



Epilepsy Course for Neurology and Pediatric Neurology Residents 2025

SUDEP

(Sudden Unexpected Death in Epilepsy)

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Disclosures

No financial disclosures relevant to this talk

Overview

- What is SUDEP & how often does it happen?
- What are the known risks for SUDEP?
- What causes SUDEP?
- How can SUDEP be prevented?

A leading cause of death in epilepsy

- Standardized Mortality Ratio (SMR): 2.2-2.6
- SMR higher for children: 5.3-9.0
- Predictors of mortality:
 - 1. Active epilepsy
 - 2. Symptomatic epilepsy
 - 3. ASM adherence
 - 4. Medical intractability
- Deaths related to epilepsy: direct/indirect, SUDEP
- Deaths related to the cause of epilepsy
- Deaths unrelated to epilepsy

"sudden death is 24-34X more likely in young PWE^{1,4}"

40-year follow-up of childhood-onset epilepsy: 30% mortality³

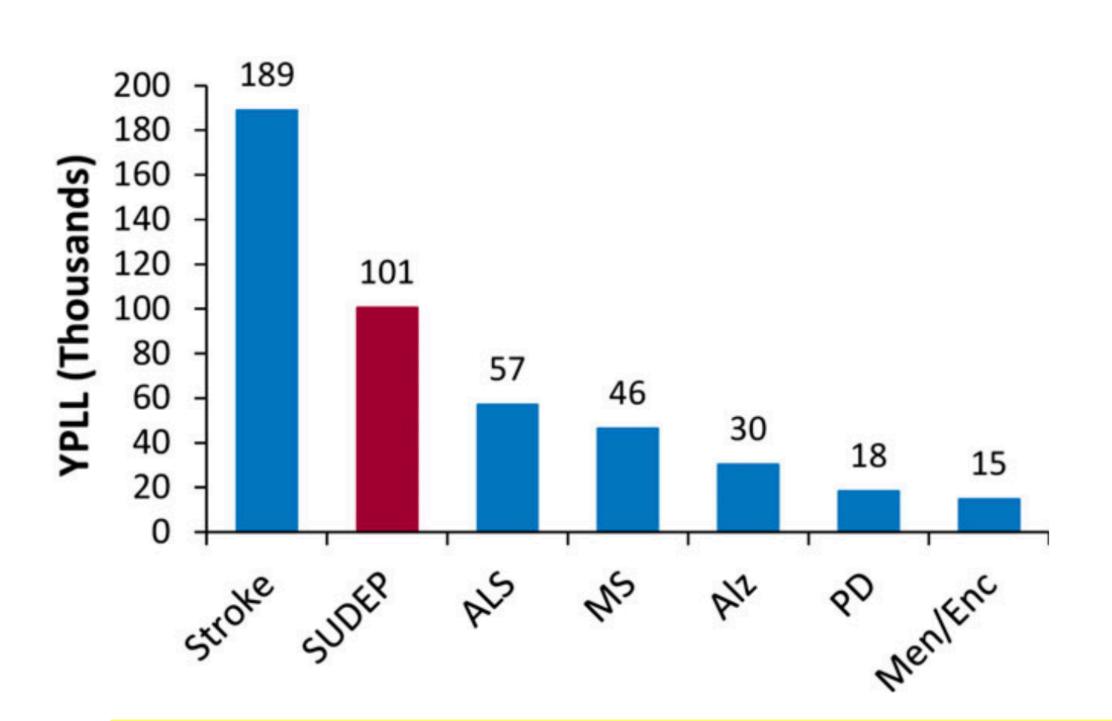
Table 2. Causes of Death.			
Variable	All Subjects (N = 245)	Subjects with Idiopathic or Cryptogenic Epilepsy (N=122)	Subjects with Epilepsy Due to Remote Symptomatic Causes (N=123)*
Total deaths — no.	60	15	45
Death related to epilepsy — no./total no. of deaths (%)	33/60 (55)	9/15 (60)	24/45 (53)
Witnessed seizure — no.	6	0	6
Status epilepticus — no.	4	0	4
Probable seizure — no.	3	2	1
Drowning — no.	6	0	6
Sudden, unexplained death — no.	18	7	11
Death not related to epilepsy — no./ total no. of deaths (%)	26 (43)	6 (40)	20 (44)
Pneumonia — no.	12	0	12
Cardiovascular disease — no.	8	2	6
Suicide — no.	2	2	0
Other cause of death — no.	4	2	2
Cause of death unknown — no.	1	0	1

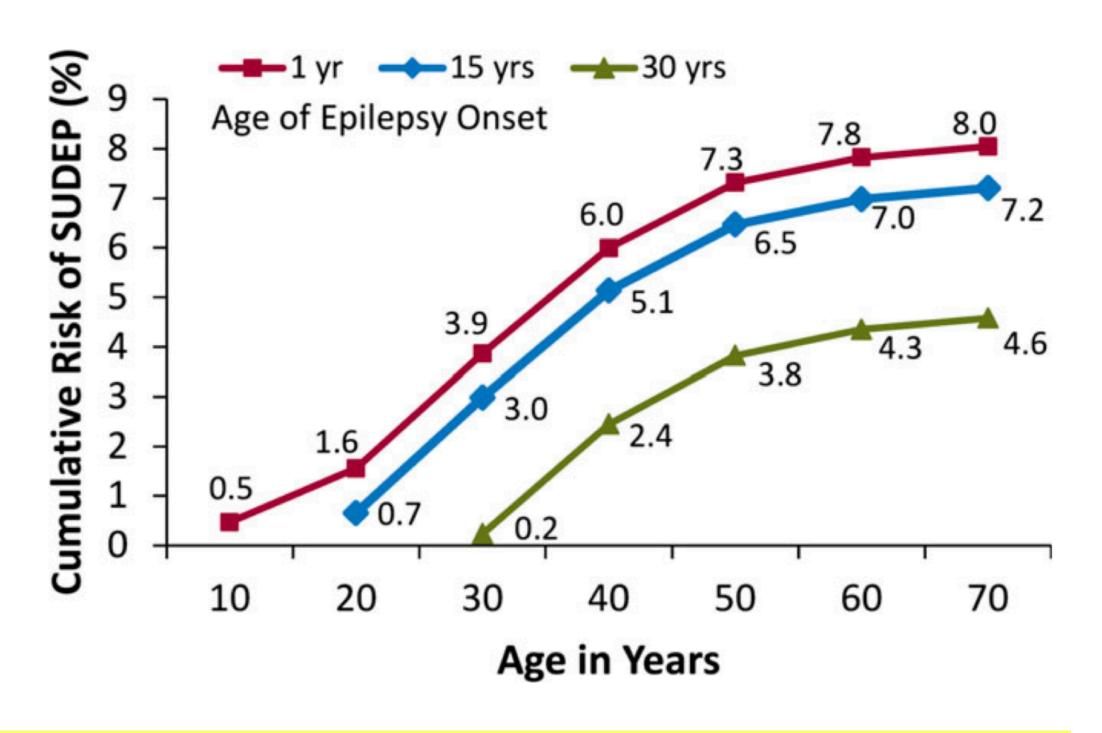
^{*} A remote symptomatic cause of epilepsy indicates epilepsy associated with a major neurologic abnormality or insult.

Sudden unexpected death in epilepsy: Assessing the public health burden

*David J. Thurman, †Dale C. Hesdorffer, and ‡Jacqueline A. French

Systematic search for epidemiologic studies of sudden death in epilepsy





- ✓ SUDEP ranks second only to stroke in term of years of potential life lost (YPLL)
- ✓ Lifetime risk of 4.6-8% by age 70

SUDEP is a diagnosis of exclusion

- Mas epilepsy, death was sudden and unexpected, in benign circumstances
- MOT a consequence of trauma, drowning, or status epilepticus
- May be witness or unwitnessed; evidence of a preceding seizure is NOT required
- Postmortem exam: NOT reveal cause of death = Definite SUDEP
- Without autopsy = *Probable SUDEP*; a competing cause of death = *Possible SUDEP*;
- Survives resuscitation >1 h = Near-SUDEP
- A clear cause of death is known = NOT SUDEP

Scenarios

- Sudden death in conjunction with witness <u>first seizure</u>; negative postmortem examination
 Does not meet criteria for epilepsy → Not SUDEP
- 2) Patient with uncontrolled epilepsy; found dead in the daytime; postmortem reveals <u>aspiration of gastric contents of unspecified amount</u>

 Possible SUDEP
- 3) Epilepsy; cardiorespiratory arrest after witnessed sz; resuscitated but dies within a few days, negative postmortem examination

Death occurred more than 1 h → Near-SUDEP

Risk of SUDEP varies within the epilepsy population

- Newly diagnosed epilepsy: 0.09 per 1000 patient-years
- Candidates for epilepsy surgery: 9 per 1000 patient-years



Practice guideline summary: Sudden unexpected death in epilepsy incidence rates and risk factors

Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology and the American Epilepsy Society

Population	SUDEP/1,000 patient-years (CI)	Confidence level
Overall	0.58 (0.31–1.08)	Low
Childhood	0.22 (0.16–0.31)	Moderate
Adulthood	1.2 (0.64–2.32)	Low

- What is the incidence of SUDEP?
- Are there specific risk factors for SUDEP?

