Appropriate Use of Technology in the Diagnosis and Management of Fits and Faints

A brief overview of

Tricky Transient Losses of Consciousness (TLOC)

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Transient Loss of Consciousness (TLOC)

Definition:
An apparent loss of consciousness with an abrupt onset, a short duration, and spontaneous, complete recovery.

TLOC poses diagnostic difficulties, as the causes are diverse, carry vastly different risks.

https://www.indiatimes.com/

Transient Loss of Consciousness (TLOC)

Typically, the **cause must be established after the event** so that a comprehensive history from the patient and witness is crucial and the most important determinant of reaching a correct diagnosis.

When there is clear clinical evidence of loss of consciousness, then fits or faints are most likely, but if there is doubt then other causes of collapse need to be considered:

- Functional seizures
- Functional faints
- Vestibular dysfunction
- Cataplexy
- Panic

Van Dijk et al Nat. Rev. Neurology 5, 438-448 (2009)

Transient Loss of Consciousness
The clues are in the history

A **detailed history and examination** provides the diagnosis in the majority of patients

This may be confirmed by **carefully chosen investigations**

These almost always include an **ECG and EEG**.

Colman, N. et al. Clin. Auton. Res. 14 (Suppl. 1), 37-44 (2004); van Dijk, N. et al. J. Cardiovasc. Electrophysiol. 19, 48-55 (2008).

Transient Loss of Consciousness
Approach to Investigation

With a detailed history and examination, and these two simple investigations (ECG and EEG), an accurate diagnosis is obvious in approximately 60% of cases

Additional investigations should be carefully considered and requested on an individual patient basis

The answer often rests on a combination of clues rather than on one or two elements.
No single symptom or sign will reliably differentiate syncope from seizures.

Colman, N. et al. Clin. Auton. Res. 14 (Suppl. 1), 37-44 (2004); van Dijk, N. et al. J. Cardiovasc. Electrophysiol. 19, 48-55 (2008).

Differentiating fits from faints:
Scoring Systems

A number of scoring systems have been developed to assist in differentiating syncope from seizures, and two, in particular, particular, are widely used:

- **Evaluation of Guidelines in Syncope Study (EGYS) score**
- **Calgary vasovagal score (VVS) score**

Sheldon et al *Emerg Med Int*. 2015; Del Rosso et al *Heart*. 2008;94(12):1620-1626.(2015):1-5; Koshman et al *Eur Heart J*. 2006;27(3):344-350. ; Romme et al . *Eur Heart J*. 2009;30(23):2888-2896

Clues to differentiating Fits from Faints:
It's almost always in the story

Detailed accounts of several attacks from patients and eyewitnesses is crucial.

Some crucial questions to ask are:

- What exactly were you doing at the time?
- What exactly did you experience before, during, and after the event?
- How long did the episode last, and how long did you take to recover?
- How many episodes have you experienced, and how similar have these been?
- At what age did the episodes begin, and how often do they occur?

Differentiating Fits from Faints:
Medical History

- All **comorbidities:** especially neurological, cardiac, otological, psychiatric, and psychosocial

- **Medications** and **recreational drug** use

- A history of **family** members with cardiac & neurological conditions, similar episodes, and **unexplained deaths** (including drownings and unusual road traffic accidents)

Differentiating Fits from Faints:
Examination

Physical examination should always include detailed **neurological-, cardiac-, and vestibular** evaluations.

Recognising Seizures:
When it's easy, it's easy

Recognition of epileptic seizures is often straightforward, except in cases where eyewitness accounts are lacking and patients have no recollection of events.

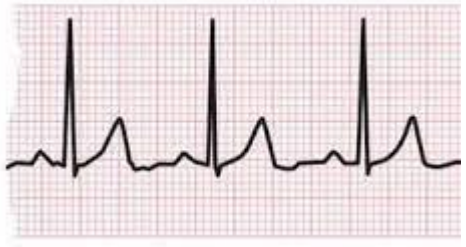
In **focal seizures with altered awareness and generalised seizures**, the following features may be helpful to confirm a fit

- ***An aura***
- ***Lateral version of the eyes and head***
- ***En guard positioning of head and upper limbs***
- ***A tonic phase and prolonged bisynchronous rhythmic jerking***
- ***Prolonged loss of consciousness (approx. 60 seconds)***
- ***Severe biting of the lateral aspect of the tongue***
- ***Post ictal confusion***

Keep in mind that virtually all of these have also been described with syncope

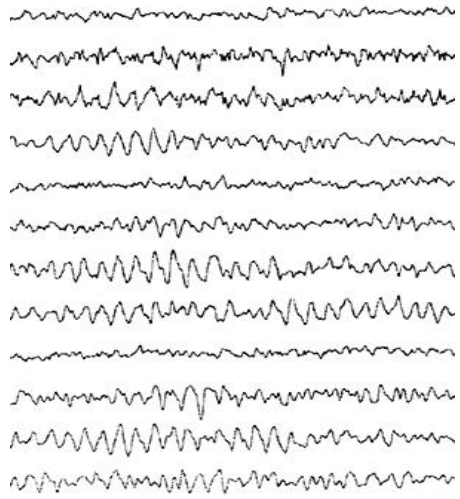
Differentiating Fits from Faints:
Investigations

Where these is still uncertainty after a careful history & examination, **first line investigations** should always include an **ECG and standard EEG**.



ECG:

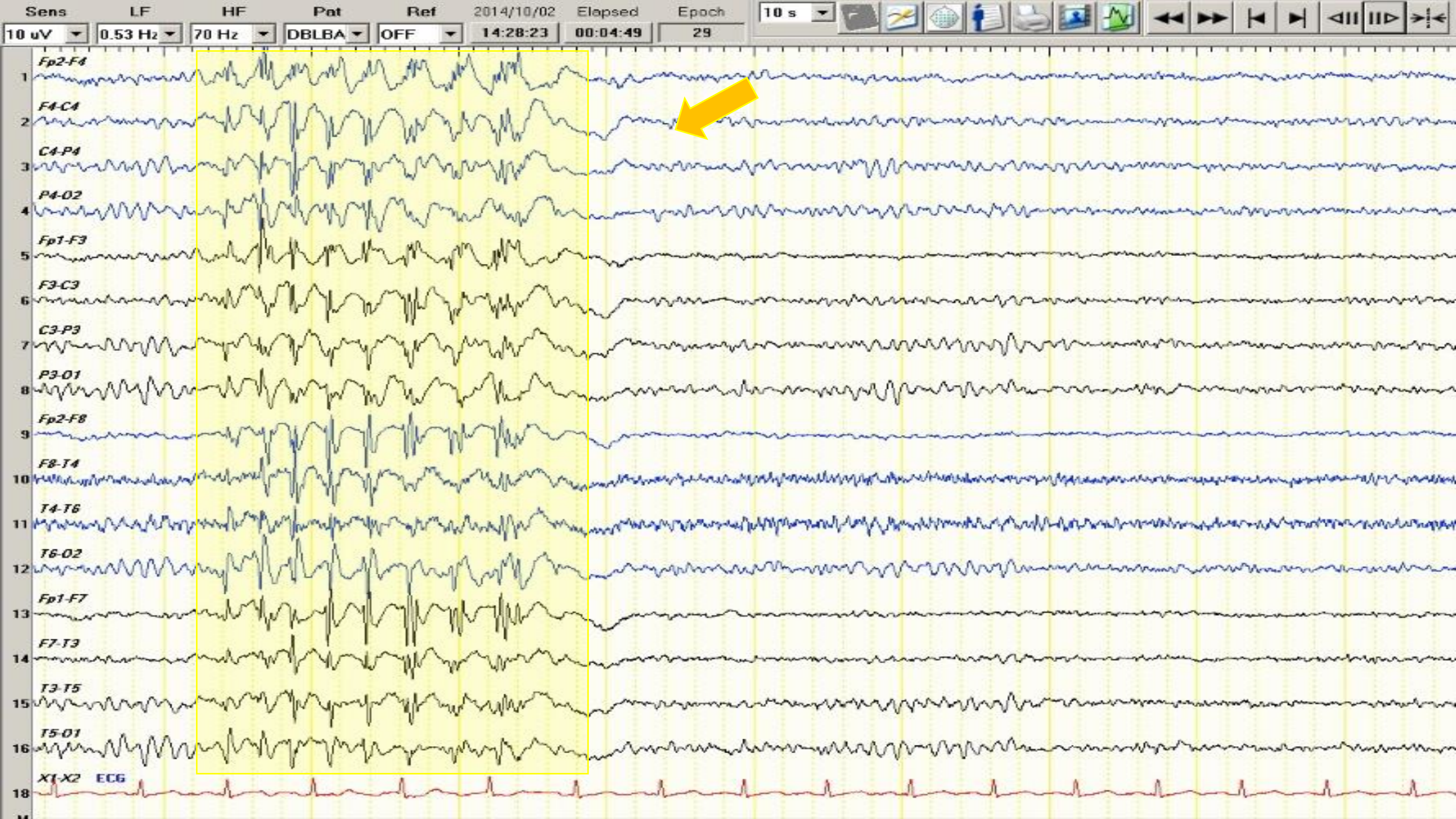
- Cardiac dysrrhymias?



