Lo stato di male (SE) in PS

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Azienda Ospedaliera Universitaria

Vittorio Emanuele - Policlinico

Catania

Sessioni Interattive

SALA 2

Discussione casi clinici
URGENZE IN REPARTO

14.00 *DISTRESS RESPIRATORIO*
Conduce *U. Loaisa Levano*
Discussant *D.M.L. Simonetti*

14.45 *STATO DI MALE*
Conduce *R. Falsaperla*
Discussant *C. Minetti - P. Striano*

15.30 *ANAFILASSI*
Conduce *E. Tappi*
Discussant *P. Bertolani*

16.15 *SINDROME EMOLITICO-UREMICA*
Conduce *R. Lubrano*
Discussant *P. Scoppi*

Perché Stato di Male ?

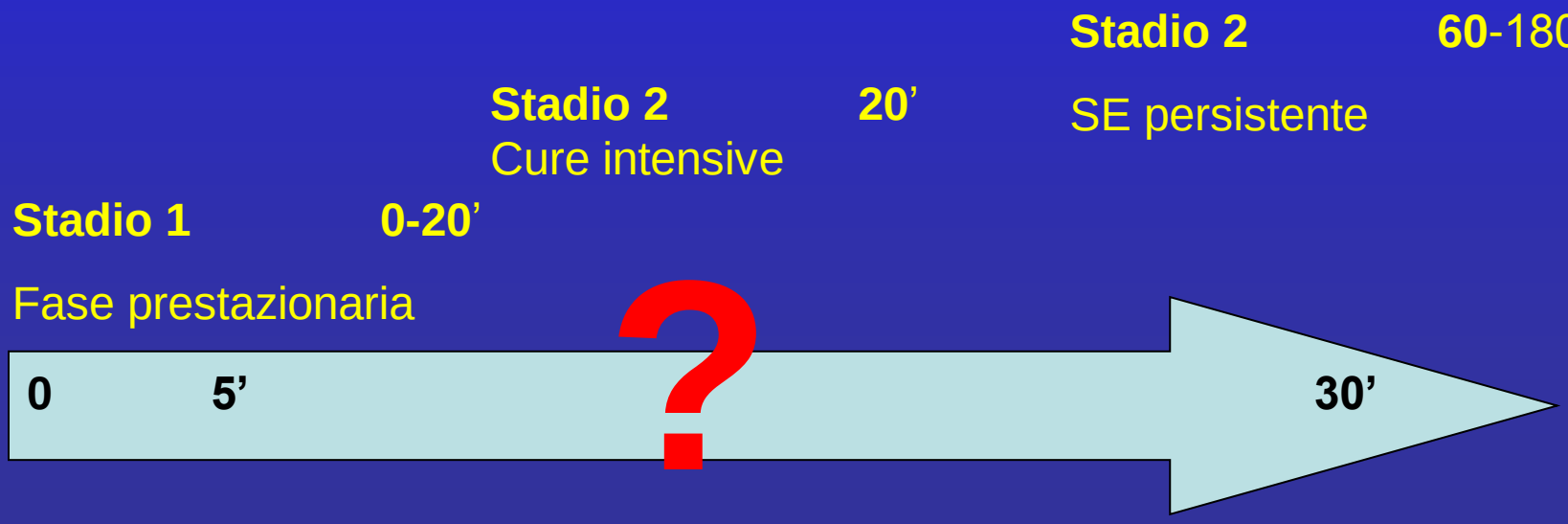
Urgenza

Diagnosi tempestiva

a. Riconoscere tempestivamente
b. Morbilità e Mortalità

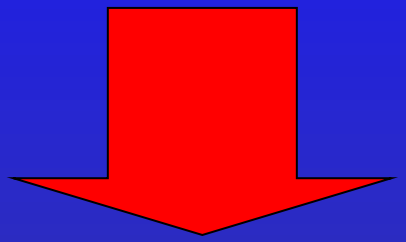
Terapia “efficace”

Non esiste una definizione universalmente accettata, in particolare per quanto riguarda la **durata delle manifestazioni cliniche epilettiche** (da **5 a 30 minuti**)



Definizione di SE

Epilepsy Foundation of America Working Group sullo SE (Epilepsy Foundation of America's Working Group on Status Epilepticus)



la somministrazione di farmaci antiepilettici deve iniziare se una crisi si protrae per più di 10 minuti (EFA 1993).

Definizione di SE

Considerando che:

una singola crisi raramente dura più di 2 minuti (Theodore et al.1994, Shinnar et al. 2001)

Definizione operativa:

negli adulti e nei bambini di età >5 anni, viene stabilita la presenza di uno stato epilettico generalizzato convulsivo (SEGC) qualora una crisi continua o due o più crisi distinte, tra le quali non vi sia un completo recupero della coscienza, si manifestino per più di 5 minuti (Lowenstein and Alldredge, 1998; Lowenstein, 1999, Lowenstein et al., 1999).

Pasquale Striano

Neurologo

Maria Finocchiaro

Pediatra PS

Hugo Loayza

Anestesista

Scenario:

- **Calogero è un bambino di 4 anni**
- **La madre riferisce che da 20 minuti non partecipa all'ambiente come di consueto**

  Risponde in modo adeguato a momenti a momenti no, se deambula cade a volte

Scenario

- **Il pediatra di PS (Maria Finocchiaro)**

All'arrivo in PS:

Anamnesi familiare e personale negativa per patologie degne di nota.
Non riferiti traumi, assunzione di farmaci e/o di sostanze tossiche.

ESAME CLINICO

A -> vie aeree pervie
B-> respiro spontaneo (FR 35 a/m, SatO2 in a.a. 99%) ->posizionare monitor
C -> polso regolare (FC 100 b/m ; PA 105/65 mm/Hg); reperire accesso venoso
D-> AVPU-> A. (alert); V. (voice); P. (pain); U.(unresponsive) -> vigile, tuttavia risponde in modo monosillabico e interagisce in maniera non sempre appropriata
E-> assenza di lesioni cutanee (manifestazioni emorragiche, macchie ipo o ipercromiche, emangiomi,) e/o temperatura febbrile

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Show additional filters

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Article types
More ...

Text availability
Abstract available
Full text available

Publication dates
5 years
10 years
Custom range...

Species
Humans

Ages clear
✓ Child: birth-18 years
Adult: 19+ years
Adult: 19-44 years
Aged: 65+ years
More ...

Clear all

Show additional filters

Display Settings: Summary, Sorted by Recently Added Send to:

Results: 4

Filters activated: Child: birth-18 years. Clear all to show 25 items.

