Neuroprognose bei Erwachsenen
Klinik, Bilder, “harte Marker"

Bettina Pfausler
Universitätsklinik für Neurologie
Medizinische Universität Innsbruck
Tirol Kliniken

Neurovaskuläre Erkrankungen (Blutung, Ischämie)
Schädelhirntrauma
Hypoxische Enzephalopathie
Neurodegenerative Erkrankungen
Genetische Erkrankungen
…
…
….

quo ad vitam

quo ad rehabilitationem

quo ad functionem

Lebensqualität

Neurovaskuläre Erkrankungen
(Blutung, Ischämie)

Schädelhirntrauma

Hypoxische Enzephalopathie

Neurodegenerative Erkrankungen

Genetische Erkrankungen

…

…

….

quo ad vitam

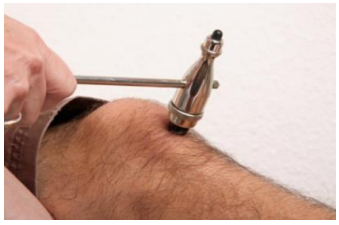
quo ad rehabilitationem

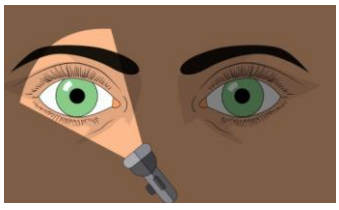
quo ad functionem

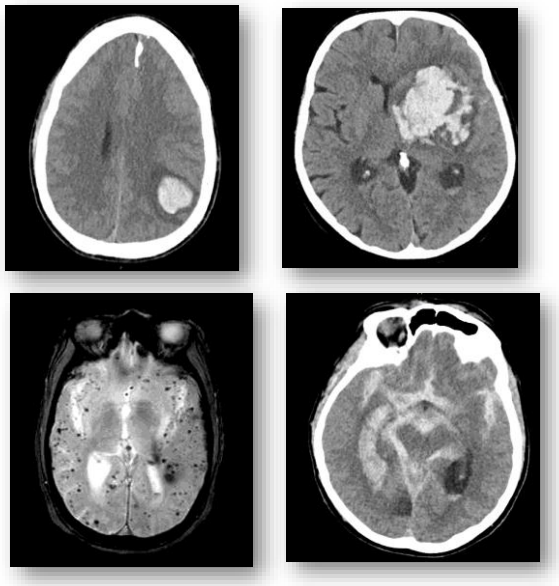
Lebensqualität

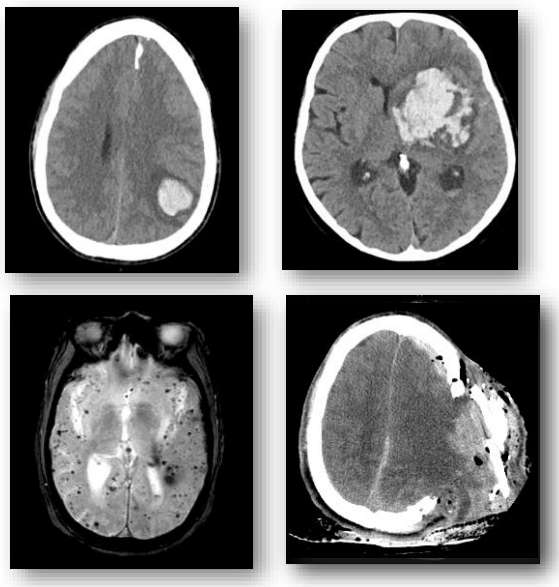
Wann?

zeitgemäß?

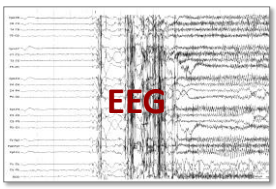


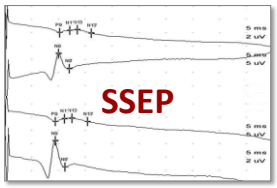






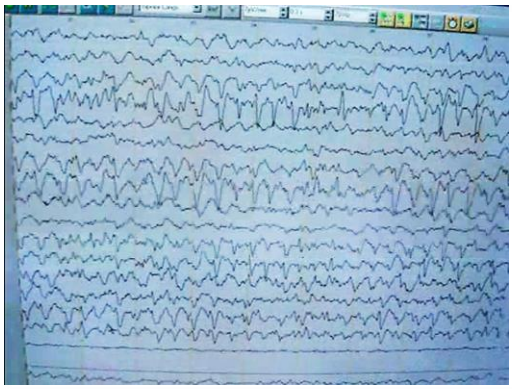
Universitätsklinik für Neurologie - Medizinische Universität Innsbruck, Austria

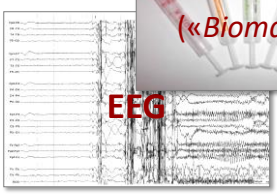


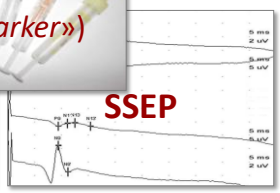


?

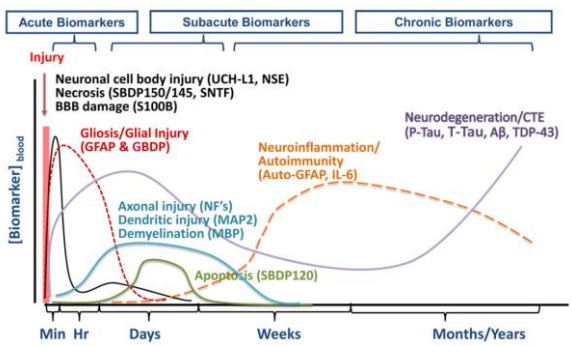
kontinuierlich
Oberflächenableitung







Kevin K. Wang, Zhihui Yang, Tian Zhu, Yuan Shi, Richard Rubenstein

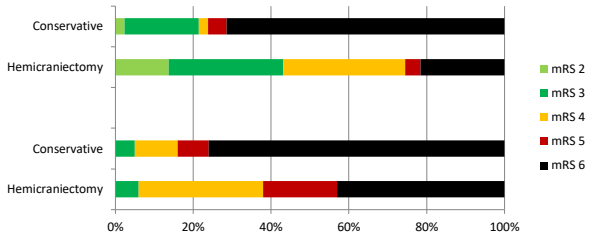


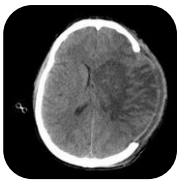
Expert Rev Mol Diagn. 2018 February ; 18(2): 165–180.