EEG:

- Epileptiform activity strongly supports the diagnosis of Epilepsy

- Distinguish between Focal vs. Generalised epileptiform discharges
  - If focal, consider brain imaging
  - Influences choice of ASM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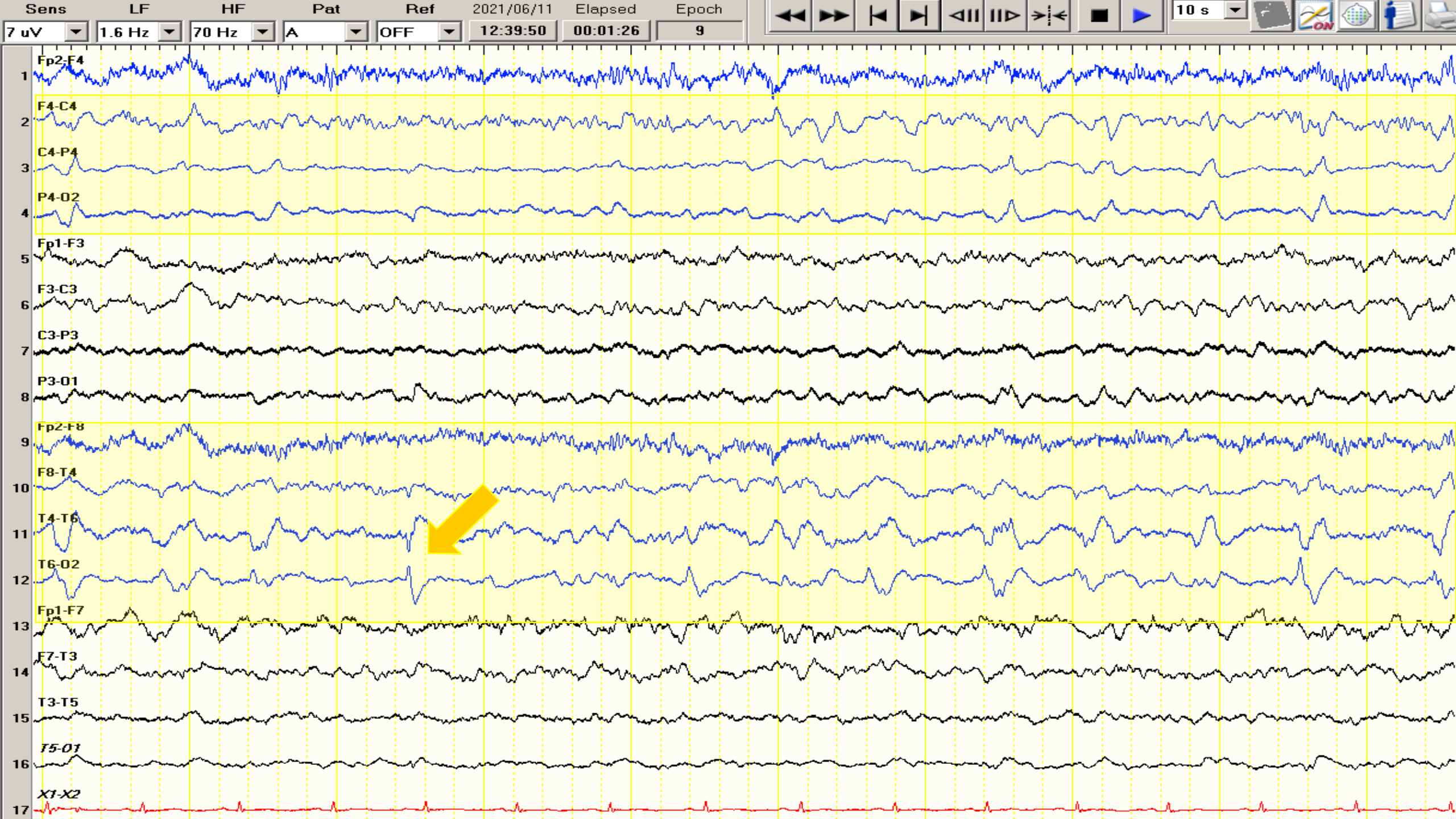
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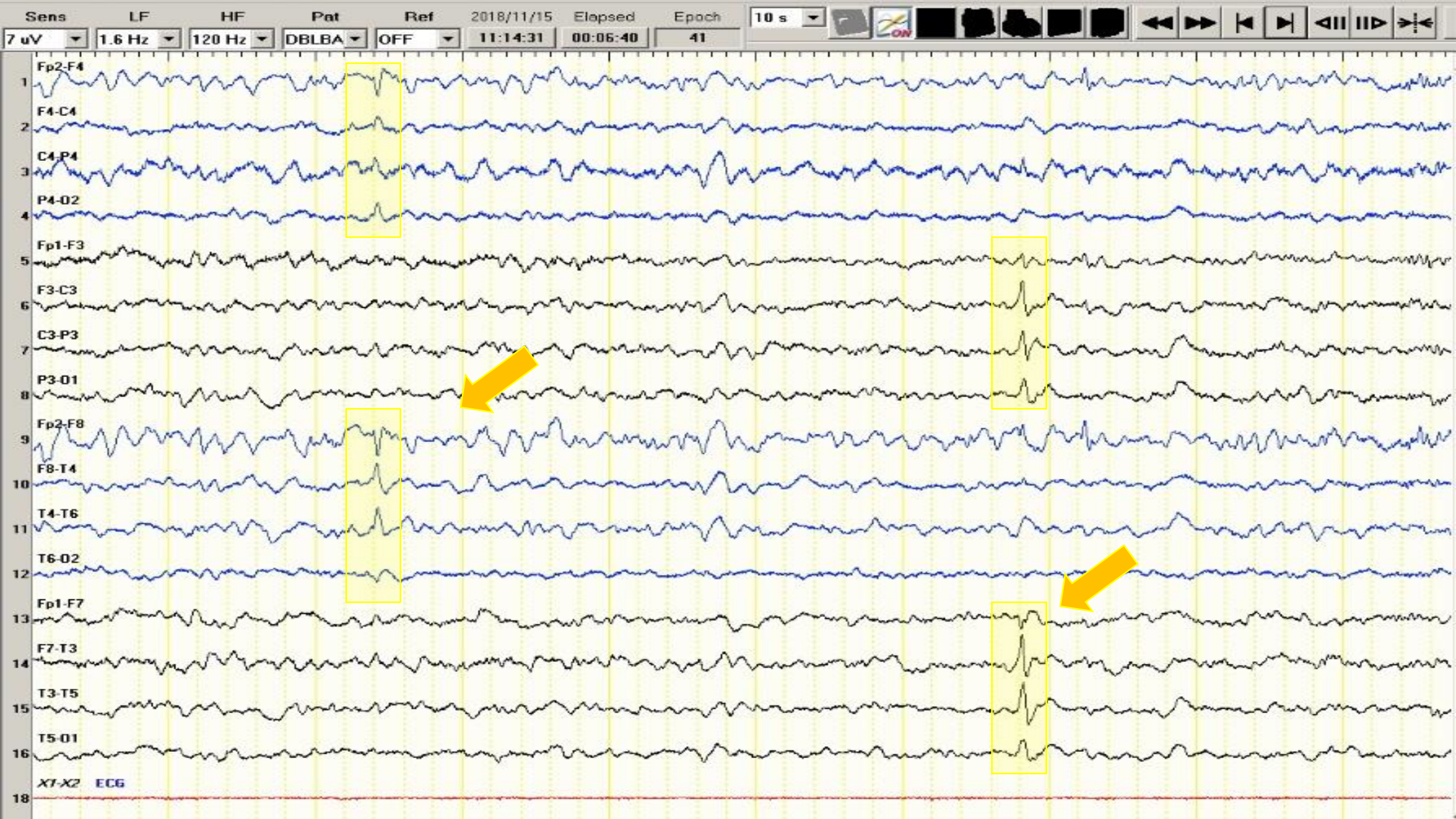
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T5-O1
X1-X2
ECG

Differentiating Fits from Faints:
Investigations

Additional investigations should be carefully considered on a case-by-case basis and strictly driven by the most likely aetiology:

Where a seizure is suspected:

- Long term video-EEG
- Brain imaging (MRI, CT, CTA, CTV)

Where syncope is suspected

- Video-tilt-table test
- ECG monitoring for arrhymias
- Cardiac ultrasound

A "shotgun" approach is expensive, generally unhelpful and false positive result are common

Functional seizures (PNES)

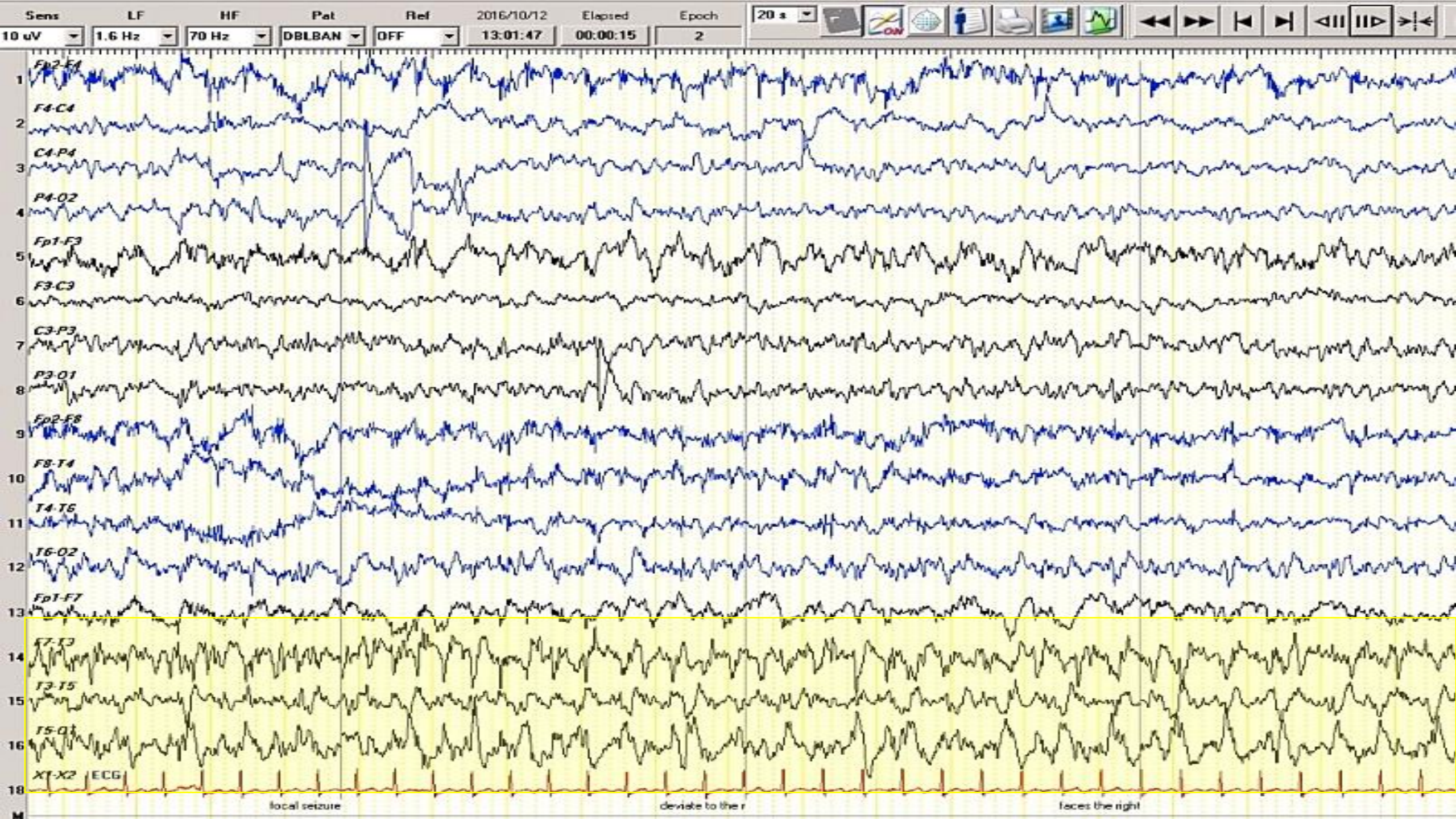
In tertiary epilepsy clinics, up to 20% of all patients might have functional seizure.

