



LUND  
UNIVERSITY



ttm2

# Status Epilepticus efter hjärtstopp -kan det behandlas?

Tobias Cronberg  
Adjungerad Professor  
Skånes Universitetssjukhus  
Lunds Universitet



# En patient har kramper efter hjärtstopp

## 2 viktiga frågor

- Vad innebär det för prognosen?
- (Hur) skall vi behandla?



# Terminologin för kramper efter hjärtstopp är förvirrande

Myoclonus*	Epileptiformt EEG	Terminologi
Ja	Ja	Myoklont status epilepticus (MSE)

\* Generaliserat > 30 min



# Kramper kan manifesteras sig kliniskt och/eller elektrografiskt

Myoclonus*	Epileptiform EEG	Terminology
Ja	Ja	Myoklont status epilepticus (MSE)
Ja	Nej/oklart	Status Myoclonus

\* Generaliserat > 30 min



# Kramper kan manifesteras sig kliniskt och/eller elektrografiskt

Myoclonus*	Epileptiform EEG	Terminology
Ja	Ja	Myoklont status epilepticus (MSE)
Ja	Nej/oklart	Status Myoclonus
Nej	Ja	Non-Konvulsivt Status Epilepticus (NCSE)

\* Generaliserat > 30 min



# Hur vanligt är kliniska kramper?

## TTM studien 2010-13

**Table 2**

Frequency of clinical seizures.

Seizure type	TTM33 (n = 473)	TTM36 (n = 466)	All (n = 939)
Any seizure	147 (31%)	121 (26%)	268 (29%)
Myoclonic	132 (28%)	108 (23%)	240 (26%)
Status myoclonus	37 (8%)	36 (8%)	73 (8%)
Focal myoclonus	48 (10%)	33 (7%)	81(8%)
Tonic-clonic	37 (8%)	34 (7%)	71 (8%)
Tonic-clonic status	12 (3%)	8 (2%)	20 (2%)
Focal tonic-clonic seizures	6 (1%)	5 (1%)	11 (1%)
Combination <sup>a</sup>	22 (5%)	21 (5%)	43(5%)



# Hur vanligt är kliniska kramper?

## TTM studien 2010-13

**Table 2**

Frequency of clinical seizures.

Seizure type	TTM33 (n = 473)	TTM36 (n = 466)	All (n = 939)
Any seizure	147 (31%)	121 (26%)	268 (29%)
Myoclonic	132 (28%)	108 (23%)	240 (26%)
Status myoclonus	37 (8%)	36 (8%)	73 (8%)
Focal myoclonus	48 (10%)	33 (7%)	81(8%)
Tonic-clonic	37 (8%)	34 (7%)	71 (8%)
Tonic-clonic status	12 (3%)	8 (2%)	20 (2%)
Focal tonic-clonic seizures	6 (1%)	5 (1%)	11 (1%)
Combination <sup>a</sup>	22 (5%)	21 (5%)	43(5%)



# Hur vanligt är kliniska kramper?

## TTM studien 2010-13

**Table 2**

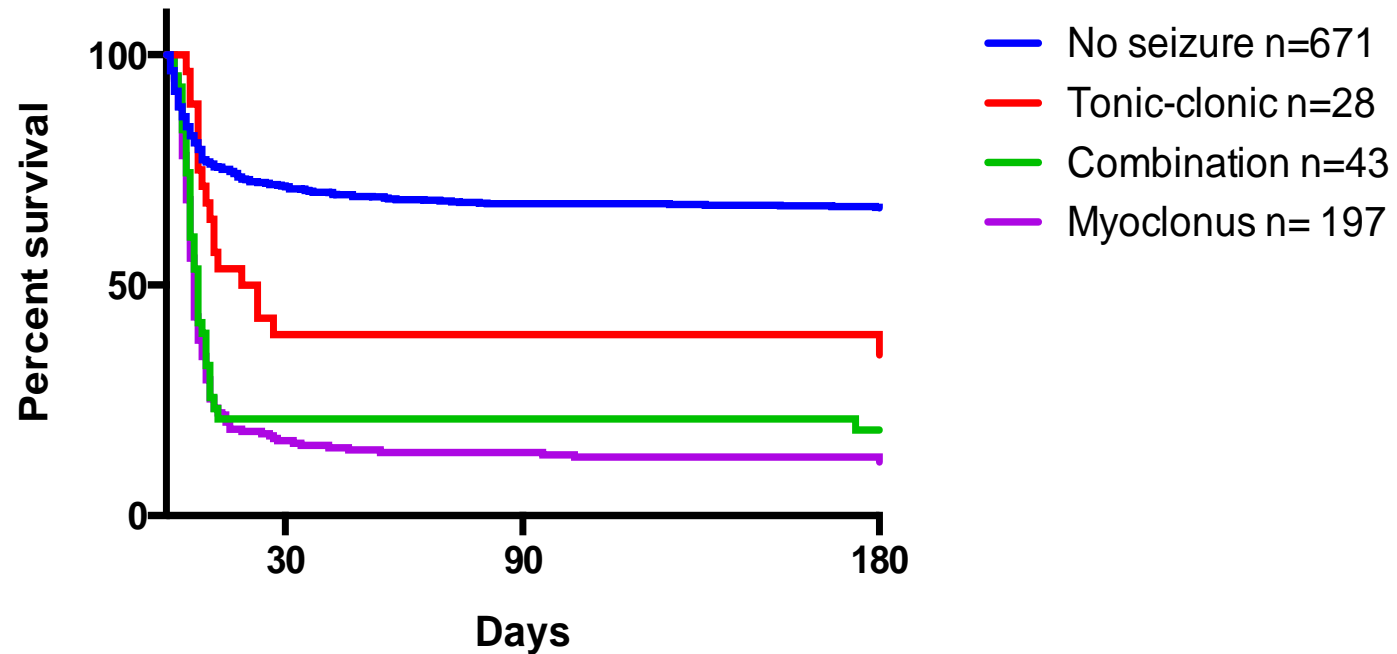
Frequency of clinical seizures.

Seizure type	TTM33 (n = 473)	TTM36 (n = 466)	All (n = 939)
Any seizure	147 (31%)	121 (26%)	268 (29%)
Myoclonic	132 (28%)	108 (23%)	240 (26%)
Status myoclonus	37 (8%)	36 (8%)	73 (8%)
Focal myoclonus	48 (10%)	33 (7%)	81(8%)
Tonic-clonic	37 (8%)	34 (7%)	71 (8%)
Tonic-clonic status	12 (3%)	8 (2%)	20 (2%)
Focal tonic-clonic seizures	6 (1%)	5 (1%)	11 (1%)
Combination <sup>a</sup>	22 (5%)	21 (5%)	43(5%)