Alter < 60y
12 Monate
N=93, DESTINY. DECIMAL. HAMLET
Huttner. Lancet Neurol 2009

Alter > 60a, (median70y)
N=112, DESTINY II 12 months
Jüttler, NEJM 2014

Dekompressive Kranietomie <48h
(HAMLET, Destiny, Decimal 2007-2009)





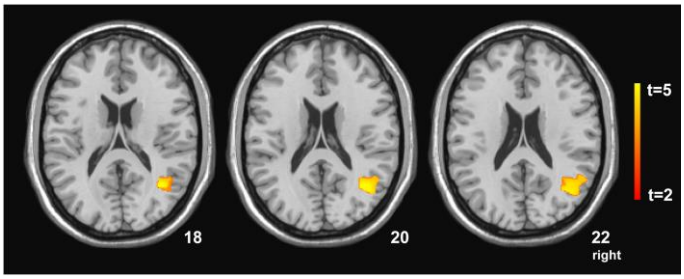
„nicht zu spät und nicht zu klein“

Kopfrechnung 23 x8
5 aufeinanderfolgenden
Tagen für je 30 Minuten

Plastizität des Gehirnes

Fähigkeit der Anpassung neuronaler Netzwerke an veränderte Umgebungsbedingungen

Trainings-induzierte Plastizität

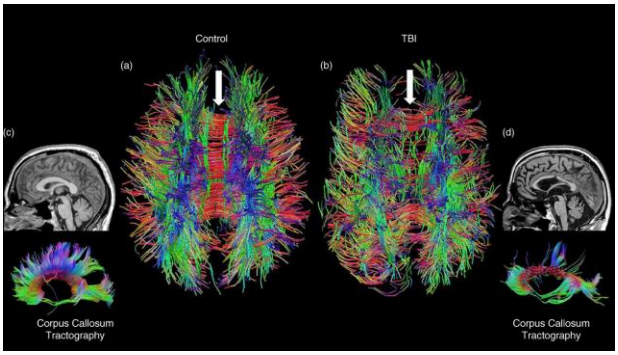


Zamarian et al., Universitätsklinik für Neurologie, Innsbruck, 2018

Plastizität des Gehirnes

Fähigkeit der Anpassung neuronaler Netzwerke an veränderte Umgebungsbedingungen

- Trainings-induzierte Plastizität
- Läsions-induzierte Plastizität



Hayes JP; J Int Neuropsychol Soc. 2016 ; 22; 120-137)

Bild aus „Vorarlberger Nachrichten“

275 000 OHCAs / Jahr in der Europäischen Union - Große regionale Unterschiede in den Daten

50% der CA PatientInnen nach Aufnahme an der Intensiv versterben < 6 Monaten

die Mehrzahl innerhalb < ersten 2 Wochen

<3 Tage cardiale Ursache + schwerer hypoxischer Hirnschaden

> 3 Tag hypoxische Hirnschädigung (50-70% OHCA; 25% IHCA)

malignes hypoxisches Hirnödem mit Hirntodsyndrom selten

< 2 % nach OHCA sind beim Eintreffen im Krankenhaus wach

< 10% erreichen ein funktionelles gutes Outcome

Aung Myat, Kyoung-Jun Song, Thomas Rea, Jan-Thorsten Gräser , EuReCa One, Josef Dankiewicz

European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2021: Post-resuscitation care☆

Jerry P. Nolan[a,b,1,], Claudio Sandroni[c,d,1], Bernd W. Böttiger[e], Alain Cariou[f], Tobias Cronberg[g], Hans Friberg[h], Cornelia Genbrugge[i,j], Kirstie Haywood[k], Gisela Lilja[l], Véronique R.M. Moulaert[m], Nikolaos Nikolaou[n], Theresa Mariero Olasveengen[o], Markus B. Skrifvars[p], Fabio Taccone[q], Jasmeet Soar[r]*

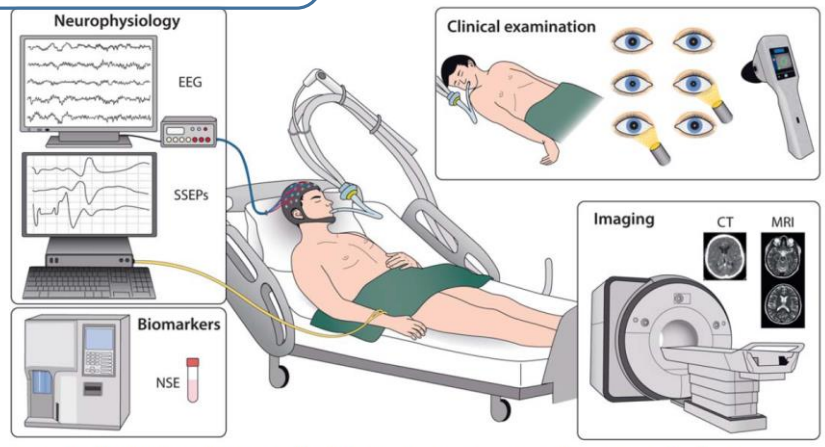
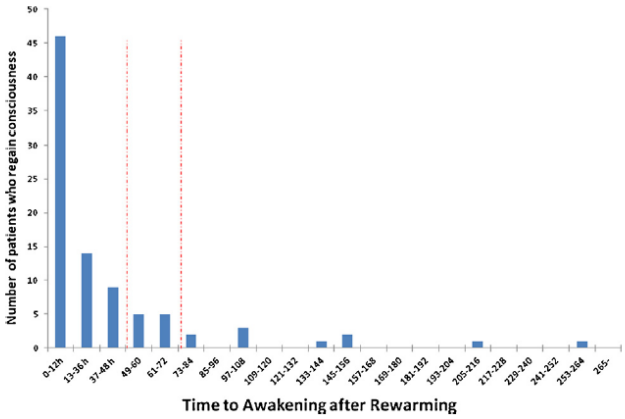


Fig. 4 – Prognostication modes. EEG electroencephalography; NSE neuron specific enolase; SSEP somatosensory evoked potential.