Incidence

- Overall 1:1,700 pt-yrs
- Children 1:4,500 pt-yrs
- Adults 1:1,000 pt-yrs

Incidence of sudden unexpected death in epilepsy in children is similar to adults

Anne E. Keller, MPH, Robyn Whitney, MD, Shelly-Anne Li, MSc, Michael S. Pollanen, MD, PhD, and Elizabeth J. Donner, MD, MSc

Table Incidence of SUDEP by analysis method as compared to reported incidence in the literature

Method	Included classifications of SUDEP	No. of SUDEP cases	Epilepsy prevalence, %	Incidence (95% CI) per 1,000 patient-years
Crude analysis	All	17	0.27	1.17 (0.68–1.88)
Canada	Definite, definite plus, probable	16	0.27	1.11 (0.63–1.79)
Sensitivity analysis	Definite, definite plus, probable	16	0.21	1.42 (0.81–2.31)
	Definite, definite plus, probable	16	0.34	0.88 (0.50–1.42)
Capture-recapture analysis	Definite, definite plus, probable	21	0.27	1.45 (0.90–2.22)
From the literature				
Source	Included classifications of SUDEP	Рор	ulation	Incidence (95% CI) per 1,000 patient-years
AAN guidelines ¹	Definite, definite plus, ^a probable	"Chil	dhood"	0.22 (0.16-0.31)
	Definite, definite plus, ^a probable	"Adu	lt"	1.22 (0.64–2.32)
Sveinsson et al. ²	Definite, definite plus, probable	<16	у	1.11 (0.45–2.29)
Sweden	Definite, definite plus, probable	16–5	0 y	1.13 (0.76–1.62)
	Definite, definite plus, probable	>50	y	1.29 (0.88–1.82)

SUDEP in the North American SUDEP Registry

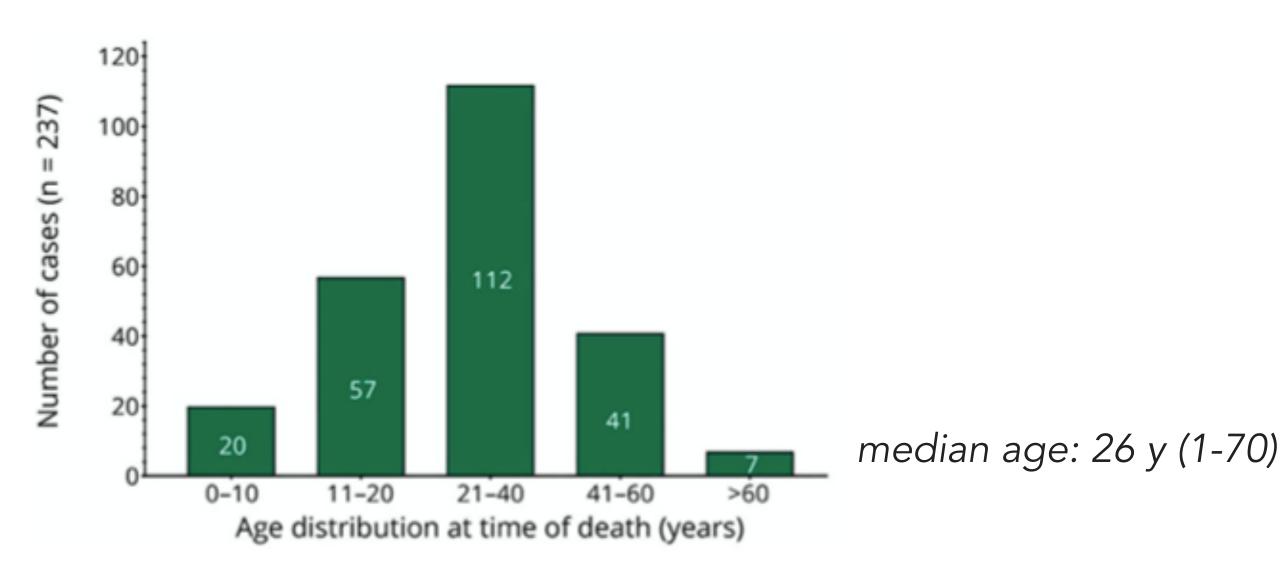
The full spectrum of epilepsies

Chloe Verducci, BA, Fizza Hussain, MS, Elizabeth Donner, MD FRCP(C), Brian D. Moseley, MD, Jeffrey Buchhalter, MD, Dale Hesdorffer, PhD, Daniel Friedman, MD, MSc, and Orrin Devinsky, MD

Table 1 Circumstances of death (n = 237)

Circumstance of death	n	N	%
Took last ASM dose?	66	180	37
Asleep at time of death	118	168	70
Known recent illness	30	175	17
Room sharing during sleep	57	161	35
CPR performed	108	212	51
Sleep deprived	24	157	15
Full autopsy performed	155	237	65
Found in prone position	128	186	69
Evidence of preceding seizure	123	167	74

Young adult, during apparent sleep, were prone



- Low rate of witnessed death 7%
- Only 16% of next of kin had heard about SUDEP before their relatives' death

SUDEP in the North American SUDEP Registry

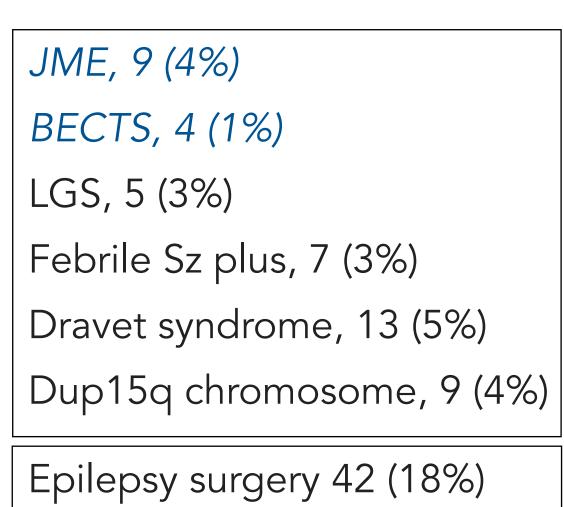
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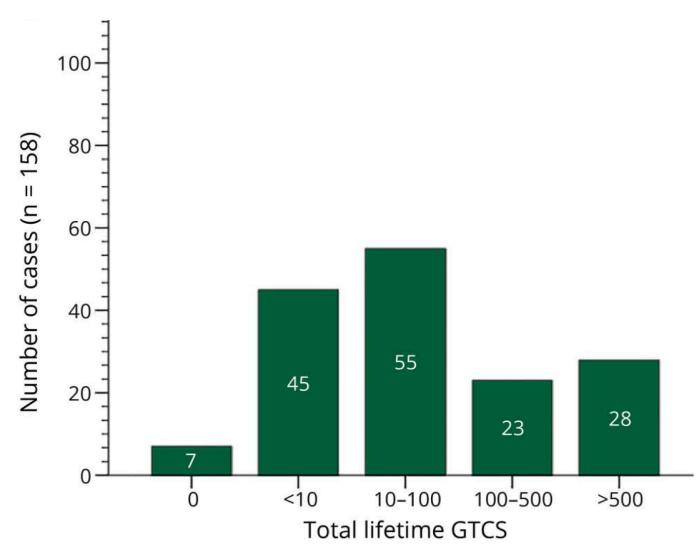
Table 3 Seizure histories in cases of SUDEP with sufficient information to adjudicate (143 of 237)

Seizure history	n	N	%
Generalized	57	143	40
Tonic-clonic	46	143	32
Focal	86	143	60
Focal to bilateral	67	143	47
Preserved awareness	20	143	14
Impaired awareness	59	143	41
Unclassified	94	237	40
Both	15	143	10

- GGE are also at risk
- SUDEP affects the full spectrum of epilepsies
- SUDEP risk is NOT limited to frequent GTCS



Neurostimulation 32 (14%)



A major risk factor is the presence & frequency of GTCS



Practice guideline summary: Sudden unexpected death in epilepsy incidence rates and risk factors

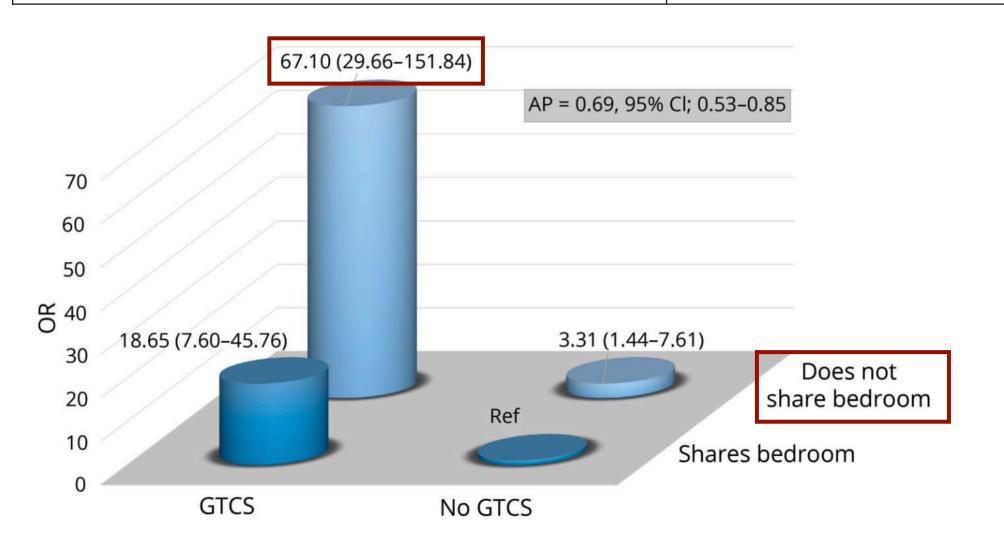
Report of the Guideline Development, Dissemination, and Implementation Subcommittee of the American Academy of Neurology and the American Epilepsy Society