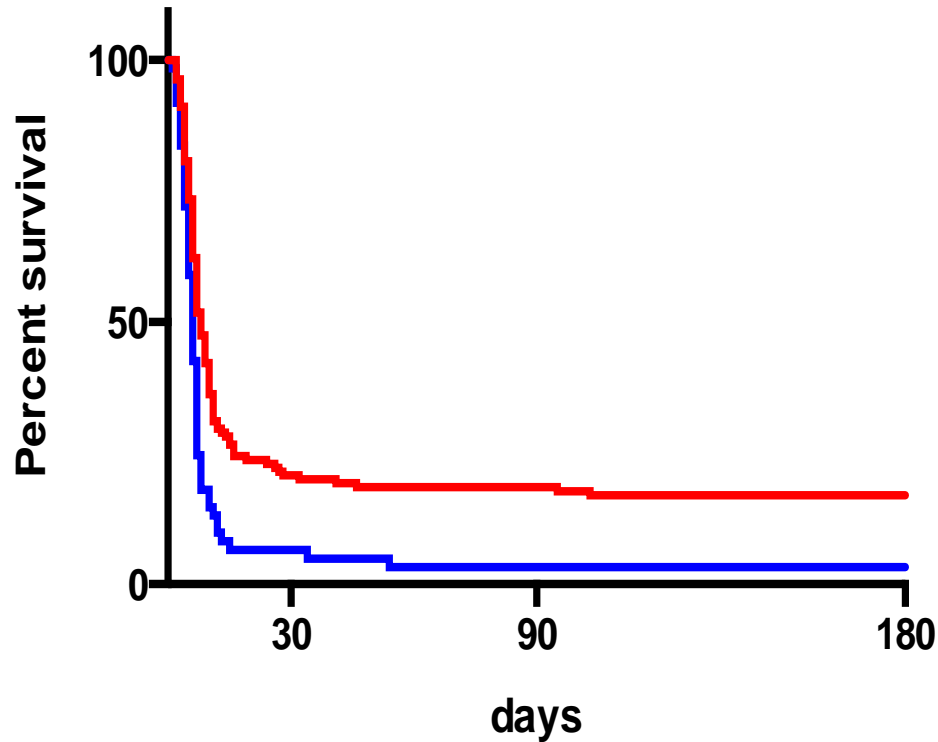




# Kramper är ett dåligt tecken



# Status myoklonus är ett **mycket** dåligt tecken



60/61 med status myoklonus fick ett dåligt utfall



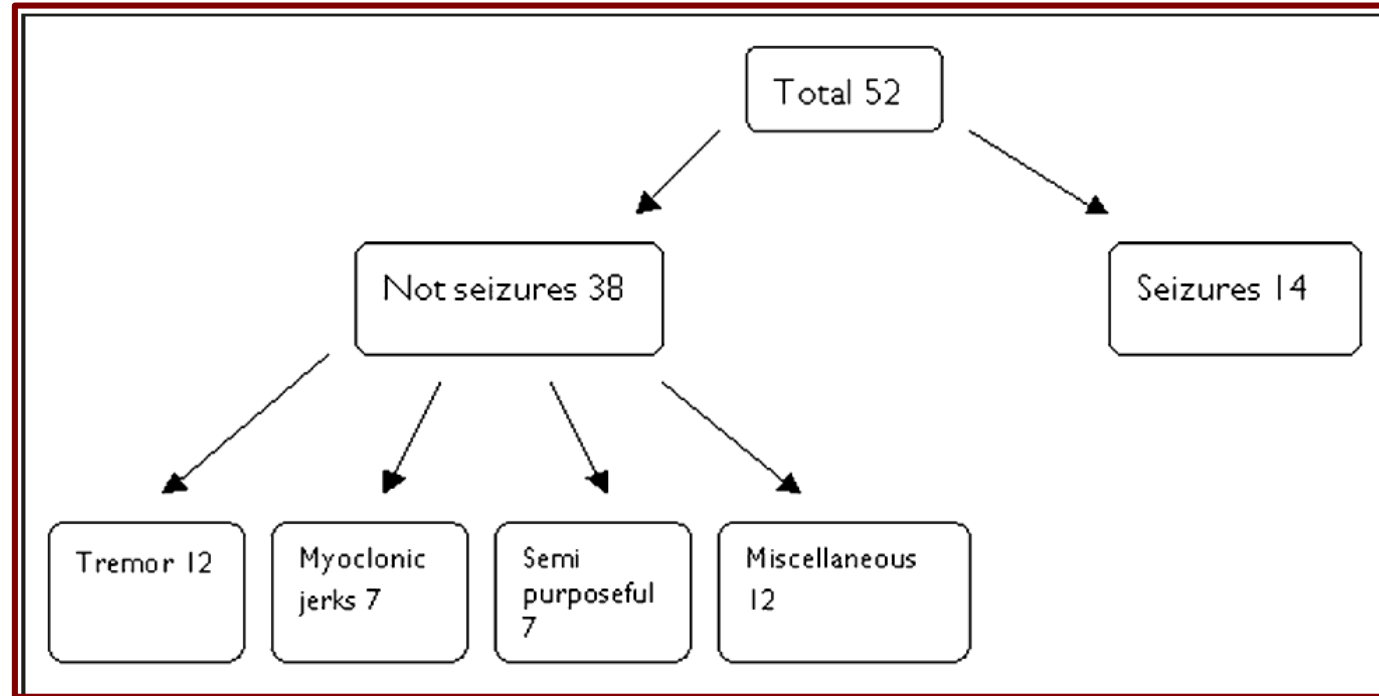
# Modern intensivvård försvårar vår neurologiska bedömning



- Temperaturreglering till 33 eller 36°C
- Sedation, analgesi och muskelrelaxation
- Förlångsammad metabolism av läkemedel
- Kliniska kramper maskeras



# What's shaking in the ICU?

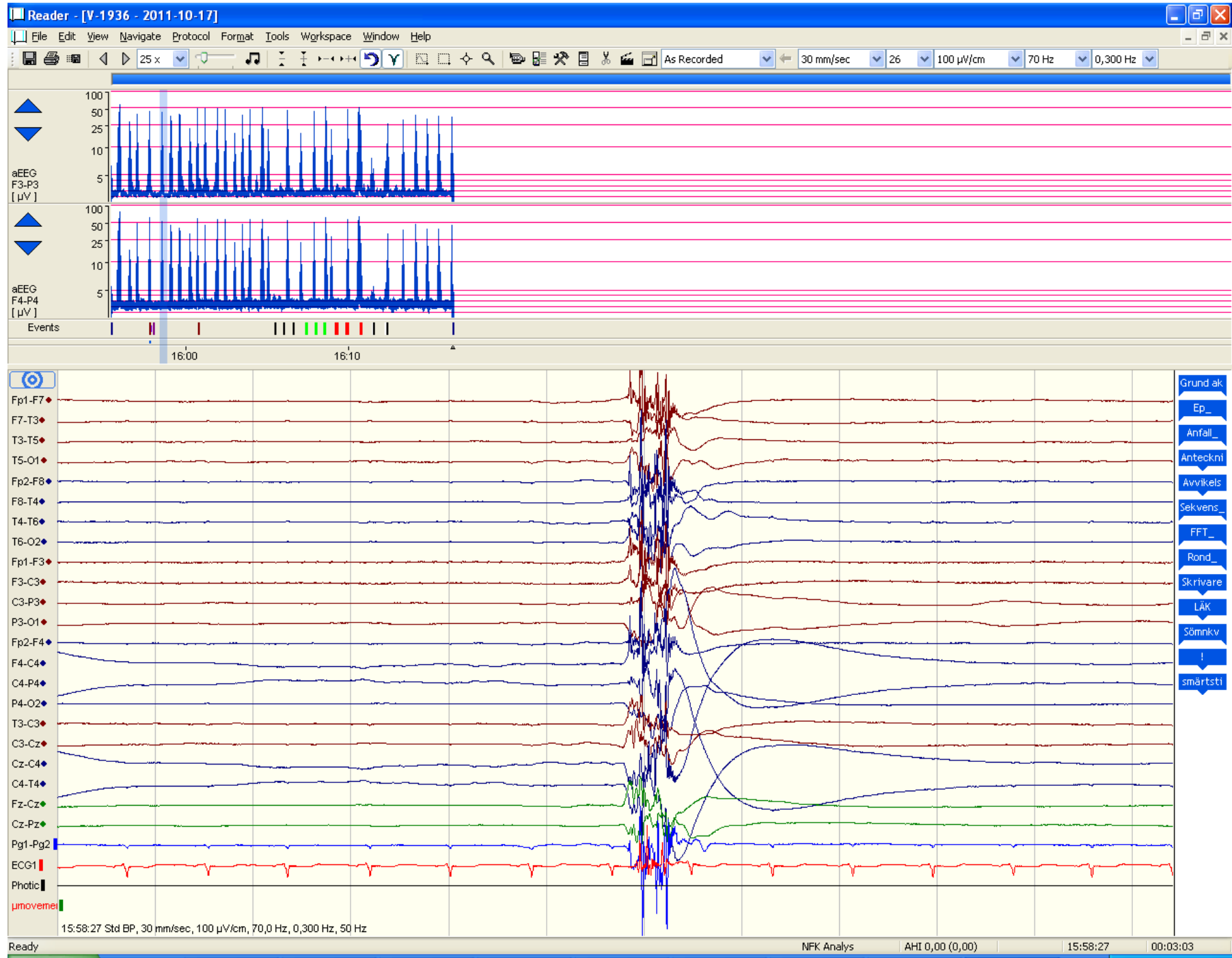


- Vuxna patienter på IVA
- Video-EEG på indikationen “misstänkt kramp”
- 73% var inte epileptiskt



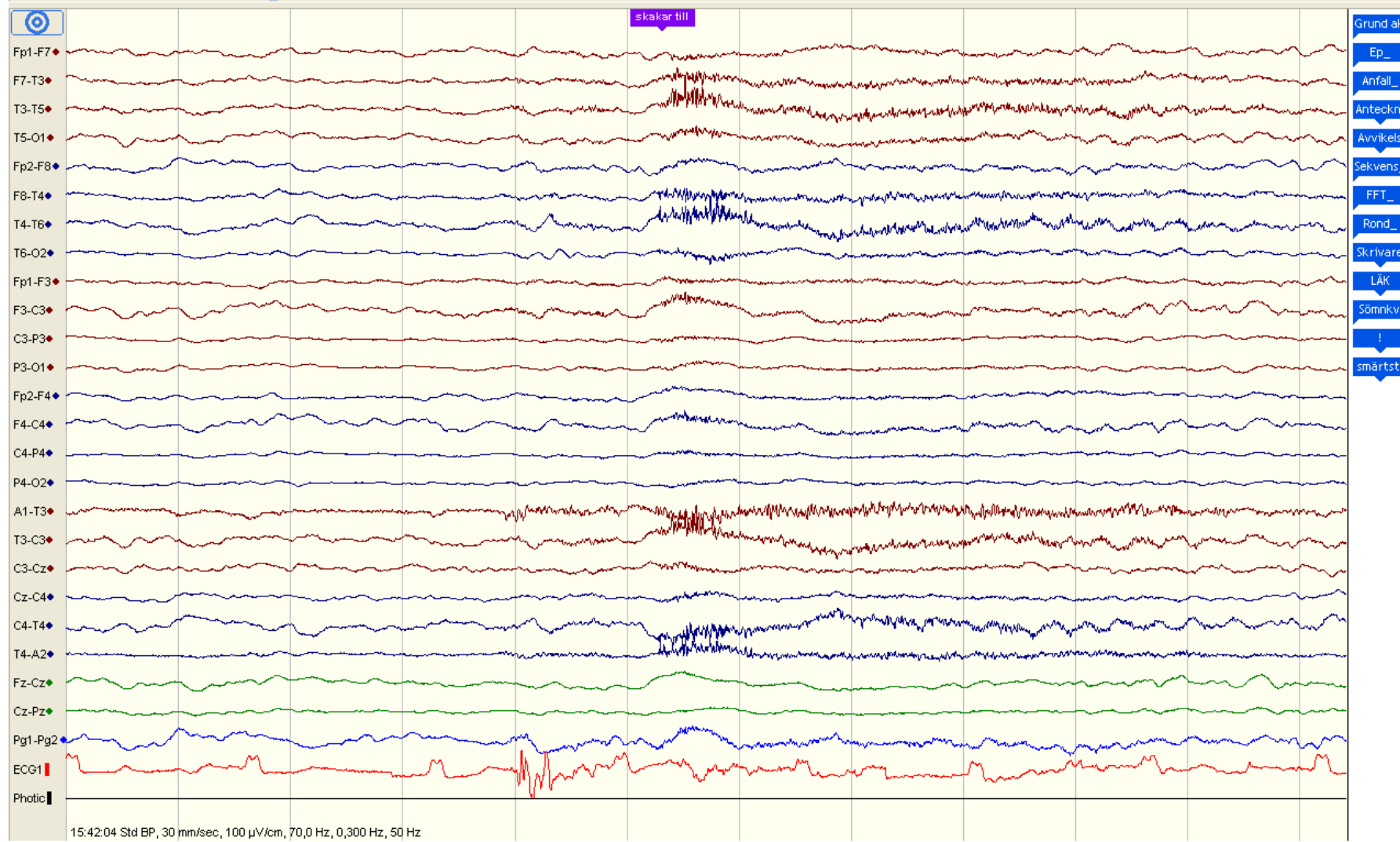
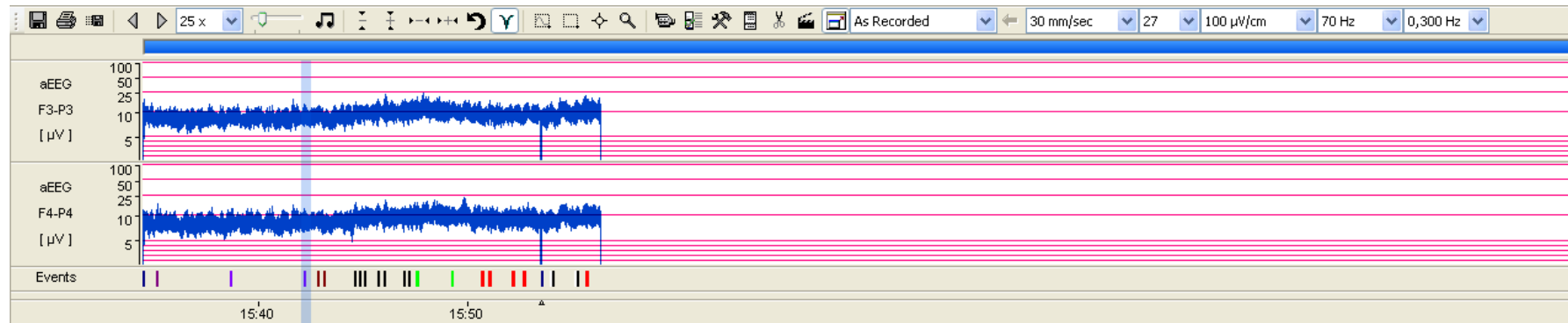
# What's shaking in the ICU?