Awakening after cardiac arrest and post resuscitation hypothermia: Are we pulling the plug too early?☆

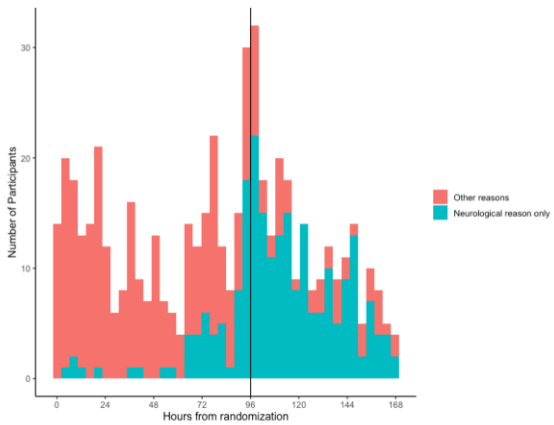
Gold B, Puertas L, Davis SP, Metzger A, Yannopoulos D, Oakes DA, Lick CJ, Gillquist DL, Holm SY, Olsen JD, Jain S, Lurie KG

Resuscitation 85 (2014) 211– 214



Conclusion: Following OHCA and TH, arbitrary withdrawal of **life support <48 h after rewarming** may prematurely terminate life in many patients with the potential for full neurological recovery. Additional clinical markers that correlate with late awakening are needed to better determine when withdrawal of support is appropriate in OHCA patients who remain comatose >48 h after rewarming

Figure S3. Withdrawal of life-sustaining therapies, by reason.



Reason and timing of WLST during the first 7 days after randomization. WLST: Withdrawal of life-sustaining therapies. The vertical black line denotes the time point from which protocolized neuroprognostication was allowed.

Dankiewicz J, Cronberg T, Lilja G et al TTM2 Trial Investigators. Hypothermia versus Normothermia after Out-of-Hospital Cardiac Arrest. N Engl J Med. 2021 Jun 17;384(24):2283-2294

European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2021: Post-resuscitation care☆

Jerry P. Nolan[a,b,1,*], Claudio Sandroni[c,d,1], Bernd W. Böttiger[e], Alain Cariou[f], Tobias Cronberg[g], Hans Friberg[h], Cornelia Genbrugge[i,j], Kirstie Haywood[k], Gisela Lilja[l], Véronique R.M. Moulaert[m], Nikolaos Nikolaou[n], Theresa Mariero Olasveengen[o], Markus B. Skrifvars[p], Fabio Taccone[q], Jasmeet Soar[r]

"residual sedation should always be considered and excluded"

GCS - Motor Score ≤ 3 at 72 h or later … **neurological prognostication may be needed**

poor neurological outcome

- comatose at 72 h or later
- bilateral absence of the standard pupillary light reflex **-**
- bilateral absence of corneal reflex
- presence of myoclonus within 96 h
- Status myoclonus within 72 h

European Resuscitation Council and European Society of Intensive Care Medicine Guidelines 2021: Post-resuscitation care☆

Jerry P. Nolan[a,b,1,*], Claudio Sandroni[c,d,1], Bernd W. Böttiger[e], Alain Cariou[f], Tobias Cronberg[g], Hans Friberg[h], Cornelia Genbrugge[i,j], Kirstie Haywood[k], Gisela Lilja[l], Véronique R.M. Moulaert[m], Nikolaos Nikolaou[n], Theresa Mariero Olasveengen[o], Markus B. Skrifvars[p], Fabio Taccone[q], Jasmeet Soar[r]

"residual sedation should always be considered and excluded"

GCS - Motor Score ≤ 3 at 72 h or later … **neurological prognostication may be needed**

poor neurological outcome

- comatose at 72 h or later
- bilateral absence of the standard pupillary light reflex **- quantitative pupillometry**
- bilateral absence of corneal reflex
- presence of myoclonus within 96 h
- Status myoclonus within 72 h

Universitätsklinik für Neurologie - Medizinische Universität Innsbruck, Austria

ORIGINAL

Quantitative versus standard pupillary light reflex for early prognostication in comatose cardiac arrest patients: an international prospective multicenter double-blinded study

Oddo M et all Intensive Care Med. 2018 Dec;44(12):2102-2111.

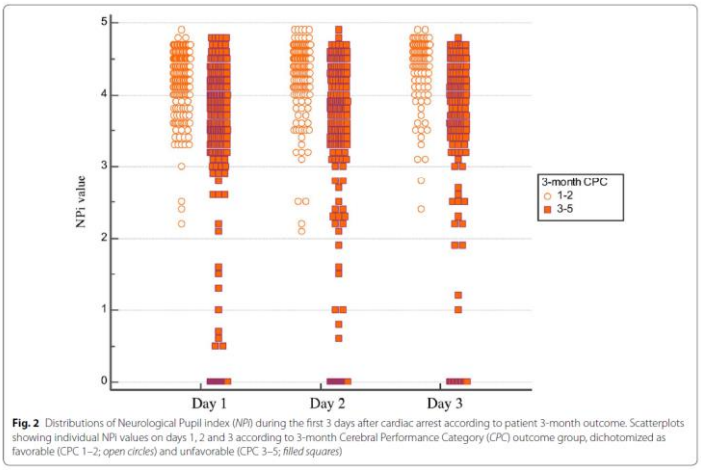


Fig. 2 Distributions of Neurological Pupil index (*NPI*) during the first 3 days after cardiac arrest according to patient 3-month outcome. Scatterplots showing individual NPi values on days 1, 2 and 3 according to 3-month Cerebral Performance Category (*CPC*) outcome group, dichotomized as favorable (CPC 1–2; *open circles*) and unfavorable (CPC 3–5; *filled squares*)

ORIGINAL

Quantitative versus standard pupillary light reflex for early prognostication in comatose cardiac arrest patients: an international prospective multicenter double-blinded study

Oddo M et all Intensive Care Med. 2018 Dec;44(12):2102-2111.

Table 2 Specificity, sensitivity, positive predictive value, negative predictive value and false-positive rate for unfavorable outcome (CPC 3–5) of the different prognostic tests

Day after cardiac arrest	Sample size (*n*)	CPC 3–5 *n* (%)	Specificity % (95% CI)	Sensitivity % (95% CI)	Positive predictive value % (95% CI)	Negative predictive value % (95% CI)	False-positive rate % (95% CI)
Neurological pupil index (NPi) ≤ 2							
Day 1–3	456	269 (59)	100 (98–100)	32 (27–38)	100 (100–100)	51 (49–53)	0 (0–2) %
Day 1	450	264 (59)	100 (98–100)	22 (17–27)	100 (100–100)	47 (46–49)	0 (0–2) %
Day 2	361	213 (59)	100 (98–100)	19 (14–25)	100 (100–100)	46 (45–48)	0 (0–2) %
Day 3	271	166 (61)	100 (97–100)	17 (12–24)	100 (100–100)	43 (41–44)	0 (0–3) %
Bilaterally absent standard pupillary light reflex (sPLR)							
Day 1	392	225 (57)	90 (85–94)	35 (29–42)	83 (75–89)	51 (48–54)	10 (6–15) %
Day 2	278	163 (59)	90 (84–95)	29 (22–36)	81 (70–89)	47 (44–50)	10 (5–16) %
Day 3	206	128 (62)	94 (86–98)	18 (12–26)	82 (65–92)	41 (39–43)	6 (2–14) %
Bilaterally absent somatosensory evoked potentials (N20 wave)							
Day 2–3	188	133 (71)	100 (94–100)	48 (39–57)	100 (100–100)	44 (40–48)	0 (0–6) %
Combination of NPi ≤ 2 and bilaterally absent somatosensory evoked potentials							
Day 2–3	188	133 (71)	100 (94–100)	58 (49–66)	100 (100–100)	55 (50–59)	0 (0–6) %