Some clinical clues:

- ***Episodes are often prolonged, and may last hours, with many events on one day,***
- ***movements wax and wane in severity and typically change in nature,***
- ***Commonly there are alternating movements of the limbs, ('arc de cercle') and pelvic thrusting are common***
- ***Eyes are almost always closed, often with forced eye closure***
- ***Tongue biting, and traumatic injury may occur but are seldom serious and***
- ***Incontinence is rare but does occur***

The gold standard for diagnosis of PNES is a video-EEG recording of an attack which demonstrates absence of electrographic ictal activity

https//Neurosymptoms.org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ECG
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deviate to the r
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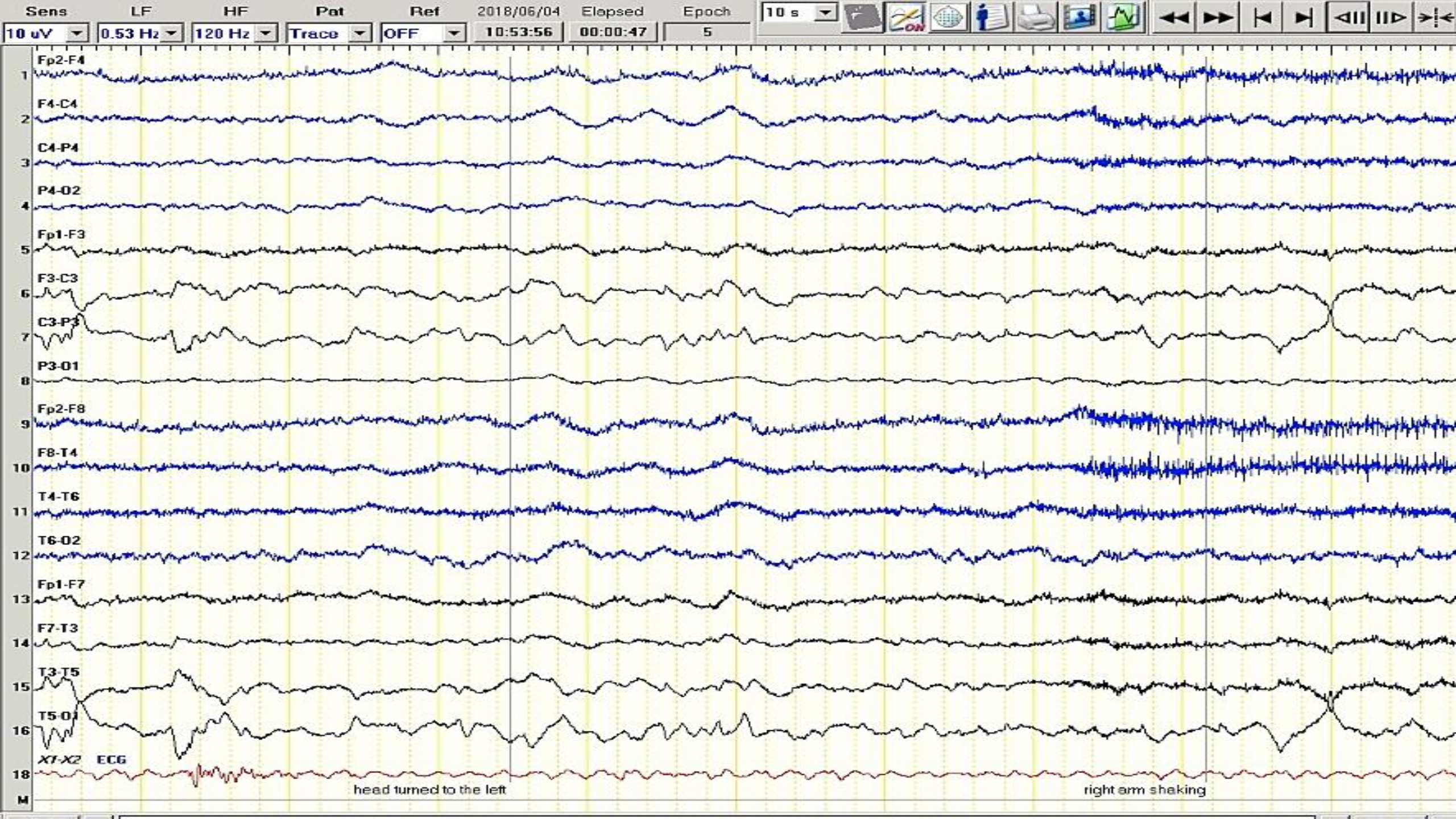
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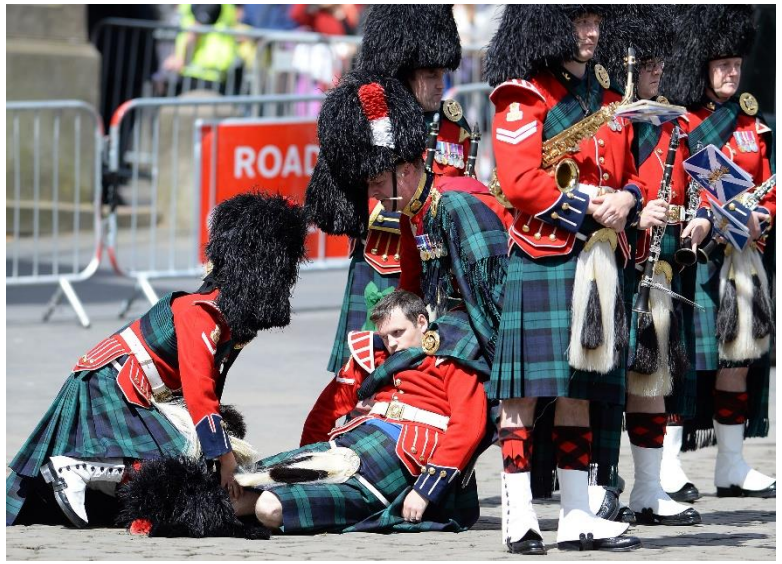
T5-O1
X1-X2
ECG
head turned to the left
right arm shaking

Syncope



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News.com.au

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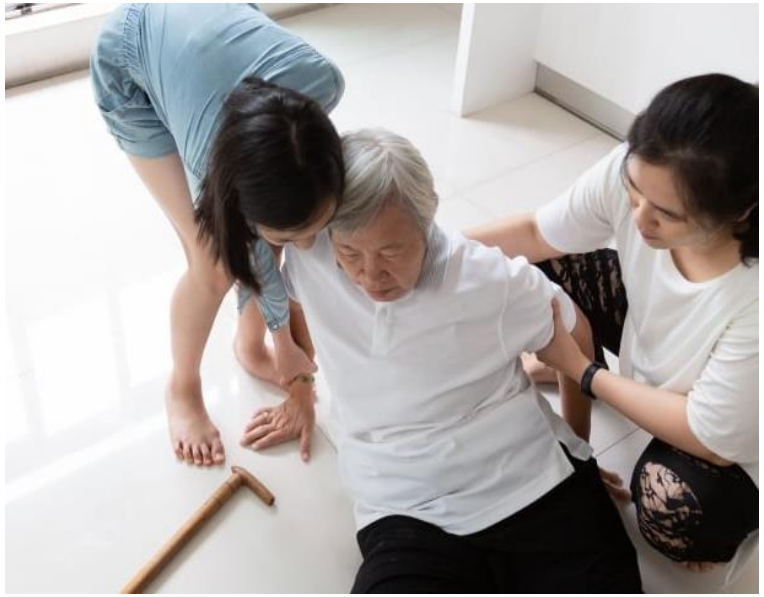
https://cbtwestport.com/

https://www.ukrunchat.co.uk/



https://www.cbc.ca/

Syncope

Sudden loss of consciousness due to hypopofusion of the brain

This **typically involves inadequate or defective baroreceptor response to a drop in blood pressure**

Importantly, **it may also be due to reduced cardiac output**

The loss of consciousness is thought to be related to reduced perfusion of the reticular activating system and / or both hemispheres

Syncope:
Clinical Features

Simultaneously with the loss of consciousness, there is an **abrupt loss of consciousness and postural muscle tone**, and the patient collapses to the ground

- **Typically :**
  - Occurs in the upright position
  - Preceded by prodromal symptoms: dizziness sweating palpitation visual symptoms
  - A rapid return to full awareness within seconds

- **Commonly Associated:**
  - Myoclonic jerks (50-70%)
  - Urinary incontinence (25%)

- **Occasionally Associated:**
  - Stiffness and transient dystonic posturing
  - Traumatic injury and tongue biting, typically related to falling

Syncope:
Clinical Features

Importantly:

- Syncope **may occur without a prodrome**, or in the seated or prone position (**especially cardiac syncope**)

- **LOC may be prolonged**, especially if the patient is kept upright during the event or if the syncope is due to a persistent cardiac dysrrhymia.

