dellinger, MD[c,d]

Crit Care Clin 27 (2011) 299–326

A SÜRGŐSSÉGI GYAKORLATBAN:

1. SÜRGŐSSÉGI UH-DIAGNOSZTIKA

   - FÓKUSZÁLT (CÉLZOTT) UH-DIAGNOSZTIKA (FOCUS)

   - „HAGYOMÁNYOS” UH-DIAGNOSZTIKA (RADIOLÓGUS)

2. UH-VEZÉRELT ESZKÖZÖS ELJÁRÁSOK

   - EREK KANÜLÁLÁSA UH-VEZÉRLÉSSEL

   - UH-VEZÉRELT (NONVASCULARIS) ESZKÖZÖS ELJÁRÁSOK

INTERVENCIÓS RADIOLÓGUS-SÜRGŐSSÉGI/INT.TER. ORVOS-TÁRSKLINIKUSOK, SZONOGRÁFUSOK

FOCUS - POCUS

- FOCUS: FÓKUSZÁLT (CÉLZOTT) UH-DIAGNOSZTIKA. *CÉLZOTT KÉRDÉSRE CÉLZOTT VÁLASZ US-SEL, NÉHÁNY PERC ALATT, FŐLEG A SÜRGŐSSÉGI ELLÁTÁSBAN.*
- POCUS (POINT OF CARE US): TÖBB ÁLLÁSPONT!

  - ÁGYMELLETTI US, A SÜRGŐSSÉGI ÉS INTENZÍV ELLÁTÁSBAN, A *FÓKUSZÁLT US-SEL SZINONIM FOGALOM*.

  - *DANSE SZERINT* (ECR 2013): POINT OF CARE US: *FŐLEG UH-VEZÉRELT ESZKÖZÖS ELJÁRÁSOK (EREK KANÜLÁLÁSA VAGY FOLYADÉK PUNCTIO), FOLYADÉKGYÜLEMEK BECSLÉSE (PL. FAST).*

  - (ACEP 2011) POINT-OF-CARE (POC) *EMERGENCY ULTRASOUND* (EUS).

ELŐNY

- PORTÁBILIS, KÖNNYEN HOZZÁFÉRHETŐ
- NININVAZÍV
- ISMÉTELHETŐ
- BEDSIDE
- OLCSÓ
- REALTIME, MULTIPLAN
- SUGÁRZÁS HIÁNYA

LIMITÁCIÓ

- VIZSGÁLÓ DEPENDENS
- BETEG ALKATÁTÓL FÜGG
- GYENGE PENTRÁCIÓ (SUBCUTIS, GÁZ …)
- EGYEDI (VARIÁBILITÁS – KÓRFOLYAMAT/BETEG)

Surg Clin N Am 91 (2011) 195–207

The RUSH Exam: Rapid Ultrasound in SHock in the Evaluation of the Critically Ill

Table 1
Rapid Ultrasound in SHock (RUSH) protocol: ultrasonographic findings seen with classic shock states

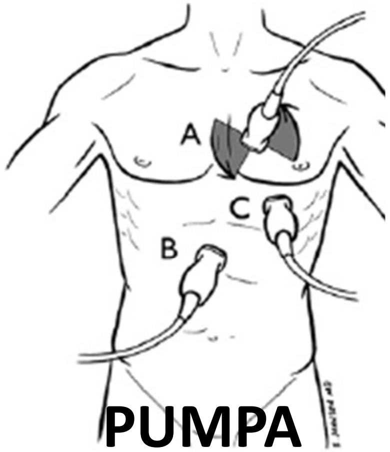
RUSH Evaluation	Hypovolemic Shock	Cardiogenic Shock	Obstructive Shock	Distributive Shock
Pump	Hypercontractile heart Small chamber size	Hypocontractile heart Dilated heart	Hypercontractile heart Pericardial effusion Cardiac tamponade RV strain Cardiac thrombus	Hypercontractile heart (early sepsis) Hypocontractile heart (late sepsis)
Tank	Flat IVC Flat jugular veins Peritoneal fluid (fluid loss) Pleural fluid (fluid loss)	Distended IVC Distended jugular veins Lung rockets (pulmonary edema) Pleural fluid Peritoneal fluid (ascites)	Distended IVC Distended jugular veins Absent lung sliding (pneumothorax)	Normal or small IVC (early sepsis) Peritoneal fluid (sepsis source) Pleural fluid (sepsis source)
Pipes	Abdominal aneurysm Aortic dissection	Normal	DVT	Normal