# What's shaking in the ICU?

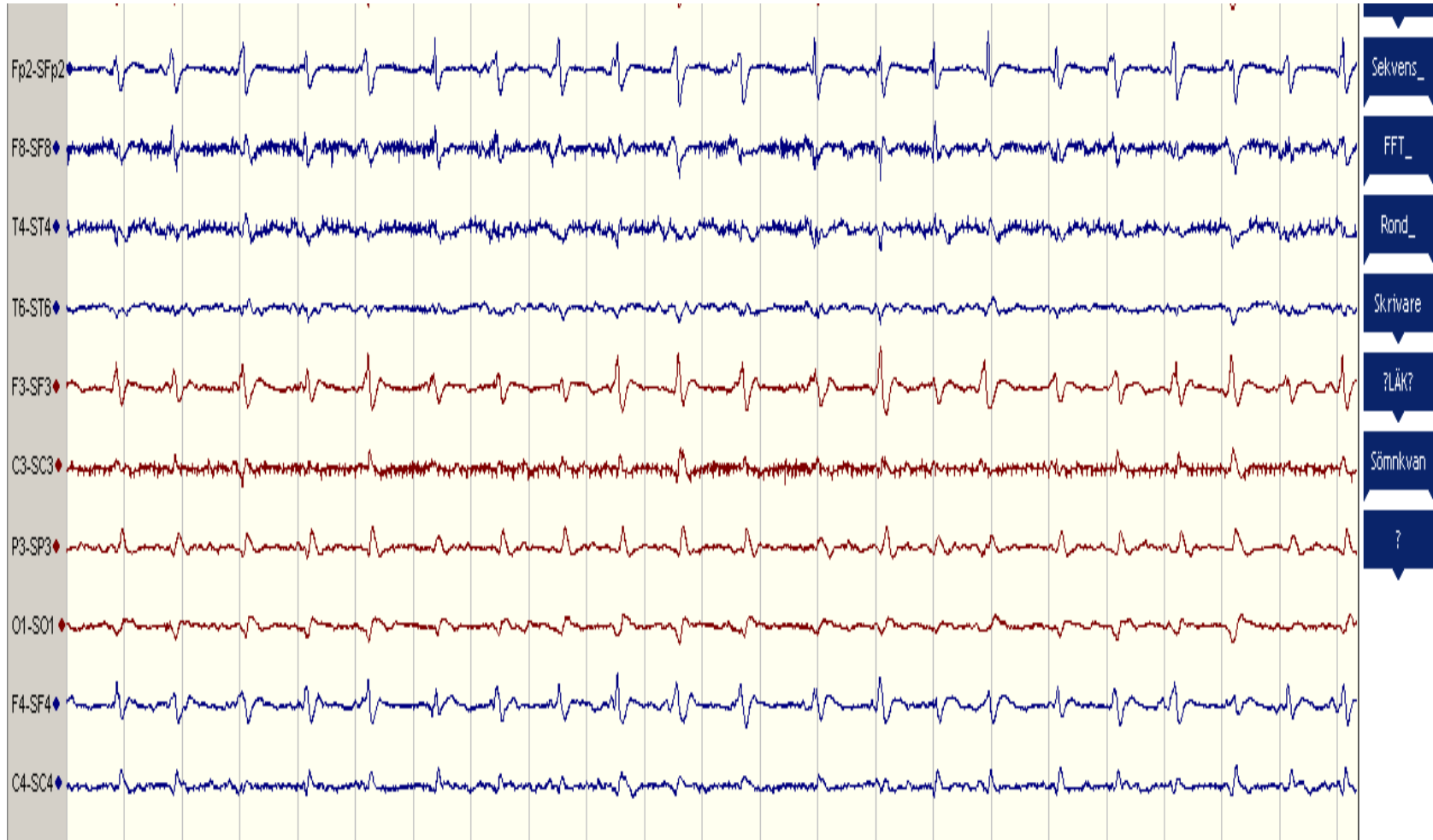






# What's shaking in the ICU?

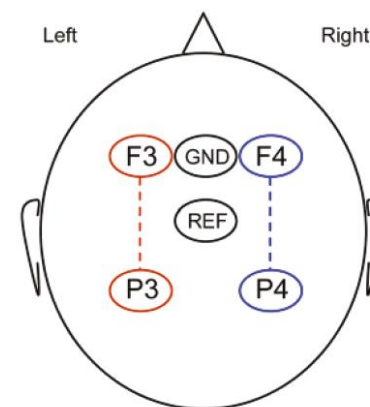




# Kontinuerligt EEG kan användas för att följa hjärnans återhämtning



- Förenklat montage appliceras av IVA-sköterskor
- Distanstolkning av neurofysiolog



# Clinically Distinct Electroencephalographic Phenotypes of Early Myoclonus after Cardiac Arrest

Jonathan Elmer, MD, MS,<sup>1,2</sup> Jon C. Rittenberger, MD, MS,<sup>1</sup> John Faro,<sup>3</sup>  
Bradley J. Molyneaux, MD, PhD,<sup>2,4</sup> Alexandra Popescu, MD,<sup>4</sup>  
Clifton W. Callaway, MD, PhD,<sup>1</sup> and Maria Baldwin, MD,<sup>5</sup>  
for the Pittsburgh Post-Cardiac Arrest Service

**Objective:** We tested the hypothesis that there are readily classifiable electroencephalographic (EEG) phenotypes of early postanoxic multifocal myoclonus (PAMM) that develop after cardiac arrest.

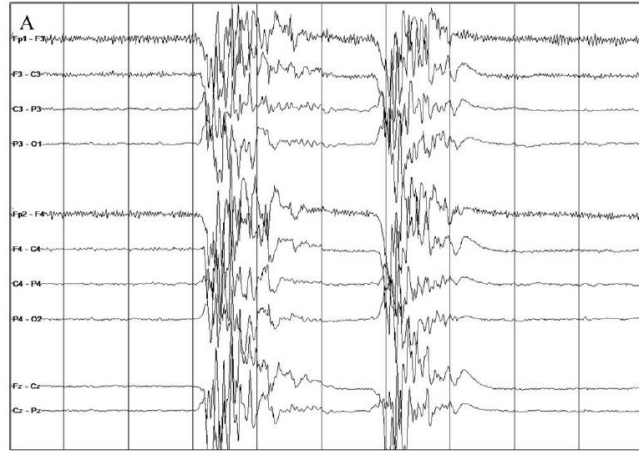
**Methods:** We studied a cohort of consecutive comatose patients treated after cardiac arrest from January 2012 to February 2015. For patients with clinically evident myoclonus before awakening, 2 expert physicians reviewed and classified all EEG recordings. Major categories included: Pattern 1, suppression-burst background with high-amplitude polyspikes in lockstep with myoclonic jerks; and Pattern 2, continuous background with narrow, vertex spike-wave discharges in lockstep with myoclonic jerks. Other patterns were subcortical myoclonus and unclassifiable. We compared population characteristics and outcomes across these EEG subtypes.