Prolonged syncope, especially when there is associated cardiac asystole, may rarely be associated with **frank clinical seizure-like activity** and/or **watershed infarcts** may be associated.

Types of Syncope

1. **Reflex** (neutrally-mediated/situational/vasovagal)
2. Orthostatic
3. Cardiogenic

or a combination of these

Distinguishing Syncope Types

It is very important to distinguish between these because they have significantly varying prognoses

While reflex syncope is typically a benign condition, orthothostatic and cardiac syncope are often associated with significant morbidity and may be life threatening.

Once again, the clues are in the history

Van Dyk et al.Nat Rev Neurol 5 438-448

Reflex (Vasovagal / Situational) Syncope

Most common form of syncope and occurs at some point in up to **40% of the population**

Generally regarded as a **benign** condition which typically occurs in otherwise normal individuals.

incidence peaks at 13-16 years of age, is relatively rare during most of adult life, although it often re-emerges in old age.

Pronounced **familial** occurrence

Grub, N Engl J Med 2005;352:1004-10

Reflex Syncope:
Mechanism

Typically associated with **central triggers** which result in an ill-understood excessive reflex in the brain causing **massive parasympathetic output**

Triggers typically involve **discomfort, often with emotional element** although these may not be severe

- claustrophobia,
- Heat
- Pain
- Fear
- Shocking news
- a bout of coughing or a sneeze
- A neck movement or tight collar (baro-receptor hypersensitivity)

Grub, N Engl J Med 2005;352:1004-10

Reflex Syncope:
Clinical Features

Symptoms related to excessive parasympathetic activity predominate

Prodrome:

- light-headedness / dizziness
- blurring or darkening of peripheral vision
- Feeling of warmth / cold
- Paleness of the face
- Nausea, abdominal discomfort or urge to defecate

➢ Followed by loss of consciousness and muscle tone, and a fall to the ground if standing

➢ Myoclonic Jerking and urinary incontinence are common

➢ Posturing may occur

Average duration of loss of consciousness is 12 sec.

Reflex Syncope:
Clinical Features

Patients with Reflex Syncope typically experience **pronounced autonomic symptoms pre- and post syncope**,

Although they may recover awareness rapidly after a few seconds, they typically feel awful and washed out, often for hours after the event.

In contrast, patients with more sinister cardiac-related syncope, typically feel normal as soon as they regain awareness.

Postural Hypotension and Orthostatic Syncope:

https://rarehistoricalphotos.com/

Postural Hypotension:
Definition and Pathophysiology

Definition:
A fall in blood pressure of >20 mm Hg systolic, or > 10 mm Hg diastolic, on standing or during 60 deg head-up tilt

Results from adequacy or dysfunction of the Baro-reflex, autonomic system and venous return mechanisms to maintain cerebral perfusion in the upright position

It may be asymptomatic, or it may cause pre-syncope or syncope

Hainsworth, R. Clin. Auton. Res. 14 (Suppl. 1), 18-24 (2004).

https://croi.ie/heart/high-blood-pressure/

Postural Hypotension:
Physiology of Standing

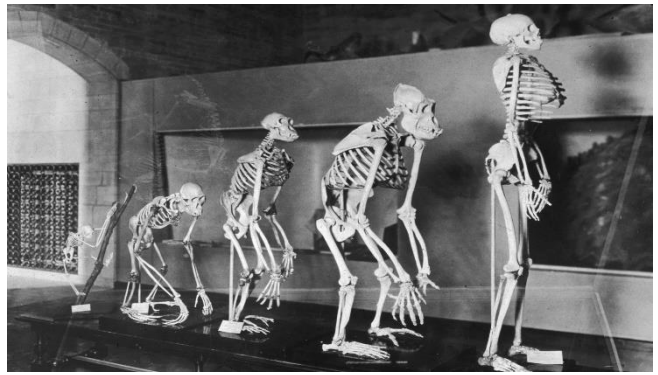
Homo Sapiens has not been upright for long

We have a **total blood volume of approx. 5000 ml** , of which **20% peruses the brain**

When we stand up, **blood pools into the lower limbs** removing between of 500-750 ml from the circulation

The **baroreflex typically accommodates** for this by reducing vagal tone and increasing sympathetic tone to shunt blood from non-vital organs such as the skin and kidneys, and maintain perfusion to the brain.

Hainsworth, R. Pathophysiology of syncope. Clin. Auton. Res. 14 (Suppl. 1), 18-24 (2004).
Gilani et al BMJ 2021;373:n922



https://www.newsweek.com/

Postural Hypotension:
Physiology of Standing

Also, after standing for approximately 30 min, **10% our plasma volume diffuses into the peripheries**

We normally accommodate for this with venous return by **contraction of the leg muscles**

But not if our knees are locked!

Hainsworth, R. Pathophysiology of syncope. Clin. Auton. Res. 14 (Suppl. 1), 18-24 (2004). Gilani et al BMJ 2021;373:n922

https://www.magzter.com/

Does Postural Hypotension Matter?

Yes!

Increased risk of:

- **falls** (odds ratio 1.73, 95% confidence interval 1.50 to 1.991),
- **heart failure** (hazard ratio 1.34, 95% CI 1.17 to 1.5219),
- **coronary heart disease** (hazard ratio 1.44, 1.18 to 1.7519),
- **stroke** (hazard ratio 1.64, 1.13 to 2.372),
- **atrial fibrillation** (hazard ratio 1.51, 1.28 to 1.7919),
- **all-cause mortality** (relative risk 1.50, 1.24 to 1.812)

Small studies also point to an increased risk of **cognitive impairment, dementia, and depression**.

Mol, et al. J Am Med Dir Assoc 2019;20:589-597; Ricci et al. et al. Eur Heart J 2015;36:1609-17. Min, et al. J Clin Hypertens 2019;21:1221-7, Cooke , et al. QJM 2009;102:335-9.

Orthostatic Syncope

Orthostatic syncope refers to syncope resulting from a postural hypotension

Affects about 20% of adults over the age of 60 years

It consistently occurs when the mean cerebral blood pressure falls to around 40 mmHg.

A fall in blood pressure usually occurs first, initially with tachycardia followed by bradycardia, which is attributable to the increased vagal activity.

Hainsworth, R. Clin. Auton. Res. 14 (Suppl. 1), 18-24 (2004).

Causes of Postural Hypotension and Orthostatic Syncope

Non-neurally mediated ***Potentially reversible***

- Hypovolaemia dehydration, diarrhoea, septicaemia, blood loss
- Medications, especially hypotensives and vasodilators

Neurally-mediated (autonomic dysfunction) ***Largely irreversible***

- primary autonomic failure'
- pure autonomic failure,
- Parkinson disease, MSA, Lewy body dementia and other parkinsonian syndromes.

'Secondary autonomic failure'

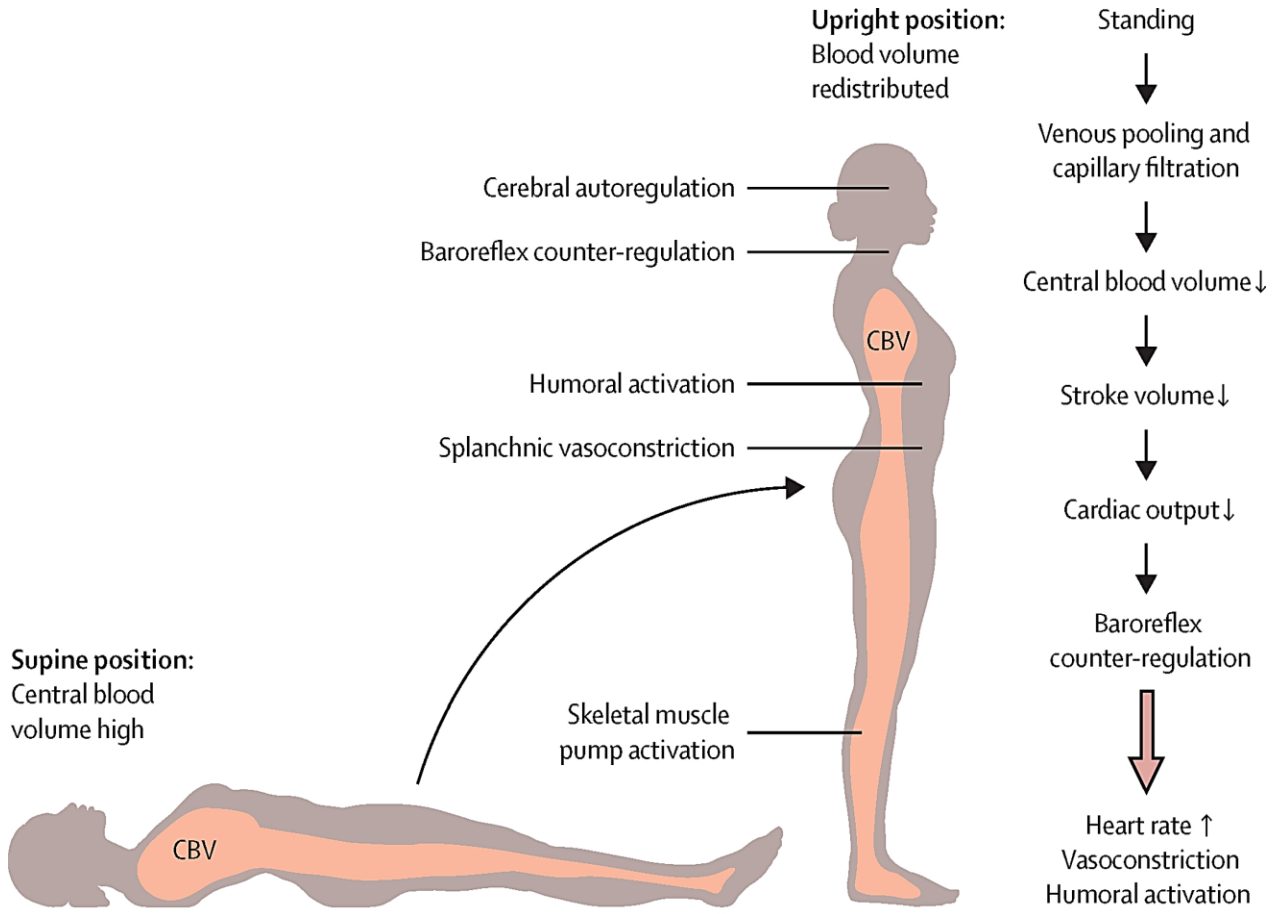
- diabetes
- Amyloidosis

Gilani et al *BMJ* 2021;373:n922 | doi: 10.1136/bmj.n922

Orthostatic Syncope:
Clinical Features

Symptoms, clinical signs and their duration are very similar to Reflex syncope

The main clue is that orthostatic syncope occurs in the **upright position**, and characteristically shortly after rising from a prone or sitting position.



