Arythmies sévères d'origine neuro-vasculaire

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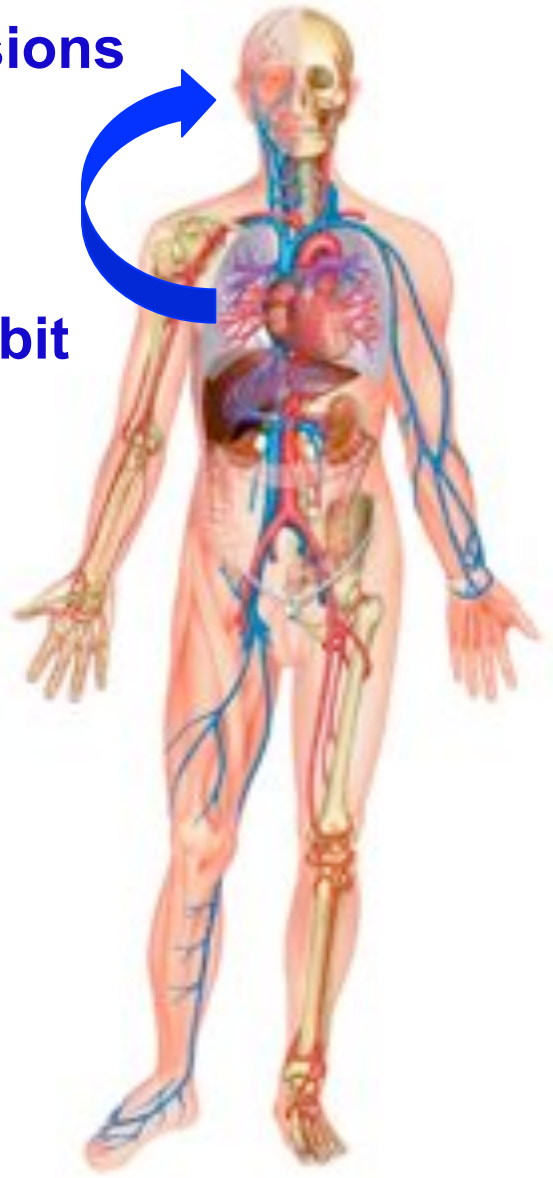
Arythmies sévères d'origine neuro-vasculaire

MERCI PATRICK !

Arythmies sévères d'origine neuro-vasculaire

Convulsions

Bas débit



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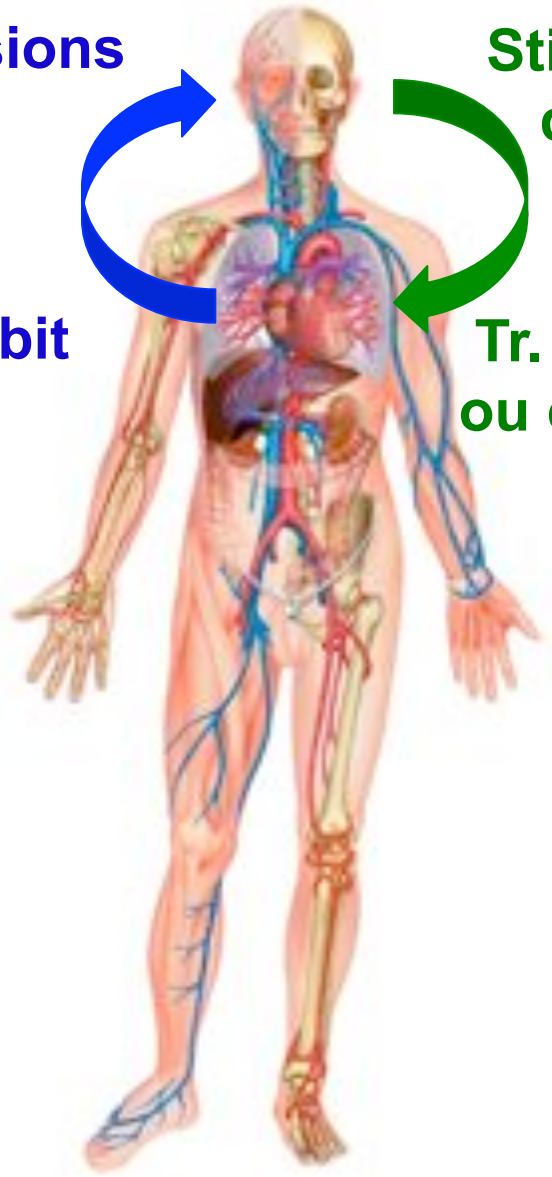
Arythmies sévères d'origine neuro-vasculaire

Convulsions

Stimulation du SNC

Bas débit

Tr. du rythme ou conduction



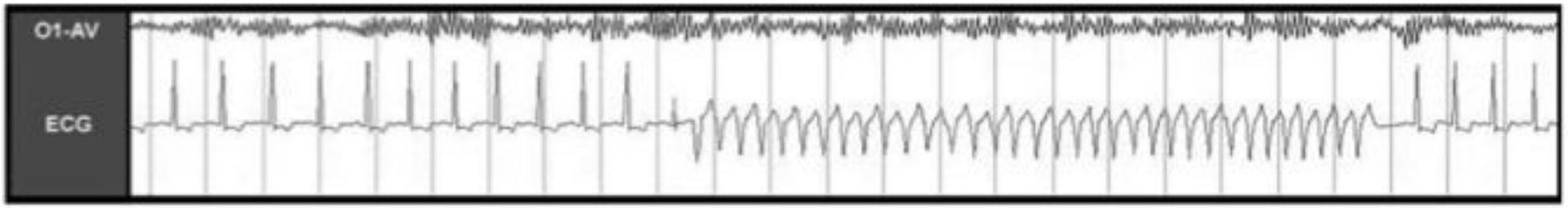
MERCI PATRICK !

CASE REPORT

Neth Heart J Published online: 05 July 2013

Loss of consciousness and convulsion induced by a ventricular tachycardia mimicking epilepsy in a patient with noncompaction cardiomyopathy: a case report

S. A. W. G. Dello · C. Kievit · P. H. Dunselman · M. Alings



Abstract Convulsions and loss of consciousness can be caused by, among other things, arrhythmias, conduction disorders or epilepsy. In clinical practice it can be difficult to distinguish between these causes of syncope, even for well-trained specialists. Patients with cardiac syncope have a substantial risk of subsequent sudden death. We present a patient with previously unknown noncompaction cardiomyopathy in whom syncope induced by ventricular tachycardia was misinterpreted as epilepsy. We present this case report in order to underline the necessity for cardiological assessment in patients with assumed mild epilepsy or syncope of unknown origin.

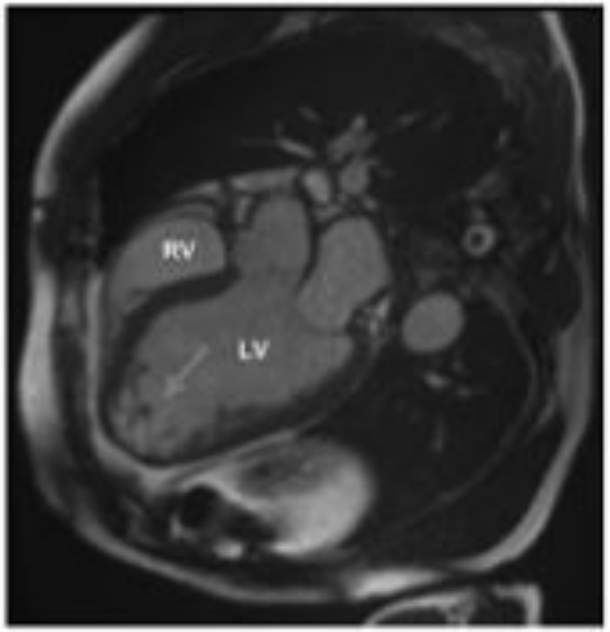


Fig. 2 MRI of the heart confirmed the diagnosis of noncompaction cardiomyopathy, excessive trabeculation (*arrow*) LV = left ventricle, RV = right ventricle

Cardiac Asystole in Epilepsy: Clinical and Neurophysiologic Features

Epilepsia, **44**(2):179–185, 2003

*R. Rocamora, *M. Kurthen, †L. Lickfett, *J. von Oertzen, and *C. E. Elger

*Departments of *Epileptology and †Medicine-Cardiology, University of Bonn, Bonn, Germany*

TABLE 3. *ECG and EEG correlates during the event*

Pat no.	ECG description	Asystole onset	Asystolic period	EEG during asystolic period	EEG normalization period
1	Sinus bradycardia followed by sinus arrest	100 s after seizure onset	60 s	Extremely flat (no activity more than 10 μV)	Longer than 2 h
2	Sinus bradycardia followed by sinus arrest	20 s after seizure onset	18 and 28 s	High-amplitude delta waves with focal ictal activity	30 s
3	Sinus bradycardia followed by sinus arrest	25 s after seizure onset	7 s	High-amplitude delta waves with focal ictal activity	35 s
4	Atrial fibrillation followed by asystole	60 s after seizure onset	7 s	Technical artifact (during seizures series)	Technical artifact
5	Sinus bradycardia followed by sinus arrest	5–14 s after seizure onset	5–9 s	Bilateral theta slow waves	2–5 s

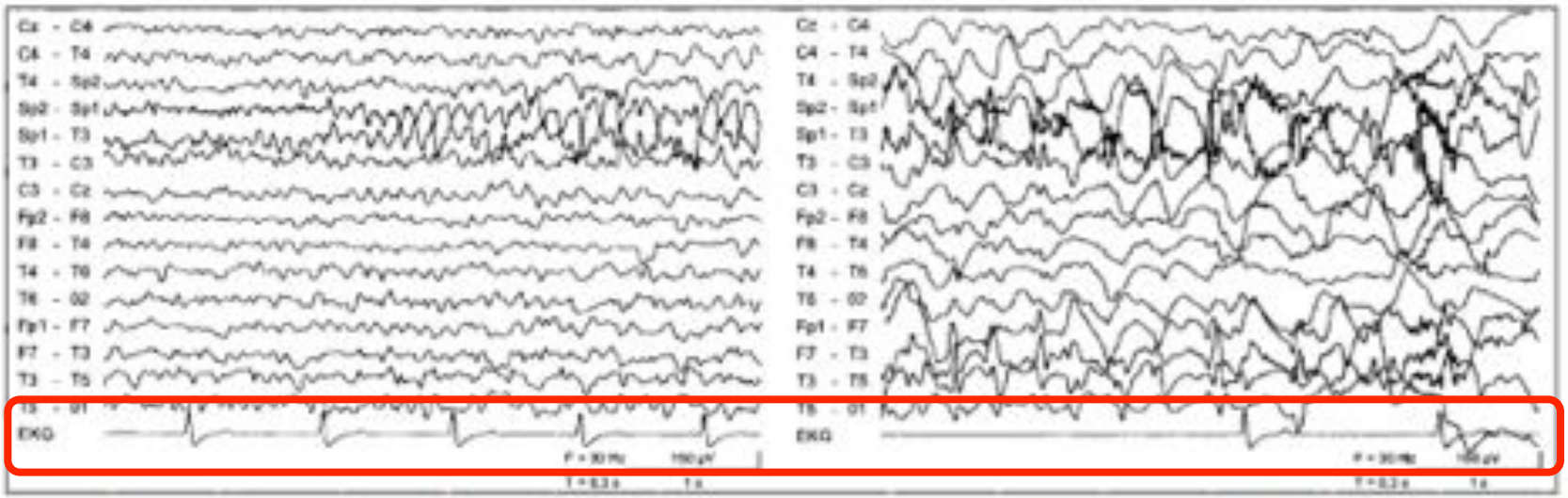


FIG. 3. EEG evolution in patient 3. Left: seizure onset; right: 7-s asystole 25 s after seizure onset. Combined generalized EEG slowing and focal ictal activity.

Out-of-hospital cardiac arrests of non-cardiac origin

Epidemiology and outcome

European Heart Journal (1997) **18**, 1122–1128

M. Kuisma and A. Alaspää *Helsinki City Emergency Medical Services, Agricolankatu, Helsinki, Finland*

- Cohorte prospective à Helsinki
  - population = 525 000 hbts
  - Système EMS + médecins

-> 809 AC réanimés en 24 mois

-> 276 AC d'origine non-cardiaque

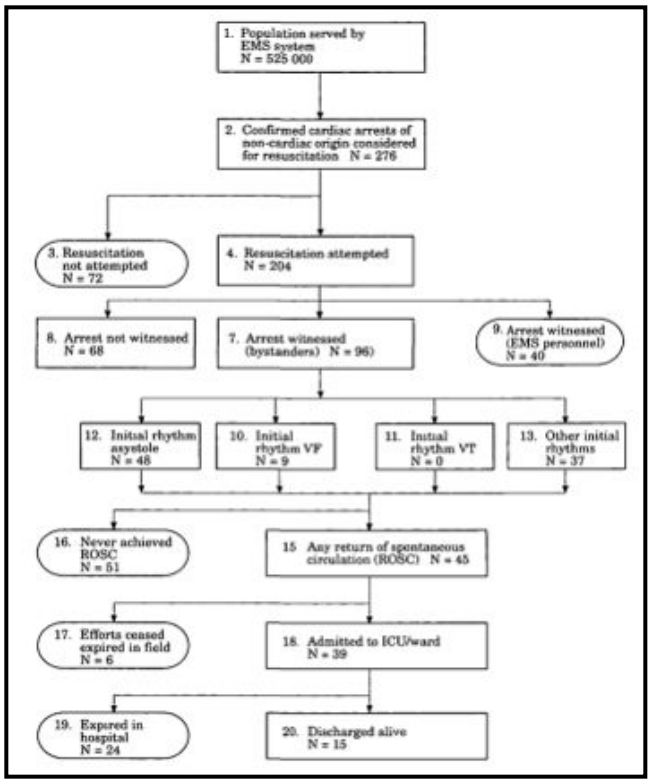


***Table 3** Detailed aetiology of 276 out-of-hospital cardiac arrests of non-cardiac origin. Cases in which the specific non-cardiac aetiology was only revealed after in-hospital investigations or autopsy are presented in the right column*

Aetiology	Total n (%)	Remained unrecognized prehospitally n
Trauma	62 (22·5)	4
Non-traumatic bleeding*	36 (13·0)	21
Intoxication	31 (11·2)	17
Near-drowning	22 (7·8)	3
Pulmonary embolism	18 (6·5)	11
Malignancy	16 (5·8)	5
Intracranial processes	14 (5·1)	7
Choking	14 (5·1)	4
Pneumonia	12 (4·4)	10
Hanging	11 (4·0)	0
Asthma	8 (3·0)	2
Convulsions	5 (1·8)	4
SIDS	5 (1·8)	2
Carbon monoxide intoxication	5 (1·8)	0
Haemorrhagic		4
Sepsis		
Other		
SIDS = su		
Aortic rupture		

Origine neurologique ~ 20%

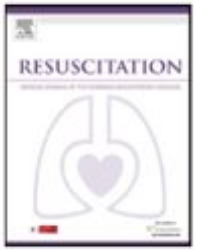
Clinical paper

Causes of in-hospital cardiac arrest and influence on outcome☆

Christian Wallmuller, Giora Meron, Istepan Kurkciyan*, Andreas Schober, Peter Stratil, Fritz Sterz

Department of Emergency Medicine, General Hospital of Vienna, Medical University of Vienna, Austria

Resuscitation 83 (2012) 1206–1211



- Etude rétrospective à Vienne
  - juil. 1991 -> déc. 2001
  - AC réanimés dans l'hôpital
    - aux urgences
    - dans les services d'hospitalisation

-> 1041 AC-IH réanimés en 17,5 ans

-> 37% origine non-cardiaque

- **hémorr. intracérébrale (32%)**
- **hémorr. méningée (23%)**
- **épilepsie (18%)**
- **encéphalite**
- **angiome**
- **AVC ischémique**
- **myasthénie gravidique**
- **méningite fungique**

Table 1
Definitions for the aetiology of cardiac arrest.