**Results:** Overall, 401 patients were included, of whom 69 (16%) had early myoclonus. Among these patients, Pattern 1 was the most common, occurring in 48 patients (74%), whereas Pattern 2 occurred in 8 patients (12%). The remaining patients had subcortical myoclonus (n = 2, 3%) or other patterns (n = 7, 11%). No patients with Pattern 1, subcortical myoclonus, or other patterns survived with favorable outcome. By contrast, 4 of 8 patients (50%) with Pattern 2 on EEG survived, and 4 of 4 (100%) survivors had favorable outcomes despite remaining comatose for 1 to 2 weeks postarrest.

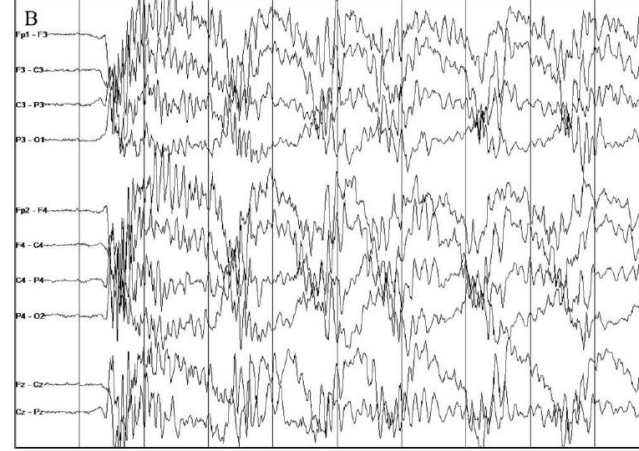
**Interpretation:** Early PAMM is common after cardiac arrest. We describe 2 distinct patterns with distinct prognostic significances. For patients with Pattern 1 EEGs, it may be appropriate to abandon our current clinical standard of aggressive therapy with conventional antiepileptic therapy in favor of early limitation of care or novel neuroprotective strategies.



# Type 1: Burst suppression with time-locked myoclonus



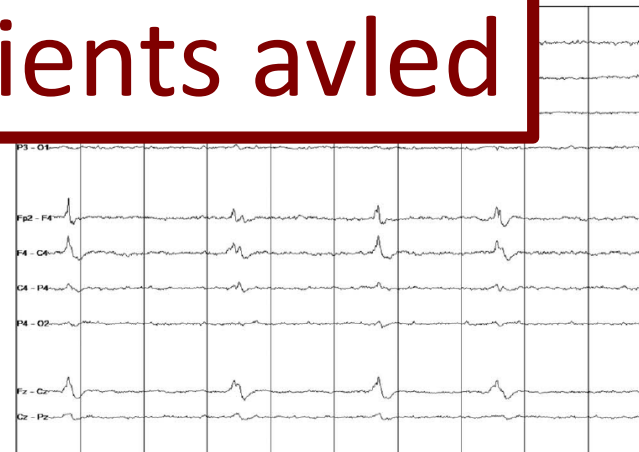
D0



D1



D2



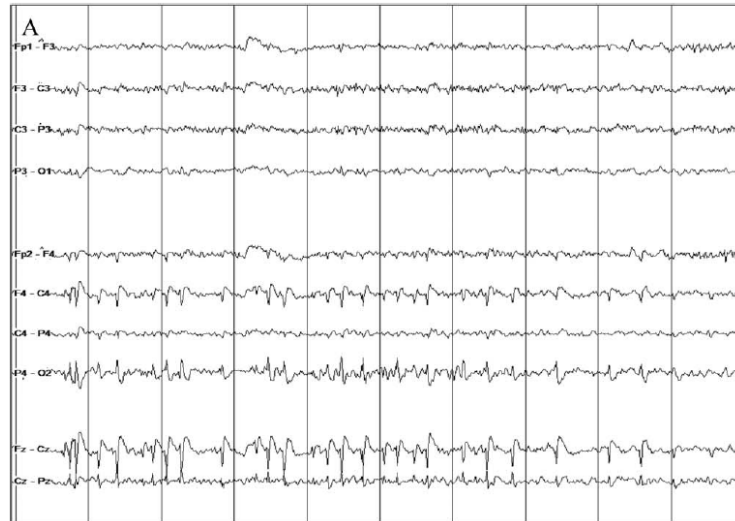
D3

48/48 patients avled

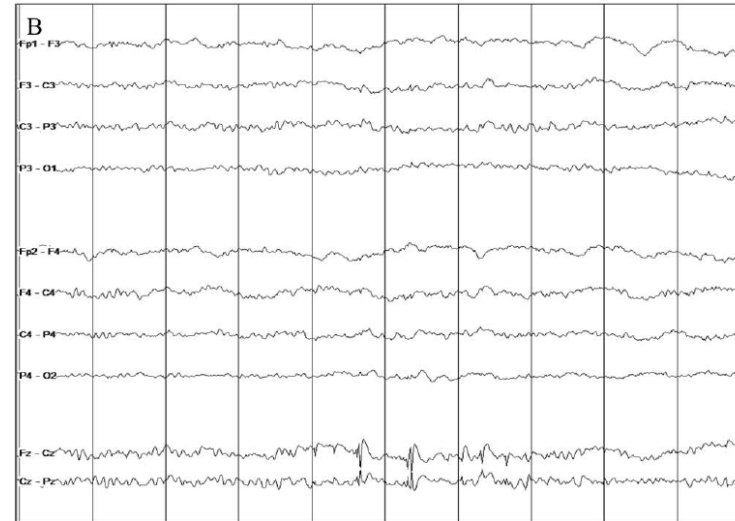




# Type 2: Spike-wave with time-locked myoclonus



D0



D40

4/8 patienter överlevde  
Alla hade Lance Adams action-myoclonus



# Postanoxiskt status epilepticus (PSE)

- Ingen konsensus rörande EEG-kriterier för PSE.
- Incidensen beror på klassifikationen.
- Kan diagnostiseras med EEG hos 18-38% under de första dagarna efter hjärtstopp

Rossetti et al, *Neurology*, 2009

Legriel et al, *Epilepsia*, 2010

Mani, *Resuscitation*, 2012

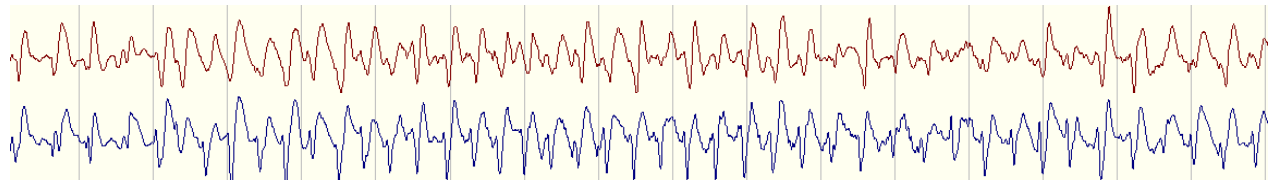
Rittenberger, *Neurocrit care*, 2012

Dragancea, *Epilepsy and behaviour*, 2015

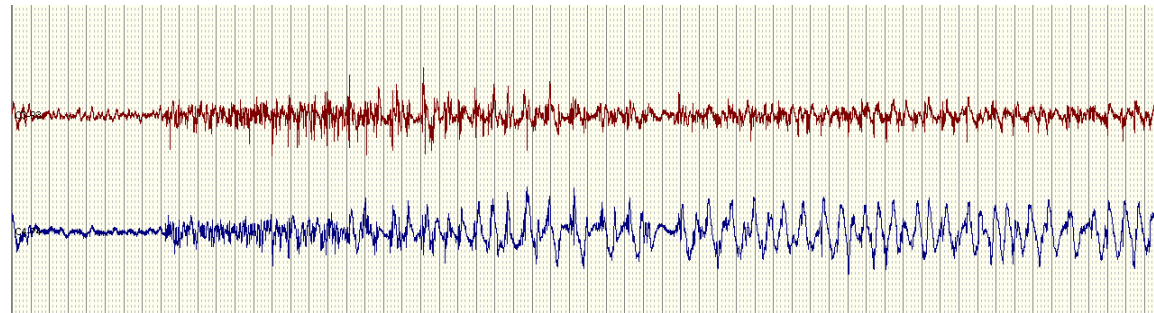


# American Clinical Neurophysiology Society's Critical Care EEG Terminology: 2012 version

- Unequivocal electrographic seizure
  - Generalized spike-wave discharges at 3/s or faster



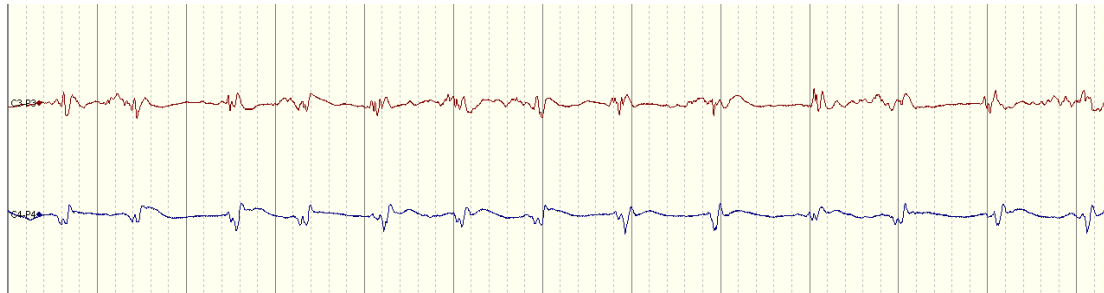
- clearly evolving discharges of any type that reach a frequency  $>4/s$





# American Clinical Neurophysiology Society's Critical Care EEG Terminology: 2012 version

- Possible ictal/interictal patterns
  - Generalized spike and wave patterns slower than 3/s; and evolving discharges that remain slower than or equal to 4/s.