Factor	Odds ratio (CI)	Confidence level
Presence of GTCS vs lack of GTCS	10 (7–14)	Moderate
Frequency of GTCS	OR 5.07 (2.94–8.76) for 1–2 GTCS per y, and OR 15.46 (9.92–24.10) for >3 GTCS per y	High
Not being seizure free for 1–5 y	4.7 (1.4–16)	Moderate
Not adding an AED when patients are medically refractory	6 (2–20)	Moderate
Nocturnal supervision (risk reduction)	0.4 (0.2–0.8)	Moderate
Use of nocturnal listening device (risk reduction)	0.1 (0.0–0.3)	Moderate

Clinical risk factors in SUDEP

A nationwide population-based case-control study

	OR (95% CI)
GTCS during the preceding year	26.8 (14.9-48.4)
Nocturnal GTCS during last year	15.31 (9.6-24.5)
Living alone	5.0 (2.9-8.6)
Sharing household, not bedroom	2.3 (1.1-4.6)



Medication adherence & intensified ASM can reduce SUDEP risk

	OR (95% CI)		
Nonadherence mentioned	2.7 (1.6-4.8) ²		
2 ASMs	0.6 (0.3-1.1) ²		
≥3 ASMs	0.3 (0.1-0.7) ²		
Efficacious adjunctive ASMs	0.2 (0.1-0.6)1		
Statin use	0.3 (0.1-0.9) ²		
SSRIs	0.6 (0.3-1.4) ²		
Alcohol dependence, substance abuse	2.3 (1.0-5.2) ² , 2.1 (1.1-4.0) ²		
No increase risk associated with any specific ASM ²			

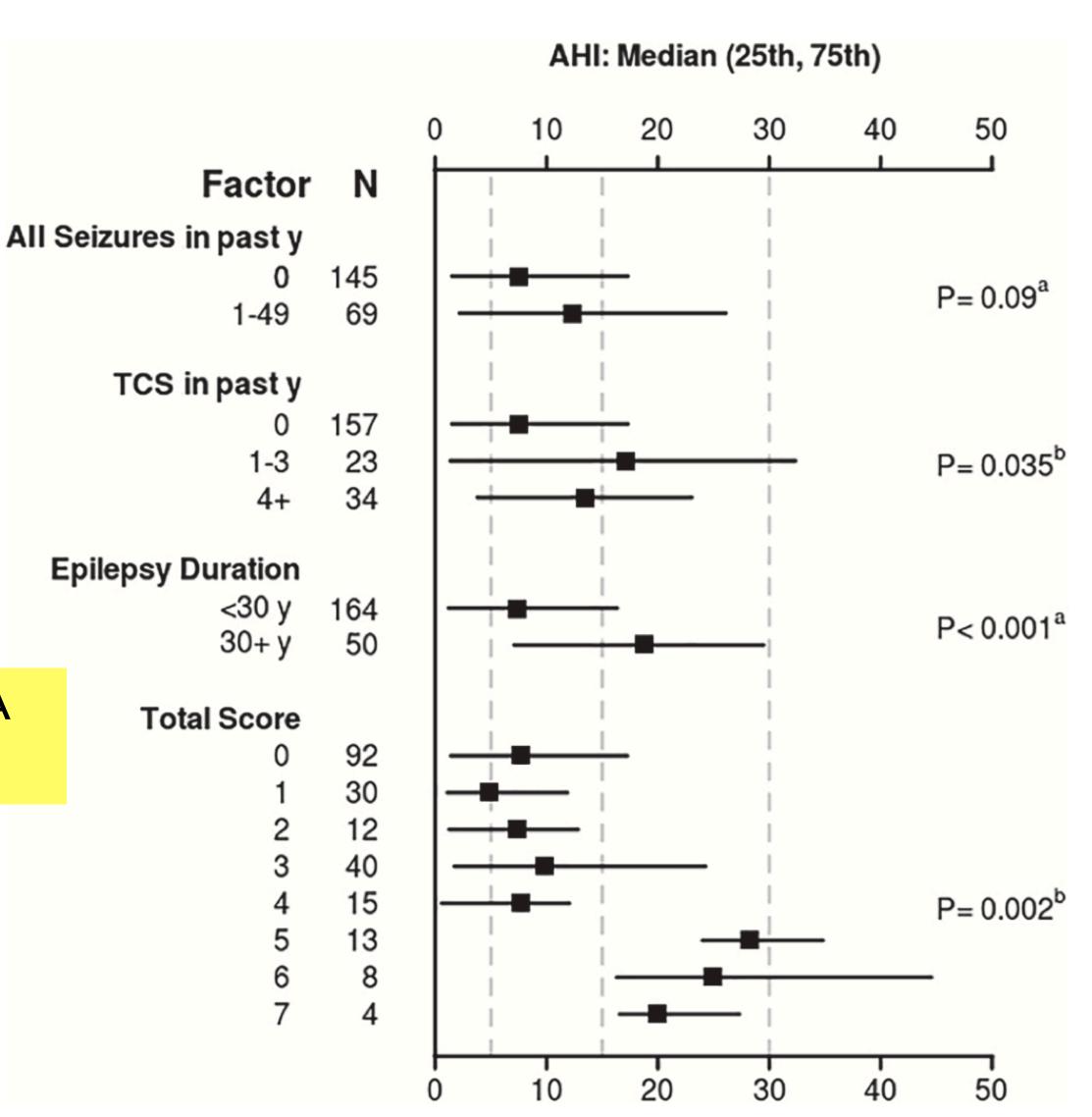


Obstructive sleep apnea is associated with risk for sudden unexpected death in epilepsy (SUDEP) using rSUDEP-7

Atiwat Soontornpun ^{a,b,1}, Christian Mouchati ^{a,1}, Noah D. Andrews ^a, Madeleine M. Grigg-Damberger ^d, Nancy Foldvary-Schaefer ^{a,*}

To evaluate relationships between OSA and SUDEP risk Adults with epilepsy who underwent PSG, N=214 SUDEP risk using revised SUDEP Risk Inventory (rSUDEP-7) (scores 0-10, higher score means greater risk)

Adults with epilepsy and moderate-to-severe OSA have higher risk of SUDEP



How does SUDEP happen?

- SUDEP is typically unwitnessed
- SUDEP is rare in a clinically monitored setting
- Hypotheses informed by indirect evidence
 - Death scene & autopsy findings
 - Clinical observations in non-fatal human seizures
 - Animal models of seizure-associated death

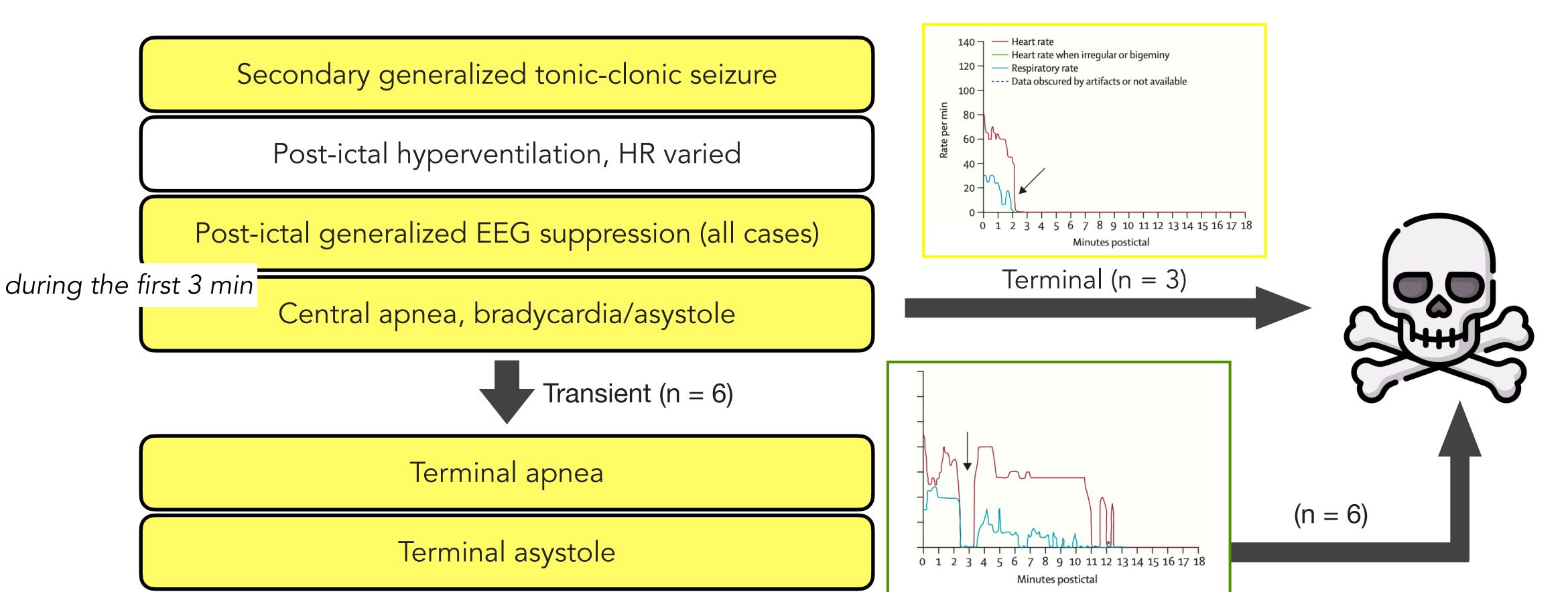


- 1. Post-ictal central apnea, neurogenic pulmonary oedema
- 2. Cardiac arrhythmia
- 3. Post-ictal cerebral electrical shutdown