An arrest is presumed to be of following aetiology if it is cause can be primary related to:

Cardiac
Heart disease such as coronary heart disease, arrhythmic mechanism or other types of structural heart disease (e.g., congenital coronary artery anomalies, myocarditis, hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy), hypertension

Pulmonary
Chronic obstructive pulmonary disease (COPD) characterized by airflow limitation that is not fully reversible, a chronic inflammatory disorder of the airways, pulmonary embolism, central airway obstruction

Aortic dissection/rupture
Tear in the aortic intima with or without blood passing into the aortic media

Intoxication and adverse drug reactions
Accidental and intentional poisonings or iatrogenic or accidental drug overdose, adverse reactions to medications

Exsanguination
Bleeding (gastrointestinal, iatrogenic or spontaneously vascular, excl. spontaneous aortic rupture, etc.)

Metabolic
Electrolyte disorders (hypo-/hyperkalaemia), hypoglycaemia, hyperglycaemia, lactic acidosis, hepatic disorders, uraemia, etc.

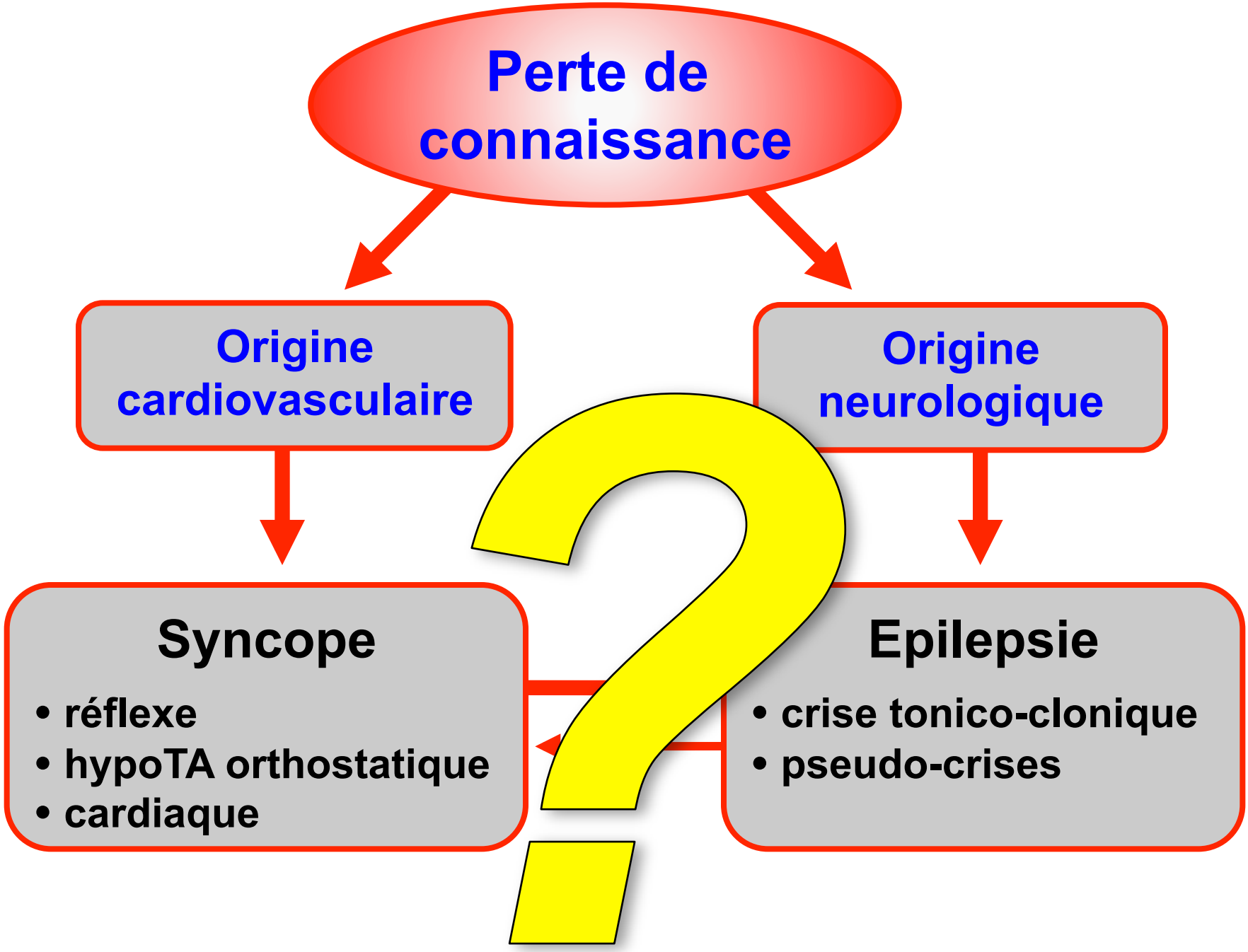
Cerebral
Intracranial/subarachnoid haemorrhage, cerebral vascular occlusion/thrombosis, contusion, tumour, meningitis/encephalitis, seizures/status epilepticus, heat stroke, etc.

Sepsis
The clinical syndrome that results from a dysregulated inflammatory response to an infection with signs of hypoperfusion or organ dysfunction

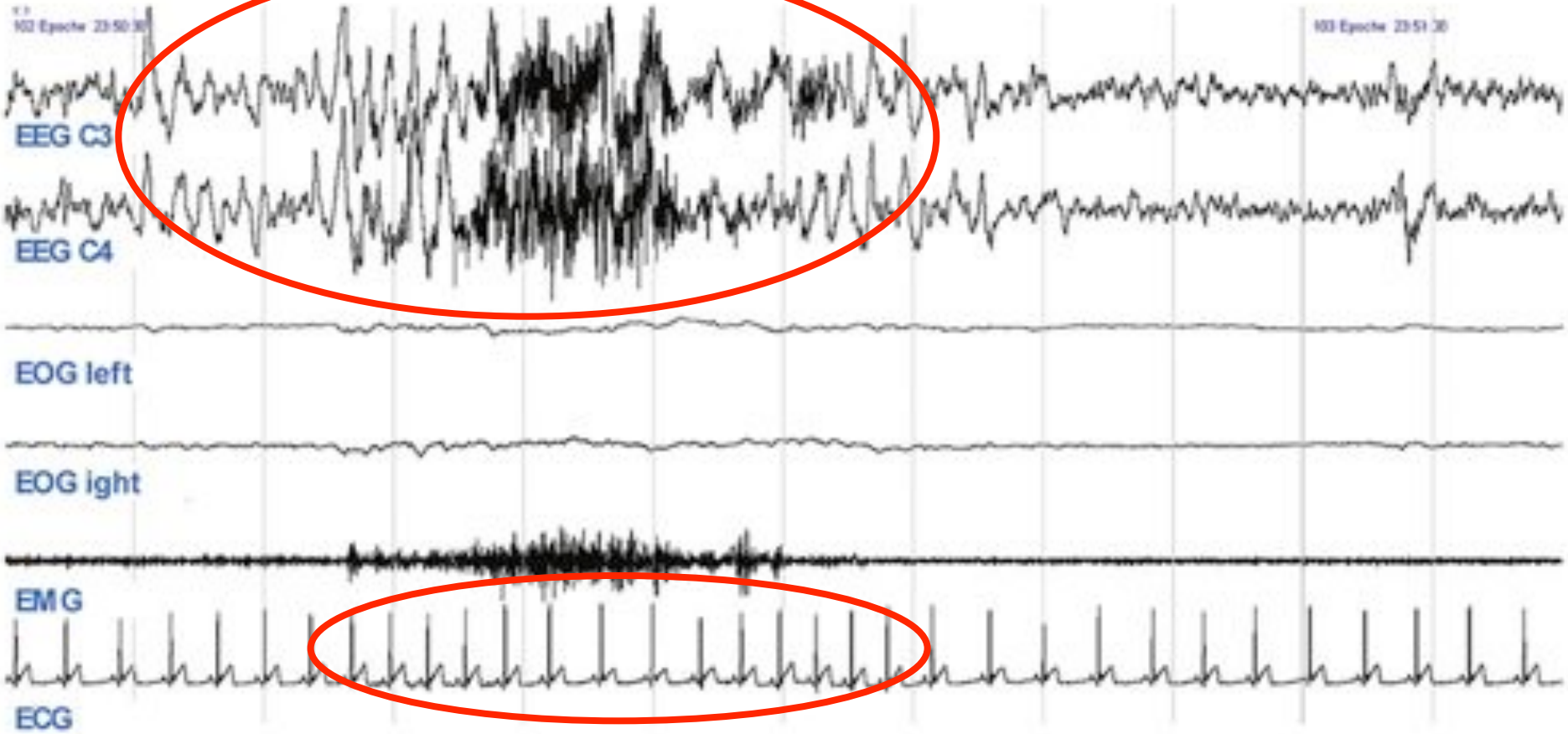
Accidental hypothermia
Defined as a core temperature [illegible] caused by evaporation, radiation, [illegible] altered cell [illegible]

(anaphy[illegible] as trauma

Origine neurologique = 22%

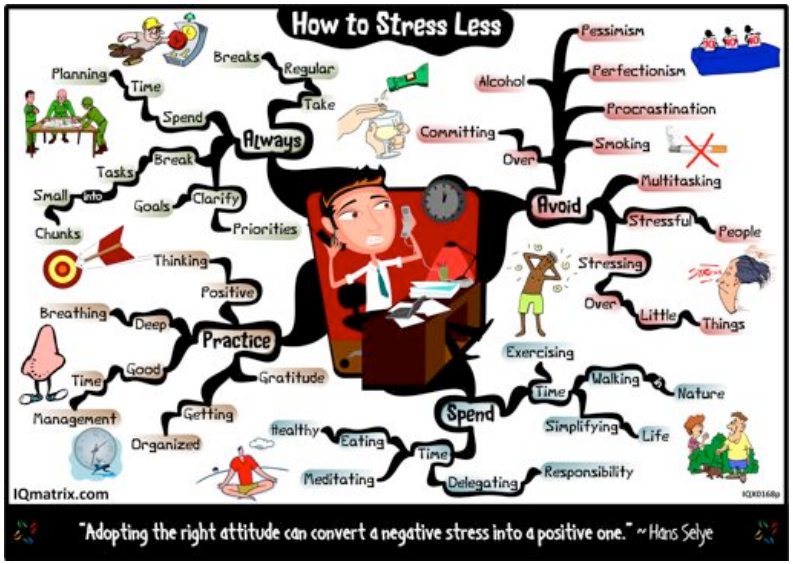

Perte de connaissance
Origine cardiovasculaire
Origine neurologique
Syncope
• réflexe
• hypoTA orthostatique
• cardiaque
Epilepsie
• crise tonico-clonique
• pseudo-crises

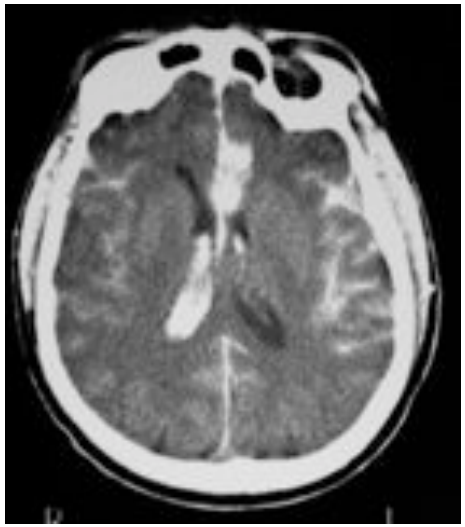
Enregistrement simultané de l'ECG et de l'EEG



Conséquences rythmiques des pathologies neurovasculaires

- Stress et émotions
- Migraine
- Epilepsie
- Hémorragies cérébrales et infarctus cérébraux
- Mort encéphalique





Stress et émotions

STANDSTILL OF THE HEART OF VAGAL ORIGIN

ALFRED M. WEDD, M.D.
CLIFTON SPRINGS, N. Y.
AND
DAVID C. WILSON, M.D.
CHARLOTTESVILLE, VA.