CI confidence interval, *CPC* Cerebral Performance Category

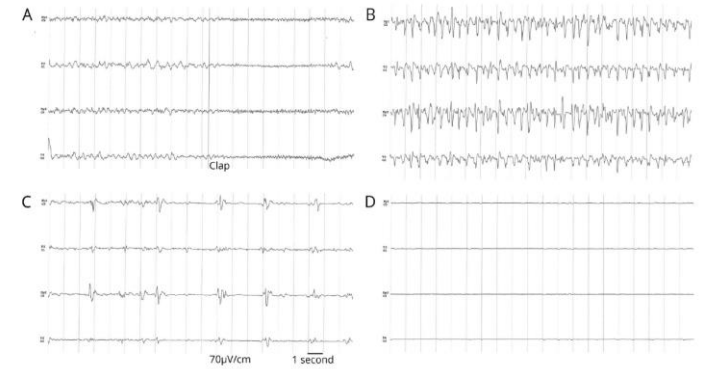
Universitätsklinik für Neurologie - Medizinische Universität Innsbruck, Austria

Neurologic outcome of postanoxic refractory status epilepticus after aggressive treatment

„maligne EEG Muster“

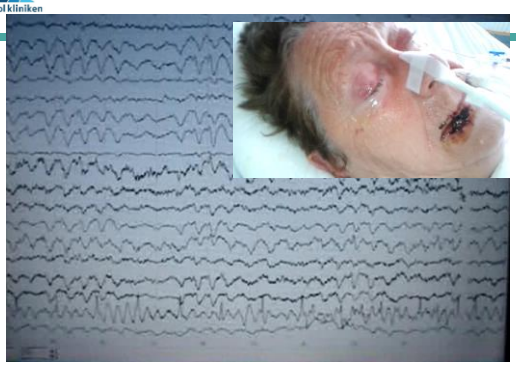
Low voltage output
Burst suppression
Generalisierte periodische Entladungen
Kontinuierliche epileptische Entladungen
Fehlende Hintergrundaktivität
α-Koma

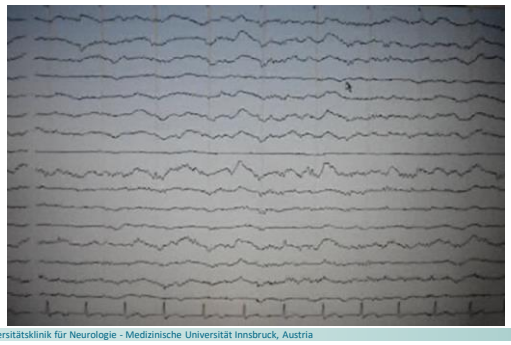
Figure 2 Prognostic EEG patterns in postcardiac arrest patients



Representative epochs from 4-channel continuous EEG monitoring of patients with (A) benign EEG pattern, (B) refractory status epilepticus pattern, (C) generalized periodic discharge pattern, and (D) malignant nonepileptiform EEG pattern.

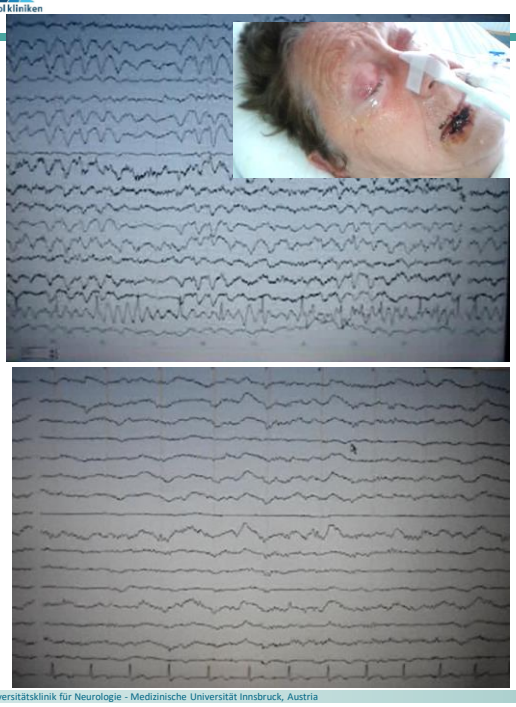
Beretta S et al: Neurologic outcome of postanoxic refractory status epilepticus after aggressive treatment. Neurology. 2018 Dec 4;91(23):e2153-e2162.





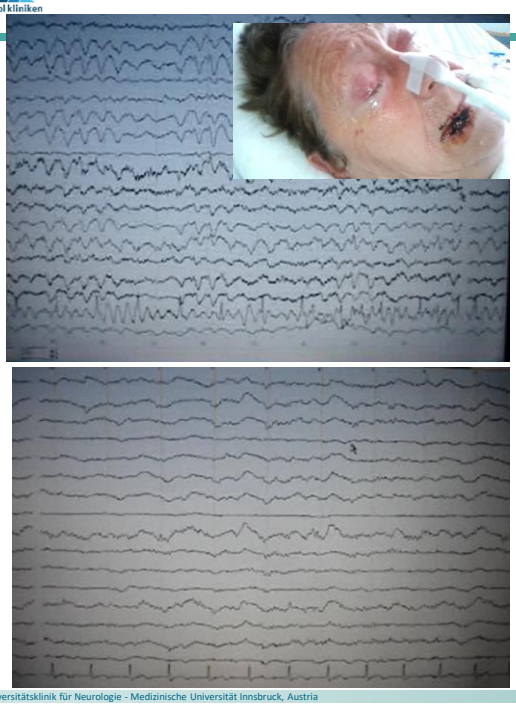
«Henne-Ei-Problem» beim postanoxischen Status epilepticus

- Postanoxischer SE als Ausdruck der schweren Hirnschädigung und somit **therapierefraktärer Folgezustand**
- Postanoxischer SE propagiert eine sekundäre neuronale Schädigung und **erfordert eine aggressive Therapie**



Eine prospektive, multizentrische, randomisierte Studie **(TELSTAR)** zur **medikamentösen Suppression ‚rhythmischer und periodischer' EEG-Muster** nach Herzstillstand und Reanimation konnte keinen Vorteil einer aggressiven Therapie mit anfallssupprimierenden Medikamenten belegen, (Ruijter et al., 2022).