Incidence and mechanisms of cardiorespiratory arrests in epilepsy monitoring units (MORTEMUS): a retrospective study

- Study pooled cardiorespiratory arrests during video-EEG admissions worldwide (147 units)
- 16 SUDEP, 9 near-SUDEP

Time to CPR: SUDEP 13-180 min; near-SUDEP ≤3 min

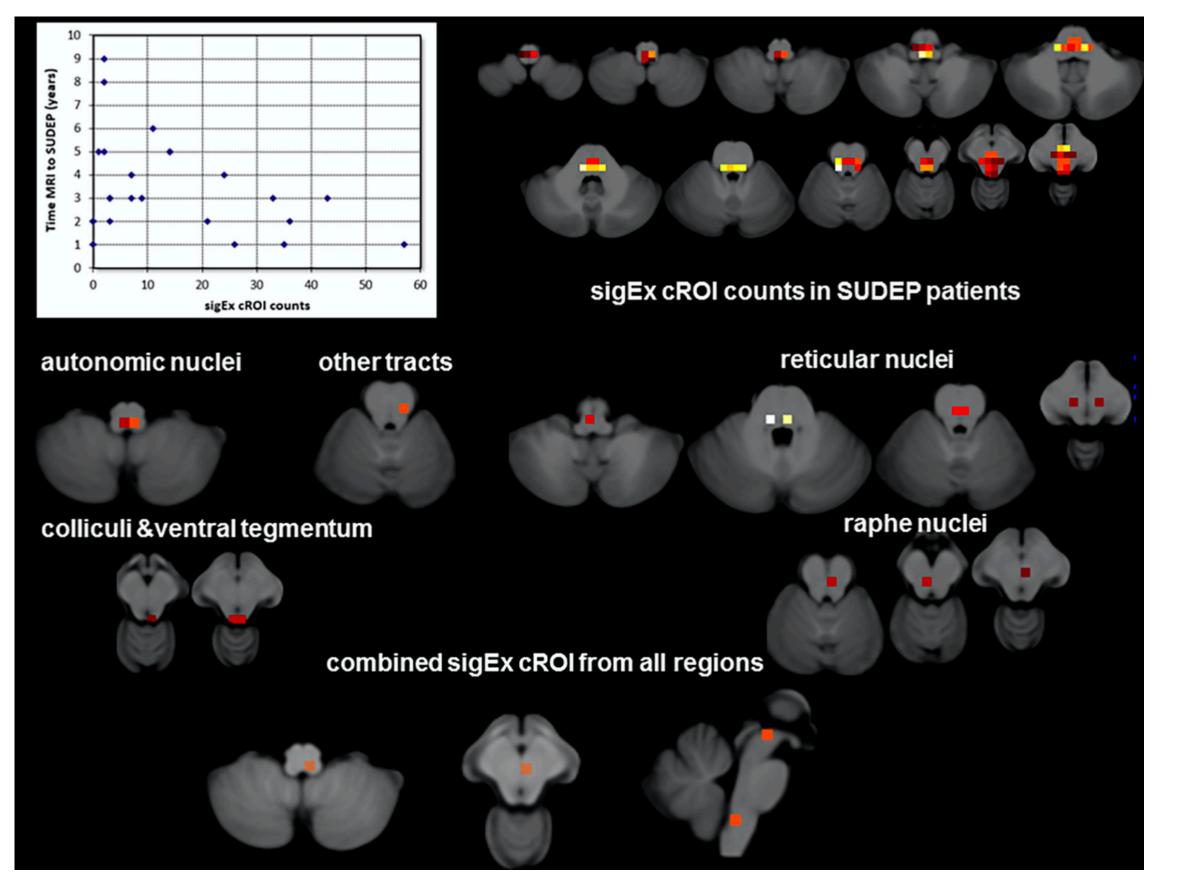


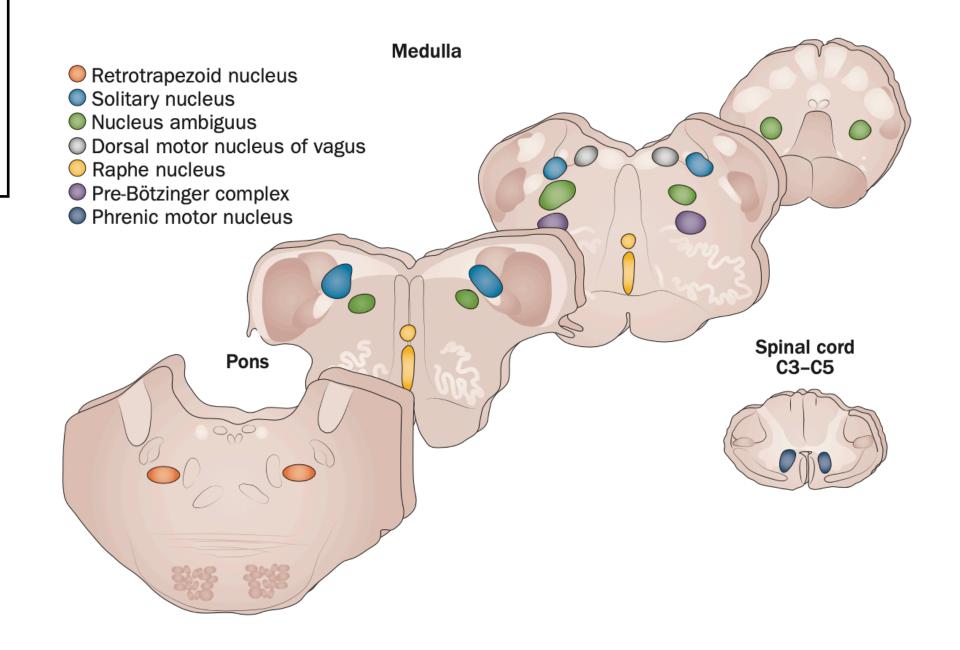
Brainstem network disruption: A pathway to sudden unexplained death in epilepsy?

Susanne G. Mueller¹ | Maromi Nei² | Lisa M. Bateman³ | Robert Knowlton⁴ | Kenneth D. Laxer⁵ | Daniel Friedman⁶ | Orrin Devinsky⁶ | Alica M. Goldman⁷

Hum Brain Mapp 2018: 39:

Two population: 18 focal epilepsy vs 11 controls; 26 SUDEP patients





- Volume loss in brainstem regions was correlates w/ autonomic dysfunction (HRV) in epilepsy patients
- Patients who died of SUDEP had widespread brainstem volume loss in their last MR exam
- More extensive damage correlated with shorter survival time

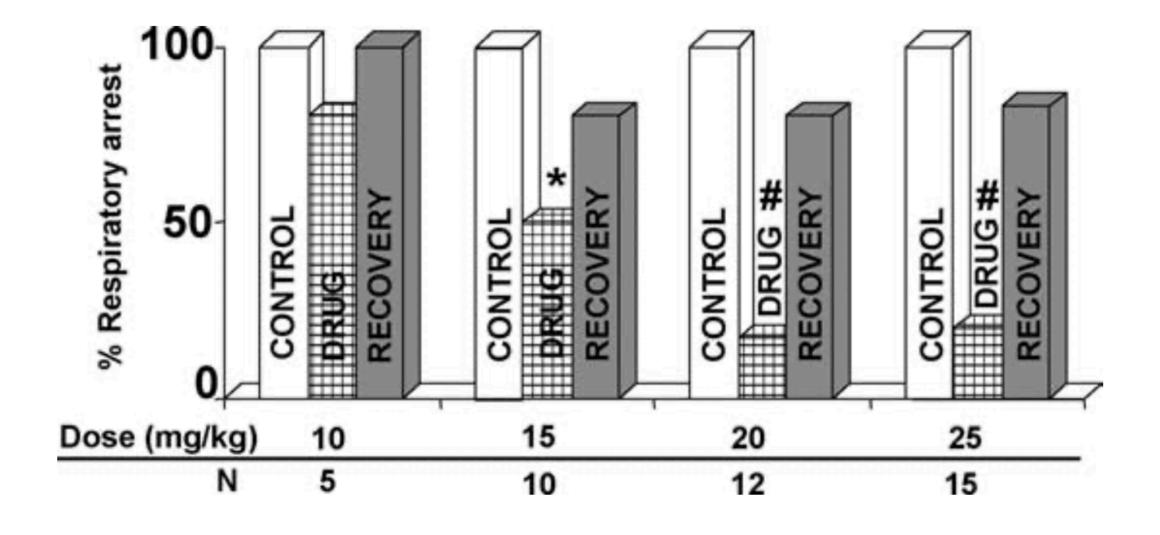
Evidence Supporting a Role of Serotonin in Modulation of Sudden Death Induced by Seizures in DBA/2 Mice

Srinivasan Tupal and Carl L. Faingold

Department of Pharmacology, Southern Illinois University School of Medicine, Springfield, Illinois, U.S.A.