Am Heart J 1930 ;
5 (4) : 493-503

- H 23 ans
  - bilan de syncopes lors d'épisodes de stress

-> pauses sinusales par hyperactivité vagale
prévention par injection d'atropine

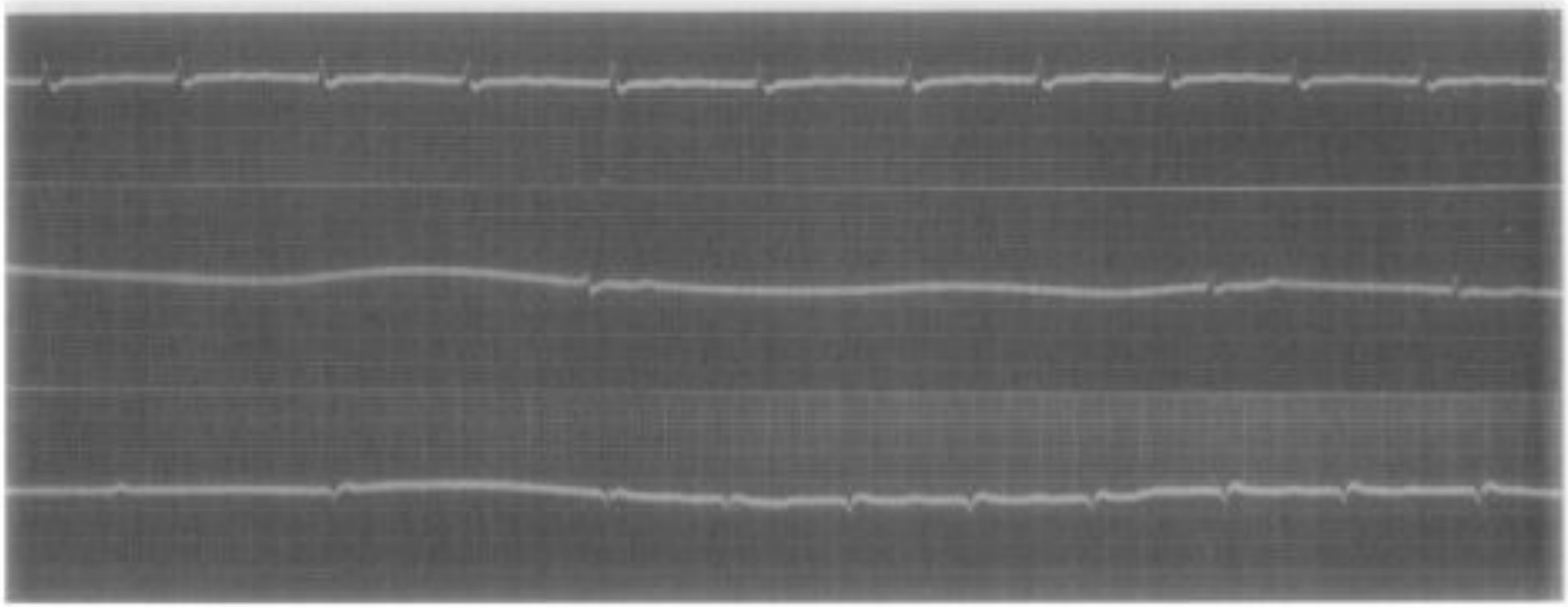
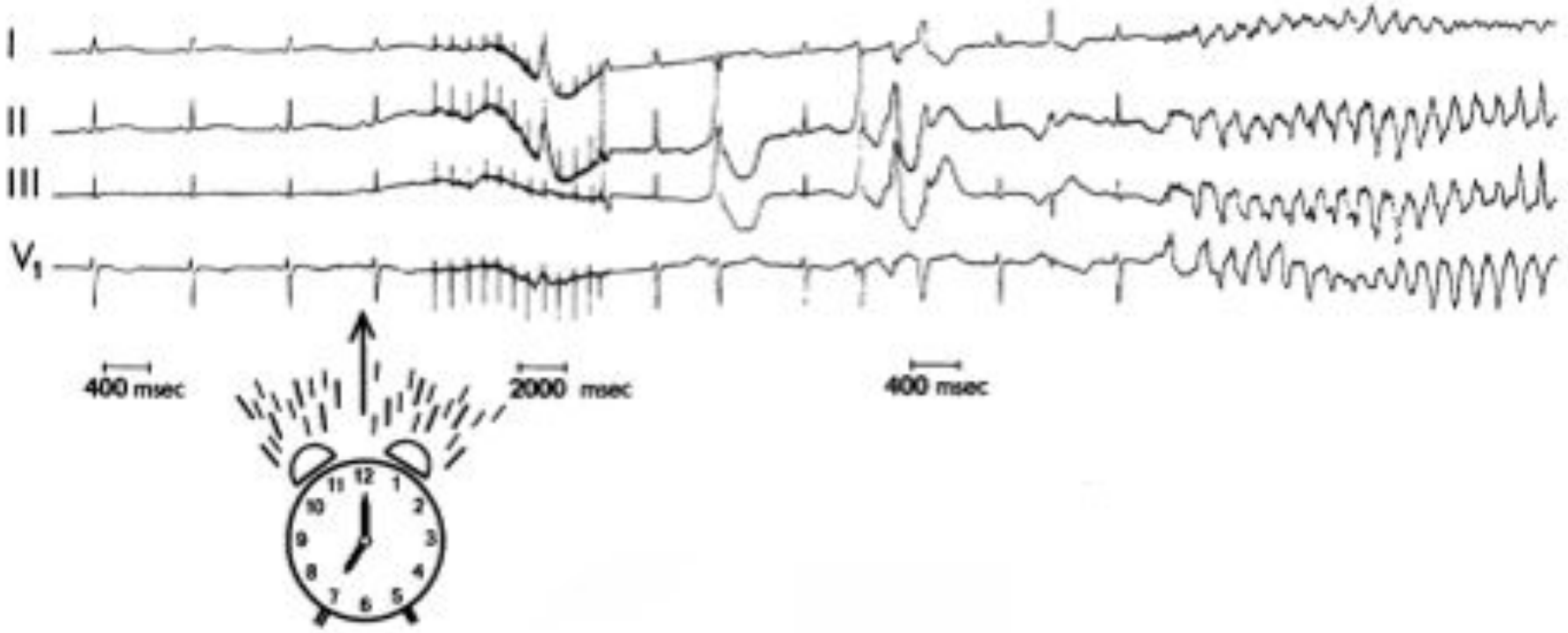


Fig. 1.—Electrocardiogram of June 22, 1928. Lead I, rate 74. Lead III, rate after pause, 83 per minute.

Ventricular Fibrillation Occurring on Arousal from Sleep by Auditory Stimuli
HEIN J. J. WELLENS, AART VERMEULEN and DIRK DURRER
Circulation. 1972;46:661-665

- F 14 ans
  - bilan de PC après coups de tonnerre ou sonnerie du réveil

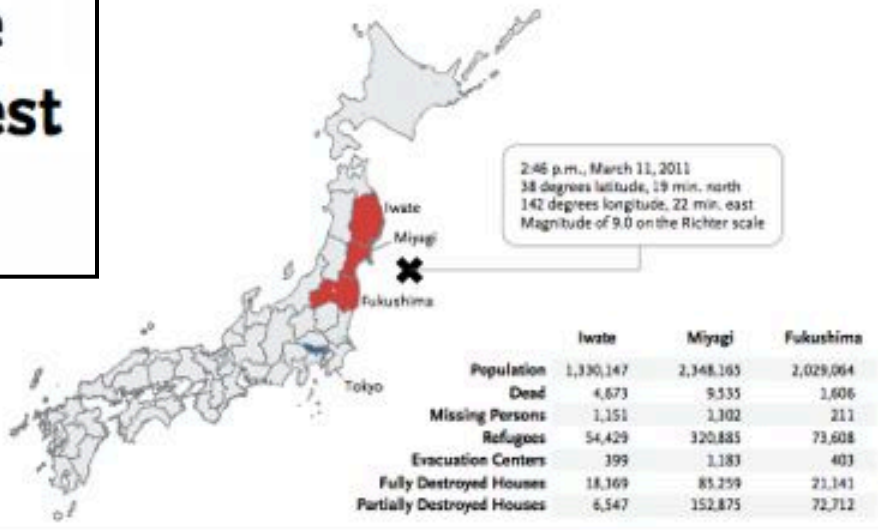
-> allongement de QT, inversion de T, ESV
puis FV (durée max. 141 sec.) de résolution spontanée



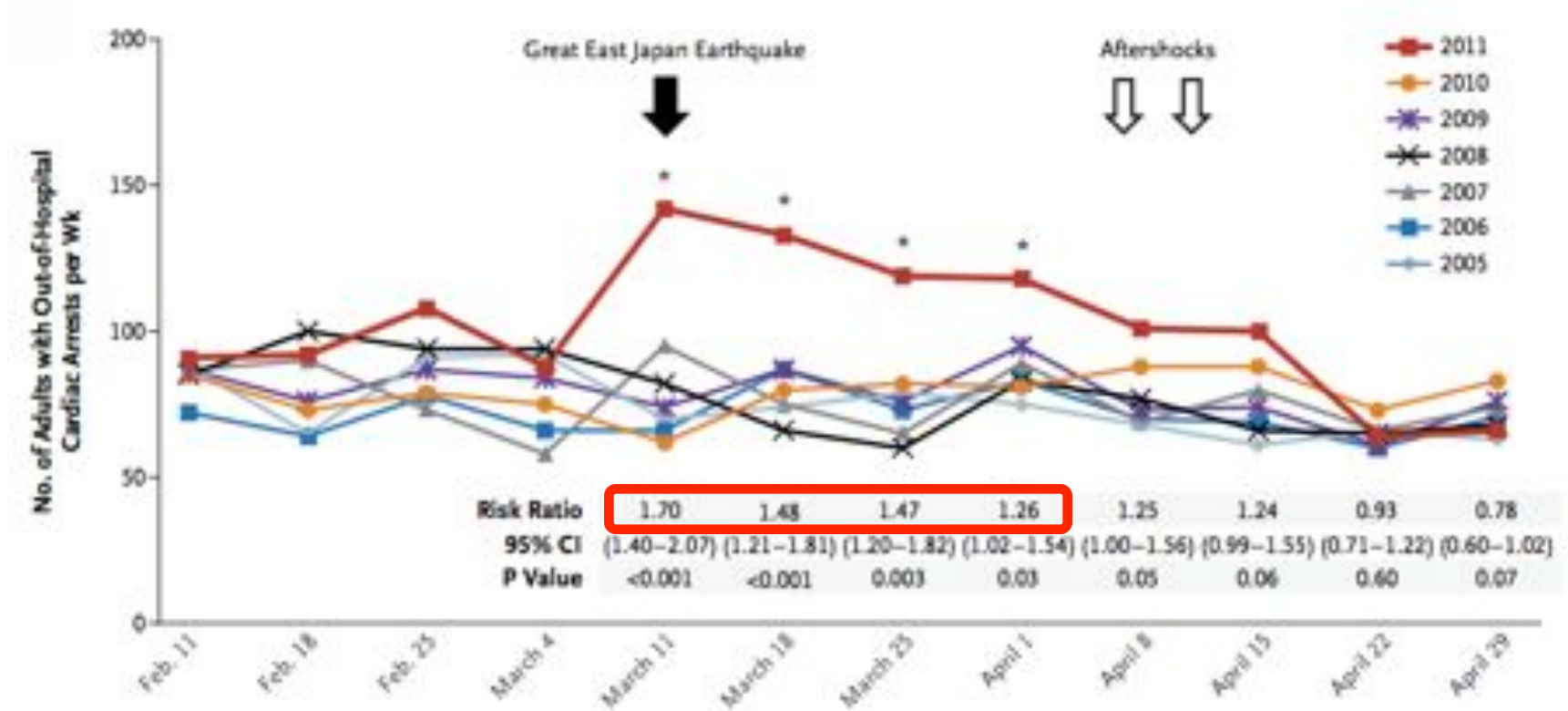
The Great East Japan Earthquake and Out-of-Hospital Cardiac Arrest

N ENGL J MED 369;22 NOVEMBER 28, 2013

Tetsuhisa Kitamura, M.D.

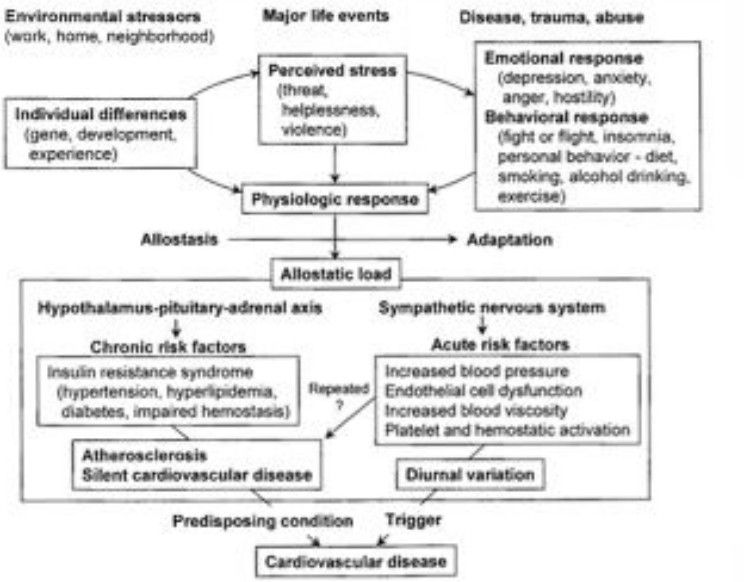


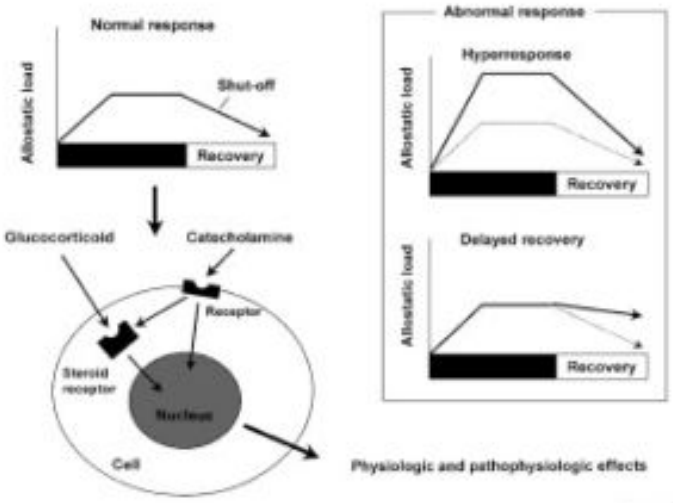
- AC d'origine cardiaque
  - chronologie des AC sur 6 ans / date du tremblement de terre