1. "Relevance vector machine" **consciousness** classifier applied to cerebral metabolism of vegetative and locked-in patients.
Phillips CL, Bruno MA, Maquet P, Boly M, Noirhomme Q, Schnakers C, Vanhaudenhuyse A, Bonjean M, Hustinx R, Moonen G, Luxen A, Laureys S.
Neuroimage. 2011 May 15;56(2):797-808. doi: 10.1016/j.neuroimage.2010.05.083. Epub 2010 Jun 4.
PMID: 20570741 [PubMed - indexed for MEDLINE]
Related citations

2. Fear of loss of vigilance: development and preliminary validation of a self-report instrument.
Tsao JC, Craske MG.
Depress Anxiety. 2003;18(4):177-86.
PMID: 14661187 [PubMed - indexed for MEDLINE]
Related citations

3. Aetiology and outcome of non-traumatic **altered states** of **consciousness** in north western Ethiopia.
Melka A, Tekie-Haimanot R, Assefa M.
East Afr Med J. 1997 Jan;74(1):49-53.
PMID: 9145579 [PubMed - indexed for MEDLINE]
Related citations

4. Evaluating sensory regulation as a method to improve awareness in patients with **altered states** of **consciousness**: a pilot study.
Wood RL, Winkowski TB, Miller JL, Tierney L, Goldman L.
Brain Inj. 1992 Sep-Oct;6(5):411-8.
PMID: 1393174 [PubMed - indexed for MEDLINE]
Related citations

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Filters: Manage Filters

New feature
Try the new Display Settings option - **Sort by Relevance**

3 free full-text articles in PubMed Central
Psychometric **evaluation of** the **altered states of consciousness** rating scale [PLoS One. 2010]
Review Improving the **clinical** assessment **of consciousness** with advance [BMC Neurol. 2010]
Altered states of consciousness profile: an Afro-centric intrapsychic **ev** [J Natl Med Assoc. 1985]
See all (3)...