Deficiency of serotonergic tone

- DBA/2 mice: respiratory arrest(RA) after audiogenic seizure(AGS)
- Fluoxetine reduced incidence of RA



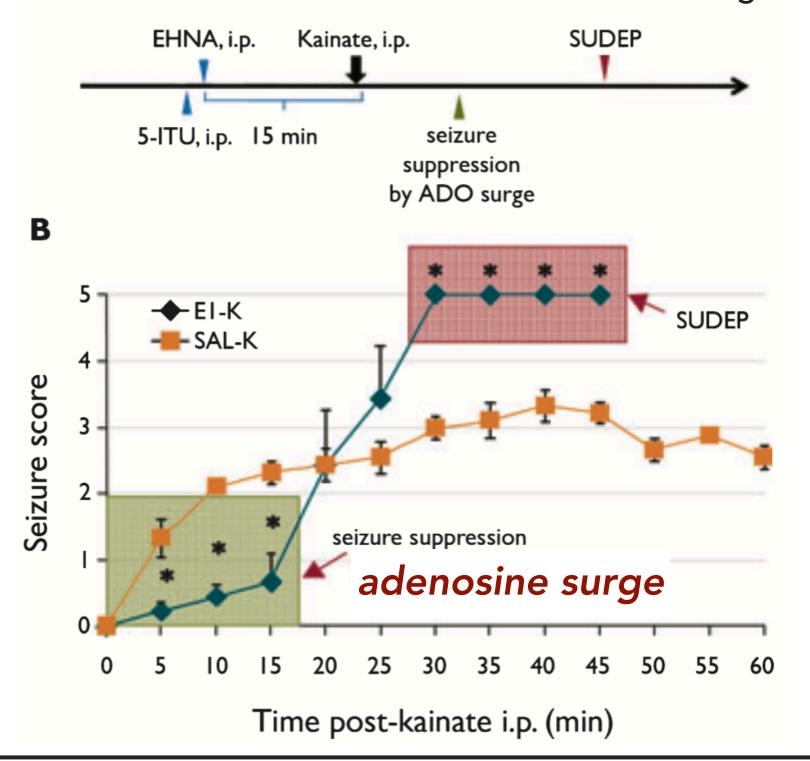
BRIEF COMMUNICATION

A novel mouse model for sudden unexpected death in epilepsy (SUDEP): Role of impaired adenosine clearance

Hai-Ying Shen, Tianfu Li, and Detlev Boison

Robert Stone Dow Neurobiology Laboratories, Legacy Research, Portland, Oregon, U.S.A.

A EHNA/ITU: inhibition of adenosine-removing enzyme



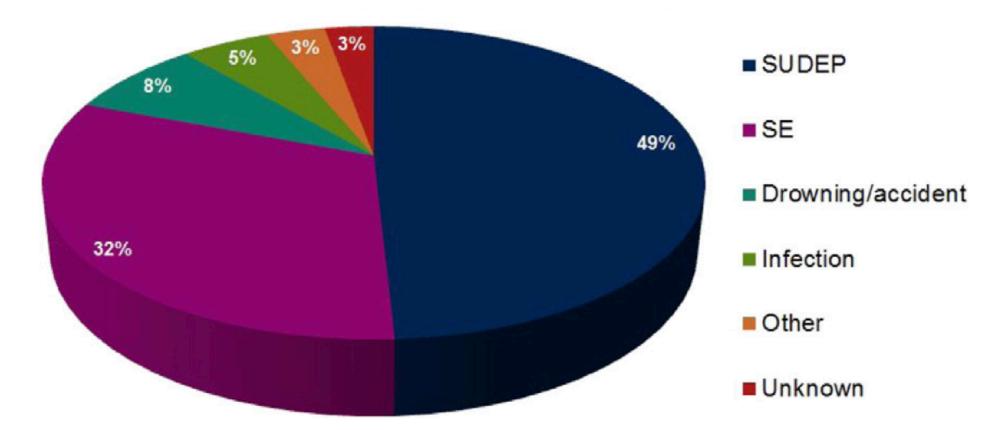
Serotonin : stimulation of breathing & arousal; defect in 5-HT system -> 1 susceptibility of SUDEP SUDEP is a consequence of an over-activation of adenosine receptor

Selected gene mutations that increase SUDEP risk

Genetic

- Genetic epilepsies with increased SUDEP risk
- Cardiac arrhythmia genes (7-15%)
- Respiratory genes

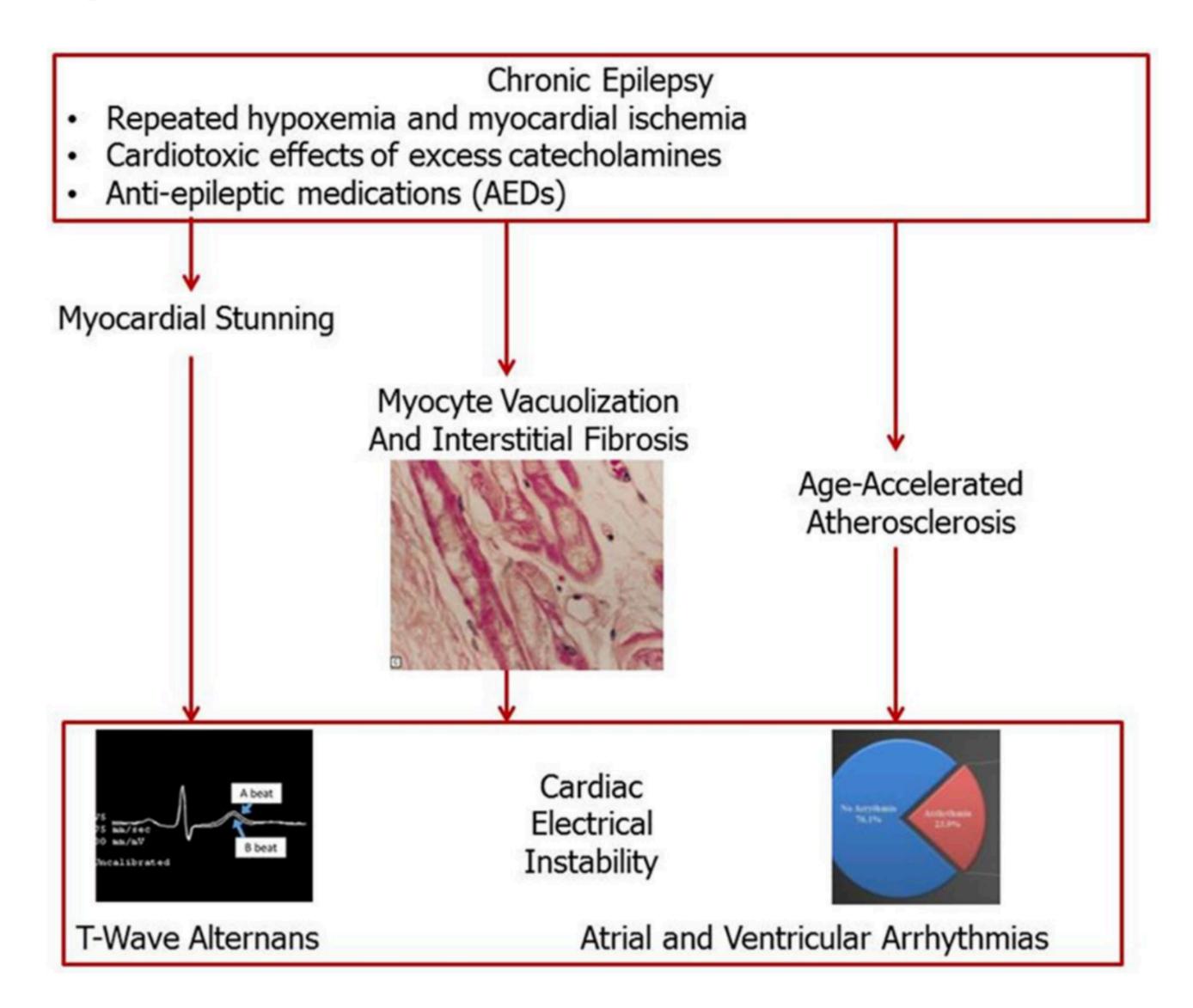
Cause of death in 177 Dravet syndrome cases