Wieling, et al. 2022 Lancet Neurology V21 (8) 735-746

Orthostatic Syncope:
Making the diagnosis

Diagnosis is made on history and confirmation of persistent reduction in blood pressure of at least 20 mmHg systolic or 10mmHg diastolic within 3 minutes of standing or being upright or on the 60 degrees head-up on a tilt table test

It is advisable to take multiple measurements lying and after standing for at least 3 minutes standing

Take note of changes in heart rate when taking lying and standing blood pressure measurements.

If postural hypotension is found:

- **an accompanying increase in heart rate of >15 beats per minute may suggest a non-neurogenic cause, while**
- **an increase in heart rate of <15 beats per minute may suggest a neurogenic cause**

Freeman, et al, N. Engl. J. Med. 358, 615-624 (2008) Freeman, et al., 2011. Clin. Auton. Res. 21 (2), 69–72. Wieling, W. & Schatz, et al J. Hypertension 27, 935-938 (2009); Naschitzet al Postgrad. Med. J. 83, 568-574 (2007)

Postural Hypotension:
Management

Depends on identifying the underlying cause

Non-neurally mediated

- **Some causes of non-neurally mediated postural hypotension are reversible and correctible:**
- Dehydration, anaemia, sepsis, anti-hypertensive drugs,

Neurally-mediated

- **In general, degenerative neutrally-mediated dysautonomias are unresponsive to treatment.**
- **The aim of management is to reduce symptoms and risk of injury, not to normalise the postural fall in blood pressure.**
- There is some evidence that pharmacological intervention may reduce some of the symptoms postural hypotension, but this is weak.
    - Fludrocortizone
    - Midodroine and
    - droxidopa

Freeman et al. Hypertension 2020;75:1325-32. Myint et al. Int J Clin Pract 2013;67:633-46. Parsaik et al. J Gen Intern Med 2013;28:1496-503. Gilani et al BMJ 2021;373:n922

Postural Hypotension:
non-Pharmacological Management

Similarly the evidence for non-pharmacological interventions is also weak

Box 4: Non-pharmacological treatments for postural hypotension

- Change position slowly and in stages (from lying to sitting to standing), rather than changing from lying to standing in a swift motion
- Maintain adequate hydration
- Avoid alcohol, large meals, very warm environments, and hot showers or baths
- Sleep with the head of the bed elevated
- Exercise programmes
- Physical manoeuvres such as crossing the legs while standing and tensing the muscles in the legs and buttocks after standing
- Lower limb compression
- Abdominal binders

Gilani et al *BMJ* 2021;373:n922 | doi: 10.1136/bmj.n922

Orthostatic Syncope:
Antihypertensive Drug Use

If a patient is taking **multiple antihypertensives**, discontinuing any one of these is likely to reduce symptoms.

Different drug classes of anti-hypertensives probably confer different risk of postural hypotension

But **reports linking particular antihypertensive drugs to postural hypotension are inconsistent**.

Of note: **It may not be necessary to compromise blood pressure targets in people with postural hypotension and, in fact, uncontrolled hypertension may worsen postural hypotension**

Rivasi, et al Journal of hypertension, 2020, Vol.38 (12), p.2356-2362; Biaggioni et al. Am J Hypertens. Milazzo, et al. J Hypertens 2012

Cardiogenic Syncope:
The one not to miss!

Rarest cause of syncope has the poorest prognosis

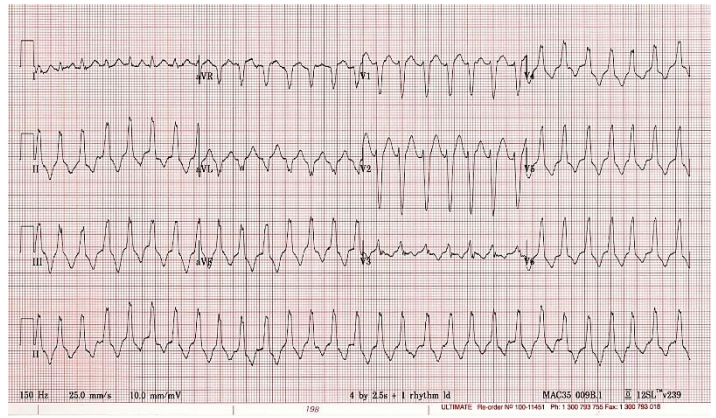
Pathophysiology: sudden reduction in cardiac output; often associated with inadequate autonomic vascular responsiveness

More common in the elderly but also seen in younger patients

Cardiogenic Syncope:
Causes

Cardiac Dysrrhymias

- **Bradycardic arrhymias (more common)**
- **Ventricular tachycardias (higher mortality)**
- Stokes-Adams
- Wolf-Parkinson White

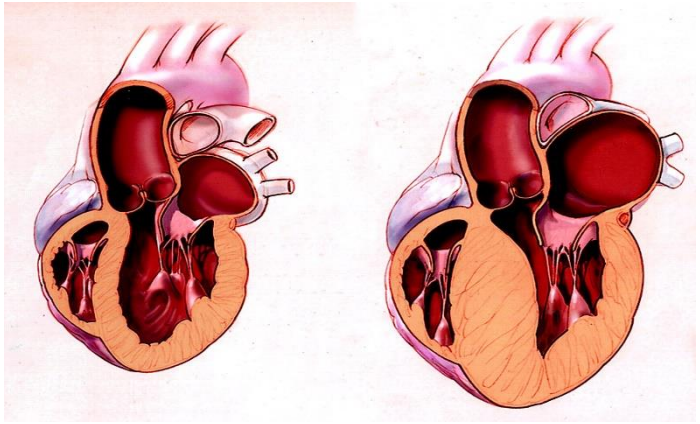


Wide complex ventricular tachycardia
https://litfl.com/

Structural Cardiac Disease

- **Valve disease** (typically aortic stenosis, mitral valve dysfunction)
- Hypertrophic obstructive cardiomyopathy (**HOCM**)
- Other causes are rare: cardiac tamponade, myocardial infarction

It is also important to consider pulmonary embolism



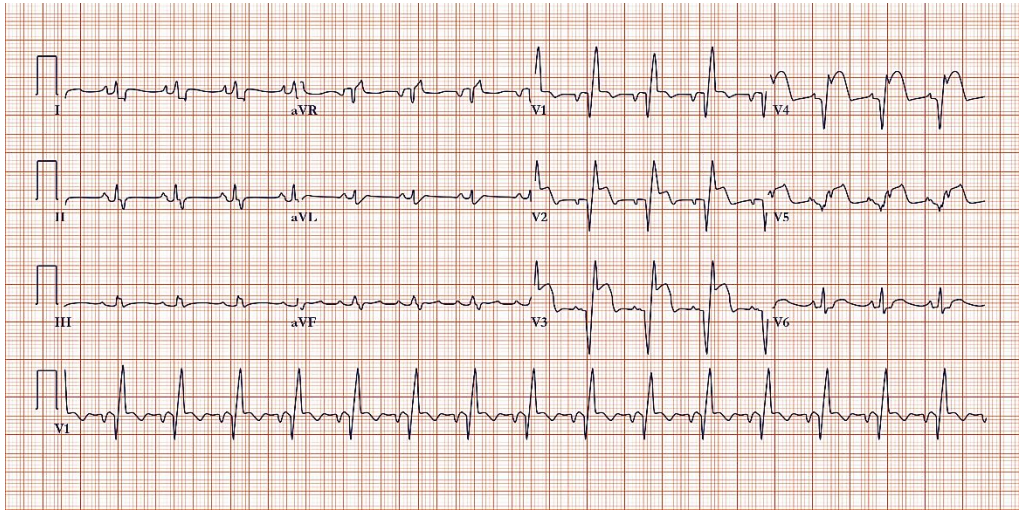
HOCM
doi/10.1161/01.CIR.0000097621.97566.96

Myocardial Infarction and Cardiac Syncope?

This is an **extremely rare cause of cardiogenic syncope**

It would require infarction of the:

- SA node, the AV node or the purkinje system, or
- A very large area of myocardium causing inadequate cardiac output



https://www.aclsmedicaltraining.com/

McDermott, et al, 2009, Vol.11 (2), p.156-160;
Rosset al, 2023, Vol.20 (5), p.S538-S539 Berg, et al, 2009, Vol.2 (1), p.8142

Cardiogenic Syncope:
Some Red Flags on History

Background:

- Fist time syncope > 35 years of age
- Family history of cardiac disease or unexplained death at a young age, including drowning unusual road traffic accidents
- Cardiac comorbidity
- Medication, usually antihypertensives and especially those which prolong ventricular repolarization (such as [beta]-adrenergic blockers, calcium channel blockers or antiarrhythmic drugs).