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Database: Select
Find items

Search details
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(altered[All Fields] AND
states[All Fields] AND
("consciousness"[MeSH
Terms]
OR "consciousness"[All
```
Search See more...

Recent Activity

Glasgow coma score pediatrico

Apertura degli occhi	Spontanea	**4**
	Alla chiamata	**3**
	Allo stimolo doloroso	**2**
	Non apre gli occhi	**1**
Risposte verbali	Giocherella, balbetta	**5**
	Pianto irritato	**4**
	Pianto da dolore	**3**
	Emette solo gemiti	**2**
	Non risponde	**1**
Risposte motorie	Si muove spontaneamente	**6**
	Si retrae al tocco	**5**
	Si retrae al dolore	**4**
	Flette al dolore	**3**
	Estende al dolore	**2**
	Non reagisce	**1**

Punteggio normale 15
Lieve alterazione del sensorio : 14-12
Trauma cranico maggiore : 12-8
Indicazione rianimatoria: <8

Alla Valutazione neurologica:

Sensorio vigile
NN cranici app. indenni
Tono e forza muscolare conservati
Andatura incerta, a larga base d'impianto
Rot presenti e simmetrici
Segni meningei assenti
Disturbo della interazione con disorientamento spazio temporale intermittente associato a comportamento inconsueto (la madre riferisce letteralmente "non lo riconosco") caratterizzato da riso immotivato, linguaggio povero, utilizzo improprio degli oggetti

Ipotesi diagnostiche:

Tumore ?

Trauma cranico ?

Avvelenamento ?

Stroke ?

Encefalite ?

Crisi convulsiva ?

Squilibrio metabolico ?

Esami di laboratorio e strumentali

Glicemia a determinazione rapida, emogasanalisi, Tac encefalo-> nella norma

Contestualmente si richiedono altri esami ematochimici di routine compresi test di coagulazione, ammoniemia, ed esame urine

Rivalutazione clinica > esame invariato

.....e se fosse una crisi convulsiva ?????

EEG URGENTE

Pasquale Striano

Neurologo

Eziologia

- convulsioni febbrili prolungate
- infezioni
- errori del metabolismo
- squilibri idroelettrolitici (ipoglicemia, iponatriemia, ipocalcemia)
- tumori
- disgenesie cerebrali (disgenesia del corpo calloso, displasia corticale)
- intossicazioni (farmaci, droghe, monossido carbonio)
- Idiopatico (genetico)

New 4-axes classification of SE

- 1) Semiology

-prominent motor systems

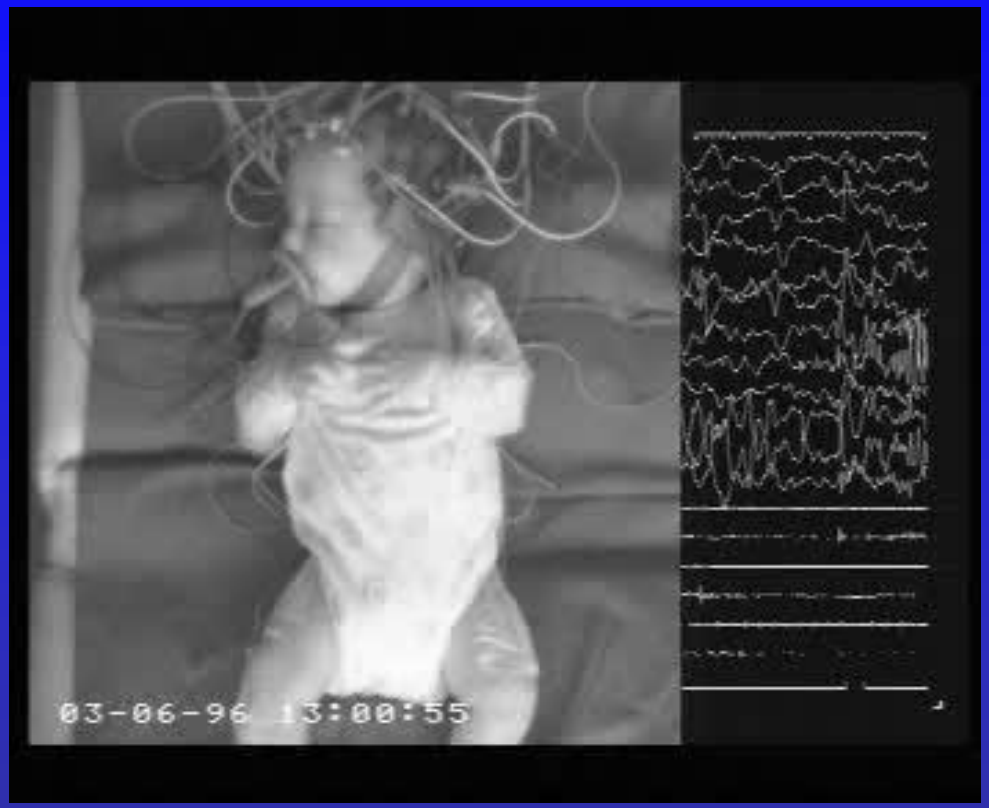
-without prominent motor systems

-indeterminate conditions (confusional states with epileptiform EEG)

- 2) Aetiology

- 3) EEG correlates

- 4) Age (neonatal, infancy, childhood, adolescent, adulthood, elderly)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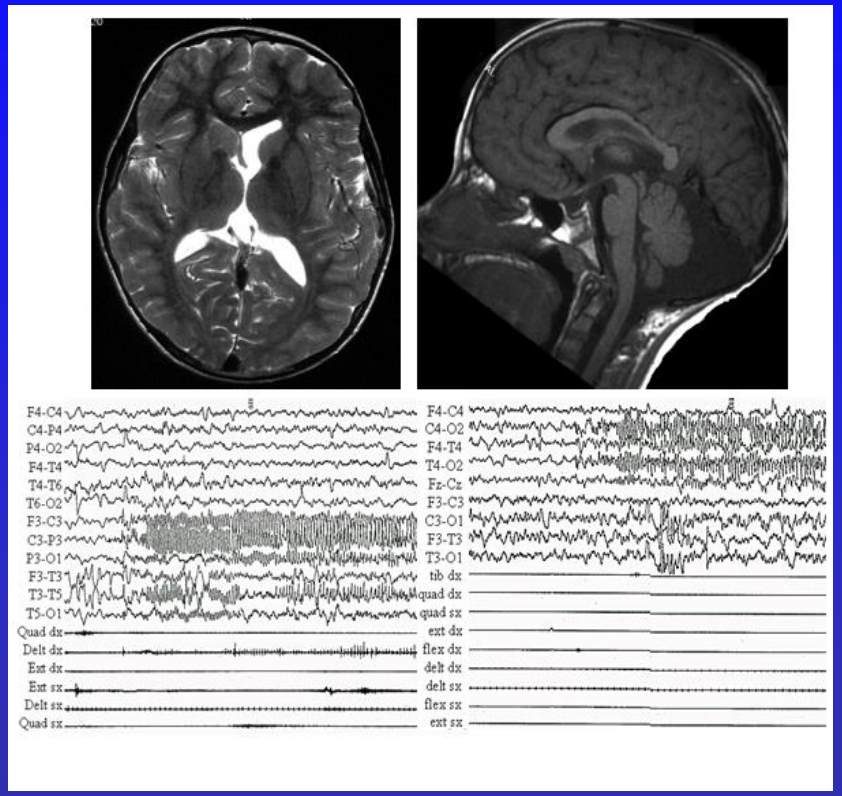
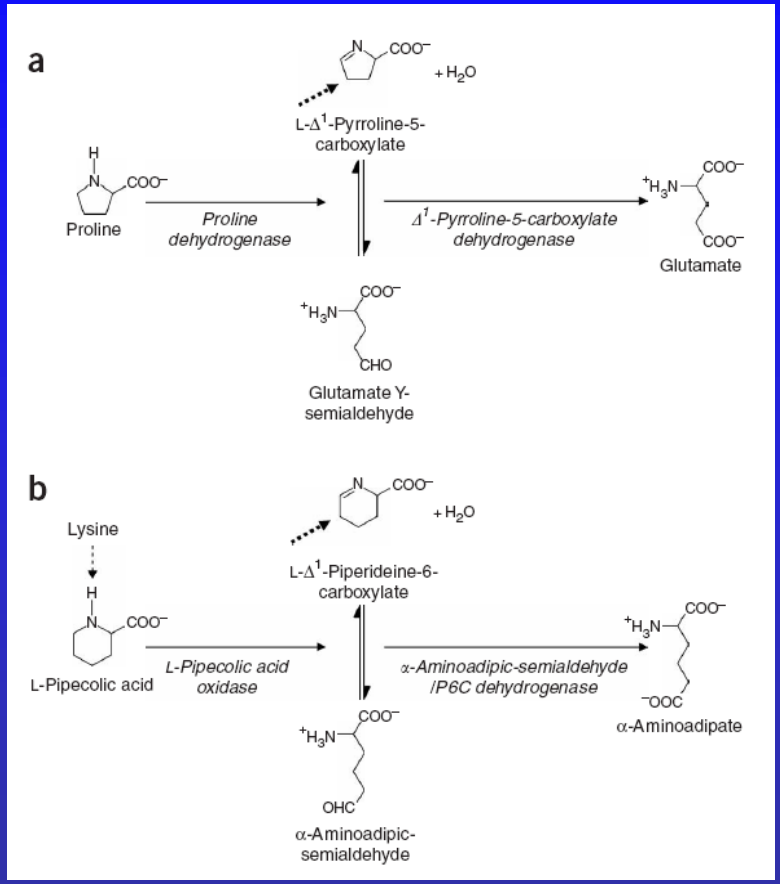

F4-C4
C4-P4
P4-O2
F4-T4
T4-T6
T6-O2
F3-C3
C3-P3
P3-O1
F3-T3
T3-T5
T5-O1
Quad dx
Delt dx
Ext dx
Ext sx
Delt sx
Quad sx
F4-C4
C4-O2
F4-T4
T4-O2
Fz-Cz
F3-C3
C3-O1
F3-T3
T3-O1
tib dx
quad dx
quad sx
ext dx
flex dx
delt dx
delt sx
flex sx
ext sx

Mutations in antiquitin in individuals with pyridoxine-dependent seizures

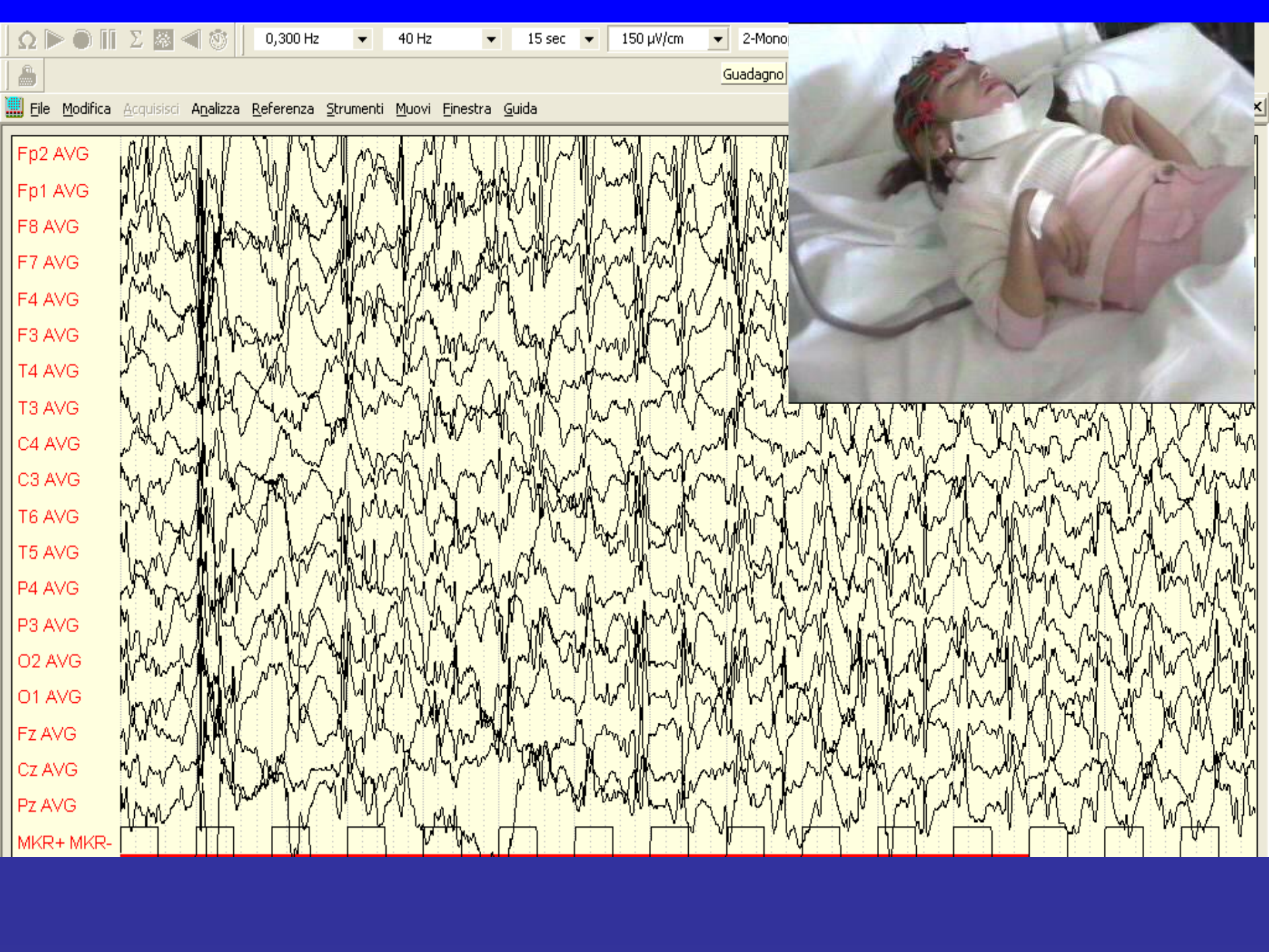
Philippa B Mills[1], Eduard Struys[2], Cornelis Jakobs[2], Barbara Plecko[3], Peter Baxter[4], Matthias Baumgartner[5], Michèl A A P Willemsen[6], Heymut Omran[7], Uta Tacke[7], Birgit Uhlenberg[8], Bernhard Weschke[8] & Peter T Clayton[1]