Gene	Protein	Associated human disease	Human disease manifestations	Mouse model phenotype	SUDEP	Reference
SCN1A	Na _v 1.1	Dravet syndrome	Febrile seizures in children; refractory seizures in adults; psychomotor regression; ataxia; sleep disturbance; cognitive impairment; premature death	Interictal heart rate variability; atropine-sensitive ictal bradycardia; premature death	Yes	Kalume (2013) ⁸⁰ Kalume et al. (2013) ⁸⁸ Auerbach et al. (2013) ¹²⁷
SCN5A*	Na _v 1.5	Brugada syndrome	ST-segment elevation in V1–V3 on electrocardiogram; syncope; seizure; disrupted sleep; premature death	Ventricular tachycardia; cardiac abnormalities	Possibly	Hedley et al. (2009) ¹²⁸ Martin et al. (2012) ¹²⁹ Derangeon et al. (2012) ¹³⁰
SCN5A‡	Na _v 1.5	Long QT syndrome type 3	Delayed repolarization; torsades de pointes; sudden death; palpitations; syncope; gastrointestinal symptoms	QT prolongation, ventricular tachycardia and early afterdepolarization in vitro	Possibly	Aurlien et al. (2009) ¹³¹ Johnson et al. (2009) ¹³²
KCNA1	K ₂ 1.1	NA	Episodes of ataxia with continuous inter-attack myokymia; partial epilepsy in some cases	Severe epilepsy; atrioventricular conduction block; bradycardia; premature ventricular contractions; premature death	Yes	Glasscock <i>et al.</i> (2010) ⁷⁹ Zuberi <i>et al.</i> (1999) ¹³³
KCNH2	K,11.1	Long QT syndrome type 2	Delayed repolarization of the heart; torsades de pointes; heart palpitations; syncope; sudden death; long QT events triggered by auditory stimuli	Kcnh2 ^{-/-} genotype is embryonic lethal	Yes	Anderson et al. (2014) ⁷⁴ Johnson et al. (2009) ¹³² Tu et al. (2011) ¹³⁴
KCNQ1	K ₂ 7.1	Long QT syndrome type 1	Delayed repolarization of the heart; torsades de pointes; palpitations; syncope; sudden death; hearing loss; long QT events during swimming	Impaired neuronal repolarization; seizures; dysregulated autonomic control of heart	Yes	Goldenberg & Moss (2008) ⁷¹ Goldman et al. (2009) ⁷⁷
HTR2C	5-HT _{2C}	NA	NA	Epilepsy; respiratory arrest; cardiac monitoring not completed	Yes	Tecott <i>et al.</i> (1995) ²³
RYR2	RyR2	Catecholaminergic polymorphic ventricular tachycardia	Tachycardia due to catecholamine release during exercise; dizziness; syncope; seizures; premature death	Exercise-induced ventricular arrhythmias; generalized tonic–clonic seizures; sudden cardiac death	Yes	Derangeon et al. (2012) ¹²⁹ Lehnart et al. (2008) ¹³⁵ Napolitano et al. (1993) ¹³⁶

Epileptic heart

"A heart and coronary vasculature damaged by chronic epilepsy as a result of repeated surges in catecholamines and hypoxemia leading to electrical and mechanical dysfunction, and of AEs of certain ASMs which may predispose to hyperlipidemia or arrhythmias"



Whether PGES is an independent marker of SUDEP is unknown

Yes:

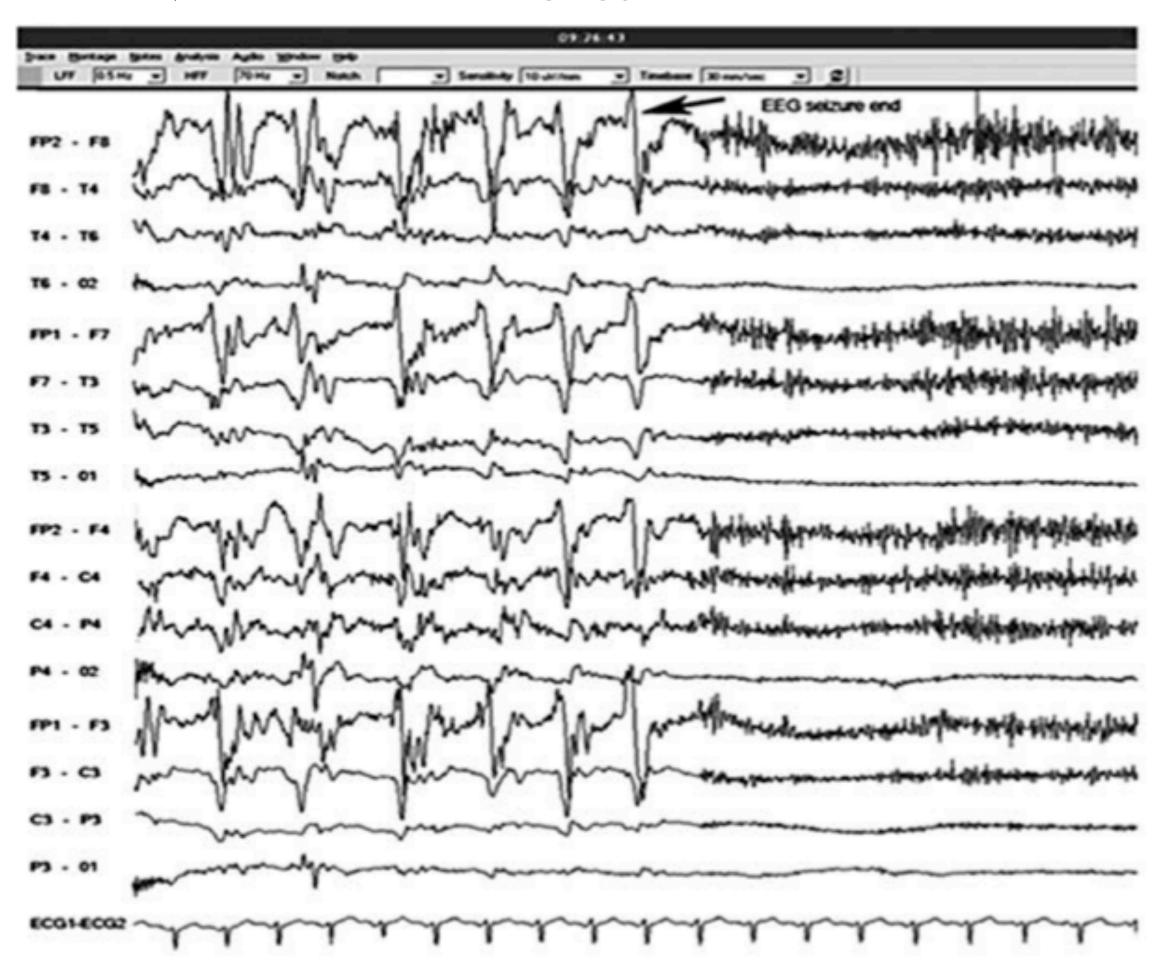
- All SUDEP cases in MORTEMUS were preceded by PGES
- 10 SUDEP cases and 30 controls (US): PGES >50 sec associated with increased risk (OR 5.22)

O No:

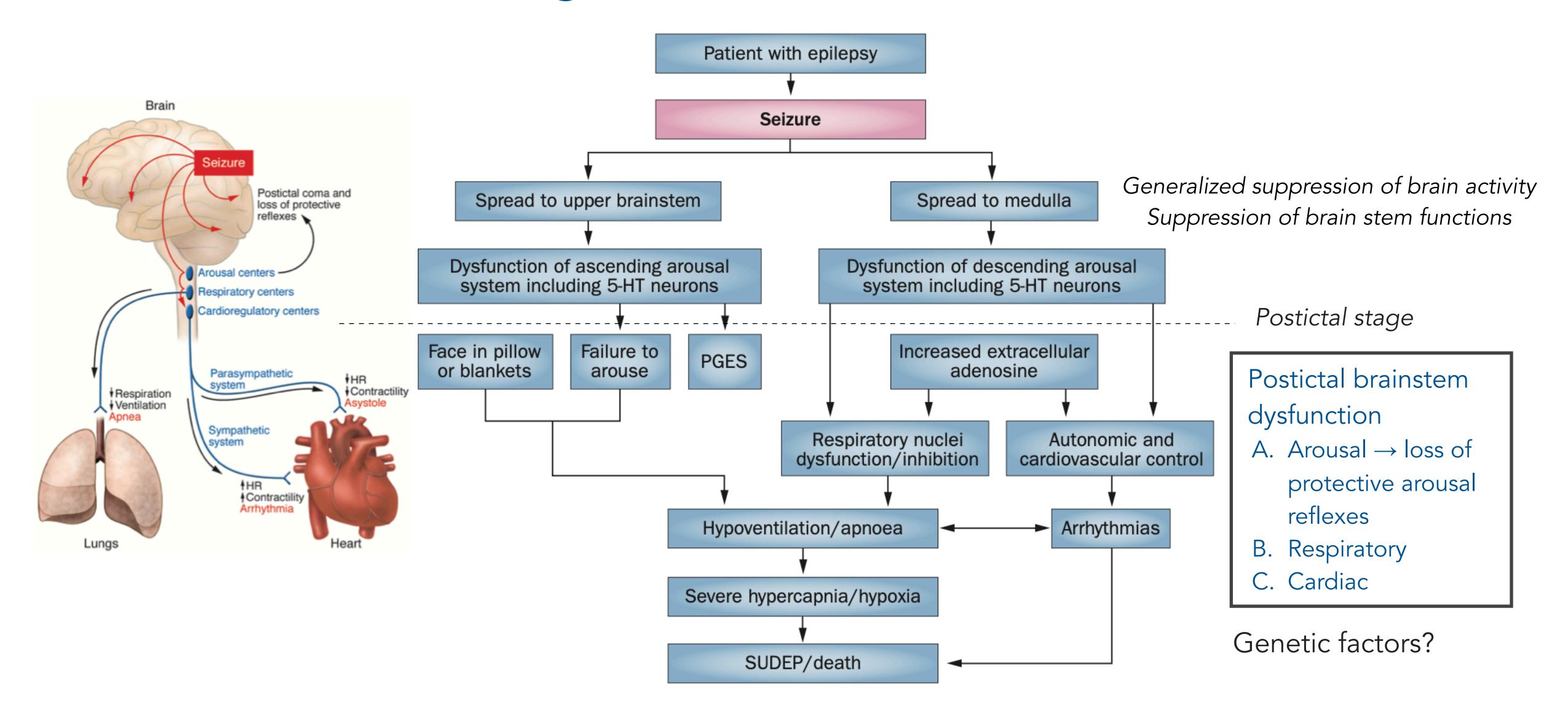
- 17 SUDEP cases and 19 controls (UK):
 PGES presence or duration did not differ between both groups
- Prior studies examine PGES and SUDEP risk have been small and single center