Albassam JAMA. 2019;321(24):2448-2457
Brain 2014: 137; 576–585

Cardiogenic Syncope:
Some Red Flags (clinical)

Typically:

- No obvious trigger
- May occur when seated or supine
- Preceding chest pain / discomfort / dyspnoea
- Little or no parasympathetic prodrome
- Rapid recovery of consciousness, with little/no post-event parasympathetic-associated symptoms
- More prolonged loss of consciousness
- Syncope which occurs during (as apposed to after) exertion

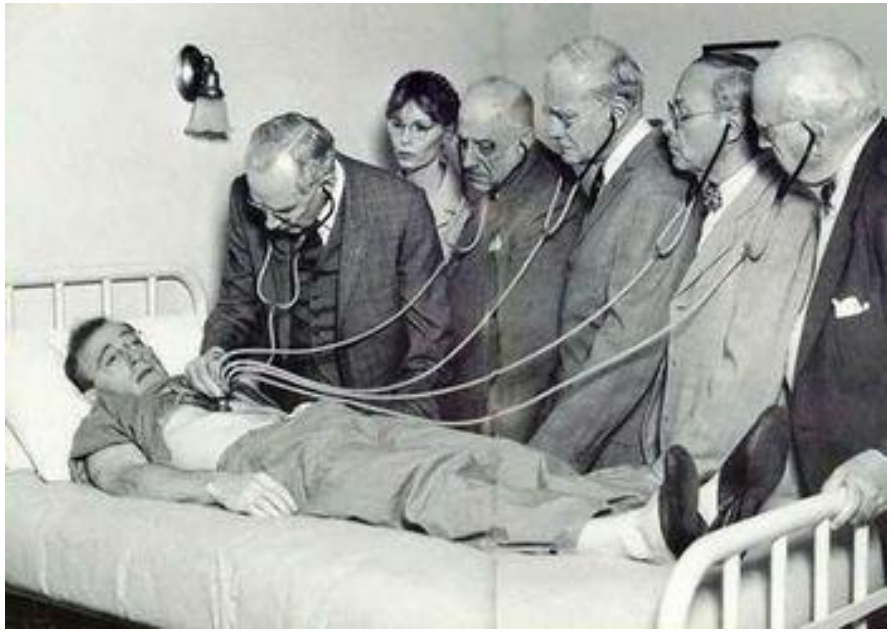
Palpitations commonly reported, but have limited diagnostic value

https://www.skysports.com/

Albassam JAMA. 2019;321(24):2448-2457

Cardiogenic Syncope:
Examination

- Cardiac dysrrhymia?
- Asymmetrical pulses?
- Abnormal cardiac auscultation?
- Cardiomegaly?
- Signs of cardiac failure?
- Postural drop in BP?



Albassam JAMA. 2019;321(24):2448-2457

https://www.healio.com/

Cardiogenic Syncope:
Investigations

ECG is obligatory!

A normal ECG is reassuring but does not exclude dysrrhymia as a cause (consider Halter)

Look carefully for any features of an unhealthy cardiac conduction system:

- Prolonged p–r interval,
- R / L bundle branch block
- Slow sinus rate
- Long Q-T interval

Brain imaging (MR & CT) and cerebral vascular studies (CTA, MRI, carotid US are typically unhelpful, and should only be requested on a case-by-case basis.

Albassam JAMA. 2019;321(24):2448-2457

Cardiogenic Syncope:
Cardiac Investigations

Cardiac investigations should be patient-specific and include a combination of:

- **Blood tests**: serum K, Mg, Ca, etc.
- **Echocardiogram** (transthoracic and/or trans-oesophageal)
- Wearable **halter-ECG monitoring**
- **Implantable ECG loop recorders**

Cardiac syncope is typically managed by the cardiologists to address its underlying aetiology

Berignole et al. International Journal of Cardiology 370 (2023) 330–337
Albassam JAMA. 2019;321(24):2448-2457

Convulsive/Ictal Syncope

i.e. Syncope Associated with Seizures

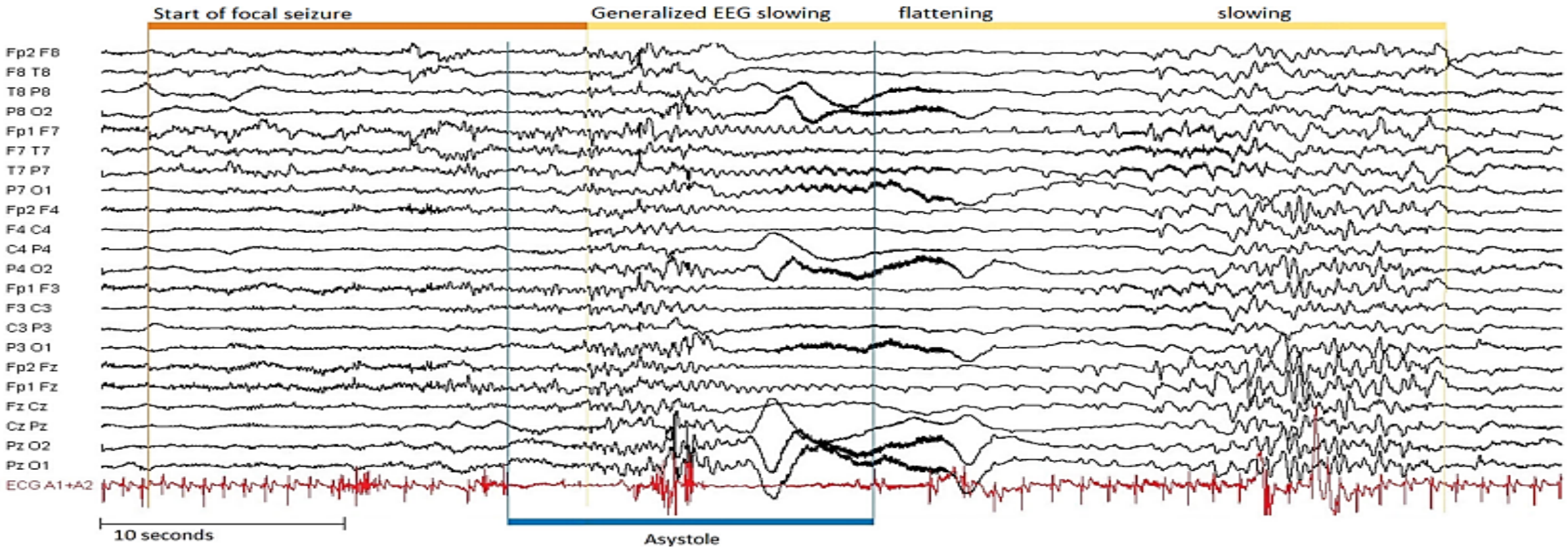


FIGURE 1 Typical EEG pattern during syncope in ictal asystole. Example of a 60 s EEG recording (filters 0.16–10 Hz, sensitivity 100 mV/cm) of a focal seizure originating in the left temporal lobe (orange bar) with ictal asystole (blue bar; duration 15 s) followed by syncope (yellow bar; duration 34 s). Syncope coincides with a slow-flat-slow pattern in the EEG (yellow bar; duration 34 s)[15,16]

Westrhenen, *et al* 2021 DOI: 10.1111/jce.15239

Convulsive/Ictal Syncope

Tachycardia is very frequent during epileptic seizures (70–90%)

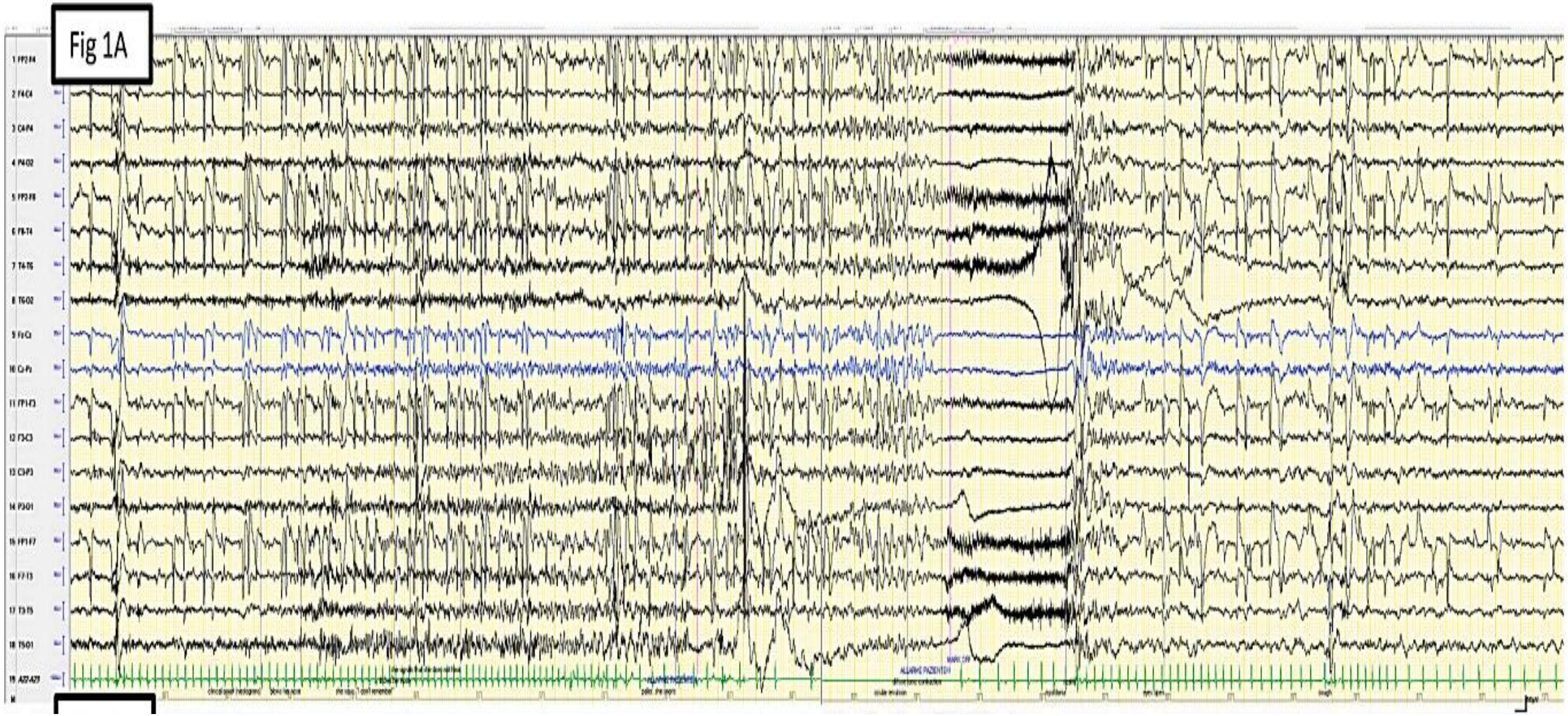
Ictal bradycardia is rare (5%)

Ictal asystole following ictal bradycardia is even less frequent (0.3-0.4%)

van der Lende et al. J Neurol Neurosurg Psychiatry 2016;87(1):69–74; Sevcencu et al Epilepsia 2010;51(5):725–37; Rocamora et al. Epilepsia 2003;44(2):179–85; Schuele et al Neurology 2007;69(5):434–41.