DGN-Leitlinien, AWMF Überarbeitung 2023 in press



Eine prospektive, multizentrische, randomisierte Studie **(TELSTAR)** zur **medikamentösen Suppression ‚rhythmischer und periodischer' EEG-Muster** nach Herzstillstand und Reanimation konnte keinen Vorteil einer aggressiven Therapie mit anfallssupprimierenden Medikamenten belegen, (Ruijter et al., 2022).

Eine Subgruppenanalyse und weitere Studien deuten aber darauf hin, dass CA-Patient:innen **mit Status epilepticus** und darüber hinaus **fehlenden Hinweisen für eine sehr schwere HIE** von einer **aggressiven anfallssupprimierenden Therapie** profitieren können (Beretta et al., 2018)

DGN-Leitlinien, AWMF Überarbeitung 2023 in press

Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest

	CPC 1–2 (n = 10)	CPC 3–5 (n = 97)	*p* Value[a]
Male sex	10/18 (56)	62/89 (70)	0.2446
Age, y	66.11 ± 14.29	63.29 ± 17.50	0.7039
Hypothermia	4/18 (22)	33/89 (37)	0.2846
Cardiac etiology	13/18 (72)	53/89 (60)	0.3132
Shockable rhythm (VF, VT)	11/18 (61)	36/89 (40)	0.1769
Time to ROSC, min	24.67 ± 13.46	25.14 ± 12.59	0.7414
Pupillary reflex present	17/18 (94)	46/89 (52)	0.0001[b]
Motor response (flexion or better) present	12/18 (67)	5/89 (6)	<0.0001[b]
Myoclonus ≤72 hours	5/18 (28)	67/89 (75)	0.0001[b]
SSEPs bilaterally absent	2/17 (12)	42/71 (59)	0.0027[b]
Serum NSE peak, µg/L, (range); no. of tested patients	25.1 (15.20–45.20); 15	72.3 (16–668.7); 56	<0.0001[b]

Barbella G et al. Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest. Neurology. 2020 Apr 21;94(16):e1675-e1683.

Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest

	CPC 1–2 (n = 10)	CPC 3–5 (n = 97)	*p* Value[a]
Male sex	10/18 (56)	62/89 (70)	0.2446
Age, y	66.11 ± 14.29	63.29 ± 17.50	0.7039
Hypothermia	4/18 (22)	33/89 (37)	0.2846
Cardiac etiology	13/18 (72)	53/89 (60)	0.3132
Shockable rhythm (VF, VT)	11/18 (61)	36/89 (40)	0.1769
Time to ROSC, min	24.67 ± 13.46	25.14 ± 12.59	0.7414
Pupillary reflex present	17/18 (94)	46/89 (52)	0.0001[b]
Motor response (flexion or better) present	12/18 (67)	5/89 (6)	<0.0001[b]
Myoclonus ≤72 hours	5/18 (28)	67/89 (75)	0.0001[b]
SSEPs bilaterally absent	2/17 (12)	42/71 (59)	0.0027[b]
Serum NSE peak, µg/L, (range); no. of tested patients	25.1 (15.20–45.20); 15	72.3 (16–668.7); 56	<0.0001[b]

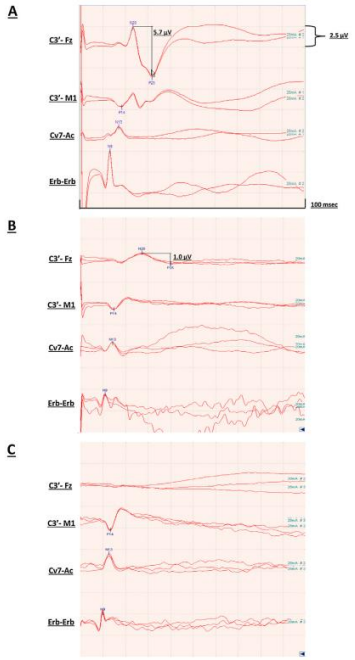
Barbella G et al. Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest. Neurology. 2020 Apr 21;94(16):e1675-e1683.

Universitätsklinik für Neurologie - Medizinische Universität Innsbruck, Austria

SSEP amplitude accurately predicts both good and poor neurological outcome early after cardiac arrest; a post-hoc analysis of the ProNeCA multicentre study

EUROPEAN RESUSCITATION COUNCIL

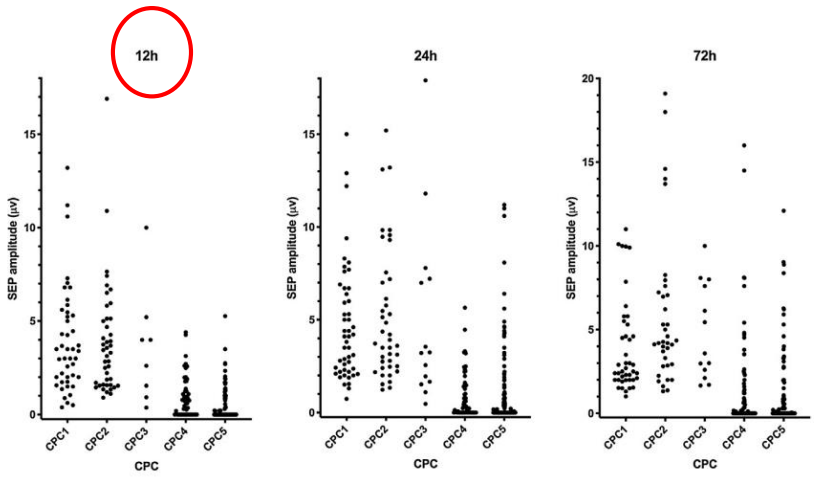
„Durch Hypothermie und Sedierung kortikale Antwort nicht beeinflusst“