Post-ictal generalized EEG suppression (PGES)

- <10 mV, >1 sec, within 30 sec of seizure cessation
- 40-60% of those who had GTCS



No single mechanism established



SUDEP risk stratification

10 points

Risk factor	SUDEP-7	Revised SUDE	P-7
GCTS in the past 12 months	$0 \text{ seizures} \rightarrow 0 \text{ points}$	$0 \text{ seizures} \rightarrow 0$	points
	1-3 seizures → 1 point	1-3 seizures →	1 point
	\geq 4 seizures \rightarrow 3 points	≥4 seizures → 2	2 points
Any seizure, frequency per month in the past 12 months	$0 \text{ seizures} \rightarrow 0 \text{ points}$	$0 \text{ seizures} \rightarrow 0$	points
	1-49 seizures → 1 point	1-49 seizures →	1 point
	>50 seizures → 3 points	>50 seizures →	2 points
Epilepsy duration	$0-29 \text{ y} \rightarrow 0 \text{ points}$		
	\geq 30 y \rightarrow 3 points		
Number of AEDs	$0-2 \text{ drugs} \rightarrow 0 \text{ points}$		
	\geq 3 drugs \rightarrow 1 point		Risk
Cognitive impairment	$IQ \ge 70 \rightarrow 0$ points		Gene
	$IQ < 70 \rightarrow 2 \text{ points}$		_1

12 points

SUDEP risk factors	SUDEP-3 weighting	OR (95% CI)
GTC seizure frequency >3 in last year	0 or 1	2.7 (0.9-7.7)
Seizure of any type >0 in last year	0 or 2	8.4 (1.0-71.1)
Intellectual disability	0 or 1	3.1 (0.7-13.4)
Total SUDEP-3 score	0 to 4	

Total score

SUDEP inventory SUDEP-7 → revised SUDEP-7 → SUDEP-3 2010 2015 2021

SUDEP-CARE 2022: drug-resistant focal epilepsy

Risk factors	Point(s) if yes			
Generalized tonic-clonic seizure frequency in the last year				
<1 per year	0			
Between 1 per month and 1 per year	1			
>1 per month	2			
Presence of intellectual disability	1			
Current or past depressive disorder	1			
Respiratory symptoms during or after seizure	1			
Sleep-related or nocturnal seizures	2			
Able to alert someone of an oncoming seizure	-1			
Seizure-related falls	1			
	Total score (sum)			

Can we prevent SUDEP?



Cochrane Database of Systematic Reviews

Treatments for the prevention of Sudden Unexpected Death in Epilepsy (SUDEP) (Review)

Maguire MJ, Jackson CF, Marson AG, Nevitt SJ

We included one cohort study and three case-control studies of serious to critical risk of bias. The 6-month prospective cohort study observed no significant effect of providing patients with SUDEP information on drug compliance and quality of life, anxiety and depression levels. The study was too short and with no deaths observed in either group to determine a protective effect. Two case control studies reported a protective effect for nocturnal supervision against SUDEP. However due to significant heterogeneity, the results could not be combined in meta-analysis. One study of 154 SUDEP cases and 616 controls reported an unadjusted odds ratio (OR) of 0.34 (95% CI 0.22 to 0.53; P < 0.0001). The same study demonstrated the protective effect was independent of seizure control, suggesting that nocturnal supervision is not just a surrogate marker of seizure control. The second case-control study of 48 SUDEP cases and 220 controls reported

Authors' conclusions

adjusted OR 0.4 (0.2-0.8)

We found limited, very low-certainty evidence that supervision at night reduces the incidence of SUDEP. Further research is required to identify the effectiveness of other current interventions — for example seizure detection devices, safety pillows, SSRIs, early surgical evaluation, educational programmes, and opiate and adenosine antagonists — in preventing SUDEP in people with epilepsy.

Approaches to SUDEP prevention

1. Optimizing epilepsy care: target GTCS and FBTCS

- Optimize medical therapy: efficacious adjective ASM, 0.9 vs 6.9 per 1000 person-years
- Timely referral for non-medical therapy:
 - 2 large studies have shown lower SUDEP and all-cause mortality in epilepsy surgery groups, 10.4 vs 5.2 and 4.6 vs 1.9 per 1000 person-years
 - VNS, RNS studies suggest SUDEP rates lower in treated patients
- 2. Early cardiopulmonary resuscitation, recommend first-aid course to relative
- 3. Nocturnal supervision, seizure detection devices, sharing household

Does talking about SUDEP reduce risk?

→ May influence adherence, lifestyle choices, treatment decision

Effect of providing sudden unexpected death in epilepsy (SUDEP) information to persons with epilepsy (PWE) and their caregivers—Experience from a tertiary care hospital

Conclusion: The present study suggests that providing information on SUDEP to PWE and their caregivers may increase drug adherence without adverse effect on quality of life or mood. Well-designed studies with high methodological quality are required to determine the precise effect size associated with disclosure of SUDEP information on drug adherence in PWE.

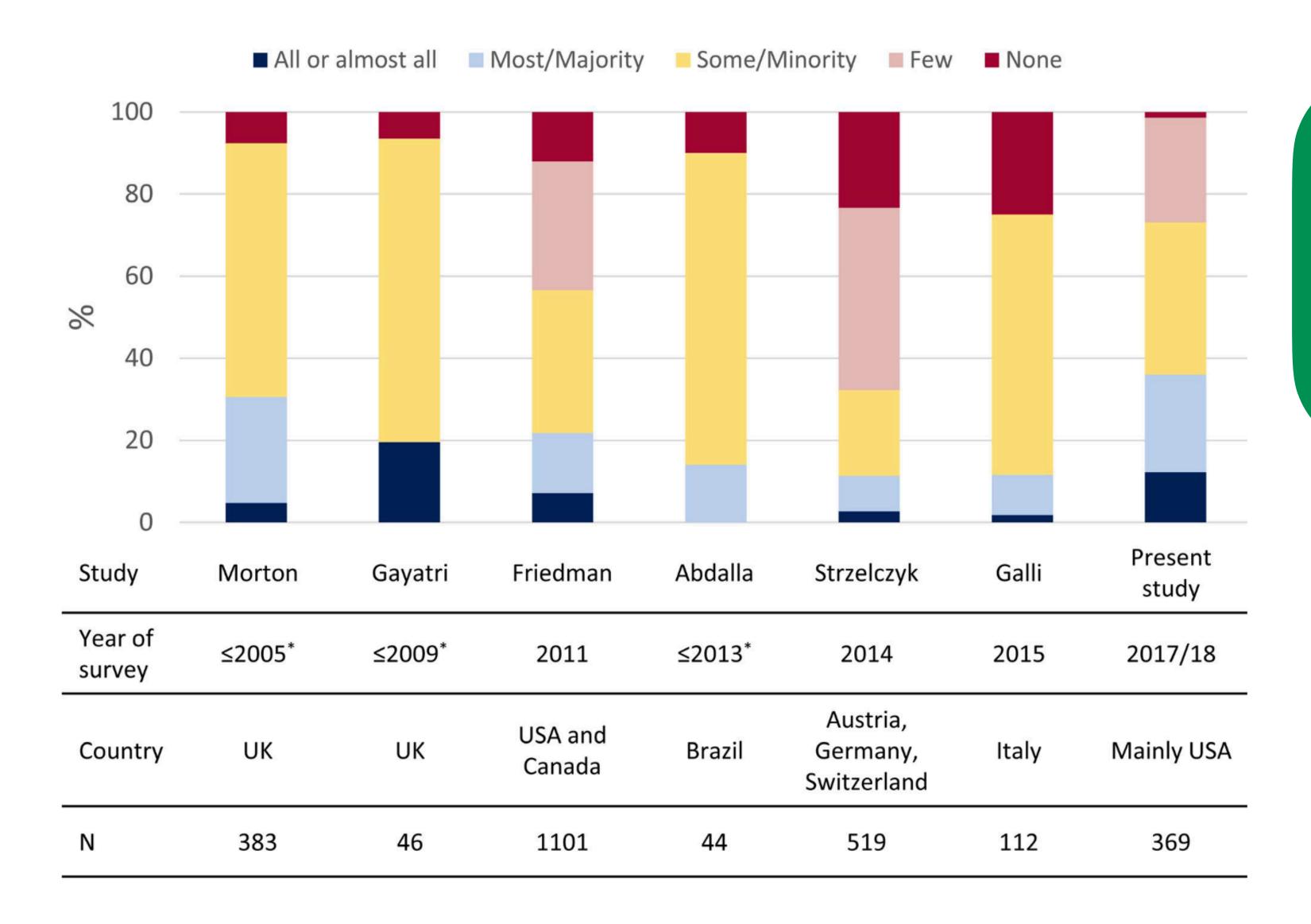
2012 Institute of Medicine Report

"To manage fears and prevent unnescessary anxiety, PWE and their families need complete & accurate information about the comorbidities and mortality risks associated with epilepsy, SUDEP"

2017 AAN/AES Recommendations

"Inform PWE and parents of children with epilepsy of the risk" "Inform PWE that seizure freedom, particularly freedom from GTCS, is strongly associated with a decreased risk of SUDEP"

How are we doing at counseling patients?