We show here that children with pyridoxine-dependent seizures (PDS) have mutations in the *ALDH7A1* gene, which encodes antiquitin; these mutations abolish the activity of antiquitin as a Δ^1-piperideine-6-carboxylate (P6C)–α-aminoadipic semialdehyde (α-AASA) dehydrogenase. The accumulating P6C inactivates pyridoxal 5′-phosphate (PLP) by forming a Knoevenagel condensation product. Measurement of urinary α-AASA provides a simple way of confirming the diagnosis of PDS and *ALDH7A1* gene analysis provides a means for prenatal diagnosis.



- Pathognomic levels of α-aminoadipic semialdehyde (α-AASA) are present have been reported in PDS.
- ALDH7A1 encodes for antiquitin, a α-AASA dehydrogenase in pipecolic acid pathway of lysine catabolism. Its deficiency causes seizures because accumulating P6C condenses with pyridoxal 5'-phosphate and inactivates this enzyme cofactor essential for metabolism of neurotransmitters and several amino acids.

NATURE MEDICINE VOLUME 12 | NUMBER 3 | MARCH 2006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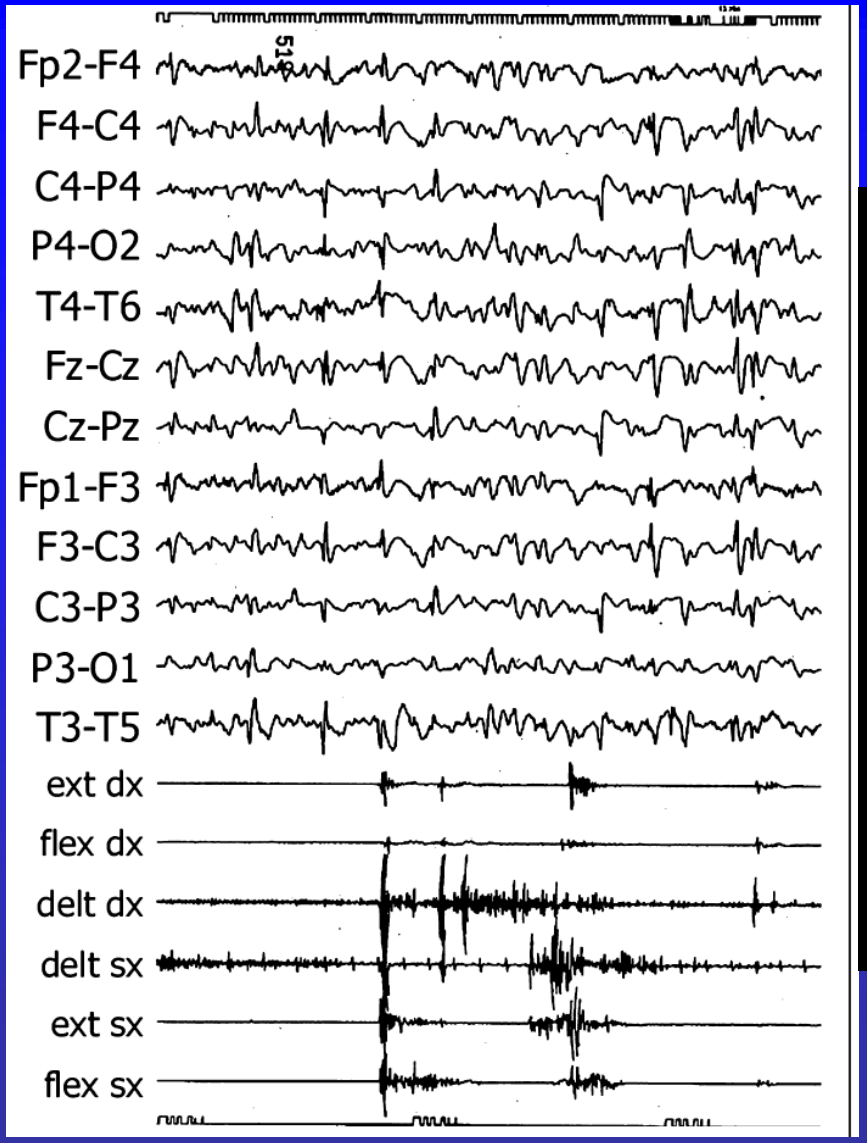
MKR+ MKR-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P3-O1
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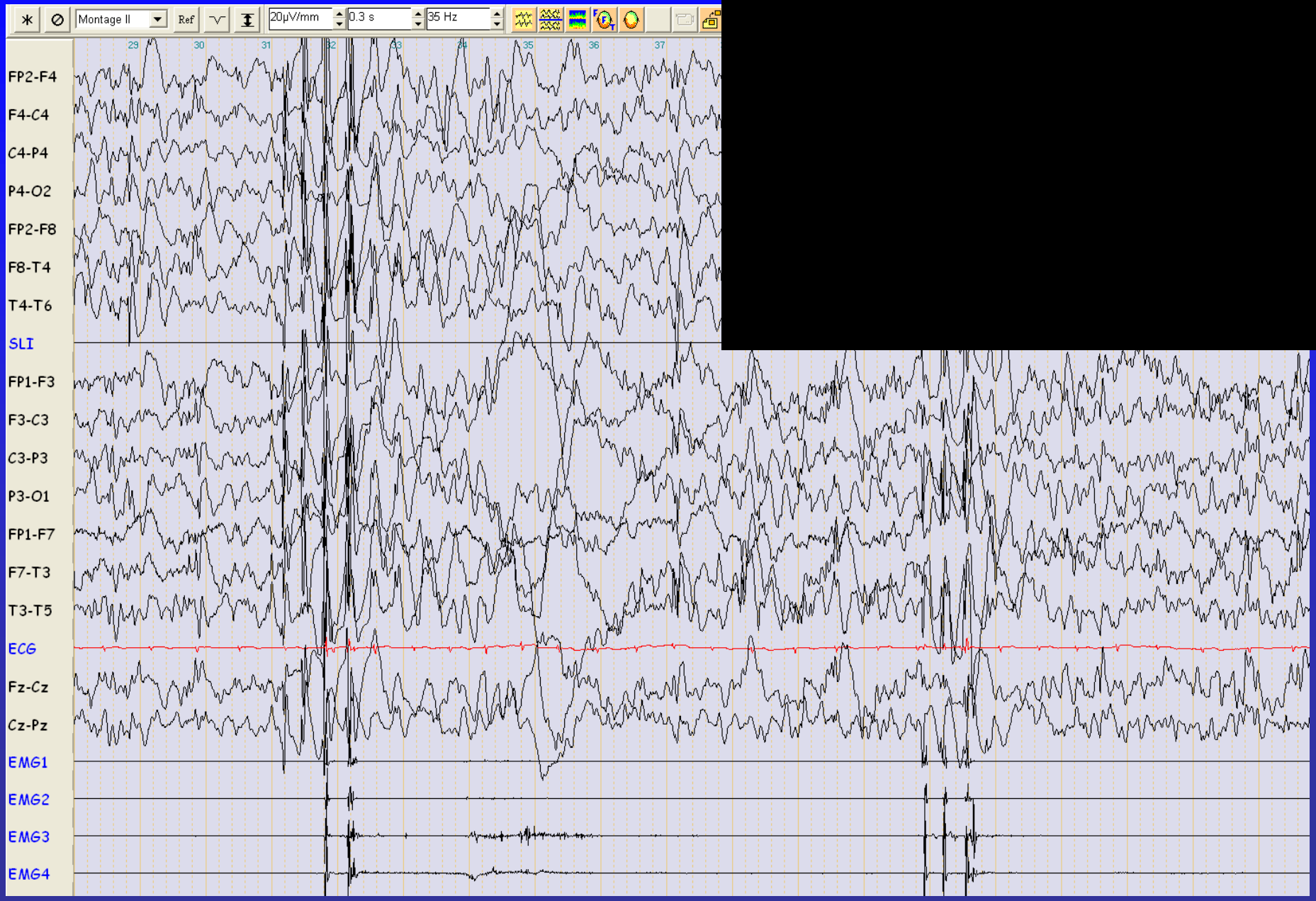
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P4-O2
FP2-F8
F8-T4
T4-T6
SLI
FP1-F3
F3-C3
C3-P3
P3-O1
FP1-F7
F7-T3
T3-T5
ECG
Fz-Cz
Cz-Pz
EMG1
EMG2
EMG3
EMG4

Severe Myoclonic Epilepsy of infancy (SMEI)

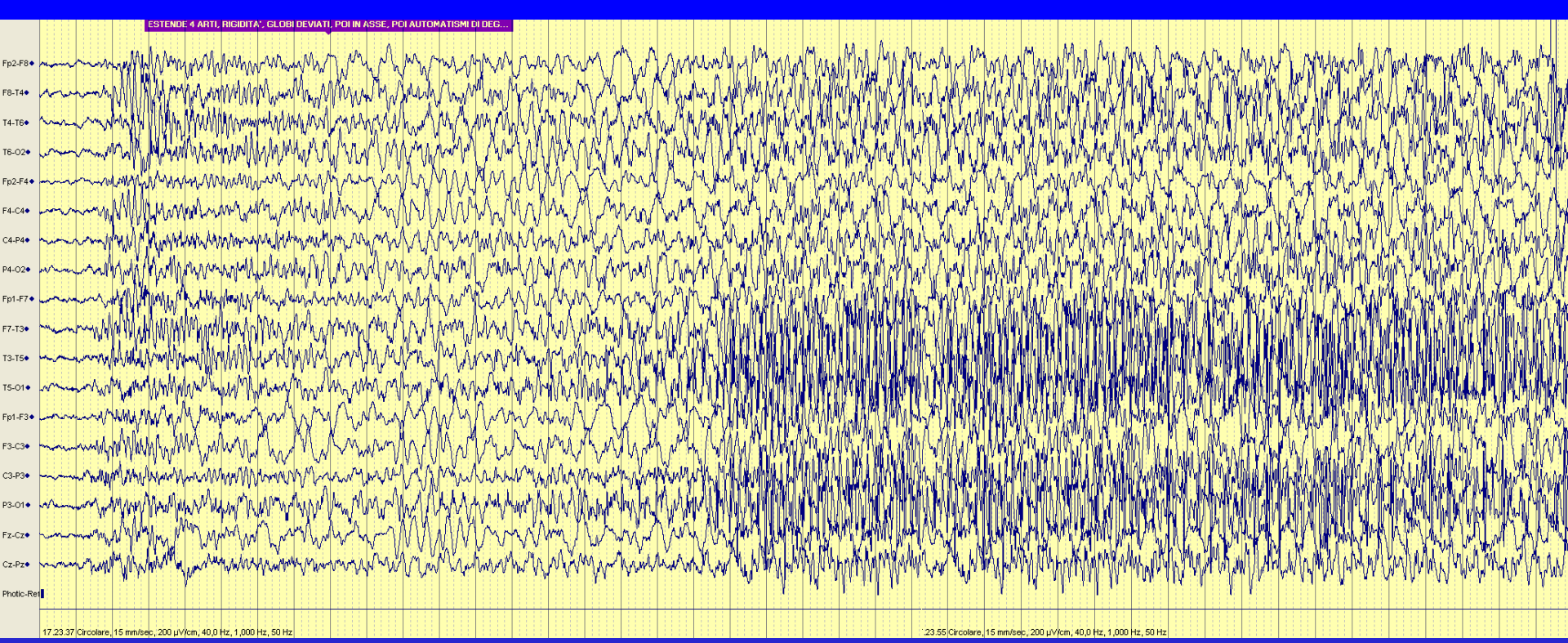
- Esordio entro un anno di vita con crisi febbrili o afebbrili, generalizzate o unilaterali
- Mioclonie
- Crisi farmacoresistenti
- Ritardo psicomotoro entro i due anni
- Storia negativa per lesioni cerebrali
- Sviluppo cognitivo e motorio all'esordio e RM normale nel primo anno di vita

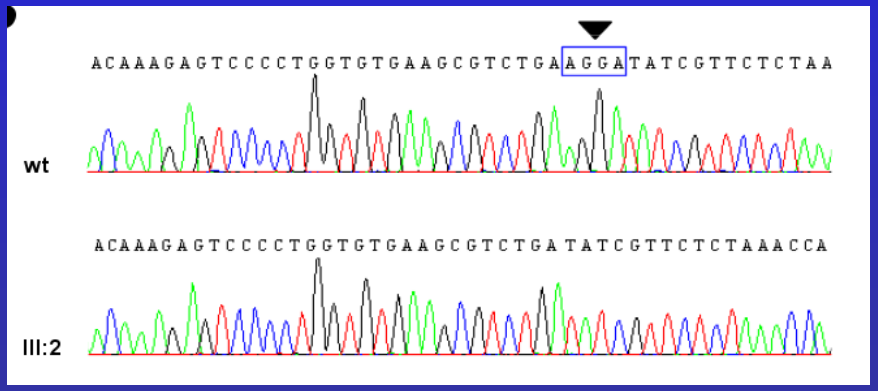
Am. J. Hum. Genet. 68:1327–1332, 2001

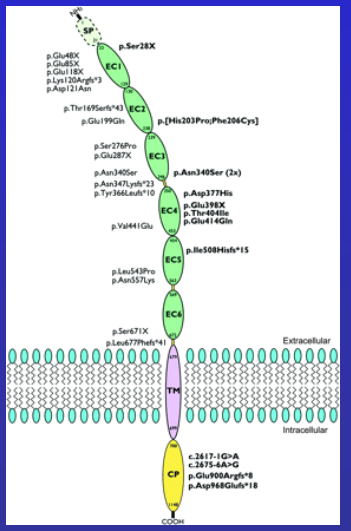
De Novo Mutations in the Sodium-Channel Gene *SCN1A* Cause Severe Myoclonic Epilepsy of Infancy

Lieve Claes,[1] Jurgen Del-Favero,[1] Berten Ceulemans,[2,3] Lieven Lagae,[3,4] Christine Van Broeckhoven,[1] and Peter De Jonghe[1,2]

[1]Department of Molecular Genetics, Flanders Interuniversity Institute for Biotechnology (VIB), University of Antwerp, and [2]Department of Neurology, University Hospital Antwerp, Antwerp; [3]Epilepsy Center for Children and Youth, Pulderbos, Belgium; and [4]Department of Child Neurology, University Hospital Gasthuisberg, Leuven, Belgium



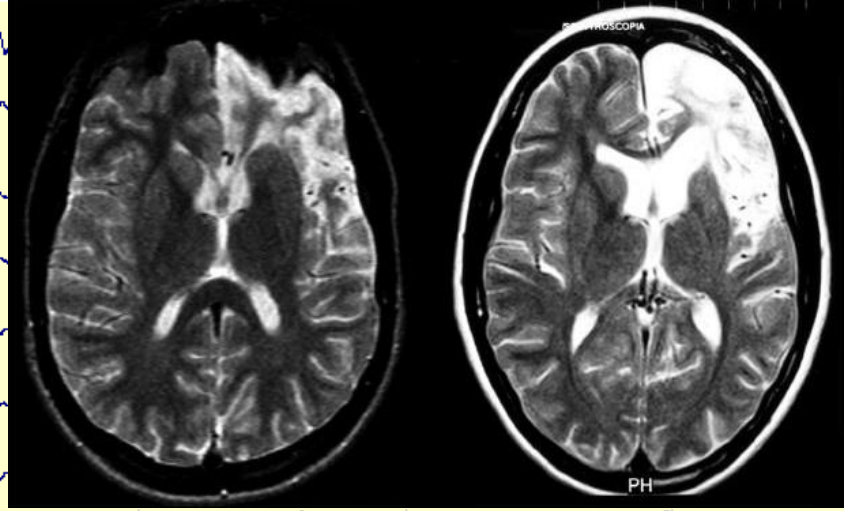




Epilepsia, 53(12):2111–2119, 2012
doi: 10.1111/j.1528-1167.2012.03649.x

FULL-LENGTH ORIGINAL RESEARCH

Focal seizures with affective symptoms are a major feature of *PCDH19* gene–related epilepsy



Fp2 AVG
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T4 AVG
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C4 AVG
C3 AVG
T6 AVG
T5 AVG
P4 AVG
P3 AVG
O2 AVG
O1 AVG
Fz AVG
Cz AVG
Pz AVG
MKR+ MKR-

Hugo Loayza Anestesista

Quando chiamare il intensivista?

- No Linee guida (LG)
- Status epillettico refrattario
- Assenza di risposta alle benzodiazepini e fenitoina