Typical PSE  
1-2 /s

“This does not imply that these patterns are not ictal, but simply that they may or may not be.”





## A definition and classification of status epilepticus – Report of the ILAE Task Force on Classification of Status Epilepticus

\*†‡Eugen Trinka, §Hannah Cock, ¶Dale Hesdorffer, #Andrea O. Rossetti, \*\*Ingrid E. Scheffer, ††Shlomo Shinnar, ‡‡Simon Shorvon, and §§Daniel H. Lowenstein

*Epilepsia*, 56(10):1515–1523, 2015  
doi: 10.1111/epi.13121

### Table 3. Currently indeterminate conditions (or “boundary syndromes”)

Epileptic encephalopathies

Coma with non evolving epileptiform EEG pattern<sup>a</sup>

Behavioral disturbance (e.g., psychosis) in patients with epilepsy

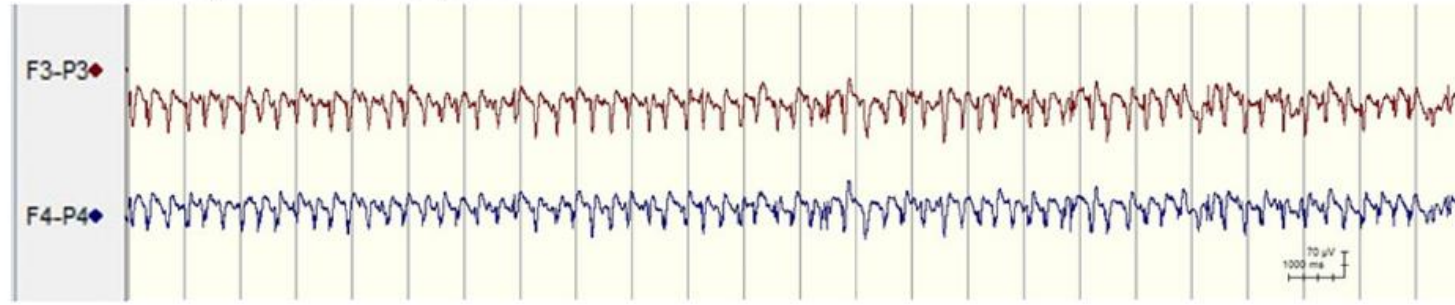
Acute confusional states, (e.g., delirium) with epileptiform EEG patterns

<sup>a</sup>Lateralized and generalized periodic discharges with monotonous appearance are not considered as evolving EEG patterns.<sup>26,27</sup>

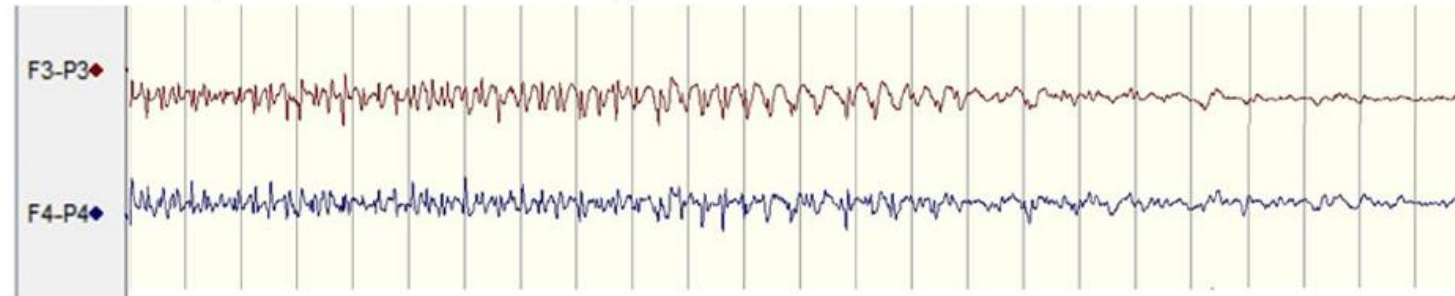


# “Otvetydigt” eller “möjligt” PSE

a Unequivocal ESE  $\geq 3\text{Hz}$



b Unequivocal ESE with evolving seizures



c Possible ESE

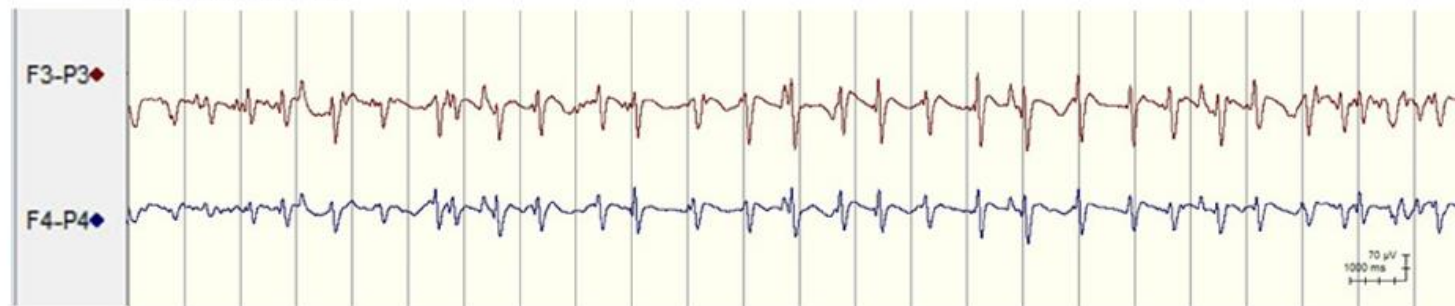
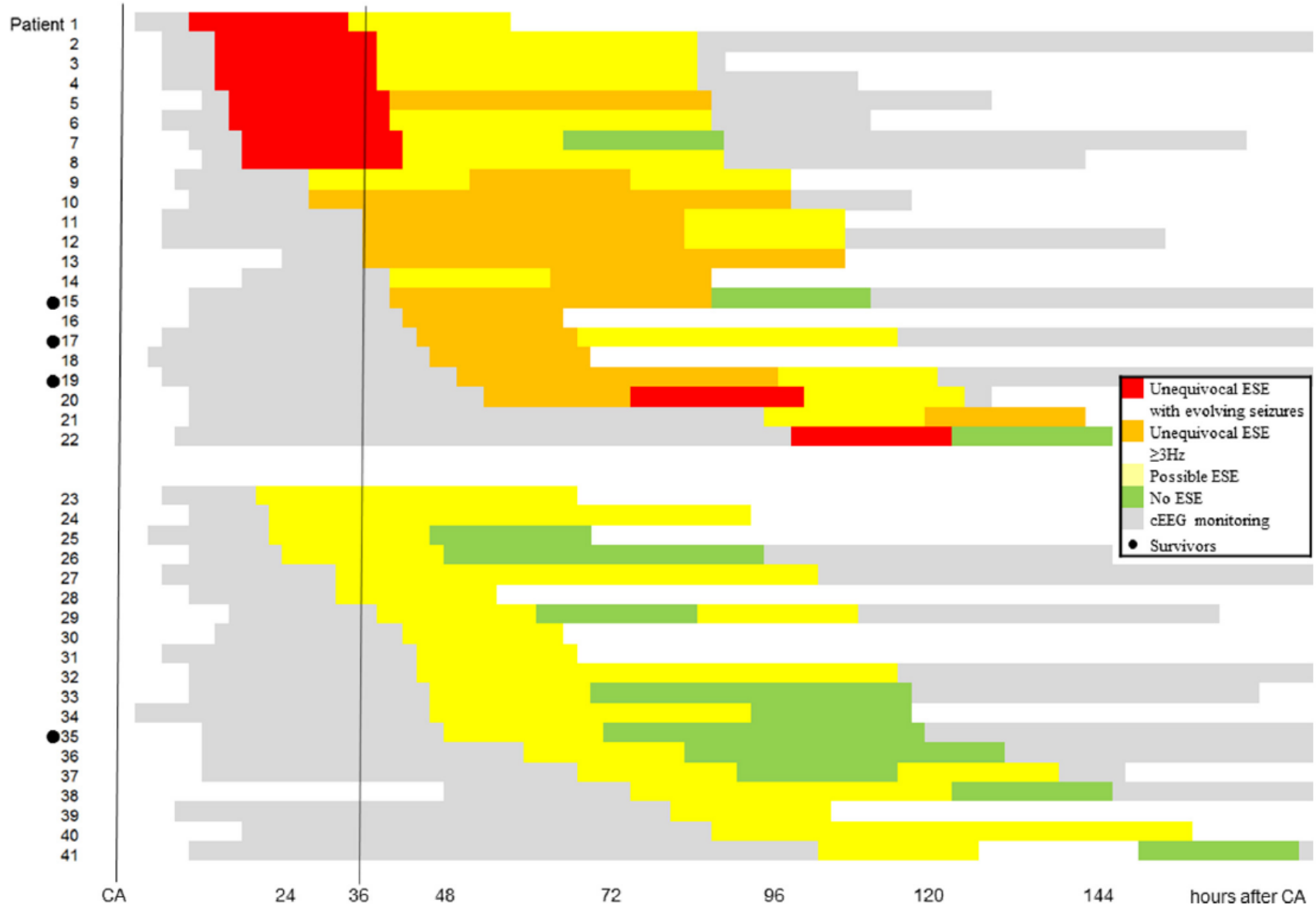


Fig. 1. Electrographic status epilepticus patterns on simplified 2-channel cEEG.