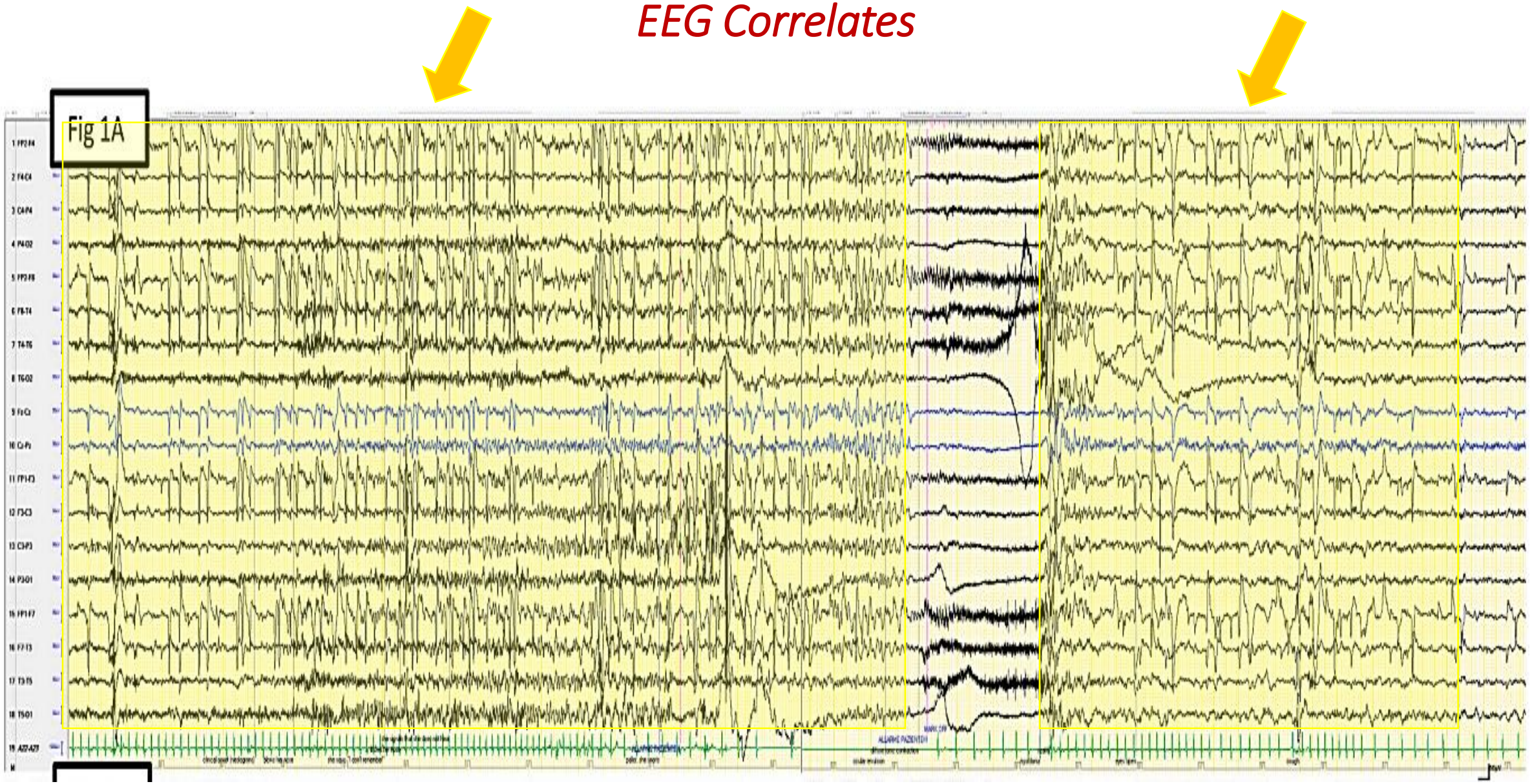
Convulsive/Ictal Syncope

EEG Correlates



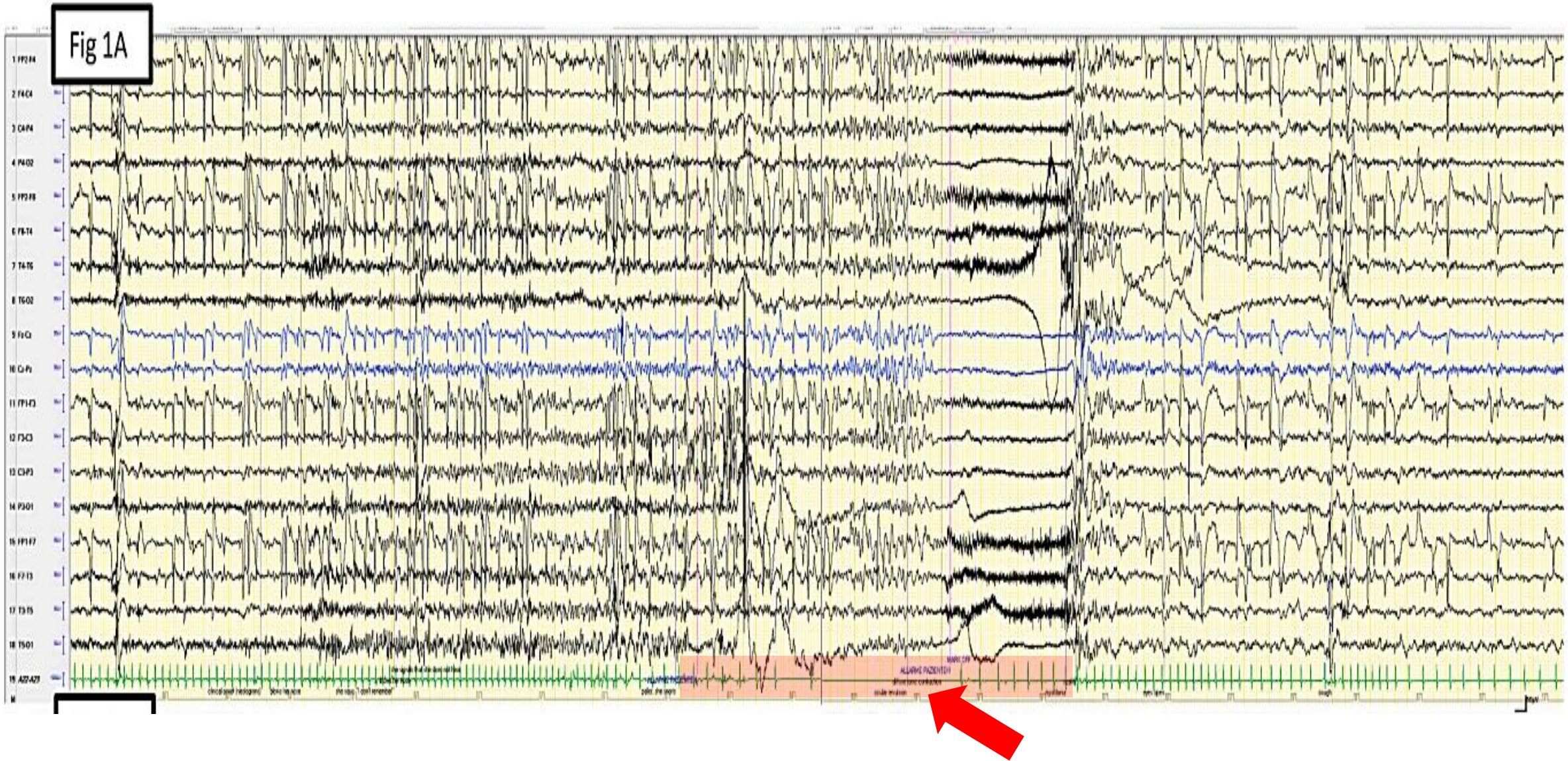
Convulsive/Ictal Syncope

EEG Correlates



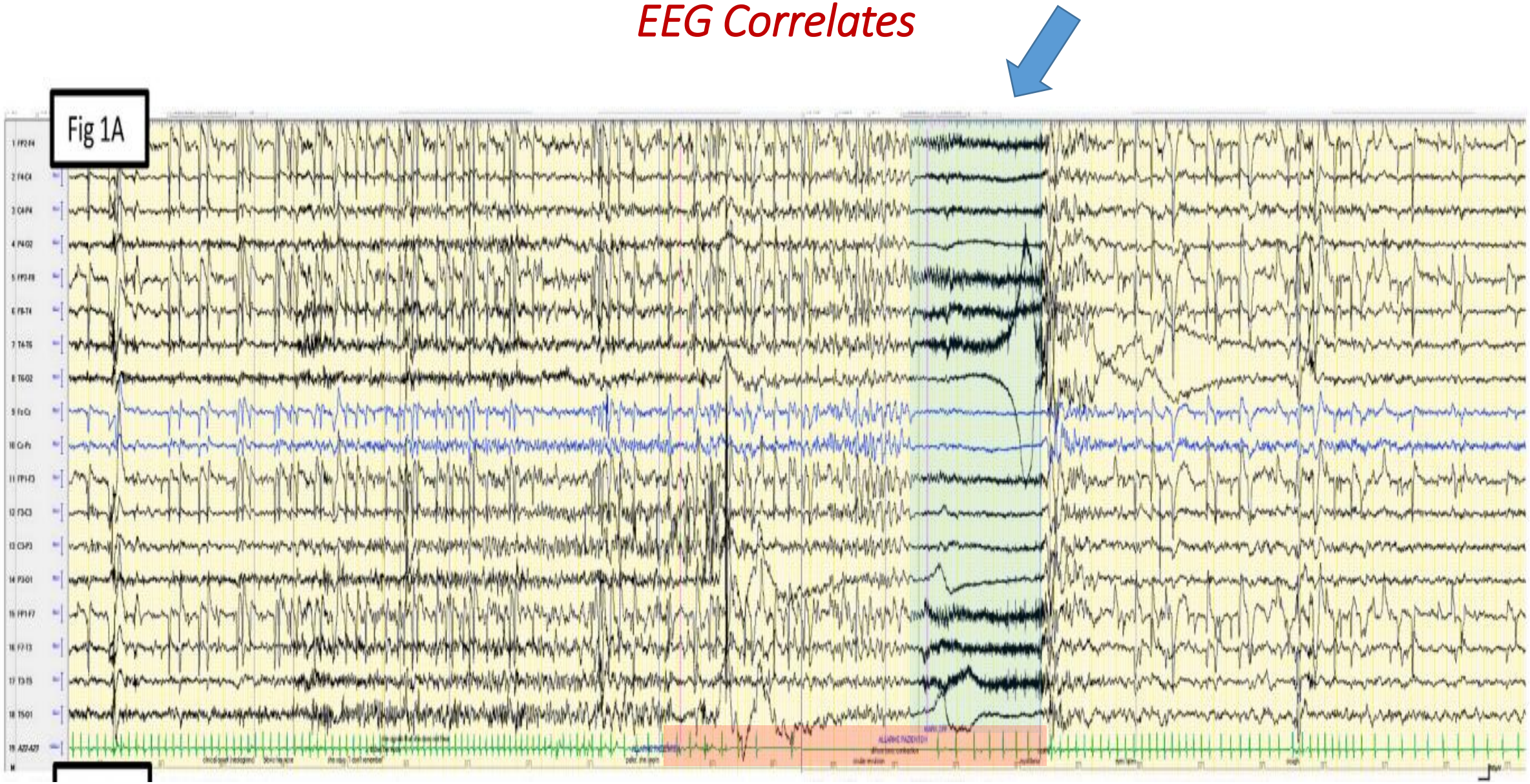
Convulsive/Ictal Syncope

EEG Correlates



Convulsive/Ictal Syncope

EEG Correlates



Ictal Asystole

Predominantly occurs during focal seizures with loss of awareness

Associated with temporal lobe epilepsy and left hemispheric lateralization (but epilepsy monitoring unit bias?)

Monté et al Epilepsy & Behavior 90 (2019) 168–171;
Sowden et al Heart Lung Circ 2021;31:25–31;
Rugg-Gunn et al Lancet. 2004;364(9452):2212–9

Ictal Asystole
Risk Factors and Associations

Risk factors include:

- focal, long-standing focal seizures
- resistance to drug therapy

Ictal asystole has rarely been described with new onset epilepsy

Statistically **associated with**:

- the duration of the seizure, and
- signs of ischemia ischaemia on the EEG

Monté et al Epilepsy & Behavior 90 (2019) 168–171
Sowden et al Heart Lung Circ 2021;31:25–31
Rugg-Gunn et al Lancet. 2004;364(9452):2212–9.

Ictal Asystole

The challenge is to distinguish seizure-related ictal bradycardia and asystole, from typical ictal seizures or cardiac syncope.

Suspect in:

- patients with a **known seizure disorder presenting with sudden falls**
- patients who present with **semiology of recurrent loss of muscle tone during seizure activity**

Ictal asystole should be considered whether or not the ECG is normal.

Giovannini et al, Epilepsy Behav Case Rep 2014;2:136–41; van Westrhenen et al. Int J Cardiol. 2019;278:147–8; Monte' et al. Epilepsy & Behavior 90 (2019) 168–171

Is there any relationship between

Ictal asystole and SUDEP?

SUDEP and Ictal Asystole

In the past, ictal asystole was considered a likely cause of SUDEP

More recently, it has been proposed that **ictal asystole may actually be protective against SUDEP,** because it has been shown to cause seizure termination on EEG, possibly on the basis of cerebral hypo perfusion

In a review of 157 cases of ictal asystole, there were no recorded deaths due to IA

There are no publications confirming ictal asystole as a cause SUDEP

Ryvlin et al. Epilepsia. 2018;59(Suppl 1):61–6]; Katz et al, Epilepsia 1983;24:248;
Sowden et al. Heart Lung Circ 2021;31:25–31; Tenyi et al. Epilepsia. 2017;58(3):356–62;
Brain Res Brain Res Rev. 2005;49(3):555–65;
Paton et al, Brain Res Brain Res Rev. 2005;49(3):555–65; N Engl J Med. 2011;365(19):1801–11.].

SUDEP:
Ictal vs. Post-ictal Asystole

Importantly, the pathophysiology of ictal asystole differs from post-ictal asystole, which is seen with prolonged tonic-clonic seizures (GTCS).

Post-ictal asystole involves cardiorespiratory depression, prolonged apnoea and parasympathetically-mediated, electro-cerebral shutdown, which are regarded as more likely implicated in the pathophysiology of SUDEP