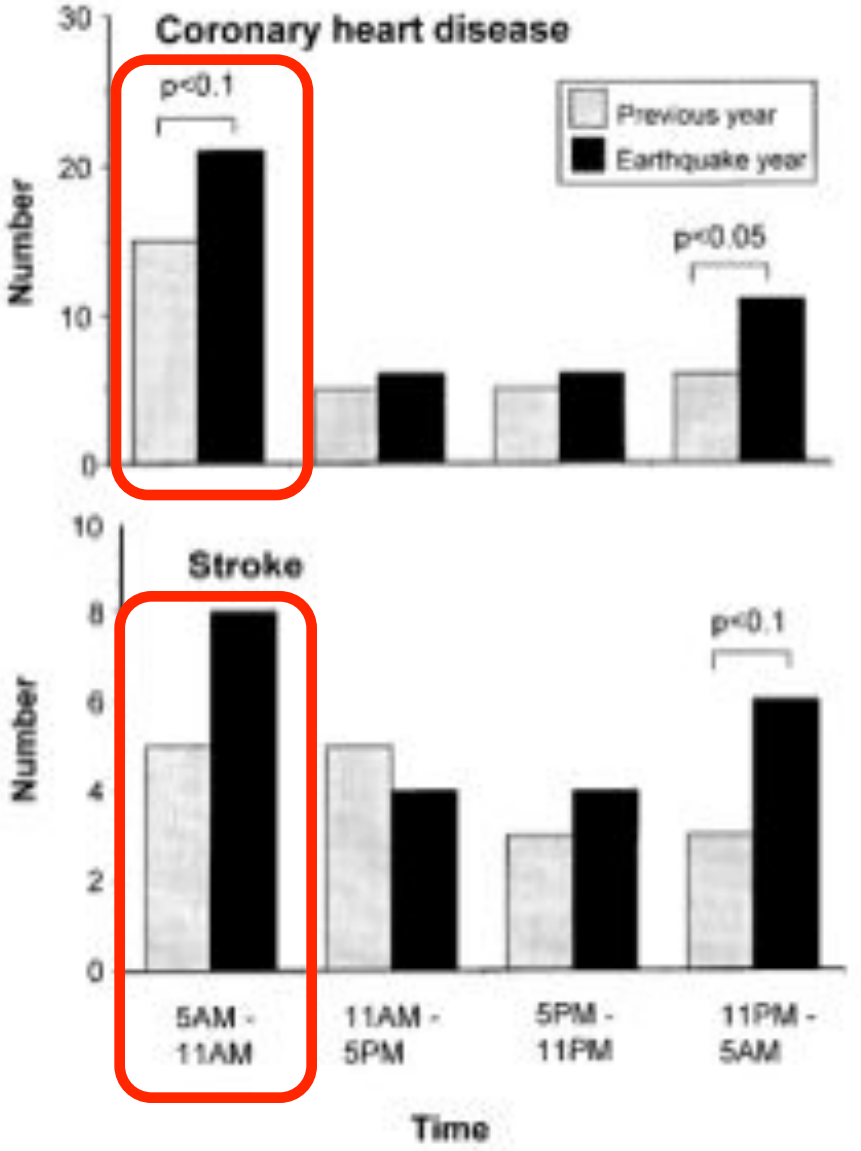


Disasters and the Heart: a Review of the Effects of Earthquake-Induced Stress on Cardiovascular Disease

Kazuomi KARIO*,**, Bruce S. McEWEN***, and Thomas G. PICKERING** *Hypertens Res* 2003; 26: 355–367







THE LANCET

Volume 302, Issue 7825, 18 August 1973, Pages 341–346

Originally published as Volume 2, Issue 7825

ELECTROCARDIOGRAM, PLASMA CATECHOLAMINES AND LIPIDS, AND THEIR MODIFICATION BY OXPRENOLOL WHEN SPEAKING BEFORE AN AUDIENCE

Peter Taggart, Malcolm Carruthers, Walter Somerville

- 30 médecins soumis à un « stress extrême » = conférence en public
  - 23 sans Atcd :
    - -> ESV fréquentes = 6 / 23
    - -> ESV multifocales = 2 / 23
    - -> ↑ taux noradrénaline = 21 / 23
    - - abolition par oxyprenolol
  - 7 avec Atcd coronariens :
    - -> ESV fréquentes = 5 / 7

Migraine

Basilar artery migraine with transient atrial fibrillation

J PETERSEN, MRCP, lecturer, Department of Therapeutics and Clinical Pharmacology
D SCRUTON, MRCP, registrar
A W DOWNIE, FRCP, senior lecturer and honorary consultant neurologist

BRITISH MEDICAL JOURNAL
29 OCTOBER 1977

- F 46 ans
  - épisodes de migraine avec troubles de conscience

Basilar artery migraine with impairment of consciousness is well recognised.[1] We wish to report a case of recurrent transient atrial fibrillation during attacks of basilar artery migraine with loss of consciousness and speculate on the mechanism.

Case report

A 46-year-old man with a family history of travel sickness and paternal migraine had had recurrent headaches with nausea and vomiting since childhood. At the age of 32 the pattern of his attacks changed. A typical attack began with a hot clammy feeling, dizziness, occasional rotatory vertigo, unsteadiness of gait, followed by penetrating bilateral occipital headache, nausea, and vomiting. His only visual disturbance has been photophobia and during attacks he has had parasthesiae of the hands and arms but no dysarthria, dysphasia, or hemiparesis.

On three separate occasions he has been admitted to hospital as an emergency semicomatose in atrial fibrillation confirmed electrocardiographically. Semicoma lasted 1-4 hours, drowsiness up to 20 hours, followed by headache and spontaneous reversion to sinus rhythm. On other occasions he has had similar attacks at home but neither he nor his wife have noticed his pulse irregular at any time.

Apart from mild high tone deafness, the results of investigations including skull *x*-ray films, lumbar puncture, left carotid angiogram, chest *x*-ray film, and protein bound iodine concentration were all normal as was his ECG between attacks. An EEG was reported as showing right-sided slow and sharp waves over the parietal occipital areas.

Treatment at intervals with digoxin, phenytoin, clonidine, amitriptyline, and psychotherapy did not alter the frequency or the nature of the attacks.

Comment

Our patient satisfies the symptoms of basilar artery migraine, as outlined by Bickerstaff.[1] During the migrainous attack he has had three documented episodes of impaired consciousness with atrial fibrillation and no evidence of other precipitating cause for the cardiac arrhythmia. Bickerstaff has speculated that the loss of consciousness in basilar artery migraine may be due to either "epilepsy" or "brain stem ischaemia."[2,3] Mauk *et al* have shown that stimulation of the reticular activating system in dogs leads to cardiac dysrhythmias including atrial fibrillation.[4] These dysrhythmias were mediated by the sympathetic nervous system.[5]

We suggest that ischaemia of the reticular activating system due to basilar artery migraine caused both impairment of consciousness and atrial fibrillation. Therefore neither epilepsy nor atrial fibrillation is likely to be the cause of impairment of consciousness in basilar artery migraine and the logical prophylaxis of the atrial fibrillation would be β-sympathetic-blocking drugs.

1 Bickerstaff, E R, *Lancet*, 1961, **2**, 1057.
2 Bickerstaff, E R, *Lancet*, 1961, **1**, 15.
3 Bickerstaff, E R, *Proceedings of the Royal Society of Medicine*, 1961, **55**, 167.
4 Mauk, H P, Hockman, C H, and Hoff, E C, *American Heart Journal*, 1964, **68**, 98.
5 Hockman, C H, Mauk, H P, and Hoff, E C, *American Heart Journal*, 1966, **71**, 695.

-> ACFA par ischémie de la zone réticulée

Chronic Paroxysmal Hemicrania: Heart Rate Changes and ECG Rhythm Disturbances. A Computerized Analysis of 24 h Ambulatory ECG Recordings

David Russell[1] and Liv Storstein[1] Cephalalgia June 1984 4: 135-144,

- 5 patients migraineux sans atcd cardiovasculaire
  - 105 attaques de migraine

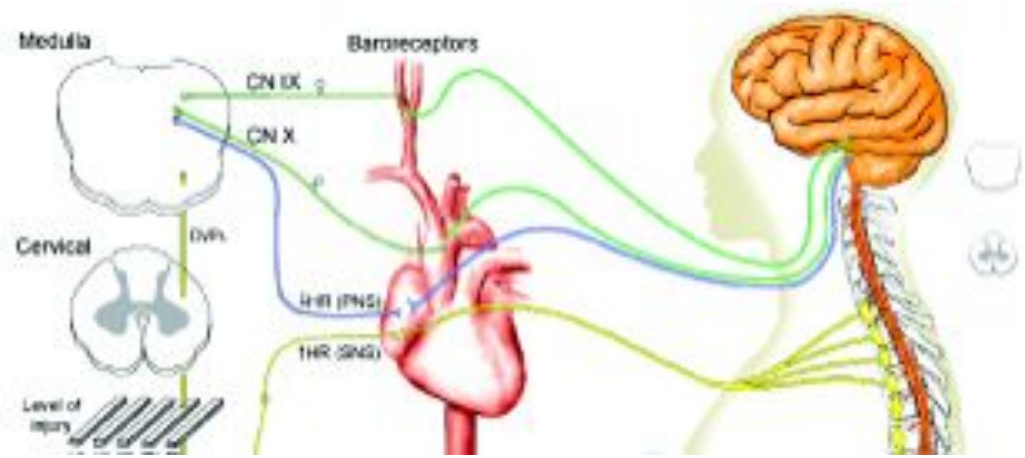
-> pas d'arythmie typique associée aux crises

-> variations majeures de la FC avant, pendant et après les crises

-> Symptômes récidivants chez 2 patients lors des crises :

- bloc sino-atrial avec échappement ventriculaire (8/10 crises)
- bloc de branche et ACFA (28/38 crises)

-> aucun symptôme chez ces 5 patients en dehors des crises



HEADACHE CURRENTS—CLINICAL REVIEW October 1, 2012.

QT Prolongation, Torsade de Pointes, Myocardial Ischemia From Coronary Vasospasm, and Headache Medications. Part 2: Review of Headache Medications, Drug–Drug Interactions, QTc Prolongation, and Other Arrhythmias

Mark J. Stillman, MD; Deborah E. Tepper, MD; Stewart J. Tepper, MD; Leslie Cho, MD

Table 3.—Drugs That Prolong QT Interval and Can Cause Torsade de Pointes in Certain Circumstances

Agent	Brand name(s)	Comment
Miscellaneous		
Galantamine	Razadyne	Cholinesterase inhibitor for Alzheimer's disease
Solifenacin	Vesicare	Bladder overactivity treatment, muscarinic receptor antagonist
Anti-infectives		
Ciprofloxacin	Cipro	Drug–drug interaction, drug metabolism inhibitor
Fluconazole	Diflucan	Drug–drug interaction, drug metabolism inhibitor; increased QT at doses >800 mg/day
Itraconazole	Sporanox	Drug–drug interaction, drug metabolism inhibitor
Ketoconazole	Nizoral	Drug–drug interaction, drug metabolism inhibitor
Ritonavir	Norvir	Protease inhibitor HIV
Trimethoprim-sulfamethoxazole	Septra; Bactrim	—
Antihistamine agents		
Diphenhydramine	Benadryl	Risk with overdosage
Psychotropic agents		
Amisulpride	Solian	Tricyclic antidepressant (TCAD), risk with overdosage
Amitriptyline	Elavil	TCAD
Clomipramine	Anafranil	TCAD
Desipramine	Pertofrane	TCAD
Doxepin	Sinequan	TCAD
Fluoxetine	Prozac, Sarafem	—
Imipramine	Norfranil	TCAD
Nortriptyline	Pamelor	TCAD
Paroxetine	Paxil	—
Protriptyline	Vivactil	TCAD
Sertraline	Zoloft	—
Trazodone	Desyrel	—
Trimipramine	Surmontil	TCAD

Zolmitriptan also has a specific warning on exacerbation of Wolf–Parkinson–White (WPW). Zolmitriptan is explicitly contraindicated in WPW and cardiac accessory conduction pathway disorders.[20] Whether other triptans trigger WPW is unknown and unreported.

Epilepsie

ELECTROCARDIOGRAPHIC ACCOMPANIMENTS OF TEMPORAL LOBE EPILEPTIC SEIZURES

L.D. Blumhardt, P.E.M. Smith, Lynne Owen

Volume 327, Issue 8489, 10 May 1986, Pages 1051–1056

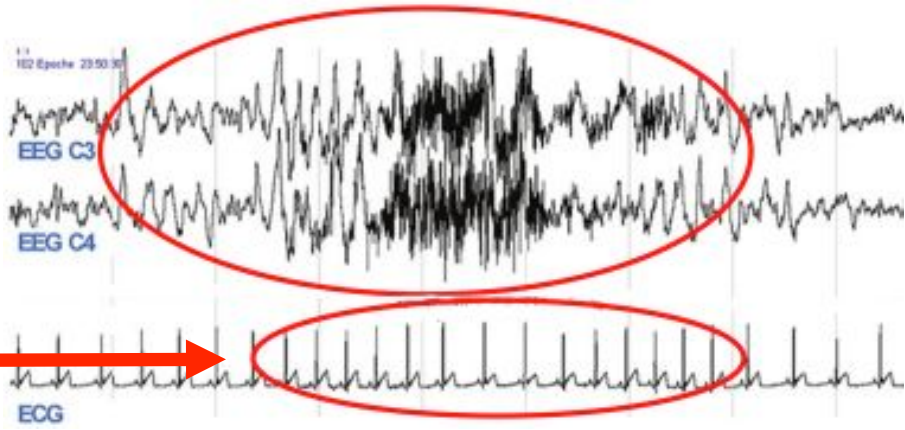
Abstract

74 spontaneous seizures in 26 patients with a clinical diagnosis of temporal lobe epilepsy (complex partial seizures) were recorded by simultaneous ambulatory cassette monitoring of the electrocardiogram and the electroencephalogram (EEG). In 24 patients (92%) seizures were associated with an increased heart rate. The maximum heart rates exceeded 120 beats/min in 67% of seizures, 140 beats/min in 30%, and 160 beats/min in 12%. The acceleration of heart rate was greater in patients under than in those over 25 years old ($p<0.01$) and in those not treated with than in those on anticonvulsant drugs ($p<0.01$). Ictal cardiac arrhythmias occured in 42% of the patients and the commonest was an irregular series of abrupt rate changes which occured towards the end of the EEG seizure discharge. Asymptomatic (clinically silent) arrhythmias occured no more frequently in the patients than in age and sex matched healthy subjects. These secondary autonomic effects of epilepsy may lead to diagnostic errors if their cerebral origins are not suspected. They seem to be reduced in severity by anticonvulsant drugs (ACD) and they may account for sudden unexplained deaths in epileptics.