- Problemi di via aerea e ossigenazione-ventilazione ineficcaci
- Dopo 10-15 min?? Dopo 60 min ? Secondo alcuni alcuni protocolli

Obiettivi di approccio intensivistico 1-Mantenere ABCDE

A- assicurare la pervieta delle vie aeree

- intubazione endotracheale ?
- Gold standar
- Obligatorio nell caso di infusione continua di anestetici EV (TIVA)
- intubazione a sequenza rapida: etomidato, pentotal, rocuronio, evitare succinilcolina per rabdomiolosi
- intubazione con anestesia inalatoria
- sondino nasogastrico dopo la intubazione

B- ossigenazione e ventilazione adeguata

Ventilazione mecanica ?

- Riduce il consumo di ossigeno
- Previene IR effetto cumulativo BZD
- Durante la sominstrazione de anestetici endovenosi
- Trattamento complicanze (polmoniti,aspirazione polmonare,)

C- circolo

- Almeno 2 acessi venosi periferici
- alternativa: via intraossea
- preferiamo : periferica e centrale
- Carico emodinamico (liquidi.farmaci vasoativi) sopratutto durante somm pentobarbital

Obbietivi approccio intesivistico

- D- monitoragio specifico
- EEG continuo
- valutazione: completo silenzio elettrico (burst suppression) o rallentamento attività elettrica patologica, sopratutto nello stato di male epilettico non convulsivo
- BIS ?

Obiettivi del approccio intensivistico2- ottimizzare terapia anticonvulsiva

- Anestetici EV: sotto monitoraggio di EEG continuo
- Infusione continua di midazolam oppure propofol
- - acidosi metabolica o sindrome da propofol?
- Infusione di tiopentale
- Dosaggio di carico + dosaggio di mantenimento
- Altri farmaci ketamina.topiramato,etc

Obiettivi approccio intesivistico

2- Identificare e trattare la causa
3- Identificare e trattare le complicanze

Staging and order of drug administration in GTC-SE*

Stage 1: stage of early SE (0–10/30 min); drugs in order of preference

Lorazepam:	4 mg (adult) or 0.1 mg/kg (child) in IV slow bolus not exceeding 2 mg/min OR
Diazepam:	10–20 mg (adult) or 0.25-0.5 mg/kg (child) in IV bolus not exceeding 5 mg/min OR
Midazolam:	0.1–0.25 mg/kg in IV bolus not exceeding 0.15 mg/kg/min or 4 mg/min OR
Clonazepam:	1–2 mg (adult) or 0.01–0.03 mg/kg (child) in IV bolus not exceeding 1 mg/min

If after 10 minutes of first drug administration seizures continue a second dose can be given

Paraldehyde:	0.4 ml/kg rectally may be used in children in whom benzodiazepines have failed or intravenous access has not been established, or in patients with respiratory distress

If seizures continue after 30 min proceed to stage 2

Stage 2: stage of established SE (10/30–60/90 min)

Fosphenytoin:	IV infusion 15–20 mg PE/kg at a max rate of 150 mg PE/min OR
Phenytoin:	IV bolus 1 g (adult) or 15-20 mg/Kg (child) at a max rate of 50 mg/min (1 mg/Kg/min) OR
Phenobarbital:	IV infusion 10–20 mg/kg (adult) or 15-20 mg/Kg (child) at a max rate of 100 mg/min OR
Valproate:	IV infusion 25 mg/kg at 3–6 mg/kg/min

If seizures continue after 30–90 min proceed to stage 3

Stage 3: stage of refractory SE (>60/90 min)[14]

Propofol:	IV bolus 2 mg/kg, repeated if necessary, and then followed by a continuous infusion of 5–10 mg/kg/hour initially, reducing to a dose sufficient to maintain a burst suppression pattern on the EEG (usually 1–3 mg/kg/hour), OR