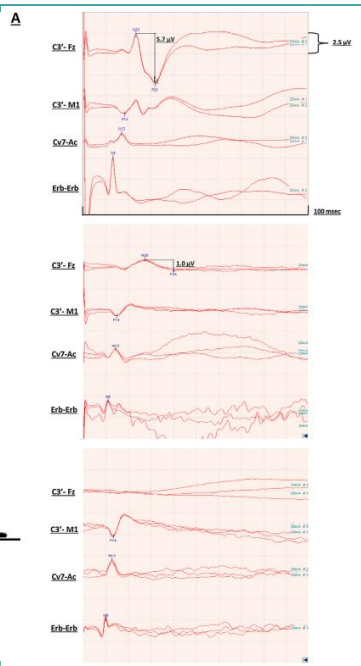


Scarpino M et aL. SSEP amplitude accurately predicts both good and poor neurological outcome early after cardiac arrest; a post-hoc analysis of the ProNeCA multicentre study. Resuscitation. 2021

gutes Outcome SSEP > 3µV (sensitivity 61 [50-72]% and FPR 11 [6-18]%)

SSEP > 10µV (sensitivity 6 [2-13]% and FPR 0 [0-3]%)





Scarpino M et aL. SSEP amplitude accurately predicts both good and poor neurological outcome early after cardiac arrest; a post-hoc analysis of the ProNeCA multicentre study. Resuscitation. 2021

SSEP amplitude accurately predicts both good and poor neurological outcome early after cardiac arrest; a post-hoc analysis of the ProNeCA multicentre study

EUROPEAN RESUSCITATION COUNCIL

Scarpino M, Lolli F, Lanzo G et al:ProNeCA study group. SSEP amplitude accurately predicts both good and poor neurological outcome early after cardiac arrest; a post-hoc analysis of the ProNeCA multicentre study. Resuscitation. 2021

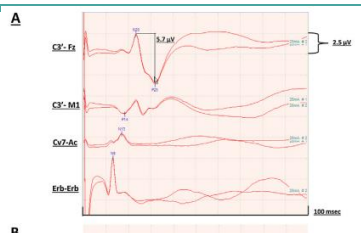
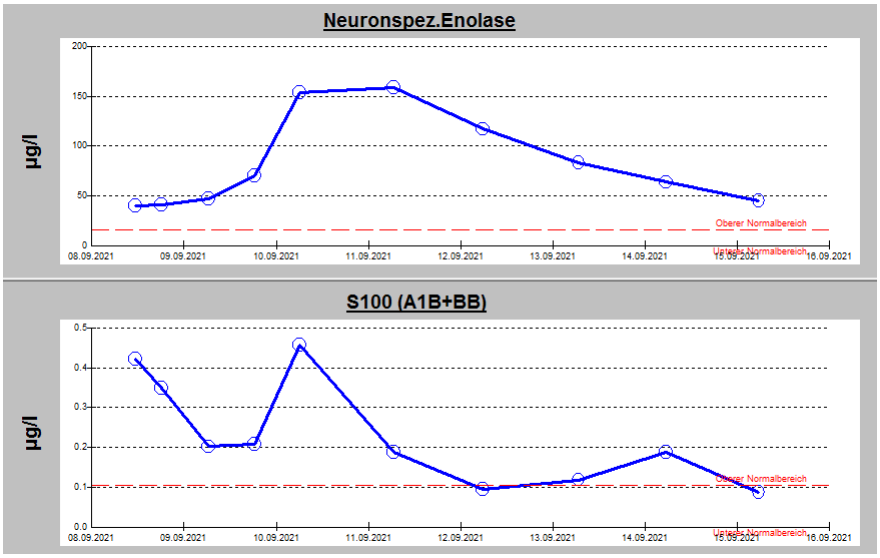


Table 3c – Prognostic accuracy of PLR, EEG, SEP patterns and their combination at 72 h for predicting poor neurological outcome (*n* = 240).

Index test	TP	FP	TN	FN	Sensitivity % (95%CI)	False positive rate % (95%CI)
AA SSEPs	87	0	77	76	53 (45–61)	0 (0–5)
AA or Low Voltage SSEPs (<1 µV)	120	0	77	43	73 (66–80)	0 (0–5)
Malignant EEG	97	0	77	66	64 (56–71)	0 (0–3)
Absent PLR	108	3	74	55	66 (58–73)	4 (1–11)
Malignant EEG and absent PLR	71	0	77	92	44 (36–52)	0 (0–5)
AA or Low Voltage SEPs and malignant EEG	87	0	77	76	53 (45–61)	0 (0–5)
AA or Low Voltage SEPs and absent PLR	71	0	77	92	44 (36–51)	0 (0–5)
AA or Low Voltage SEPs and malignant EEG and absent PLR	65	0	77	98	40 (32–48)	0 (0–5)

Abbreviations—CI: confidence interval; EEG: electroencephalogram; PLR: pupillary light reflex; SSEPs: short-latency somatosensory evoked potentials. TP, true positive; FP, false positive; TN, true negative; FN, false negative.



tirol kliniken

Neuron-Specific Enolase Predicts Poor Outcome After Cardiac Arrest and Targeted Temperature Management: A Multicenter Study on 1,053 Patients

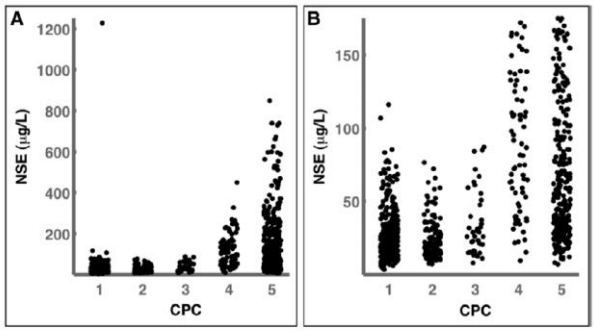


Figure 1. Neuron-specific enolase (NSE) serum concentration and outcome. The left figure (**A**) indicates an upper limit for NSE serum concentration for patients with Cerebral Performance Category (CPC) 1–3, except for one patient with CPC 1 and NSE of 1,227 µg/L. This patient had an NSE-producing neuroendocrine tumor of the pancreas. The right figure (**B**) shows a zoom into **A**.

Measurements and Main Results: A neuron-specific enolase serum concentration greater than 90 µg/L predicted Cerebral Performance Category 4–5 with a positive predictive value of 99%, false positive rate of 0.5%, and a sensitivity of 48%. All three patients with neuron-specific enolase greater than 90 µg/L and Cerebral Performance Category 1–2 had confounders for neuron-specific enolase elevation. An neuron-specific enolase serum concentration less than or equal to 17 µg/L excluded Cerebral Performance Category 4–5 with a negative predictive value of 92%. The majority of 14 patients with neuron-specific enolase less than or equal to 17 µg/L who died had a cause of death other than hypoxic-ischemic encephalopathy. Specificity and sensitivity for prediction of poor outcome were independent of age, sex, and initial rhythm but higher for out-of-hospital cardiac arrest than for in-hospital cardiac arrest patients.