-> tachycardie sinusale, arythmies

-> ESA, ESV, variation des ondes P et T

-> Variation de l'intervalle R-R

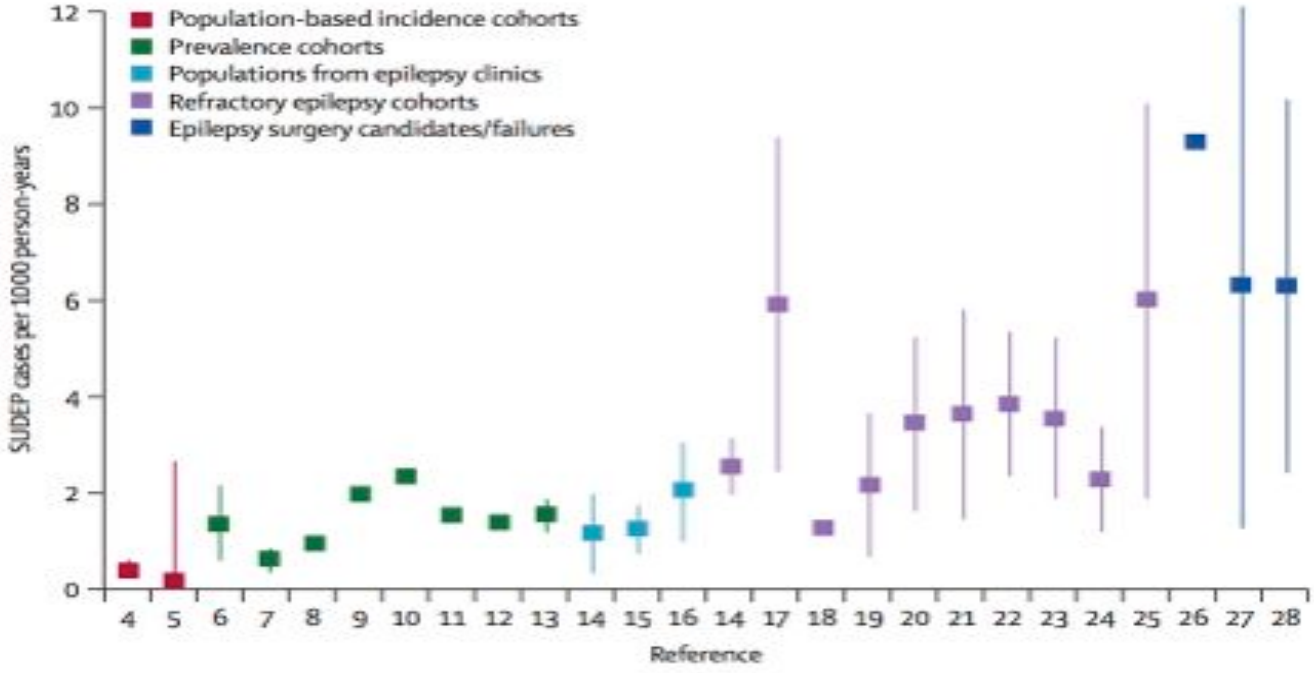


Sudden unexpected death in epilepsy: current knowledge and future directions

Lancet Neurol 2008; 7: 1021–31

Torbjörn Tomson, Lina Nashef, Philippe Ryvlin

- « Sudden Unexpected Death in EPilepsy » (SUDEP)
  - de 0,9 à 9 ‰ patients-ans
  - ~10% des décès liés à l'épilepsie (1ère cause)
  - Mortalité x 20-40 / population générale
  - Risque > 1 / 250 / an si épilepsie réfractaire

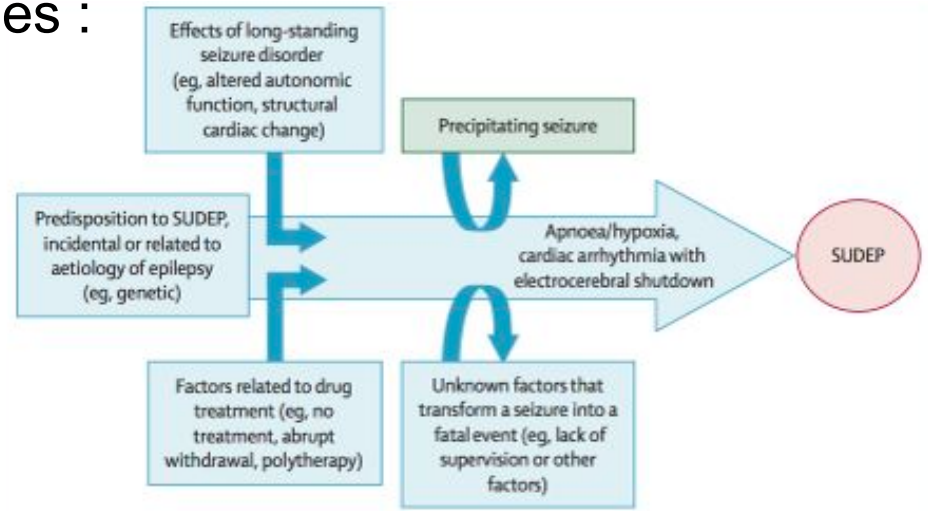


Sudden unexpected death in epilepsy: current knowledge and future directions

Lancet Neurol 2008; 7: 1021–31

Torbjörn Tomson, Lina Nashef, Philippe Ryvlin

- « Sudden Unexpected Death in EPilepsy » (SUDEP)
  - Mécanisme physiopathologique exact non identifié
  - Quelques facteurs de risque identifiés :
    - homme jeune
    - mauvaise observance du ttt
    - épilepsie mal contrôlée
    - Certains Ttt antiépileptiques :
      - *carbamazépine*
      - *lamotrigine*
    - Pathologie cardiaque pré-existante



=> Meilleure prévention = suppression définitive des crises

-> Ttt mdc et/ou chirurgical

Cardiac Asystole in Epilepsy: Clinical and Neurophysiologic Features

Epilepsia, **44**(2):179–185, 2003

*R. Rocamora, *M. Kurthen, †L. Lickfett, *J. von Oertzen, and *C. E. Elger

*Departments of *Epileptology and †Medicine-Cardiology, University of Bonn, Bonn, Germany*

- Etude rétrospective sur 1244 patients

  -> Enregistrements de longue durée EEG et ECG simultanés

  -> Asystoles chez 5 patients

TABLE 1. *Information on admission*

Pat no.	Age (yr)	Sex	AED (on admission)	ECG on admission	Previous cardiac disease	Long postictal period
1	30	F	LTG, PB, VPA, CBZ	Normal	No	Yes
2	53	M	CBZ	Normal	MI	Yes
3	27	F	VPA, CBZ	Normal	No	No
4	31	M	CBZ	Sinus bradycardia, first-degree A-V block	Aberrant condution defect	No
5	16	M	LTG; CBZ, CZP	Normal	No	No

TABLE 2. *Seizure onset and lateralization*

Pat. no.	Seizure lat.	Seizure onset	Type and number of registered seizures	Asystolic events	Ictal apnea	MRI	Ictal recording
1	Bifrontal	Bifrontal	1 sec. GS	1	Yes	Unclear signal right frontal	Video-EEG/ECG
2	L	Left-temporal	2 CPS	2	No	Unremarkable	Scalp EEG/ECG
3	L	Left-temporal	2 CPS	1	No	Small lesions in the left insula	Video-EEG/ECG
4	L	Left-frontal	Series of sec. GS	1	No	DNT left frontal	Video-EEG/ECG
5	L	Left-frontal	13 SPS	6	Yes	Unremarkable (SISCOM activation in the left insula)	Video-EEG/ECG

TABLE 3. *ECG and EEG correlates during the event*

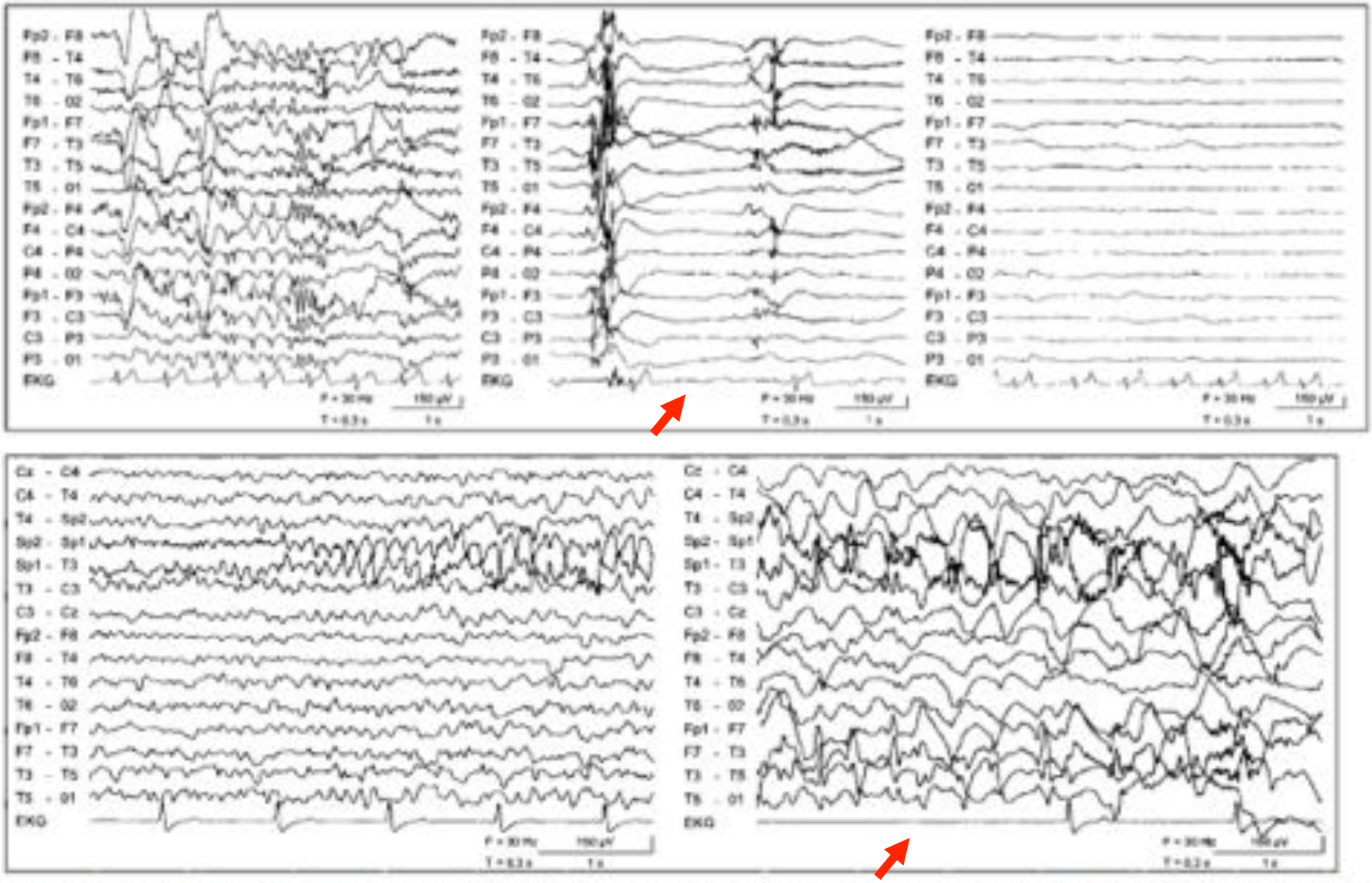
Pat no.	ECG description	Asystole onset	Asystolic period	EEG during asystolic period	EEG normalization period
1	Sinus bradycardia followed by sinus arrest	100 s after seizure onset	60 s	Extremely flat (no activity more than 10 μV)	Longer than 2 h
2	Sinus bradycardia followed by sinus arrest	20 s after seizure onset	18 and 28 s	High-amplitude delta waves with focal ictal activity	30 s
3	Sinus bradycardia followed by sinus arrest	25 s after seizure onset	7 s	High-amplitude delta waves with focal ictal activity	35 s
4	Atrial fibrillation followed by asystole	60 s after seizure onset	7 s	Technical artifact (during seizures series)	Technical artifact
5	Sinus bradycardia followed by sinus arrest	5–14 s after seizure onset	5–9 s	Bilateral theta slow waves	2–5 s

Cardiac Asystole in Epilepsy: Clinical and Neurophysiologic Features

Epilepsia, **44**(2):179–185, 2003

*R. Rocamora, *M. Kurthen, †L. Lickfett, *J. von Oertzen, and *C. E. Elger

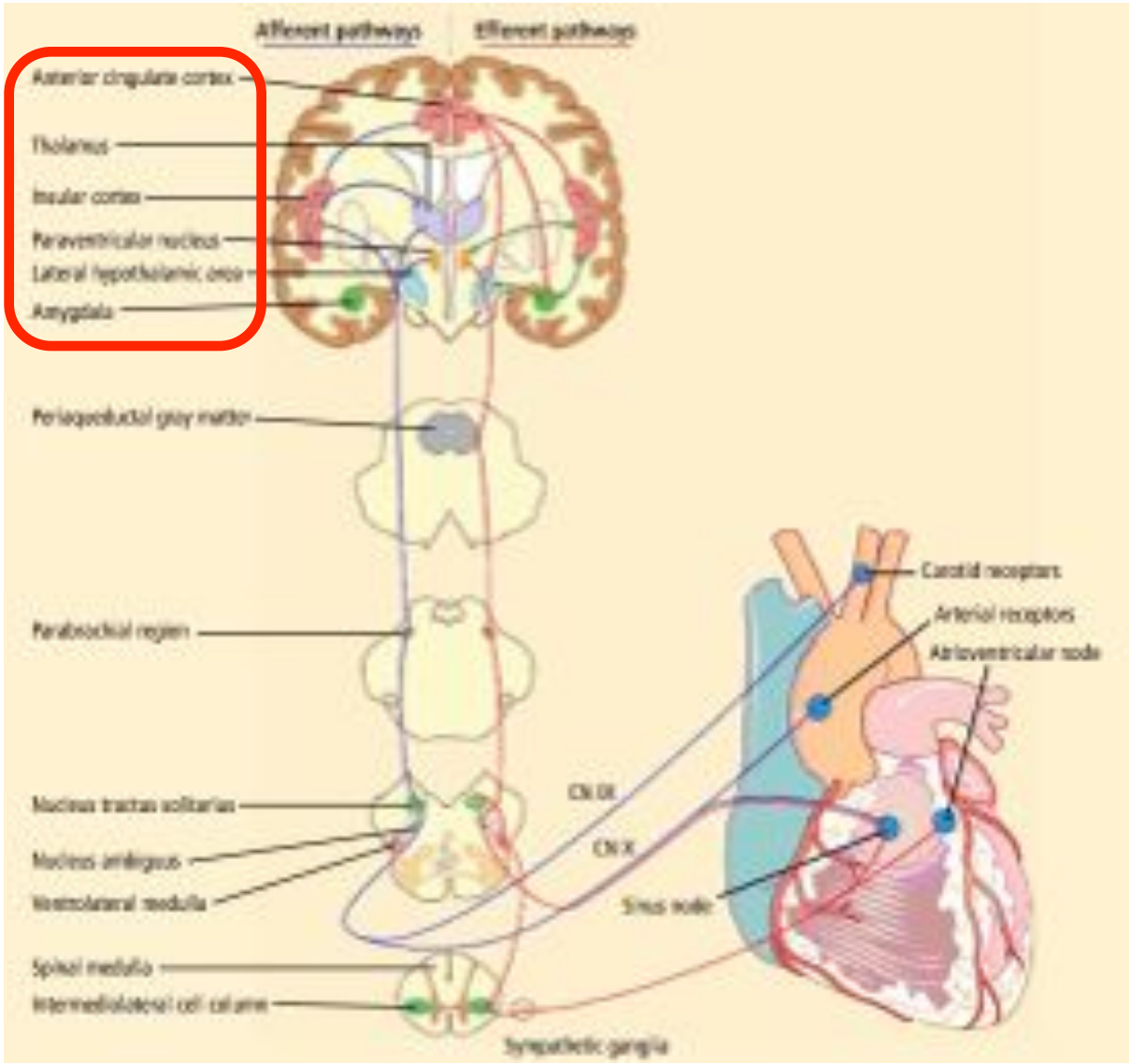
*Departments of *Epileptology and †Medicine-Cardiology, University of Bonn, Bonn, Germany*



Sudden death in epilepsy, surgery, and seizure outcomes: The interface between heart and brain

LARA JEHI, MD

CLEVELAND CLINIC JOURNAL OF MEDICINE VOLUME 77 • SUPPLEMENT 3 JULY 2010 S51



SOCIETY PROCEEDINGS
DEUTSCHE EEG GESELLSCHAFT
Munich, April 27–29, 1967
Electroencephalography and Clinical Neurophysiology
Electroenceph. clin. Neurophysiol., **1969,** *26*: 433–449

- Mesure de la fréquence cardiaque lors de stimulations lumineuses
  - Comparaison sujets sains *versus* épileptiques photo-sensibles

-> bradycardie : - 0,8 à 3,3 bpm *versus* - 1,2 à 14,2 bpm (P<0,005)

44. Alterations of heart rate induced by photic stimulation in healthy subjects and epileptics. — G. Rabending and D. Krell (Magdeburg).