Thiopental:	IV bolus 100–250 mg given over 20 s with further 50 mg boluses every 2–3 min until seizures are controlled, followed by a continuous IV infusion at a dose sufficient to maintain a burst suppression pattern on the EEG (usually 3–5 mg/kg/hour), OR
Midazolam:	IV bolus 0.1–0.3 mg/kg at a rate not exceeding 4 mg/min initially, followed by a continuous IV infusion at a dose sufficient to maintain a burst suppression pattern on the EEG (usually 0.05–0.4 mg/kg/hour).

When seizures have been controlled for 12 hours, the drug dosage should be slowly reduced over a further 12 hours. If seizures recur, the drug infusion should be given again for another 12 hours, and then withdrawal attempted again. This cycle may need to be repeated every 24 hours until seizure control is achieved.

Confirmation of seizure termination with EEG may be mandatory because electroclinical dissociation is associated with a poor prognosis.

STATO EPILETTICO

ABC
Ossigenazione, protezione vie aeree eventuale intubazione e sostegno emodinamico

SatO2
ECG
dextrostick
vena periferica (quando possibile ottenere secondo accesso venoso)

MIDAZOLAM BUCCALE all'ingresso

se accesso venoso presente all'ingresso

se persiste

dopo 5 minuti - appena disponibile accesso venoso **MIDAZOLAM** EV

se persiste

dopo 5 minuti ripetere dose **MIDAZOLAM** EV

2 dosi totali ev di benzodiazepine compreso territorio

*IN PZ IN TERAPIA CON ANTICONVULSIVANTI
vedere programma individuale o consultare neurologo

se persiste

se eta` < 2 anni **PIRIDOSSINA** bolo 200 mg ev

dopo 5 minuti **FENITOINA** EV

attivazione EEG

se persiste

dopo 10 minuti **ACIDO VALPROICO** EV oppure **LEVETIRACETAM** EV (secondo indicazione del neurologo)

attivazione RM urgente se primo episodio di SE

se persiste

dopo 5 minuti **FENOBARBITALE** EV

TRASFERIMENTO IN TERAPIA INTENSIVA

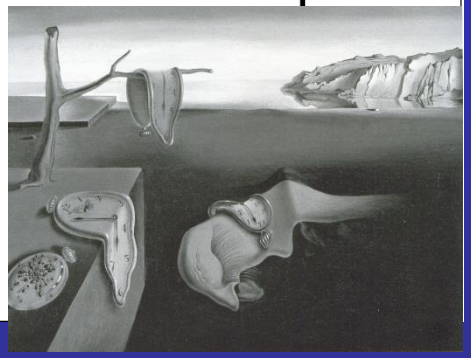
Indagini:

emogasanalisi
emocromo
coagulazione
elettroliti, Ca, Mg
AST ALT GGT
CPK LDH
bilirubina
creatinina
azotemia

livelli AEDs se in terapia

se indicato:
es. tossicologici
emocoltura
liquor

conservare campione di sangue (tappo rosso)



Cause di SE persistente.

1. dose iniziale (loading dose) troppo bassa
2. dose di mantenimento troppo bassa
3. terapia dello SE con un medicamento
   a) già utilizzato dal paziente → tolleranza
   b) a cui lo SE è resistente
4. sovradosaggio di un antiepilettico
5. fattori scatenanti insoliti
   – INH
   – epilessie vitamina B_6 dipendenti (nei bambini)
6. interpretazione errata di crisi non epilettiche
7. mancato riconoscimento di malattie primarie associate a lesioni organiche del cervello
8. mancato riconoscimento di fattori scatenanti sistemici
   – scompenso elettrolitico e metabolico
9. encefalopatia post-anossica
   – stato di male mioclonico

passaggio senza problemi alla terapia a lungo termine per via orale

Svantaggi

insicurezza posologica, la dose indicata sul foglietto illustrativo (20 mg/kg pc) probabilmente è troppo bassa, secondo noi meglio 25–30 mg/kg pc, nella letteratura fino a 100 mg/kg pc!