72%
of people who had lost a loved one to SUDEP wished it had been discussed with them!!!

Mode of counseling - patients/caregiver preferences

- At the time of epilepsy diagnosis or soon after
- Preferably discussed by the treating neurologist
- Face-to-face discussion



- Written information provided in addition to the verbal discussion
- Opportunities to follow up about SUDEP discussion afterwards
- Caregivers to decide if and when the child be informed about the risk of SUDEP

Talk about SUDEP

- Recognize opportunities to start conversation:
 - "Is epilepsy dangerous?", "What can happen?", "Is there something else that we should know?"
 - Seizure free: when you're discussing the importance of continued adherence: "keep up the good work...missing a dose can cause breakthrough seizure, lifethreatening seizure and may increase the risk called SUDEP"
 - "My goal in giving you this information is not to scare you, but to educate you"
- Explain the risk and modulating factors: typically affects 1 in 1000 adults per year.
 Therefore, annually 999 of 1000 adults with not be affected
- Stress that measures to be taken can decrease the risk of SUDEP

Conclusions

- SUDEP is the cause of significant premature mortality & potentially preventable
- Risk factors: TCS, nocturnal seizures, medication non-adherence, and lack of night-time supervision
- Prevent SUDEP: focus on risk reduction
 - exciting studies exploring interventions for improving post-ictal safety
 - seizure detection devices are currently available, however, their efficacy to reduce SUDEP is not confirmed
- Patients want to be told about SUDEP risk ⇒ time to talk about SUDEP!!!





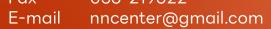
LGORITHMS: THE ART OF CLINICAL REASONING

11TH-12TH DEC 2025

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THURSDAY 11 st December 2025		
08:00 u 09:30 u.	Workshop in Neurology part 1: Unseen cases by guru	Speakers: รศ.นพ.พรชัย สถิรปัญญา มหาวิทยาลัยสงขลานครินทร์ ผศ.นพ.สุพจน์ ตุลยาเดชานนท์ โรงพยาบาลรามาธิบดี Moderator: รศ.พณ.ศิวาพร จันทร์กระจ่าง มหาวิทยาลัยเชียงใหม่
09:30 u 09:45 u.	Opening Remark	Moderator. 3M. acg. No las variorise viv amortalidated viria
09:45 u 10:30 u.	"When Guidelines Fall Short: Clinical Reasoning in Atypical Stroke Presentations"	Speakers: อ.พญ.อังคณา นัดสาสาร ศูนย์โรคสมองภาคเทนือ มทาวิทยาลัยเชียงใทม่ ผศ.นพ.กิตติ เทียนขาว มทาวิทยาลัยเชียงใทม่
10:30 u 10:45 u.	Break	
10:45 u 11:30 u.	Beyond the Algorithm: The Role of Clinical Insight in CNS Infections	Speakers: ผศ.นพ.สุพจน์ ตุลยาเดชานนท์ โรงพยาบาลรามาธิบดี อ.นพ.อาคม อารยาวิชานนท์ โรงพยาบาลสรรพสิทธิประสงค์
11:30 u 12:15 u.	Symposium 1: Early Optimization of Anti-seizure Medication (ASM) Use across All Age Groups	Speakers: ผศ.พญ.กมรวรรณ กตัญญูวงค์ มหาวิทยาลัยเชียงใหม่ รศ.นพ.อธิวัฒน์ สุนทรพันธ์ มหาวิทยาลัยเชียงใหม่
12:15 u 13:00 u.	Symposium 2: Advancing Stroke Care: Evidence and Experience with Long-Term Dual Antiplatelet Therapy	Speakers: ศ.พญ.นิจศรี ชาญณรงค์(สุวรรณเวลา) จุฬาลงกรณ์มหาวิทยาลัย ผศ.พญ.ดวงนภา รุ่งพิบูลโสภิษฐ์ มหาวิทยาลัยนเรศวร
13:00 u 13:15 u.	Break	
13:15 u 14:00 u.	"Is It MS or Not? Clinical Reasoning in Gray Zones"	Speakers: ศ.พญ.นาราพร ประยูรวิวัฒน์ คณะแพทยศาสตร์ศิริราชพยาบาล อ.นพ.เมธา อภิวัฒนากุล สถาบันประสาทวิทยา Moderator:อ.พญ.ผกามาศ พสกภักดี ศูนย์ศรีพัฒน์ มช.
14:00 u 14:45 u.	From Ear to Brain: Peripheral or Central Vertigo?	Speakers: ผศ.นพ.พินิจ ลิ้มสุดนธ์ โรงพยาบาลสมิติเวช รศ.พญ.สุวิชา แก้วศิริ มทาวิทยาลัยเชียงใหม่
14:45 u 15:00 u.	Break	
15:00 u 15:45 u.	Clinical Headache Matters: Why the Art of Thinking Still Outshines the Algorithm	Speaker: รศ.นพ.เสกข์ แทนประเสริฐสุข จุฬาลงกรณ์มหาวิทยาลัย Moderator:พศ.นพ.สุรัตน์ ตันประเวช มหาวิทยาลัยเชียงใหม่
15:45 u 16:30 u.	Highlights in Neurology	Speakers: รศ.นพ.อธิวัฒน์ สุนทรพันธ์ มทาวิทยาลัยเชียงใหม่ อ.นพ.ประกาศิต คูสุวรรณ ศูนย์ศรีพัฒน์ มช. อ.นพ.ธีรดนย์ พินิชกชกร ศูนย์โรคสมองภาคเหนือ มทาวิทยาลัยเชียงใหม่
17:00 น. เป็นต้นไป	ร่วมรับประทานอาหารเย็น (อาหารเมืองเหนือ)	
FRIDAY 12ND DECEMBER 2025		
08:00 u 09:30 u.	Workshop in Neurology part 2: Amazing cases by guru	Speakers: อ.นพ.สมศักดิ์ ลัพธิกุลธรรม โรงพยาบาลราชวิถี ศ.พญ.นาราพร ประยูรวิวัฒน์ คณะแพทยศาสตร์ศิริราชพยาบาล Moderator: รศ.พญ.ศิวาพร จันทร์กระจ่าง มหาวิทยาลัยเชียงใหม่
09:30 u 10:15 u.	Phinit Neuro-radio-pathology Conference	Speakers: รศ.นพ.พัฒน์ ก่อรัตนคุณ มหาวิทยาลัยสงขลานครินทร์ อ.นพ.ธีรดนย์ พินิชกชกร ศูนย์โรคสมองภาคเหนือ มหาวิทยาลัยเชียงใหม่ Panelists: อ.นพ.สุชาติ พุทธิเจริญรัตน์ สถาบันประสาทวิทยา ผศ.นพ.พินิจ ลิ้มสุคนธ์ โรงพยาบาลสมิติเวช ศ.นพ.กัมมันต์ พันธุมจินดา จุฬาลงกรณ์มหาวิทยาลัย อ.นพ.สมศักดิ์ ลัพธิกุลธรรม โรงพยาบาลราชวิถี
10:15 u 10:30 u.	Break	
10:30 u 11:15 u.	Siwaporn Oration Lecture	Speaker: ศ.นพ.กัมมันต์ พันธุมจินดา จุฬาลงกรณ์มหาวิทยาลัย
11:15 u 12:15 u.	Challenging Cases in Neuromuscular Diseases	Speakers: ผศ.คร.นพ.จรุงไทย เดชเทวพร โรงพยาบาลรามาธิบดี รศ.พญ.กนกวรรณ บุญญพิสิฎฐ์ คณะแพทยศาสตร์ศิริราชพยาบาล ศ.นพ.ก้องเกียรติ กูณฑ์กันทรากร มหาวิทยาลัยธรรมศาสตร์
12:15 u 13:00 u.	Symposium 3: Does Safinamide Surpass Older Agents as Add-on PD Therapy?	Speakers: รศ.ภก.ธนรัตน์ สรวลเสน่ท์ มทาวิทยาลัยมทิดล รศ.นพ.อธิวัฒน์ สุนทรพันธ์ มทาวิทยาลัยเชียงใหม่
13:00 u 13:45 u.	Symposium 4: "Optimizing Patient Care in MCI with BPSD: The Role of EGb 761 in Clinical Practice and National Guidelines"	Speakers: รศ.นพ.พงศธร พทลภาคย์ มทาวิทยาลัยขอนแก่น อ.นพ.นพดนัย ศิริมทาราช มทาวิทยาลัยเชียงใทม่
13:45 u 14:00 u.	Break	
14:00 u 14:45 u.	Symposium 5: "Integrating Anxiety and Sleep Management in Neurological Patients"	Speaker: รศ.นพ.เสกข์ แทนประเสริฐสุข จุฬาลงกรณ์มหาวิทยาลัย Moderator:อ.นพ.กิตติธัช บุญเจริญ ศูนย์ความเป็นเลิศทางการแพทย์ มช.
14:45 u 15:30 u.	Biomarkers in Alzheimer's Disease: Practical Case Studies for Clinical Application.	Speakers: ผศ.ดร.นพ.ยุทธชัย ลิขิตเจริญ จุฬาลงกรณ์มหาวิทยาลัย อ.นพ.กิตติธัช บุญเจริญ ศูนย์ความเป็นเลิศทางการแพทย์ มช.
15:30 น. เป็นต้นไป	ເฉลยคำถาม	

ติดต่อสอบถาม คุณศิริทุล ทุณาปาน

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ขอบคุณครับ



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