Emerg Med Clin N Am 28 (2010) 29–56

A) Parasternal Views
Long / Short Axis

B) Subxiphoid View

C) Apical View



PUMPA

1

2

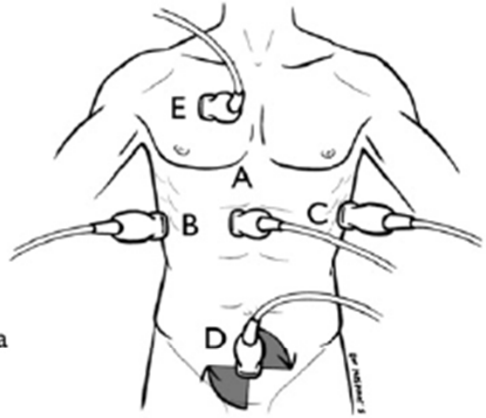
A) IVC Long Axis

B) FAST / RUQ
Add Pleural View

C) FAST / LUQ
Add Pleural View

D) FAST / Pelvis

E) Pneumothorax
Pulmonary Edema



TANK

A) Suprasternal Aorta

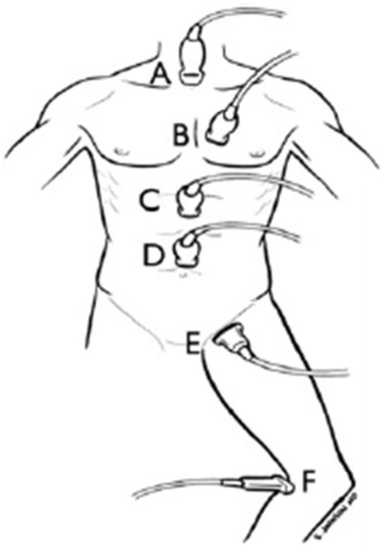
B) Parasternal Aorta

C) Epigastric Aorta

D) Supraumbilical Aorta

E) Femoral DVT

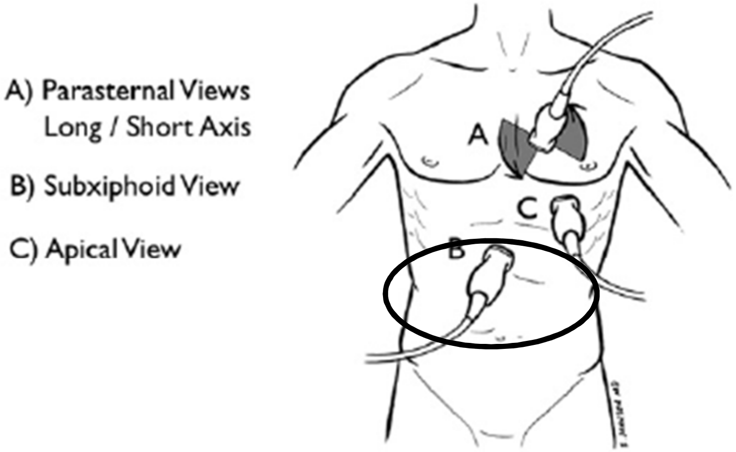
F) Popliteal DVT



3

CSÖVEK

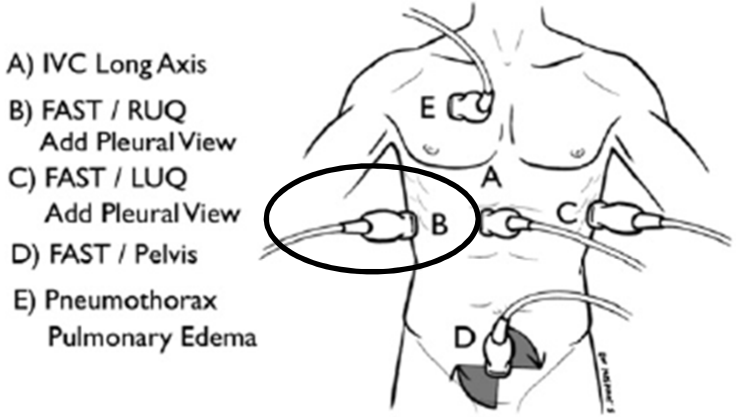
Emerg Med Clin N Am 28 (2010) 29–56



PUMPA

1

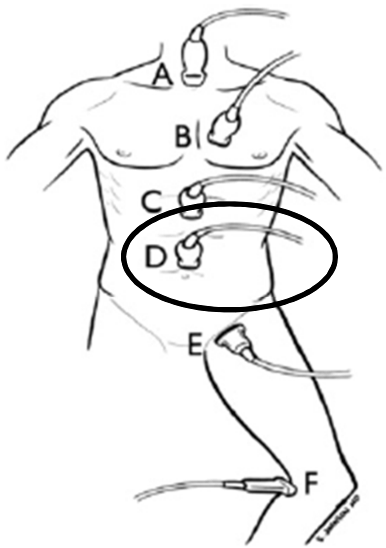
Emerg Med Clin N Am 28 (2010) 29–56



TANK 2

Emerg Med Clin N Am 28 (2010) 29–56

A) Suprasternal Aorta
B) Parasternal Aorta
C) Epigastric Aorta
D) Supraumbilical Aorta
E) Femoral DVT
F) Popliteal DVT



CSÖVEK **3**

Emerg Med Clin N Am 28 (2010) 29–56

Emergency Bedside Ultrasound to Detect Pneumothorax

Stewart Siu Wa Chan, MBBS (Syd), FRCSEd, FHKAM (Emergency Medicine)

Resuscitation 81 (2010) 1527–1533

Contents lists available at ScienceDirect

Resuscitation

journal homepage: www.elsevier.com/locate/resuscitation

Clinical paper

Focused echocardiographic evaluation in life support and peri-resuscitation of emergency patients: A prospective trial☆,☆☆

Raoul Breitkreutz[a,e,*], Susanna Price[b], Holger V. Steiger[c], Florian H. Seeger[d], Hendrik Ilper[e], Hanns Ackermann[f], Marcus Rudolph[g], Shahana Uddin[h], Markus A. Weigand[i], Edgar Müller[j], Felix Walcher[k], from the Emergency Ultrasound Working Group of the Johann Wolfgang Goethe-University Hospital, Frankfurt am Main[1]

FOCUSED ECHOCARDIOGRAPHIC EVALUATION IN LIFE SUPPORT (FEEL)