Streitberger KJ, Leithner C, Wattenberg M, Tonner PH, Hasslacher J, Joannidis M, Pellis T, Di Luca E, Födisch M, Krannich A, Ploner CJ, Storm C. Neuron-Specific Enolase Predicts Poor Outcome After Cardiac Arrest and Targeted Temperature Management: A Multicenter Study on 1,053 Patients. Crit Care Med. 2017 Jul;45(7):1145-1151.

tirol kliniken

Serum tau as a predictor for neurological outcome after cardiopulmonary resuscitation

Julia Hasslacher[a], Verena Rass[b], Ronny Beer[b], Hanno Ulmer[c], Christian Humpel[d], Alois Schiefecker[b], Georg Lehner[a], Romuald Bellmann[a], Michael Joannidis[a,], Raimund Helbok[b]*

[a] *Division of Intensive Care and Emergency Medicine, Department of Internal Medicine, Medical University Innsbruck, Anichstr. 35, 6020 Innsbruck, Austria*
[b] *Department of Neurology, Neurological Intensive Care Unit, Medical University Innsbruck, Anichstr. 35, 6020 Innsbruck, Austria*
[c] *Department of Medical Statistics, Informatics and Health Economics, Medical University Innsbruck, Schöpfstraße 41/1, 6020 Innsbruck, Austria*
[d] *Department of Psychiatry, Psychotherapy and Psychosomatics, Medical University Innsbruck, Anichstr. 35, 6020 Innsbruck, Austria*

Abstract

Aim: We evaluated serum tau protein as biomarker for poor neurological outcome over an extended observation period in patients after successful cardiopulmonary resuscitation (CPR) treated with mild therapeutic hypothermia (MTH) or normothermia (NT).

Methods: This is a retrospective analysis of a prospective observational study including 132 patients after successful CPR. Serum tau was determined in 24 h intervals for up to 168 h after CPR. Patients were treated with MTH targeting a temperature of 33 °C for 24 h or NT according to current guidelines. Neurological outcome was assessed with the Cerebral Performance Categories Scale (CPC) at hospital discharge.

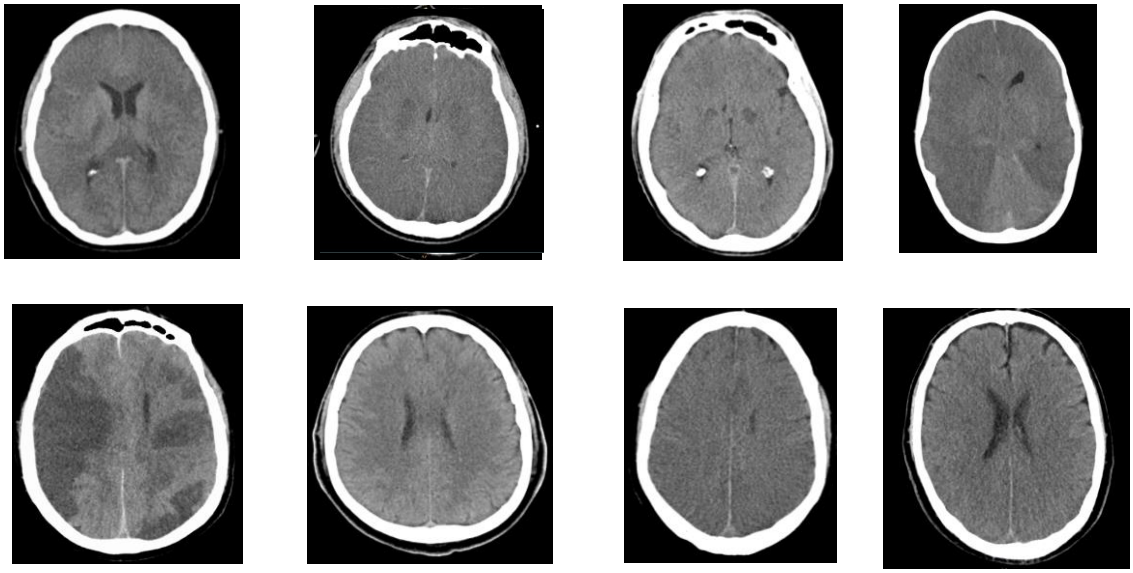
Results: Forty-three percent of the patients were treated with MTH. Serial serum tau levels (pg/ml) showed a peak between 72–96 h after CPR (159 (IQR 27–625). Patients with poor neurological outcome (CPC 3–5) at hospital discharge (n = 68) had significantly higher serum tau levels compared to patients with good neurological outcome at 0–24 h (164 (48–946) vs. 69 (12–224); p = 0.009), at 24–48 h (414 (124–1049) vs. 74 (0–215); p < 0.001), at 48–72 h (456 (94–1225) vs. 69 (0–215); p < 0.001) and at 72–96 h (691 (197–1173) vs. 73 (0–170); p < 0.001). At 72–96 h the AUC to predict poor neurological outcome was 0.848 (95% CI: 0.737–0.959). Serum tau levels were not significantly different between patients with MTH and NT in multivariate analysis after adjusting for clinical relevant covariates.

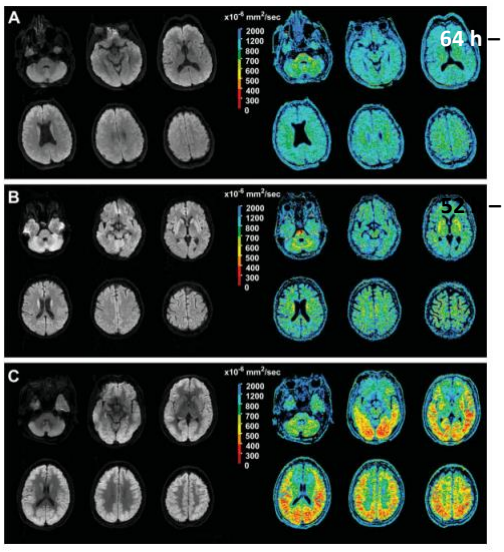
Conclusion: Serum tau showed highest values and the best prognostic discrimination of poor neurological outcome at 72–96 h after CPR. Prolonged elevation may indicate ongoing axonal damage in patients with hypoxic encephalopathy.