The influence of photic stimulation on the cardiac rhythm was investigated in photosensitive and non-photosensitive epileptics, as well as in healthy control subjects. The respiratory movements of the thorax were recorded simultaneously. The interval between the R waves was automatically measured before, during and after photic stimulation, after which estimation of the mean value was performed in order to reduce the influence of respiratory arrhythmia on the result, and to make more recognizable the systematic effects of photic stimulation on the cardiac rhythm.

The results were as follows: (a) bradycardia as an on-effect; (b) bradycardia as an on-effect with subsequent tachycardia; (c) tachycardia as an on-effect; (d) tachycardia as an on-effect with subsequent bradycardia; (e) tachycardia as an on-effect; (f) indifferent behaviour.

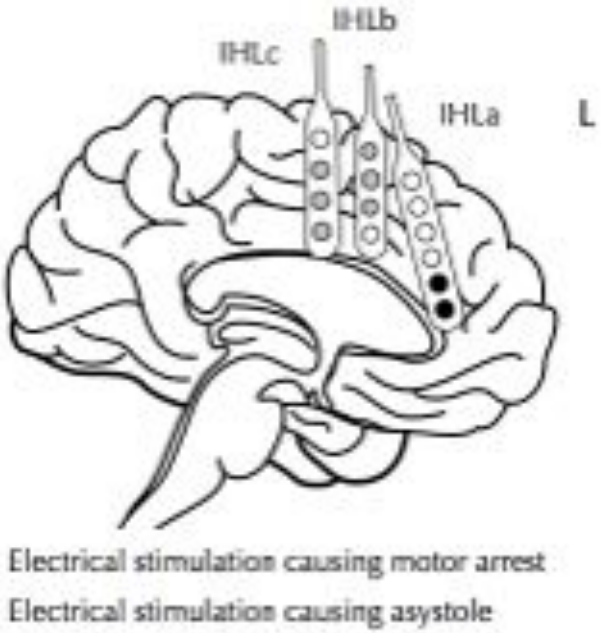
Healthy subjects and photosensitive and non-photosensitive epileptics gave only quantitative differences. Bradycardia was the commonest reaction. Its degree (maximal mean deviation from the mean initial frequency) lay in healthy subjects between 0.8 and 3.3 beats/min, in photosensitive epileptics between 1.2 and 14.2/min. This difference is well within statistical significance ($P < 0.005$). In photosensitive epileptics there was a linear relationship between the mean initial frequency and the degree. In different patients the bradycardia showed differences in time course. Bradycardia and tachycardia were independent of the partial influences of respiration.

From the results it seems that bradycardia and tachycardia are non-specific reactions to the light stimulus. Their degree and duration achieve the greatest values in photosensitive epileptics. Differences in degree and duration in comparison with healthy subjects are statistically significant when the groups are compared.

Asystole induced by electrical stimulation of the left cingulate gyrus

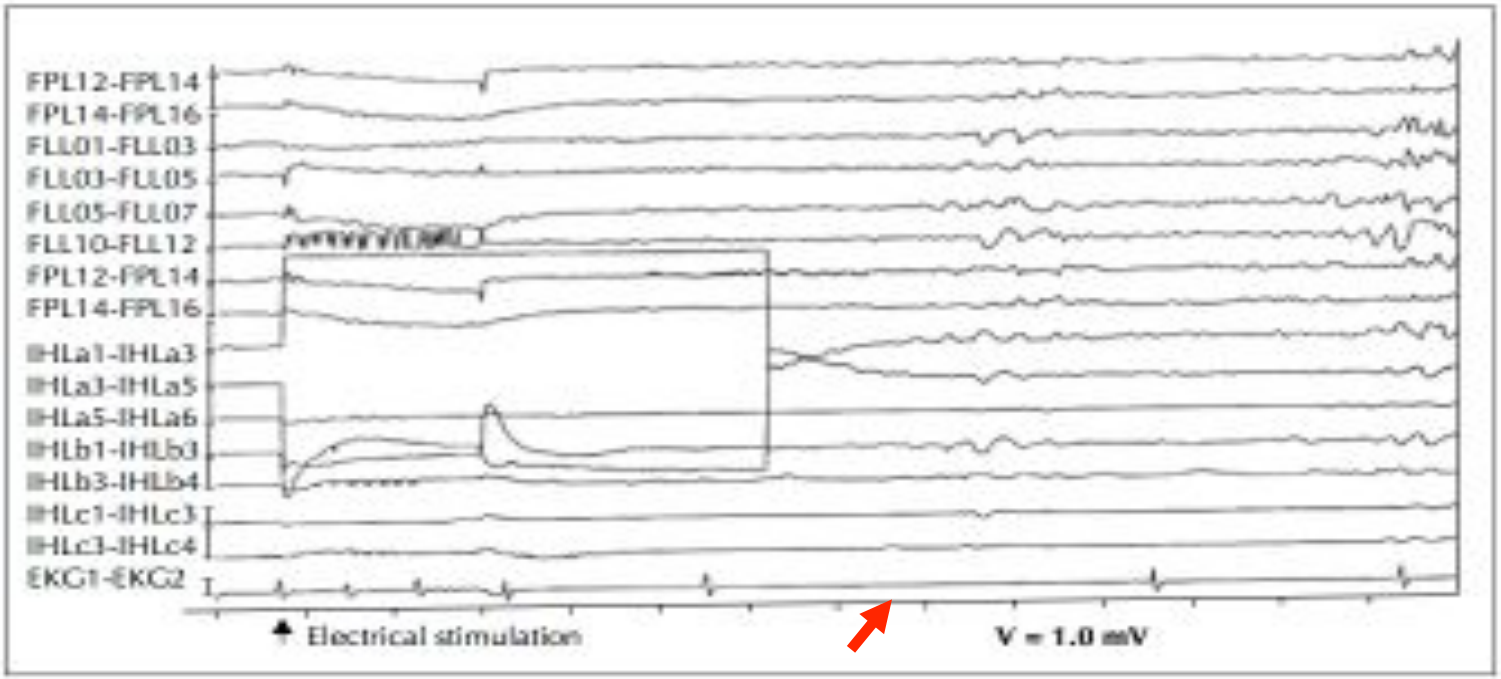
Howan Leung[1,2], Kasper Schindler[1], Patrick Kwan[2], Christian Elger[1]

Epileptic Disord 2007; 9 (1): 77-81



- H 18 ans, épilepsie frontale
  - électrodes de stimulation intracérébrales

=> Déclenchement simultané crise d'épilepsie et asystole



Hémorragies Cérébrales et Infarctus Cérébraux

A New Electrocardiographic Pattern Observed in Cerebrovascular Accidents

G. E. BURCH, ROBERT MEYERS and J. A. ABILDSKOV

Circulation. 1954;9:719-723

Circulation
JOURNAL OF THE AMERICAN HEART ASSOCIATION

- 17 patients avec hémorr. cérébrale, sous-arachnoïdienne, AVC
  - 12 patients avec ECG de référence ≤ 48h.

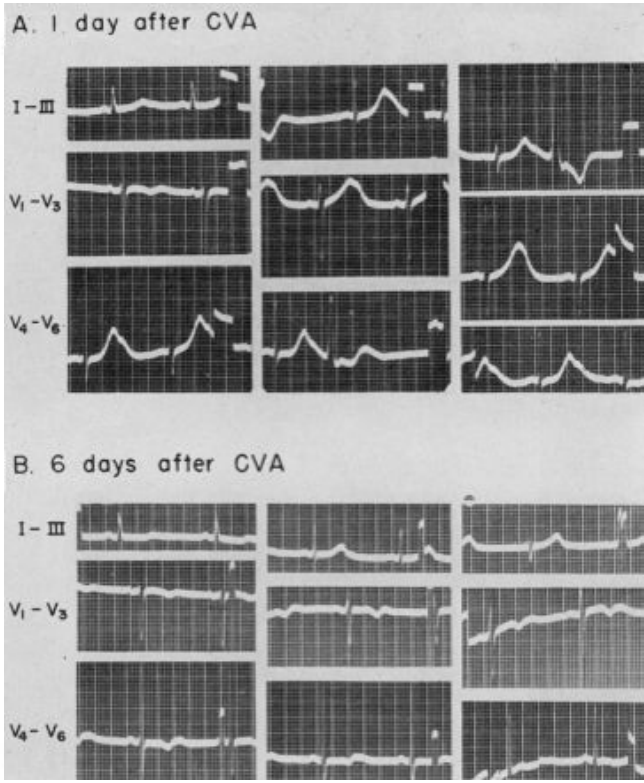
    -> larges ondes T,

    -> ± larges ondes U

    -> allongement de l'espace QT (« QU »)

TABLE 1.—*Pertinent Clinical and Electrocardiographic Data*

Patient No.	Age (yrs.)	Sex	Diagnosis	Interval Before 1st ECG	Duration Q-T Interval (sec.)	Normal Q-T Interval (sec.)	% Increase	U Wave
1	78	M	Cerebral hemorrhage	—	0.80	0.48	66	Not prominent
2	79	M	Cerebral hemorrhage	24 hours	0.44	0.37	19	V_1 and V_2
3	50	M	Cerebral hemorrhage	24 hours	0.60	0.48	25	V leads
4	47	M	Cerebral hemorrhage	24 hours	0.48	0.41	17	V_2
5	65	F	Cerebral hemorrhage	24 hours	0.46	0.40	15	Not prominent
6	63	F	Cerebral hemorrhage	7 days	0.52	0.34	53	Not prominent
7	74	F	Cerebral hemorrhage	24 hours	0.44	0.40	10	V_2-V_4
8	34	F	Subarachnoid hemorrhage	24 hours	0.48	0.36	33	Not prominent
9	58	M	Subarachnoid hemorrhage	24 hours	0.48	0.40	20	V leads
10	32	F	Subarachnoid hemorrhage	48 hours	0.48	0.44	9	V_2-V_4
11	43	F	Subarachnoid hemorrhage	24 hours	0.64	0.45	42	V_2
12	70	M	Subarachnoid hemorrhage	4 days	0.60	0.47	27	V_2
13	63	F	Subarachnoid hemorrhage	48 hours	0.62	0.38	63	Not prominent
14	71	F	Subarachnoid hemorrhage	72 hours	0.44	0.35	26	All leads
15	65	M	C.V.A.	24 hours	0.44	0.34	30	III and V_4
16	47	M	C.V.A.	48 hours	0.44	0.41	7	V_1
17	58	M	C.V.A.	72 hours	0.44	0.37	19	Not prominent



Electrocardiographic Changes Simulating Myocardial Ischemia and Infarction Associated with Spontaneous Intracranial Hemorrhage

G. J. CROPP and G. W. MANNING

Circulation. 1960;22:25-38

Circulation

JOURNAL OF THE AMERICAN HEART ASSOCIATION

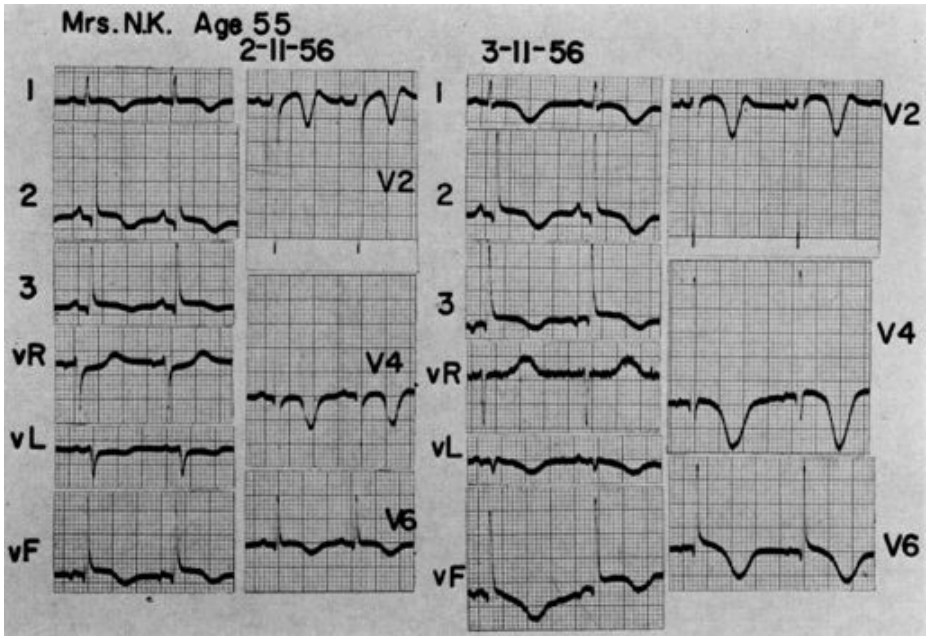
- 29 patients avec hémorragie sous-arachnoïdienne
  - ECG avant angiographie cérébrale (n=28) et/ou craniotomie (n=19)