effetto emorragico:
a) trombocitopenia allergica
b) malattia di von Willebrand
c) calo isolato del fibrinogeno

ipotensione arteriosa [46]

non omologato per la terapia dello SE in Svizzera (tuttavia ora ha ottenuto l'omologazione in Norvegia)

Svolta epocale ……..

Neurocrit Care. 2012 Sep;17 Suppl 1:73-8.

Emergency neurological life support: status epilepticus.

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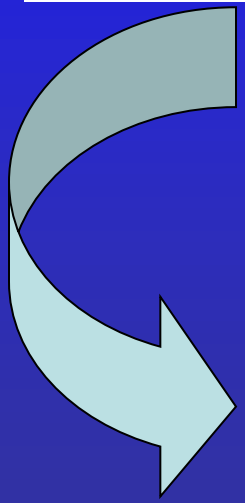
Abstract

Patients with prolonged or rapidly recurring convulsions lasting more than 5 min are in status epilepticus (SE) and require immediate resuscitation. Although there are relatively few randomized clinical trials, available evidence and experience suggest that early and aggressive treatment of SE improves patient outcomes, for which reason it was chosen as an Emergency Neurologic Life Support protocol. The current approach to the emergency treatment of SE emphasizes rapid initiation of adequate doses of first line therapy, as well as accelerated second line anticonvulsant drugs and induced coma when these fail, coupled with admission to a unit capable of neurologic critical care and electroencephalography monitoring. This protocol not only will focus on the initial treatment of SE but also review subsequent steps in the protocol once the patient is hospitalized.



Stato epilettico: 5 minuti

Rapido e tempestivo con dosi adeguate come first line therapy

Accelerare l’instaurarsi secondo farmaco anti convulsivante

Neurol Sci. 2012 Mar 7. [Epub ahead of print]

Association of antibiotics with status epilepticus.

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Abstract

The objective of this study is to evaluate clinical picture, radiological findings and response to treatment in patients with antibiotic associated status epilepticus (SE). In a retrospective review, 12 out of 117 (10%) patients with SE had temporal association with antibiotic administration. Their medical history, clinical findings, and duration and type of SE were recorded. Serum chemistry, blood counts, cranial MRI, EEG and CSF examination were carried out. The offending antibiotics were withdrawn and the patients were treated with phenytoin, lorazepam, sodium valproate or midazolam. The response to treatment was recorded and death during hospital stay noted. The median age of the patients was 36 (18 and 74) years and 5 were females. Eight patients had convulsive and four nonconvulsive SE. The median duration of status was 12 h. The antibiotics related to SE included intravenous cephalosporin (ceftazidime 5, amoxyclavulenic acid 2, piperacillin 2, cefepime 1) and quinolones (levofloxacin 3, ofloxacin 1, ciprofloxacin 2) in isolation or in combinations. Five patients had hepatic (41.7%) and 6 (50%) renal failure; the later received higher than the recommended dose of antibiotics. Cranial MRI was abnormal in 7 out of 9 (77.8%) patients that include cortical lesion in one, corticosubcortical in three and subcortical in three. SE responded to first antiepileptic drug in four and to second in five patients. Three patients (25%) had refractory SE. Eight (66.7%) patients died and death was related to SE in 2 patients. 10% SE patients may be related to antibiotics. Hence the antibiotic should be carefully chosen in patients with hepatic and renal failure, and the dose should be modified.