Keywords: Cardiac arrest, Cardiopulmonary resuscitation, Biomarker, Serum tau, Outcome, Prognostication, Inflammation, Hypothermia

Julia Hasslacher
Georg Franz Lehner
Ulrich Harler
Ronny Beer
Hanno Ulmer
Rudolf Kirchmair
Reiner Fischer-Colbrie
Romuald Bellmann
Stefan Dunzendorfer
Michael Joannidis

Secretoneurin as a marker for hypoxic brain injury after cardiopulmonary resuscitation



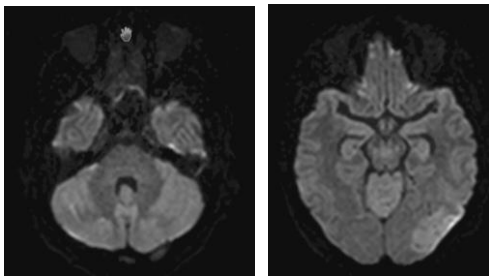


– good

– impaired

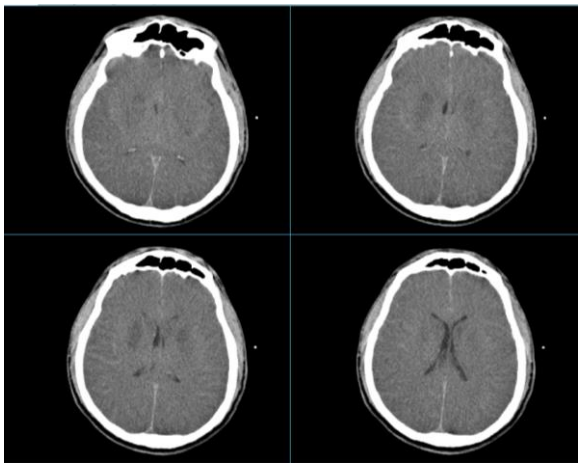
The **ideal time** window for prognostication using DWI was **between 49 and 108 hours** after the arrest. When comparing DWI in this time window with the 72-hour neurological examination, DWI improved the sensitivity for predicting poor outcome by 38% while maintaining 100% specificity (p 0.021).

Wijman CA, Mlynash M, Caulfield AF et al Prognostic value of brain diffusion-weighted imaging after cardiac arrest. Ann Neurol. 2009 Apr;65(4):394-402.

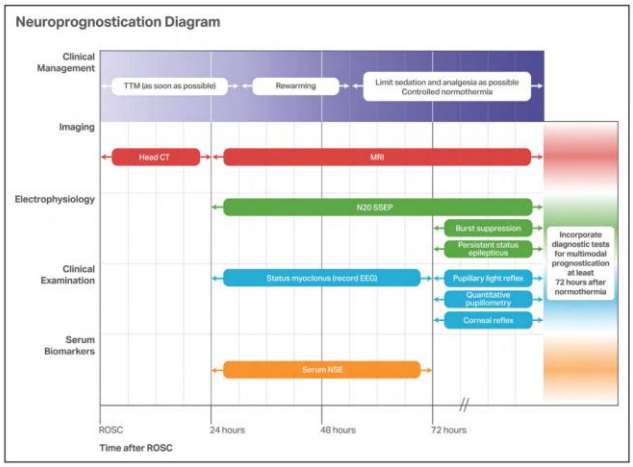


Eine multizentrische, prospektive Studie zur Quantifizierung der HIE mittels ‚diffusion tensor imaging' in der **zerebralen MRT** ergab eine sehr hohe Spezifität und Sensitivität für die Vorhersage **eines schlechten neurologischen** Outcomes bei Patient:innen, die
sieben Tage nach der Reanimation noch komatos waren. Die Methodik der Quantifizierung ist aber komplex und aktuell **nicht fur die klinische Routine verfugbar** Velly et al., 2018).

DGN-Leitlinien, AWMF Überarbeitung 2023 in press

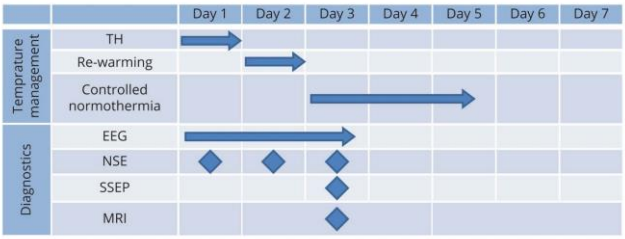


Die in der **zerebralen Computertomografie** darstellbaren Veränderungen bei schwerer hypoxisch-ischamischer Enzephalopathie entwickeln sich im Verlauf über die ersten Stunden/Tage nach Reanimation. Ein CCT zur neurologischen Prognose-Einschatzung sollte daher möglichst **>24 Stunden nach Herzstillstand und Reanimation** durchgeführt werden (Streitberger et al., 2019).



Panchal AR et al, Circulation. 2020;142(suppl 2):S366–S468.

Figure 1 Timing of targeted temperature management and clinical diagnostics



Hirsch K et al, Neurology 2020;94:e1684-1692. doi:10.1212

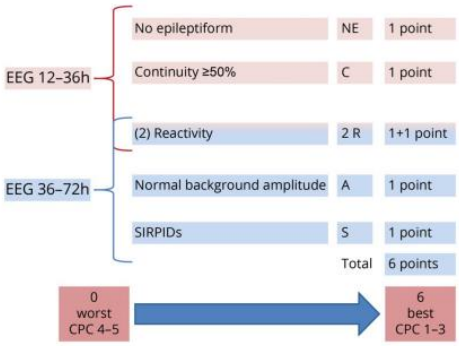
Take home massage

Die neurologische Prognoseeinstufung ist eine Summe aud Klinik, Bildgebung, Elektrophysiologie und Biomarker

Zeit ist zusätzlich ein entscheidende Faktor um die Dynamik einer Pathologie besser beurteilen zu können

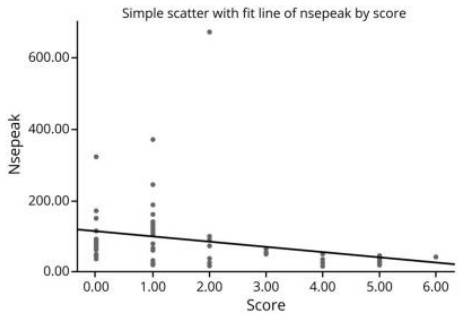
Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest

Figure 1 Algorithm summarizing the prognostic score



CPC = Cerebral Performance Category; SIRPID = stimulus-induced rhythmic periodic or ictal discharge.

Figure 3 Spearman rank correlation chart



Correlation between EEG score for Cerebral Performance Category 1–3 and neuron-specific enolase peak values in the derivation cohort is shown.

Barbella G et al. Prediction of regaining consciousness despite an early epileptiform EEG after cardiac arrest. Neurology. 2020 Apr 21;94(16):e1675-e1683.