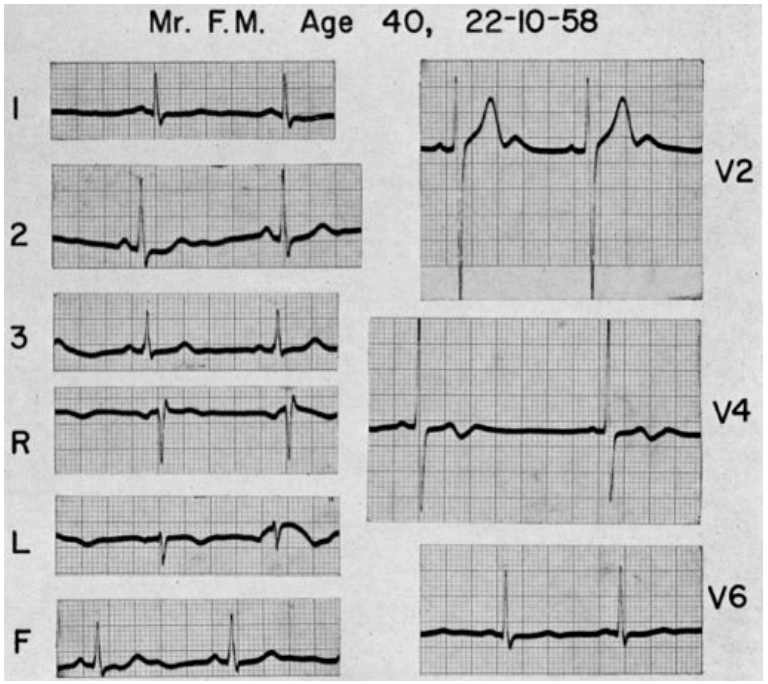
    -> allongement de l'espace QT = 67 %

    -> ondes T aplaties ou inversées = 55%

    -> ± large ondes U
  - 8 décès

    -> 5 autopsies : cœur tous normaux





Cardiac Arrhythmias in Subarachnoid Haemorrhage

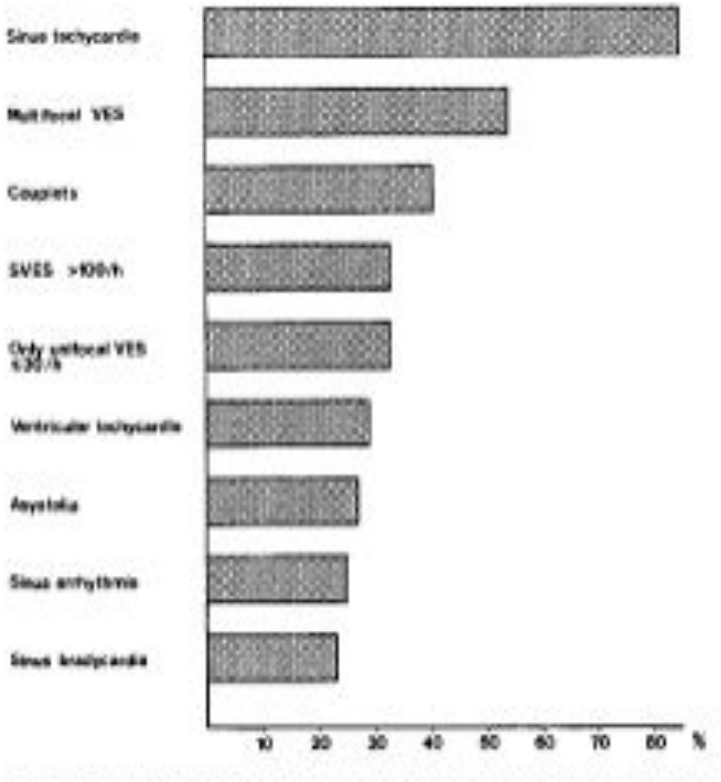
T. Stober[1], Th. Anstätt[1], S. Sen[2], K. Schimrigk[1], and H. Jäger[1]

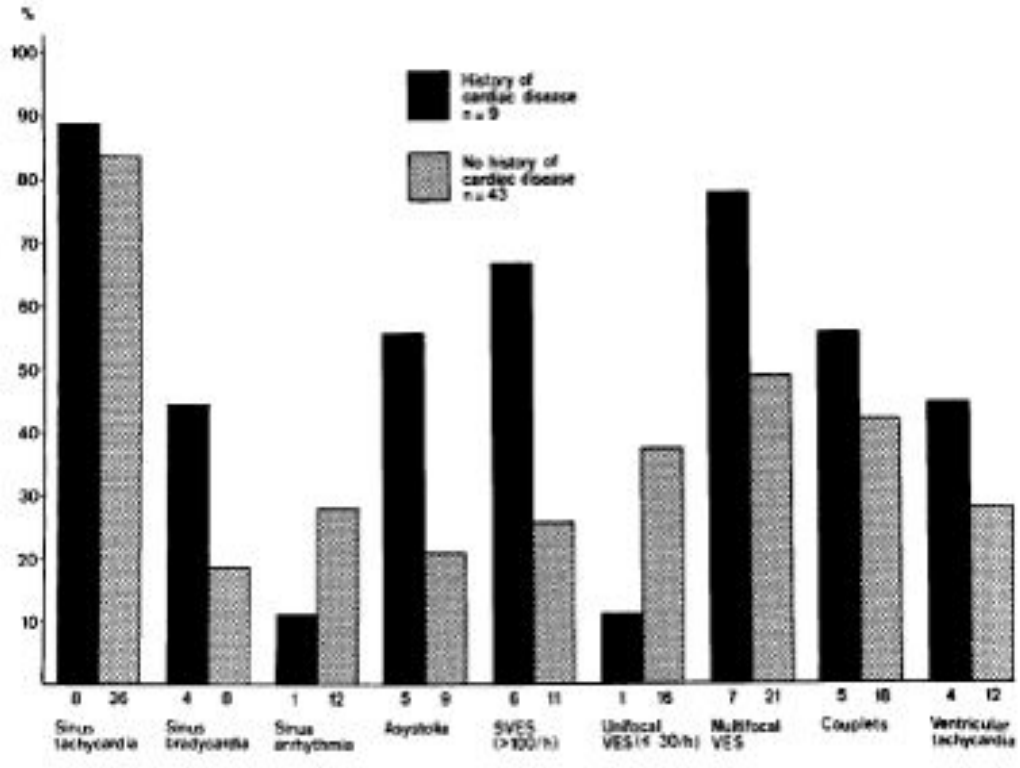
Acta Neurochir (Wien) (1988) 93: 37–44

- 52 patients ayant une hémorragie sous-arachnoïdienne
  - Enregistrement ECG continu sur 5 jours

    -> multiples troubles du rythme et/ou de conduction

    -> favorisés si pathologie cardiaque pré-existante



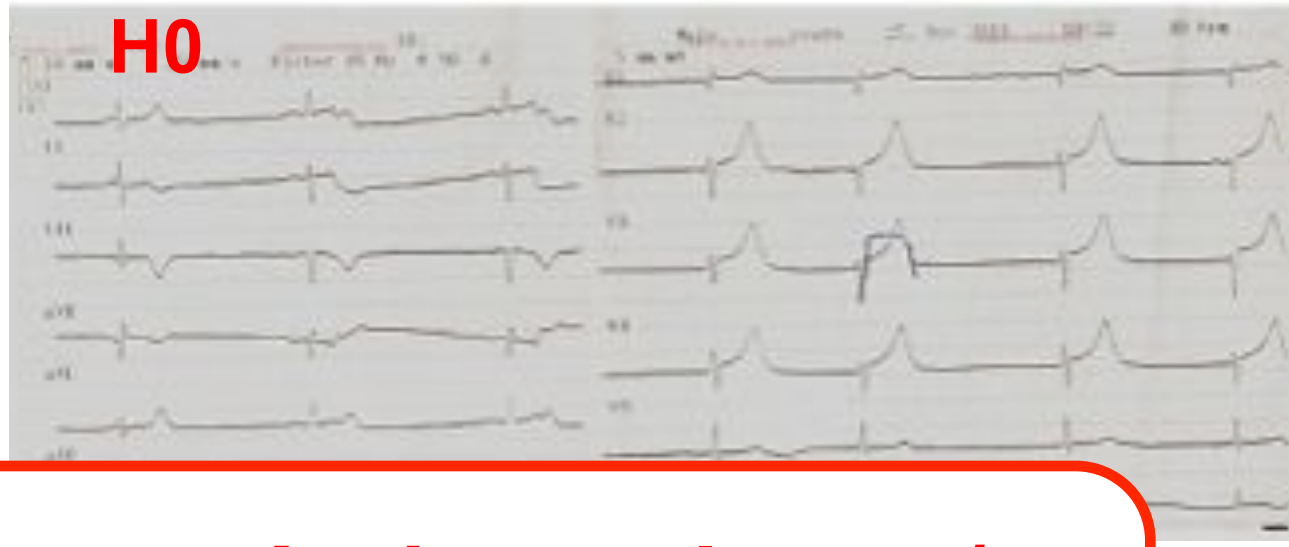


Case Report Murat Saritemur

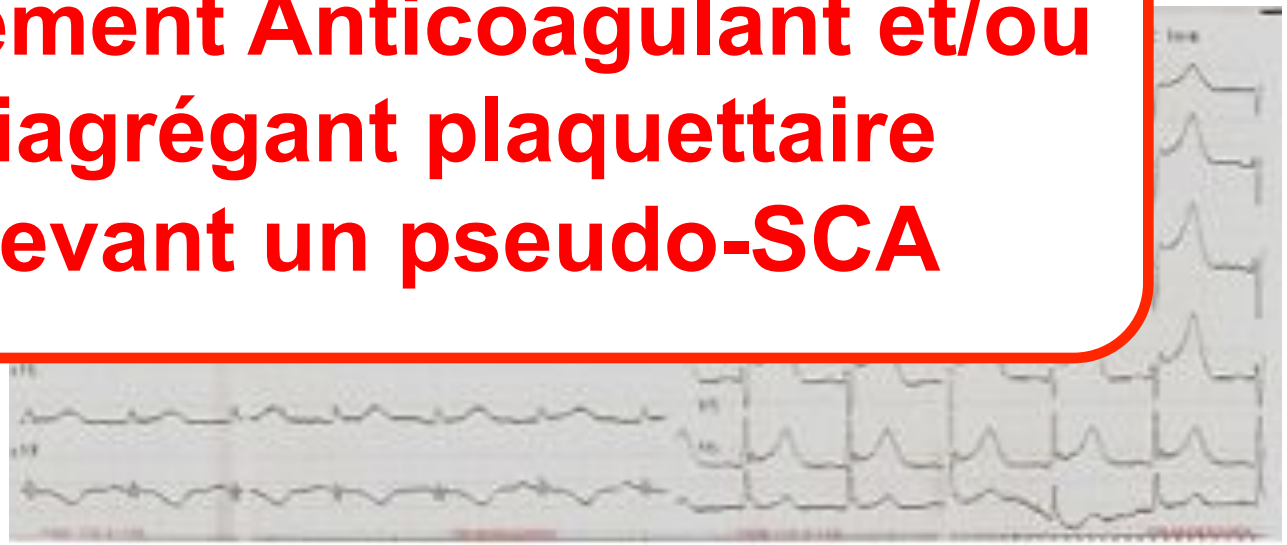
Intracranial hemorrhage with electrocardiographic abnormalities and troponin elevation

American Journal of Emergency Medicine (2013) **31**, 271.e5–271.e7

- H 45 ans au SAU
  - Anomalies ECG
  - Tropo I = 10 mg/l
  - Tr. de conscience



Traitement Anticoagulant et/ou Antiagrégant plaquettaire devant un pseudo-SCA



The electrocardiogram in stroke: relationship to pathophysiological type and comparison with prior tracings.

D S Goldstein *Stroke*. 1979;10:253-259

- 150 patients avec AVC ischémique ou hémorragiqiue
  - Comparaison *versus* 150 témoins

-> 92 % anomalies ECG précoces

-> ACFA= 47% si infarctus cérébral

-> valeur pronostique négative +++

TABLE 2 *New ECG Abnormalities in Stroke and Control Patients with Prior, Available ECGs*

Finding	Stroke	Control	Chi-square
Prolonged QT	17 (32%)	1 (2%)	18.40***
T wave inversion	8 (15%)	0 (0%)	7.12**
U waves	7 (13%)	0 (0%)	6.78**
ST depression	7 (13%)	1 (2%)	4.47*
Arrhythmia	13 (25%)	2 (3%)	10.01**
Sinus	2 (4%)	0 (0%)	N.S.
Atrial fib.	5 (9%)	0 (0%)	4.25*
Ventricular	4 (8%)	1 (2%)	N.S.
Other	2 (4%)	1 (2%)	N.S.
PACs	3 (6%)	0 (0%)	N.S.
PVCs	3 (6%)	0 (0%)	N.S.
Either ST depression or T inversion	11 (21%)	2 (3%)	7.40**
Bradycardia	4 (8%)	0 (0%)	N.S.
Tachycardia	1 (2%)	2 (3%)	N.S.
Overall number changed	39 (74%)	9 (14%)	41.71***

TABLE 3 *Factors Related to Mortality in Patients with Stroke*

Factor	Mortality	Chi-square
Level of consciousness		
Alert	3/81 (4%)	
Lethargic	5/24 (21%)	
Stuporous	8/17 (47%)	
Comatose	21/28 (75%)	57.05***
Type of stroke		
Cerebral thrombosis	4/49 (8%)	
Indeterminate	3/38 (8%)	
Cerebral embolus	6/19 (32%)	
Subarachnoid hemorrhage	15/28 (54%)	
Intracerebral hemorrhage	9/16 (56%)	30.40***
Level of CPK		
Less than 100	0/13 (0%)	
101–499	4/14 (29%)	
500 or more	6/9 (67%)	8.63**
Admission systolic pressure		
Less than 100 mm Hg	6/9 (67%)	
More than 100 mm Hg	30/135 (22%)	6.09**
Ventricular arrhythmias		
Tachycardia, fibrillation, or asystole	4/5 (80%)	
None	33/145 (23%)	4.82*

*$p < 0.05$
**$p < 0.01$
***$p < 0.001$

Possible role of catecholamines, corticosteroids, and potassium in production of electrocardiographic abnormalities associated with subarachnoid haemorrhage

British Heart Journal, 1974, **36**, 697–706.