ELSEVIER

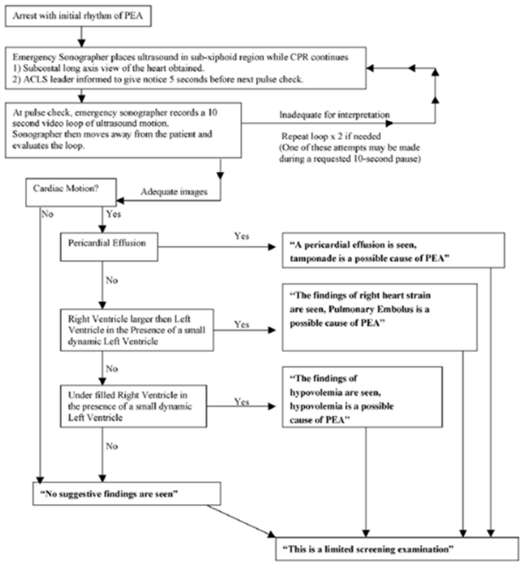
Resuscitation 67 (2005) 81–87

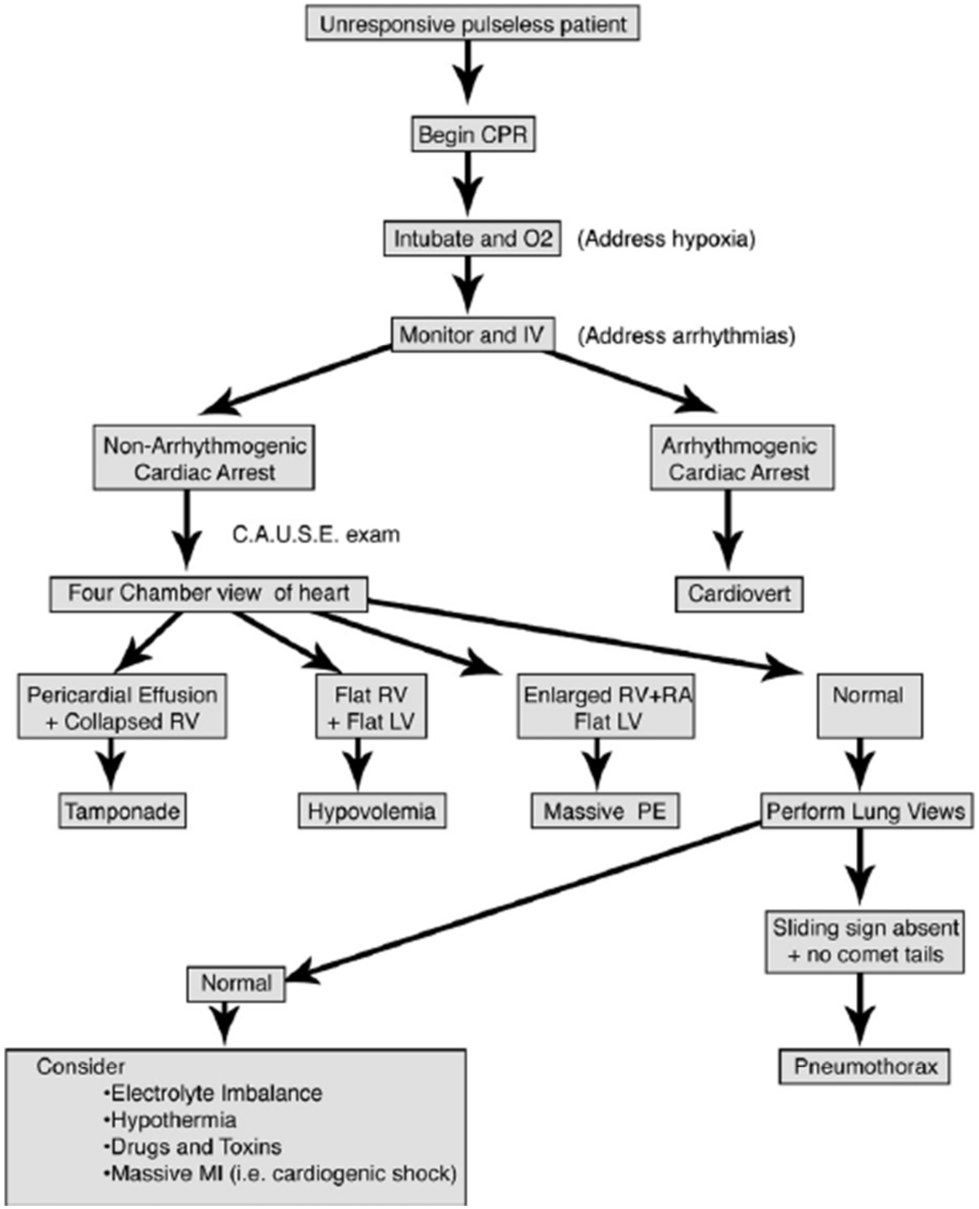
RESUSCITATION

www.elsevier.com/locate/resuscitation

Rapid cardiac ultrasound of inpatients suffering PEA arrest performed by nonexpert sonographers☆

Daniel F. Niendorff*, Athos J. Rassias, Robert Palac, Michael L. Beach,





Resuscitation (2008) 76, 198–206

available at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/resuscitation

ELSEVIER

RESUSCITATION

CLINICAL PAPER

C.A.U.S.E.: Cardiac arrest ultra-sound exam— A better approach to managing patients in primary non-arrhythmogenic cardiac arrest☆

Caleb Hernandez[a], Klaus Shuler[a], Hashibul Hannan[a], Chionesu Sonyika[a], Antonios Likourezos[a,*], John Marshall[a,b]