MIDAZOLAM *VERSUS* **DIAZEPAM** FOR THE TREATMENT **OF STATUS EPILEPTICUS IN CHILDREN AND YOUNG ADULTS: A META-ANALYSIS**

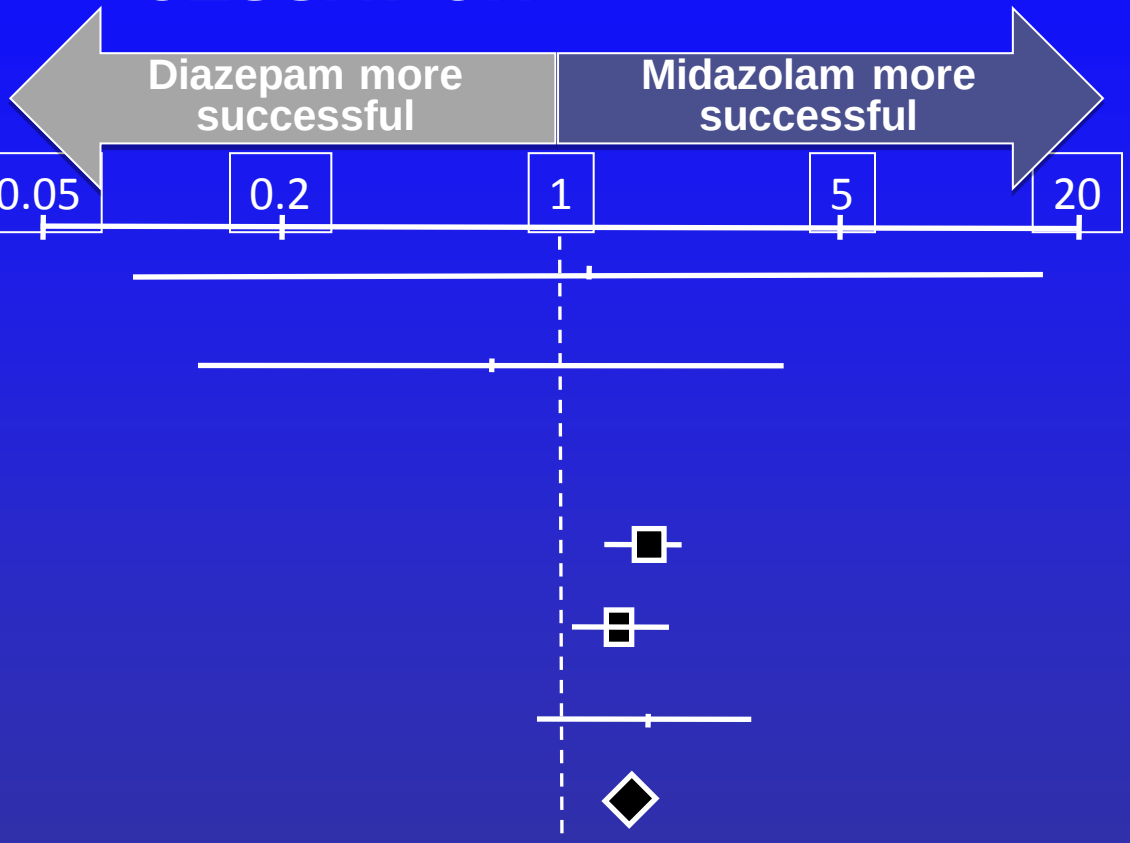
McMullan J, *et al*. *Acad Emerg Med* 2010; 17(6):575–582

META-ANALYSIS OF MIDAZOLAM *VS* DIAZEPAM: OVERVIEW OF INCLUDED STUDIES

Study	Patients, n	Age range	Diazepam dose; route	Midazolam dose; route	Definition of 'status'
Chamberlain *et al*. 1997	**24**	**0 months to 18 years**	0.3 mg/kg; IV	0.2 mg/kg; intramuscular	Seizing >10 minutes
Lahat *et al*. 2000	**52**	**6 months to 5 years**	0.3 mg/kg; IV	0.2 mg/kg; nasal	Seizing >10 minutes
Mahmoudian and Zadeh 2004	**70**	**2 months to 15 years**	0.2 mg/kg; IV	0.2 mg/kg; nasal	Seizing on arrival at ED
McIntyre *et al*. 2005	**219**	**6 months to 15 years**	0.5 mg/kg; rectal	0.5 mg/kg; oromucosal	Seizing on arrival at ED
Mpimbaza *et al*. 2008	**330**	**3 months to 12 years**	0.5 mg/kg; rectal	0.5 mg/kg; oromucosal	Seizing at arrival to ED or >5 minutes
Scott *et al*. 1999	**79**	**5 to 22 years**	10 mg; rectal	10 mg; oromucosal	Seizing >5 minutes

ED, emergency department
McMullan *et al*. 2010

META-ANALYSIS OF MIDAZOLAM *VS* DIAZEPAM: RISK RATIO OF FAILURE TO ACHIEVE SEIZURE CESSATION



Study (diazepam *vs* midazolam)
Chamberlain *et al*. 1997 (IV *vs* intramuscular)
Lahat *et al*. 2000 (IV *vs* intranasal)
Mahmoudian and Zadeh 2004[a] (IV *vs* intranasal)
McIntyre *et al*. 2005 (rectal *vs* oromucosal)
Mpimbaza *et al*. 2008 (rectal *vs* oromucosal)
Scott *et al*. 1999 (rectal *vs* oromucosal)
Total (95% confidence interval)

[a]Risk ratio could not be estimated for this study
McMullan *et al*. 2010

META-ANALYSIS OF MIDAZOLAM *VS* DIAZEPAM: KEY FINDINGS

- Varietà somministrazione
  - Midazolam è superiore al diazepam nel STOP crisi (RR 1.52, 95% CI 1.27–1.82)
- Non-IV somministrazione della somministrazione di midazolam
  - Non-IV midazolam più efficace della IV diazepam (RR 0.79, 95% CI 0.19–3.26)
  - Non-IV midazolam può essere somministrato più velocemente del IV diazepam (mean difference 2.46 minutes; 95% CI 1.52–3.39)
- Midazolam oromucosale
  - Oromucosal midazolam è superiore al diazepam rettale nel STOP crisi (RR 1.54, 95% CI 1.29–1.85)
- Tempo tra somministrazione farmaco e STOP crisi è similare tra non-IV midazolam and IV diazepam
  - Mean difference 0.68 minutes (95% CI –0.03 to 1.39)

CI, confidence interval; RR, risk ratio
McMullan *et al*. 2010

Conclusioni:

Alterazione cognitiva e/o comportamentale che dura per almeno **30 minuti** con una evidenza di crisi all'**EEG**

- La **Diagnosi è difficile** e richiede alto grado di sospetto diagnostico (spesso è sottodiagnosticata)
- **Non vi sono chiari e netti criteri diagnostici** EEGrafici
- Si osserva in **sindrome epilettiche, encefalopatie e condizioni acute**
- **Ulteriori dati** sono necessari per analizzare dati clinici ed EEG soprattutto in età pediatrica