J. M. Cruickshank, G. Neil-Dwyer, and A. W. Stott

- 40 patients avec hémorragie sous-arachnoïdienne
  - Dosage métanephrines urinaires, cortisol plasmatique et potassium

-> corrélation entre l'apparition d'anomalies sur l'ECG et :

- ↑ taux urinaires de métanéphrines, normétanéphrines, catécholamines
- ↑ cortisol plamatique
- ↓ kaliémie

TABLE 2 *Relation between electrocardiogram and 24-hour urinary normetanephrin, metanephrin, total catecholamines, and plasma cortisol.*

Electrocardiogram	*Mean urinary normetanephrin (µg/24 hr)*	*Mean urinary metanephrin (µg/24 hr)*	*Mean urinary total catecholamines (µg/24 hr)*	*Mean plasma cortisol (µg/100 ml)*
All normal	267 (±153)	218 (±125)	485 (±192)	15·0 (±6·33)
Changing	320 (±143)	290 (±127)	610 (±232)	22·9 (±9·41)
All abnormal	479 (±282)	376 (±158)	855 (±357)	25·8 (±9·41)

Mort Encéphalique

Brain death assessment using instant spectral analysis of heart rate variability

Crit Care Med 2002 Vol. 30, No. 2

Christophe Baillard, MD; Benoit Vivien, MD; Pascale Mansier, PhD; Laurence Mangin, MD; Sylvain Jasson, PhD; Bruno Riou, MD, PhD; Bernard Swynghedauw, MD, PhD

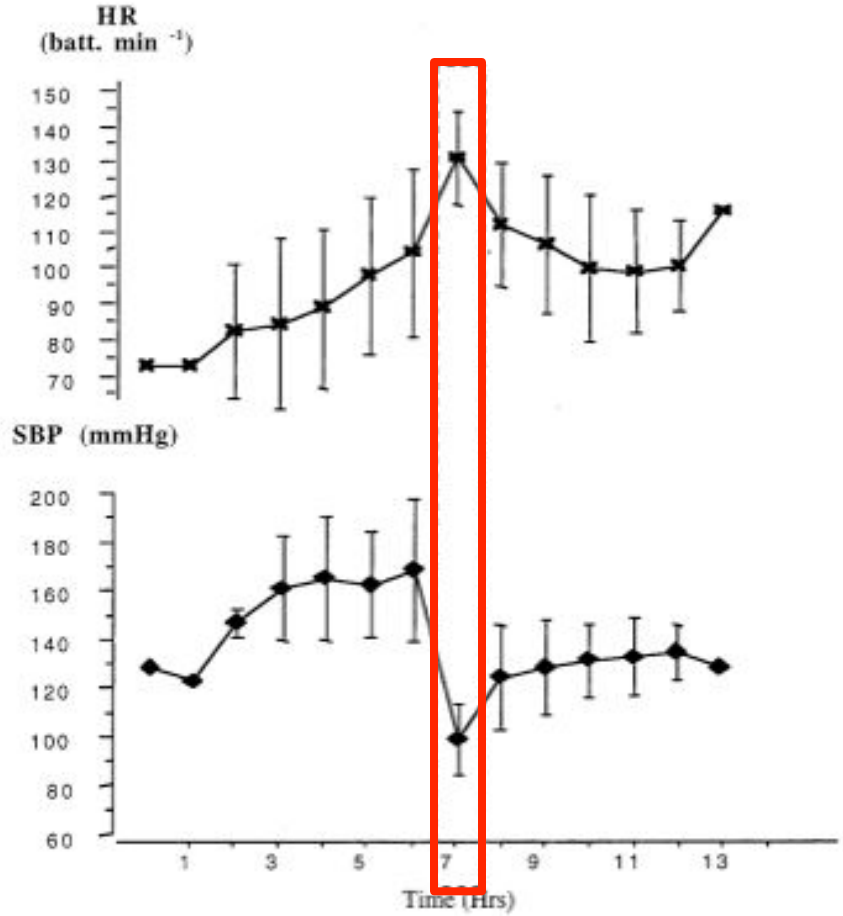
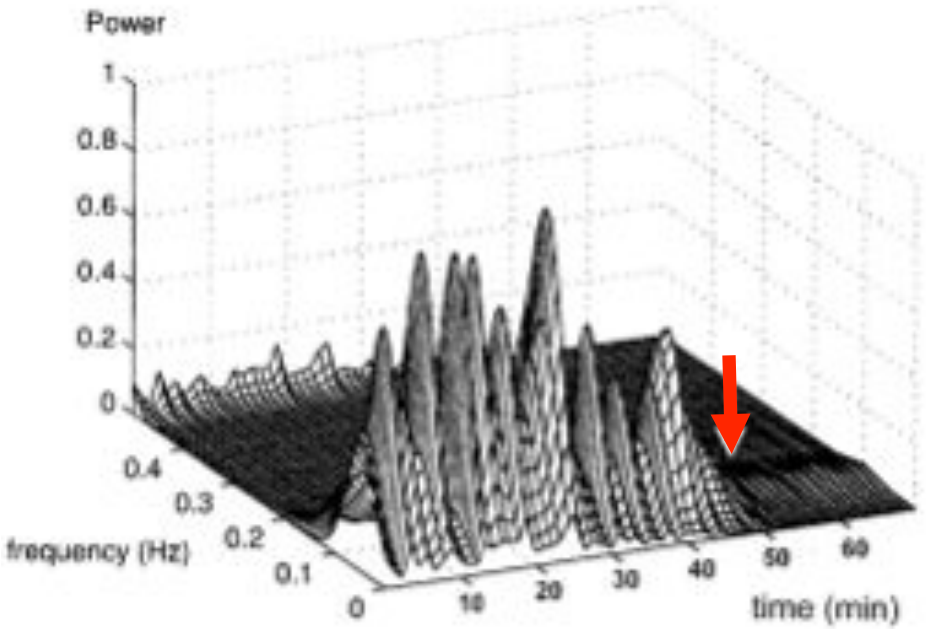


Figure 1. Systolic blood pressure (*SBP*) and heart rate (*HR*) during the study period (n = 10).



« Orage catécholaminergique »

ORIGINAL ARTICLE

Transplant International

Brain death provokes very acute alteration in myocardial morphology detected by echocardiography: preventive effect of beta-blockers

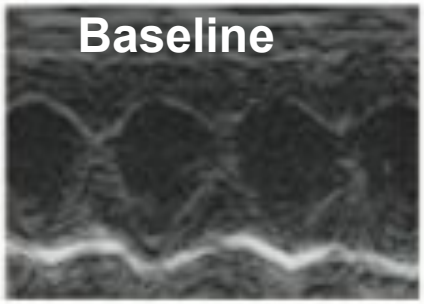
Transplant International ISSN 0934-0874

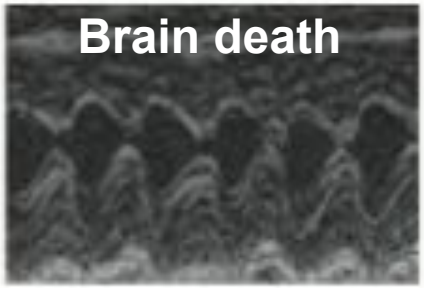
René Ferrera,[1] Guylaine Hadour,[1] Fabienne Tamion,[2] Jean-Paul Henry,[2] Paul Mulder,[2] Vincent Richard,[2] Christian Thuillez,[2] Michel Ovize[1] and Geneviève Derumeaux[1]

- Modèle expérimental de mort encéphalique (rats Wistar)
  - inflation ballon intra-crânien (300 ml)
  - évaluation HD, échocardiographique et dosage de catécholamines
  - test α-bloquant, β-bloquant, inh. calcique

-> Conséquences ME :
- ↑ FC et PA
- ↑ épaisseur paroi VG
- ↑ taux catécholamines = Nadré x 3, adré x 50

-> Prévention si β-bloquant





	Systolic blood pressure (mmHg)	Heart rate (beats/min)
Baseline		
Control	137 ± 8	336 ± 22
β-group	145 ± 5	372 ± 10
Ca-group	137 ± 7	367 ± 18
α-group	124 ± 7	327 ± 20
Treatment		
Control	137 ± 8	336 ± 22
β-group	100 ± 7*	243 ± 8*
Ca-group	81 ± 9*	232 ± 24*
α-group	104 ± 8*	340 ± 31
Brain death induction		
Control	232 ± 10	440 ± 24
β-group	234 ± 6	290 ± 15*
Ca-group	165 ± 9*	304 ± 8*
α-group	170 ± 10*	374 ± 7*
5 min after brain death		
Control	139 ± 16	417 ± 29
β-group	158 ± 16	317 ± 18*
Ca-group	104 ± 15	310 ± 10*
α-group	96 ± 20	420 ± 11
15 min after brain death		
Control	101 ± 26	360 ± 21
β-group	99 ± 16	297 ± 17
Ca-group	77 ± 12	327 ± 19
α-group	73 ± 11	355 ± 30

*$P < 0.05$ versus control values at the same stage.

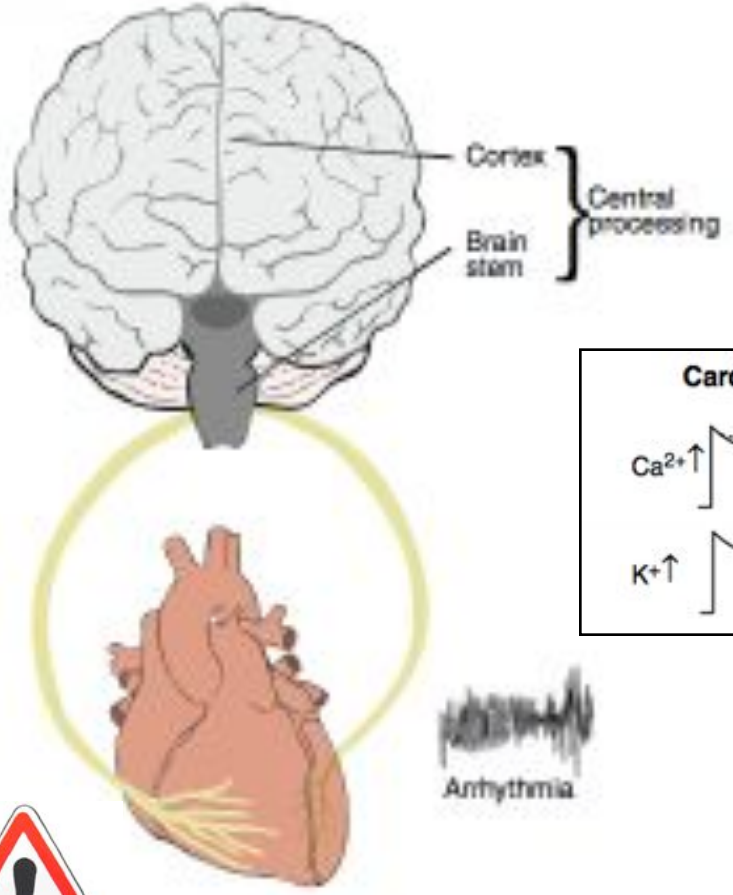
Physiopathologie de l'interaction cerveau - cœur

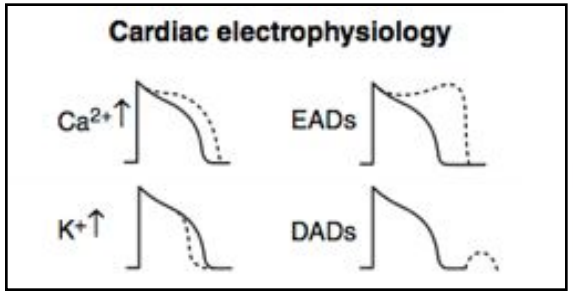
Brain-heart interactions and cardiac ventricular arrhythmias

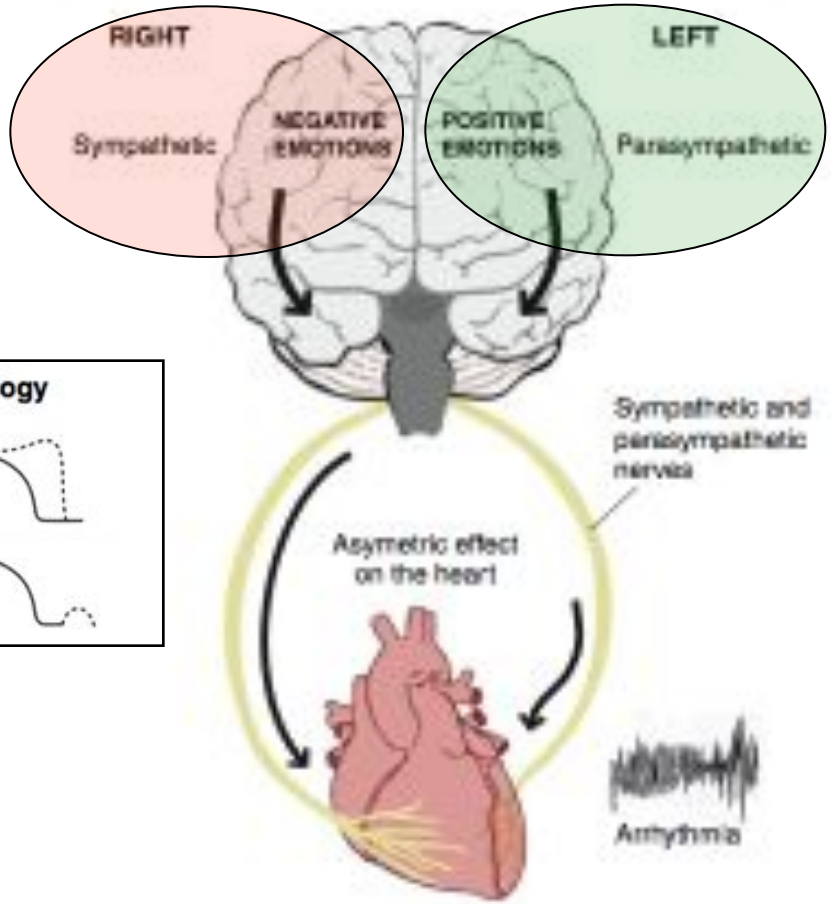
P. Taggart

Neth Heart J (2013) 21:78–81

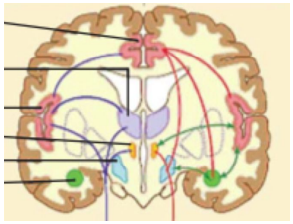
- Rôle SNA dans les troubles du rythme et la mort subite +++
  - Syst. Σ : pro-arythmique sur oreillettes et sur ventricules
  - Syst. pΣ : pro-arythmique sur oreillettes, anti-arythmique sur ventricules



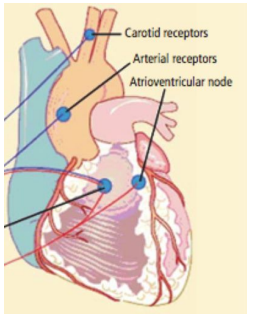




Risque majoré si pathologie cardiaque pré-existante



Conclusion



- Anomalies ECG fréquentes lors des pathologies neurologiques aiguës : stress … mort encéphalique
- Troubles du rythme, de conduction et de repolarisation
- Responsable de 20% des morts subites non cardiaques
- Fréquence et risque spécifique de l'allongement du QT
  => Instabilité électrique => arythmies ventriculaires
- Hypothèses physiopathologiques
  - ↑ taux de catécholamines circulantes
  - implication des centres régulateurs du SNA (hypothalamus)
- Terrain favorisant = anomalie cardiaque pré-existante