American Journal of Emergency Medicine (2011) **29**, 216–223

ELSEVIER

The American Journal of Emergency Medicine

www.elsevier.com/locate/ajem

Review

Usefulness of emergency ultrasound in nontraumatic cardiac arrest

Giovanni Volpicelli MD*

Abstract Treatment of nontraumatic cardiac arrest in the hospital setting depends on the recognition of heart rhythm and differential diagnosis of the underlying condition while maintaining a constant oxygenated blood flow by ventilation and chest compression. Diagnostic process relies only on patient's history, physical findings, and active electrocardiography. Ultrasound is not currently scheduled in the resuscitation guidelines. Nevertheless, the use of real-time ultrasonography during resuscitation has the potential to improve diagnostic accuracy and allows the physician a greater confidence in deciding aggressive life-saving therapeutic procedures. This article reviews the current opinions and literature about the use of emergency ultrasound during resuscitation of nontraumatic cardiac arrest. Cardiac and lung ultrasound have a great potential in identifying the reversible mechanical causes of pulseless electrical activity or asystole. Brief examination of the heart can even detect a real cardiac standstill regardless of electrical activity displayed on the monitor, which is a crucial prognostic indicator. Moreover, ultrasound can be useful to verify and monitor the tracheal tube placement. Limitation to the use of ultrasound is the need to minimize the no-flow intervals during mechanical cardiopulmonary resuscitation. However, real-time ultrasound can be successfully applied during brief pausing of chest compression and first pulse-check. Finally, lung sonographic examination targeted to the detection of signs of pulmonary congestion has the potential to allow hemodynamic noninvasive monitoring before and after mechanical cardiopulmonary maneuvers.

An evaluation of echo in life support (ELS): is it feasible? What does it add?

C Hayhurst,[1] C Lebus,[1] P R Atkinson,[1,2] R Kendall,[1] R Madan,[1] J Talbot,[2] P Ross,[2] D Lewis[3]

Emerg Med J 2011;**28**:119–121.

Table 1 Technical aspects of echo in life support

View	Number (% of total)	Adequate view (n)	Adequate view (%)	Number within single 10 s window	Success rate (adequate view and within 10 s)
Subxiphoid	40 (80%)	38	95%	38	95%
Parasternal	20 (40%)	19	95%	17	85%
Apical	4 (8%)	3	75%	2	50%
Combined	50	47	94%	45	90%

Table 2 Initial rhythm, clinical findings and number of interventions performed as a result of ultrasound

	Initial rhythm				Findings			
	Asystole	PEA	VF/VT	Data missing	VWM	Pericardial effusion	Data missing	Further intervention as a result of ELS
Traumatic arrests (n=7)	5 (71%)	2 (29%)	0	0	2 (29%)	0	1 (14%)	2 (29%)
Non-traumatic arrests (n=43)	15 (35%)	21 (49%)	6 (14%)	1 (2%)	18 (42%)	3 (7%)	0	5 (12%)
Total (n=50)	20 (41%)	23 (45%)	6 (12%)	1 (2%)	20 (40%)	3 (6%)	1 (2%)	7 (14%)

ELS, echo in life support; PEA, pulseless electrical activity; VF/VT, ventricular fibrillation/tachycardia; VWM, ventricular wall motion detected on ultrasound.

An evaluation of echo in life support (ELS): is it feasible? What does it add?

C Hayhurst,[1] C Lebus,[1] P R Atkinson,[1,2] R Kendall,[1] R Madan,[1] J Talbot,[2] P Ross,[2] D Lewis[3]

Emerg Med J 2011;**28**:119–121.