# Otvetydiga mönster ses tidigt



# PSE-patienter har dålig prognos

- 0-10% vaknar
- De flesta överlevare har en funktionsnedsättning (CPC 2-3)
- Bra utfall (CPC 1-2) hos 0-8%

Rossetti et al, *Neurology*, 2009

Legriel et al, *Epilepsia*, 2010

Rittenberger, *Neurocrit care*, 2012

Mani, *Resuscitation*, 2012

Dragancea, *Epilepsy and behaviour*, 2015



# Vad utmärker överlevarna?

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Data collection	Retrospective	Retrospective	Retrospective	Prospective	Prospective	Prospective
Age, y	60	61	55	53	68	58
Female	Yes	Yes	No	No	No	No
CA due to VF	Yes	Yes	No	Yes	Yes	Yes
Time to ROSC, min	32	45	17	20	20	21
Cardiac etiology	Yes	Yes	Yes	Yes	Yes	Yes
Preserved BR	Yes	Yes	NA	Yes	Yes	Yes
Absent MR	Yes	No	NA	No	No	Yes
SE diagnosis delay from CA, d	2	4	2	2	3	9
SE clinically (myoclonus)	Yes	No	Yes	Yes	No	No
EEG description	Repetitive diffuse sharp waves (1.5 Hz), bilateral	Repetitive diffuse sharp waves (2-3 Hz), bilateral	Repetitive diffuse sharp waves (2 Hz), bilateral	Sharp waves, spike-waves (2 Hz), L>R; SIRPID	Frontal evolving rhythmic sharp waves (3 Hz), bilateral	Frontal poly-spike-waves (2-3 Hz), R>L
<b>EEG reactivity</b>	<b>Yes</b>	<b>Yes</b>	<b>Yes</b>	<b>Yes</b>	<b>Yes</b>	<b>Yes</b>
PSE duration (d, on EEG)	3	2	2	2	5	3
Preserved N20 on SSEP	Yes	NA	Yes	Yes	Yes	Yes
AEDs	PRO, VPA, CLZ	VPA, CLZ, LEV	PRO, PHT, LEV, CLZ	VPA, PHT, LEV, CLZ	PRO, VPA, LEV, CLZ	VPA, LEV, CLZ
CPC outcome at 6 mo	3	2	2	1	3; death on day 18 (sepsis)	2
LA syndrome	Yes	No	No	Yes	No	No



# Vad utmärker överlevarna?

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Data collection	Retrospective	Retrospective	Retrospective	Prospective	Prospective	Prospective
Age, y	60	61	55	53	68	58
Female	Yes	Yes	No	No	No	No
CA due to VF	Yes	Yes	No	Yes	Yes	Yes
Time to ROSC, min	32	45	17	20	20	21
Cardiac etiology	Yes	Yes	Yes	Yes	Yes	Yes
Preserved BR	Yes	Yes	NA	Yes	Yes	Yes
Absent MR	Yes	No	NA	No	No	Yes
SE diagnosis delay from CA, d	2	4	2	2	3	9
SE clinically (myoclonus)	Yes	No	Yes	Yes	No	No
EEG description	Repetitive diffuse sharp waves (1.5 Hz), bilateral	Repetitive diffuse sharp waves (2-3 Hz), bilateral	Repetitive diffuse sharp waves (2 Hz), bilateral	Sharp waves, spike-waves (2 Hz), L>R; SIRPID	Frontal evolving rhythmic sharp waves (3 Hz), bilateral	Frontal poly-spike-waves (2-3 Hz), R>L
EEG reactivity	Yes	Yes	Yes	Yes	Yes	Yes
PSE duration (d, on EEG)	3	2	2	2	5	3
Preserved N20 on SSEP	Yes	NA	Yes	Yes	Yes	Yes
AEDs	PRO, VPA, CLZ	VPA, CLZ, LEV	PRO, PHT, LEV, CLZ	VPA, PHT, LEV, CLZ	PRO, VPA, LEV, CLZ	VPA, LEV, CLZ
CPC outcome at 6 mo	3	2	2	1	3; death on day 18 (sepsis)	2
LA syndrome	Yes	No	No	Yes	No	No





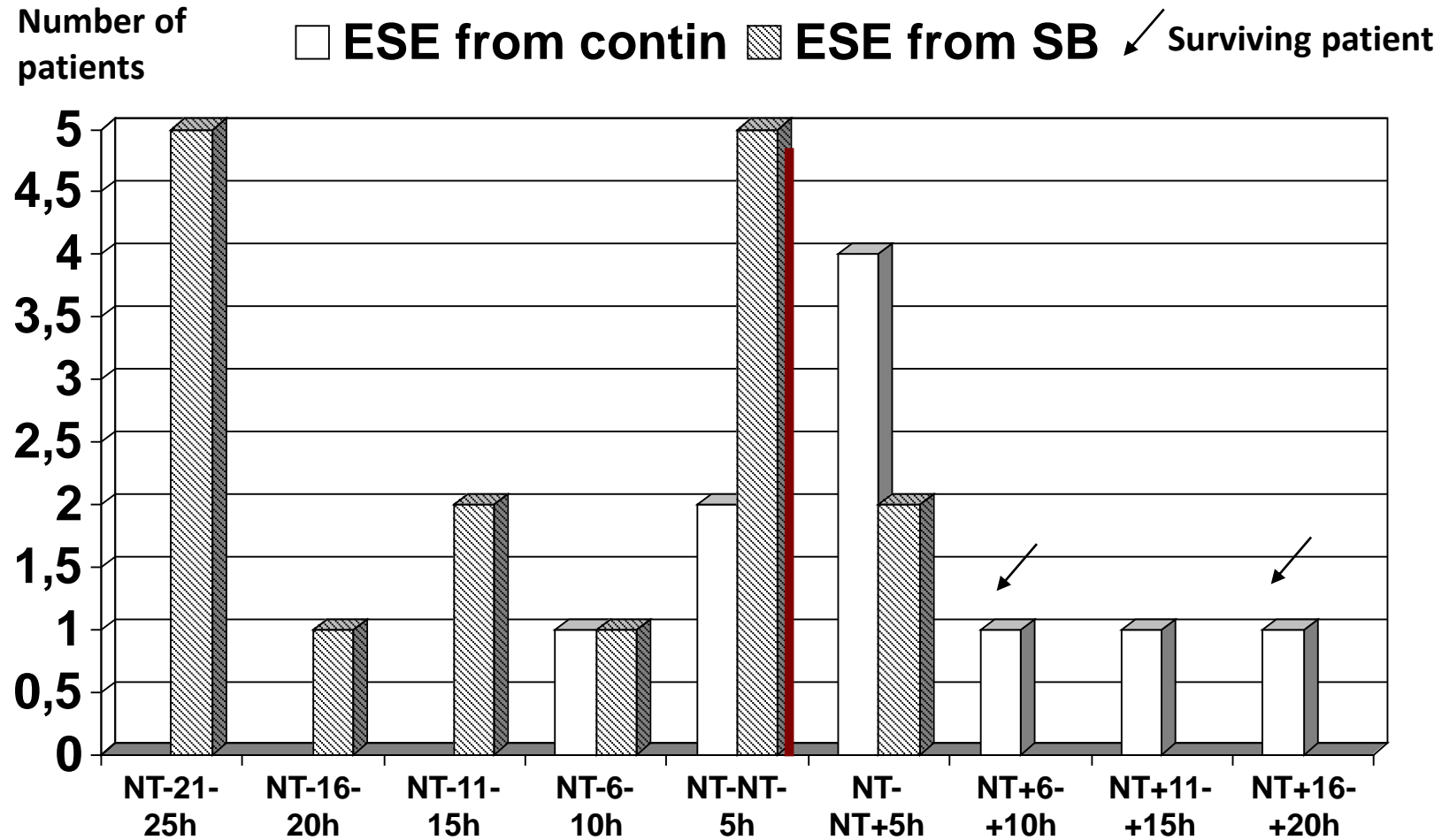
# Vad utmärker överlevarna?

	Patient 1	Patient 2	Patient 3	Patient 4	Patient 5	Patient 6
Data collection	Retrospective	Retrospective	Retrospective	Prospective	Prospective	Prospective
Age, y	60	61	55	53	68	58
Female	Yes	Yes	No	No	No	No
CA due to VF	Yes	Yes	No	Yes	Yes	Yes
Time to ROSC, min	32	45	17	20	20	21
Cardiac etiology	Yes	Yes	Yes	Yes	Yes	Yes
Preserved BR	Yes	Yes	NA	Yes	Yes	Yes
Absent MR	Yes	No	NA	No	No	Yes
SE diagnosis delay from CA, d	2	4	2	2	3	9
SE clinically (myoclonus)	Yes	No	Yes	Yes	No	No
EEG description	Repetitive diffuse sharp waves (1.5 Hz), bilateral	Repetitive diffuse sharp waves (2-3 Hz), bilateral	Repetitive diffuse sharp waves (2 Hz), bilateral	Sharp waves, spike-waves (2 Hz), L>R; SIRPID	Frontal evolving rhythmic sharp waves (3 Hz), bilateral	Frontal poly-spike-waves (2-3 Hz), R>L
EEG reactivity	Yes	Yes	Yes	Yes	Yes	Yes
PSE duration (d, on EEG)	3	2	2	2	5	3
Preserved N20 on SSEP	Yes	NA	Yes	Yes	Yes	Yes
AEDs	PRO, VPA, CLZ	VPA, CLZ, LEV	PRO, PHT, LEV, CLZ	VPA, PHT, LEV, CLZ	PRO, VPA, LEV, CLZ	VPA, LEV, CLZ
CPC outcome at 6 mo	3	2	2	1	3; death on day 18 (sepsis)	2
LA syndrome	Yes	No	No	Yes	No	No