Key points

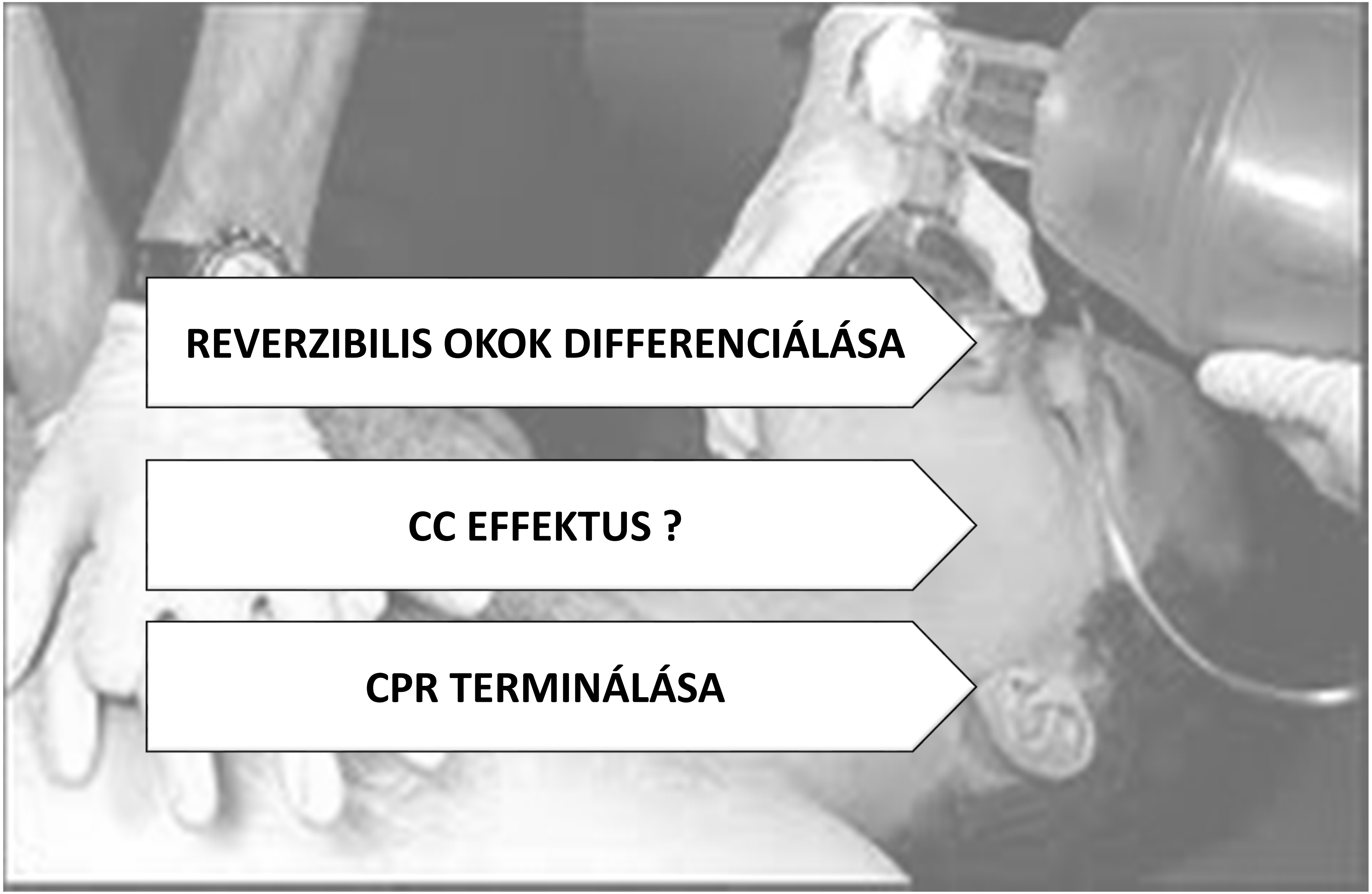
- Echo in life support
  —Is feasible
  —Can be performed within ALS guidelines
  —Does not compromise ALS delivery
  —Provides evidence of potentially reversible causes
- The absence of ventricular wall motion on a single ultrasound scan alone is not sufficient to direct stopping resuscitation; this remains a clinical decision.

REVERZIBILIS OKOK DIFFERENCIÁLÁSA

REVERSIBLE CAUSES

- Hypoxia
- Hypovolaemia ✓
- Hypo-/hyperkalaemia/metabolic
- Hypothermia

- Thrombosis - coronary or pulmonary ✓
- Tamponade - cardiac ✓
- Toxins
- Tension pneumothorax ✓


REVERZIBILIS OKOK DIFFERENCIÁLÁSA
CC EFFEKTUS ?
CPR TERMINÁLÁSA

The End?