# Lund 2004-2008, 26/95 patienter (27%) hade PSE, 2 överlevde



# Lund 2008-13, 41/127 patienter (32%) med PSE

## 4 överlevare

	Survivors N = 4	Nonsurvivors N = 37	
Time to ROSC	28 (20–76)	27 (21–40)	0.930
TTM			1.000
33 °C	4 (100%)	34 (92%)	
36 °C	0 (0%)	3 (8%)	
Time from CA to cEEG initiation (h)	8 (6–51)	8 (6–11)	0.707
Initial cEEG patterns			0.419
Flat	1 (25%)	16 (43%)	
Burst suppression	2 (50%)	18 (49%)	
Discontinuous	0 (0%)	2 (5%)	
Continuous	1 (25%)	1 (3%)	
Time from CA to debut of ESE (h)	46 (41–62)	36 (21–50)	0.210
Background prior to ESE			0.038
Burst suppression	0 (0%)	15 (40%)	
Discontinuous	0 (0%)	10 (27%)	
Nearly continuous	0 (0%)	2 (5%)	
Continuous	4 (100%)	10 (27%)	
ESE duration (h)	203 (137–269)	58 (16–113)	0.004
NSE at 48 h	22 (18–34)	47 (26–82)	0.060
SSEP			0.435
Absent N20 bilaterally	0 (0%)	7 (19%)	
Present N20 bilaterally	4 (100%)	22 (60%)	
Not performed	0 (0%)	8 (22%)	
ICU stay (h)	420 (390–540)	150 (86–212)	0.002
Hospital stay (days)	51 (26–74)	8 (4–12)	0.003
WLST	0 (%)	34 (92%)	≤0.001

- Enligt ACNS klassifikation
- 4/4 utvecklade kontinuerlig bakgrund före ESE
- Alla hade låga eller måttliga nivåer av NSE
- Alla hade SSEP-N20 svar



# Alla överlevare fick 3-4 AED

	Patient 1	Patient 2	Patient 3	Patient 4
Age	61	69	52	54
Cause of CA	Cardiac	Cardiac	Cardiac	Cardiac
Time(min ) from CA to ROSC	20	36	20	85
Time(hours) from CA to ESE diagnosis	46	42	40	58
Clinical seizures/myoclonus	Yes, after normothermia	Yes, after normothermia	No	No
EEG characteristics	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE
Anticonvulsant drugs	Fosphenytoin Clonazepam, Levetiracetam, Phenobarbital	Valproic acid , Levetiracetam Phenobarbital	Valproic acid Levetiracetam Phenobarbital Phenytoin	Valproic acid Levetiracetam Phenobarbital
Time from ESE (days) to ability to follow commands	20	24	Unknown	15
Length of sedation (day)	11	3,5	8	14
CPC at 6 months	2	3	1	2



# ..och 3-20 dagar sedation

	Patient 1	Patient 2	Patient 3	Patient 4
Age	61	69	52	54
Cause of CA	Cardiac	Cardiac	Cardiac	Cardiac
Time(min ) from CA to ROSC	20	36	20	85
Time(hours) from CA to ESE diagnosis	46	42	40	58
Clinical seizures/myoclonus	Yes, after normothermia	Yes, after normothermia	No	No
EEG characteristics	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE
Anticonvulsant drugs	Fosphenytoin Clonazepam, Levetiracetam, Phenobarbital	Valproic acid , Levetiracetam Phenobarbital	Valproic acid Levetiracetam Phenobarbital Phenytoin	Valproic acid Levetiracetam Phenobarbital
Time from ESE (days) to ability to follow commands	20	24	Unknown	15
Length of sedation (day)	11	3,5	8	14
CPC at 6 months	2	3	1	2



# De vaknade efter 2-4 veckor

	Patient 1	Patient 2	Patient 3	Patient 4
Age	61	69	52	54
Cause of CA	Cardiac	Cardiac	Cardiac	Cardiac
Time(min ) from CA to ROSC	20	36	20	85
Time(hours) from CA to ESE diagnosis	46	42	40	58
Clinical seizures/myoclonus	Yes, after normothermia	Yes, after normothermia	No	No
EEG characteristics	Continuous background →ESE	Continuous background →ESE	Continuous background → ESE	Continuous background → ESE
Anticonvulsant drugs	Fosphenytoin Clonazepam, Levetiracetam, Phenobarbital	Valproic acid , Levetiracetam Phenobarbital	Valproic acid Levetiracetam Phenobarbital Phenytoin	Valproic acid Levetiracetam Phenobarbital
Time from ESE (days) to ability to follow commands	20	24	Unknown	15
Length of sedation (day)	11	3,5	8	14
CPC at 6 months	2	3	1	2



# Lätt-måttlig funktionsnedsättning hos 3/4

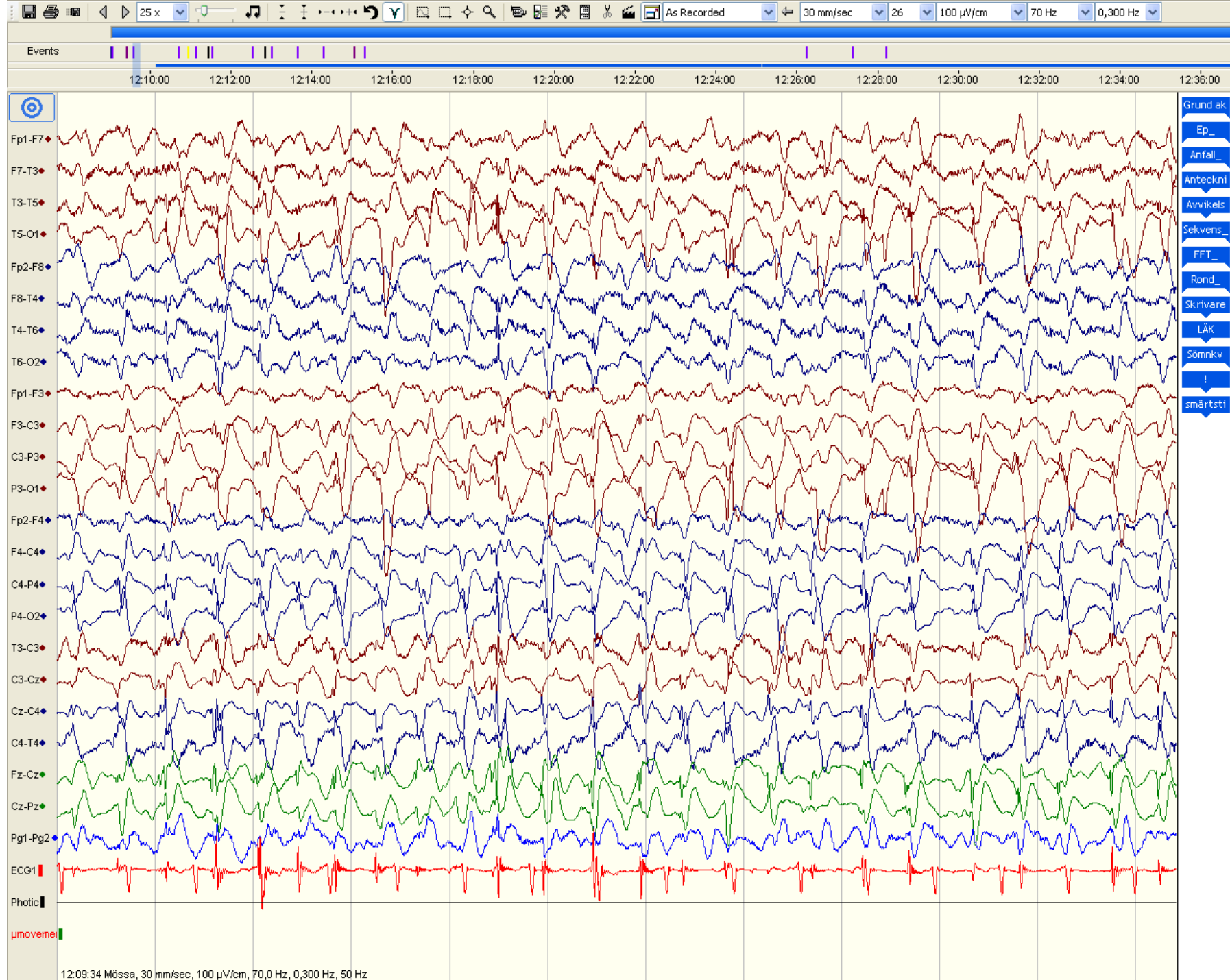
	Patient 1	Patient 2	Patient 3	Patient 4
Age	61	69	52	54
Cause of CA	Cardiac	Cardiac	Cardiac	Cardiac
Time(min ) from CA to ROSC	20	36	20	85
Time(hours) from CA to ESE diagnosis	46	42	40	58
Clinical seizures/myoclonus	Yes, after normothermia	Yes, after normothermia	No	No
EEG characteristics	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE	Continuous background → ESE
Anticonvulsant drugs	Fosphenytoin Clonazepam, Levetiracetam, Phenobarbital	Valproic acid , Levetiracetam Phenobarbital	Valproic acid Levetiracetam Phenobarbital Phenytoin	Valproic acid Levetiracetam Phenobarbital
Time from ESE (days) to ability to follow commands	20	24	Unknown	15
Length of sedation (day)	11	3,5	8	14
CPC at 6 months	2	3	1	2



# 59-årig man på thoraxintensiven

- Bilateral lungtransplantation på grund av fibros
- D2 Massiv lungembolism
- D10 Hjärtstopp. Kylbehandling 33°C
- D12 “kramper” när sederingen fasas ut
  - EEG







# 4 veckors behandling...

- SSEP med bevarade N20-svar bilat. MR-hjärna normal.
- Ökande epileptiform aktivitet när man försöker fasa ut propofol
- valproat, klonazepam, levetiracetam, fenobarbital, propofol, midazolam
- Långsamt minskande epileptiform aktivitet, reaktiv EEG-bakgrund
- Minskande doser AED.....



# Långsam återhämtning...

- D 32 grimaserar
- D 36-38 Börjar röra händer, huvud och ben, grimaserar och nickar som svar
- D 43 Vaken, kommunicerar, generaliserad muskelatrofi och svaghet
  - GTK
  - Ökande myoklonus
  - valproat, levetiracetam och klonazepam



# Lance-Adams syndrome

**Action myoclonia**  
4 weeks after CA  
and Status Epilepticus

Lance & Adams, *Brain*, 1963



# Evidens för behandling av PSE saknas

- Rapporterade överlevare har behandlats aggressivt
- PSE är självbegränsande (hos överlevare)
- Behandling kan vara skadlig
  - Pneumoni
  - Critical illness polyneuropathy/myopathy
  - Pessimism pga iatrogen koma
- Vi behöver randomiserade studier!



**STUDY PROTOCOL**

**Open Access**

# Treatment of electroencephalographic status epilepticus after cardiopulmonary resuscitation (TELSTAR): study protocol for a randomized controlled trial

Barry J Ruijter<sup>1\*</sup>, Michel JAM van Putten<sup>1,2</sup>, Janneke Horn<sup>3</sup>, Michiel J Blans<sup>4</sup>, Albertus Beishuizen<sup>5</sup>, Anne-Fleur van Rootselaar<sup>6</sup>, Jeannette Hofmeijer<sup>1,7</sup>, on behalf of the TELSTAR study group



# Vår behandlingsmodell

- cEEG med förenklat montage
- Vanligen kombination av valproat och levetiracetam med dagliga koncentrationsbestämningar
- Vi eftersträvar anfallssuppression (< 1 Hz)
- Fenobarbital kan övervägas



# Konklusioner

- Kliniska kramper och epileptiformt EEG efter hjärtstopp innebär en sämre prognos
- En del patienter återhämtar sig väl
- Det finns ingen evidensbaserad behandlingsstrategi
- Bedöm prognosen med andra metoder (SSEP, MRI, NSE)
- Ge **alltid** patienten en chans att vakna upp ur sedationen





4<sup>TH</sup> INTERNATIONAL SYMPOSIUM ON

# Post Cardiac Arrest Care

FOCUSING ON neuroprognostication, follow-up, quality-of-life, rehabilitation

May 23<sup>rd</sup>-24<sup>th</sup>, 2019

SKÅNE UNIVERSITY HOSPITAL, LUND, SWEDEN

An Interactive Symposium

- scientific highlights •
- challenging lectures •
  - panel debates •
  - case sessions •
  - skills training •
- industry exhibition •



Register & submit your abstract at:  
[www.postcare.se](http://www.postcare.se)



EUROPEAN  
RESUSCITATION  
COUNCIL  
[www.erc.edu](http://www.erc.edu)



LUND  
UNIVERSITY

# Post Cardiac Arrest Care

FOCUSING ON neuroprognostication, follow-up, quality-of-life, rehabilitation

## PROGRAM - Thursday May 23<sup>rd</sup>, 2019

08.15-09.00	Registration		
09.00-10.10	<b>Welcome</b> <i>Hans Friberg, Tobias Cronberg, Gisela Lilja</i>		
Chairs: Marcus Skrifvars & Hans Friberg			
09.10-09.30	<b>Surviving cardiac arrest - what are the critical decisions?</b> <i>Jerry Nolan, UK</i>		
09.30-09.50	<b>In the field - early stopping rules</b> <i>Laurie Morrison, Canada</i>		
09.50-10.00	Discussion		
10.00-10.30	Coffee break & exhibition		
Chairs: Jeannette Hofmeyer & Mauro Oddo			
10.30-10.45	<b>Are we making critical decisions too early - the NORCAST study</b> <i>Kjetil Sunde, Norway</i>		
10.45-11.00	<b>In the emergency room - early prediction models</b> <i>Florence Dumas, France</i>		
11.00-11.15	<b>In the ICU - Multimodal neurological prognostication</b> <i>Tobias Cronberg, Sweden</i>		
11.15-11.30	<b>Machine learning for prediction of outcome</b> <i>Niklas Nielsen, Sweden</i>		
11.30-12.00	Discussion		
12.00-13.00	Lunch break with posters & exhibition		
Chairs: David Seder & Tobias Cronberg			
13.00-14.30	<b>Best abstracts; prognostication</b> <b>Simplified monitoring and interpretation of continuous EEG after cardiac arrest - a useful bedside tool for the ICU physician</b> <i>Anna Lybeck, Sweden</i> <b>Factors associated with abnormal EEG after out-of-hospital cardiac arrest: a post hoc analysis of a randomised clinical trial</b> <i>Pekka Jakkula, Finland</i> <b>Are providers overconfident in predicting outcome after cardiac arrest?</b> <i>Alexis Steinberg, USA</i> <b>Neurocardiac risk stratification 6 hours after resuscitation from cardiac arrest</b> <i>David Seder, USA</i>		
	<b>Routine EEG versus continuous monitoring-pro/con debate</b> Routine EEG is superior - <i>Andrea Rossetti, Switzerland</i> Continuous EEG monitoring is superior - <i>Jeannette Hofmeijer, The Netherlands</i>		
14.30-15.00	Coffee break & exhibition		
Chairs: Irina Dragancea & Jerry Nolan			
15.00-15.45	<b>Novel methods of prognostication</b> Pupilometry - <i>Mauro Oddo, Switzerland</i> Novel blood biomarkers - <i>Niklas Mattsson, Sweden</i> MRI - diffusion tensor imaging - <i>Lionel Velly, France</i>		
15.45-16.00	Short break		
16.00-17.00	<b>Interactive training session</b>		
	<b>Routine neurological prognostication</b> <i>Clifton Callaway, Mauro Oddo, Irina Dragancea</i>	<b>Clinical and electrographic seizures</b> <i>Andrea Rossetti, Jeannette Hofmeijer, Tobias Cronberg</i>	<b>My worst case</b> <i>Jerry Nolan, Laurie Morrison, Kjetil Sunde</i>
18.30	Conference Get together		

## PROGRAM - Friday May 24<sup>th</sup>, 2019

08.10-08.30	Registration		
08.30-08.40	<b>Welcome</b> <i>Hans Friberg, Tobias Cronberg, Gisela Lilja</i>		
Chairs: Giesla Lilja & Jerry Nolan			
08.40-09.10	<b>The chain of recovery. From cardiac arrest to social integration</b> <i>Clifton Callaway, USA</i>		
09.10-09.30	<b>What's wrong with the good outcome; to measure outcome after cardiac arrest</b> <i>Kirstie Haywood, UK</i>		
09.30-10.00	<b>Patient reported outcomes (PRO) in cardiac arrest registers. Yes it works.</b> <i>Karen Smith, Australia</i>		
10.00-10.30	Coffee break & exhibition		
Chairs: Thomas Keeble & Erik Blennow Nordström			
10.30-11.00	<b>Rehabilitation of the patient with a prolonged Disorder of Consciousness</b> <i>Alison Goodbolt, Sweden</i>		
11.00-11.30	<b>Memory rehabilitation</b> <i>Barbra Wilson, UK</i>		
11.30-12.00	<b>Fatigue after cardiac arrest</b> <i>Gisela Lilja, Sweden</i>		
12.00-13.00	Lunch break with posters & exhibition		
Chairs: Karen Smith & Clifton Callaway			
13.00-14.00	<b>Best abstracts; outcome, follow up and rehabilitation</b> <b>LOCAS I - Understanding Long-term Outcomes after Cardiac Arrest from the Survivor and Family Member Perspective</b> <i>Katie Dainty, Canada</i> <b>Cardiac arrest and hospitalization induced-hyperarousal symptoms are associated with 1-year risk of major adverse cardiovascular events and all-cause mortality</b> <i>Sachin Agarwal, USA</i> <b>Quality of Life in a long term follow up in out-of-hospital cardiac arrest survivors</b> <i>Henning Wimmer, Norway</i> <b>Patient and public involvement in the development of rehabilitation interventions for survivors of cardiac arrest in Denmark</b> <i>Vicky Joshi, Denmark</i>		
14.00-14.30	<b>Patient and partner support groups</b> <i>Thomas Keeble &amp; Paul Swindell, UK</i>		
14.30-14.50	Coffee break & exhibition		
Chairs: Laurie Morrison & Kjetil Sunde			
14.50-15.10	<b>To form a clinical center of excellence for rehabilitation after cardiac arrest</b> <i>Paulien Goossens, The Netherlands</i>		
15.10-15.30	<b>What we learned from the ALASCA trial; focusing on follow-up after cardiac arrest</b> <i>Caroline van Heughten, The Netherlands</i>		
15.30-15.45	Short break		
15.45-16.30	<b>Minisymposiums</b>		
	<b>To build a follow up system after CA</b> <i>Paulien Goossens, Thomas Keeble</i>	<b>To assess long-term outcome in clinical trials and register studies</b> <i>Kirstie Haywood, Karen Smith</i>	<b>Cognitive rehabilitation in practice, clinical examples</b> <i>Caroline van Heughten, Barbra Wilson</i>