Ryvlin et al. Epilepsia. 2018;59(Suppl 1):61–6]; Katz et al, Epilepsia 1983;24:248; Sowden et al. Heart Lung Circ 2021;31:25–31; Tenyi et al. Epilepsia. 2017;58(3):356–62; et al Brain Res Brain Res Rev. 2005;49(3):555–65; Paton et al, Brain Res Brain Res Rev. 2005;49(3):555–65; N Engl J Med. 2011;365(19):1801–11.].

Confirmation of Ictal Asystole

High index of suspicion, a detailed history and examination

Diagnosis is based on simultaneous EEG-ECG MONITORING, during which asystole always occurs concurrently with diffuse flattening of the EEG activity

The **estimated short-term recurrence risk of IA is estimated at 40.4%.** Thus IA may go unnoticed during routine short-term EEG monitoring.

Consider:

- Simultaneous long-term Video-EEG/ECG capturing several seizures
- Wearable/implanted ECG monitoring devices

Hampel et al Neurology 2017;89: 785–791

Ictal Asystole:
Management

There are still **not enough data** to guide therapeutic management of IA

First line:

- **Optimise ASM therapy**
- **Evaluation for possible epilepsy surgery**

It has been suggested that **anti-seizure drugs (ASMs) with negative inotropic or pro-arrhythmic properties** (e.g. phenytoin and carbamazepine) should be avoided, although correlation of ictal asystole with a specific ASMs has not yet to be shown.

What about pacing?

Pasini et al ;Journal of the Neurological Sciences 434 (2022)

Cardiac Pacing for Ictal Asystole?

Cardiac pacing reduces falls and injuries due to seizure-induced syncope

No good evidence that pacing

- **prevents SUDEP**
- **reduces ictal asystole**

Class IIa recommendation for pacemaker implantation in patients with epilepsy associated with severe symptomatic bradycardia where seizures are resistant to anti-seizure medications.

Bianco et al, Journal of Electrocardiology 73 (2022) 76–78; Sowden N, et al, Heart Lung Circ 2021;31:25–31; Morita et al Neurology 2017;89:756–757; Giovannini G, et al, Epilepsy Behav Case Rep 2014;2:136–41;Kusumoto FM, et al. 2018 ACC/AHA/HRS guideline, J Am Coll Cardiol. 2019;74(7); van der Lende, et al JNNP 2016;87:69–74; Bestawros et al. Circ Arrhythm Electrophysiol 2015;8:159–164.;

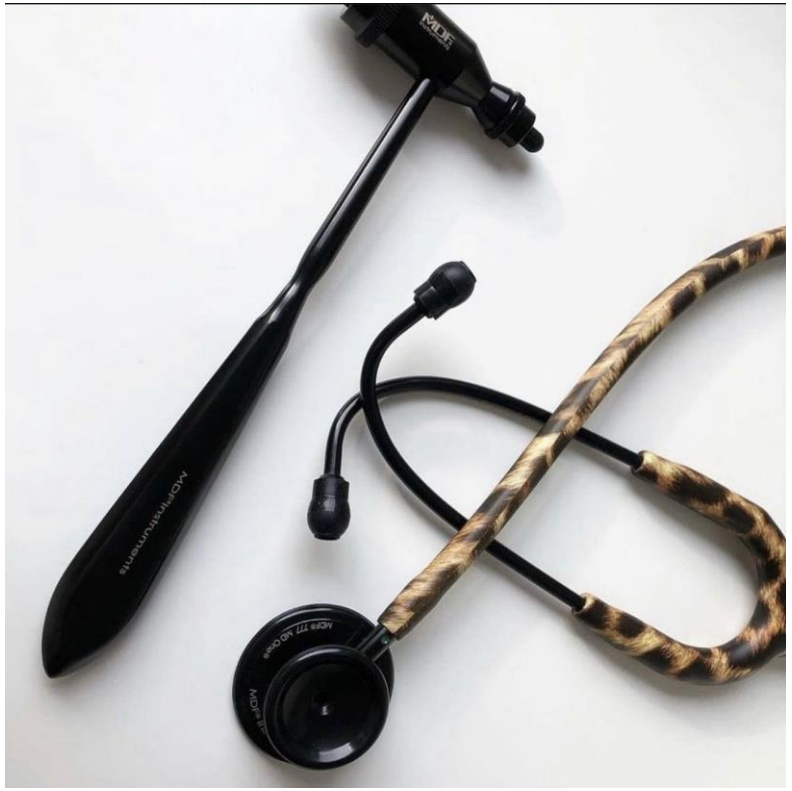
Take home messages

When assessing transient losses of consciousness, the diagnosis is almost always in the **history and clinical examination.**

An **ECG and standard EEG** should be sufficient to confirm the diagnosis in most cases.

All other investigations should be carefully considered with the most likely diagnosis in mind, and to answer a specific diagnostic question.

Close collaboration with your cardiologist colleagues may be necessary in more difficult cases



Functional Syncope

In two studies, functional attacks accounted for **6% of presumed syncope episodes**, and other studies have suggested even higher proportions.

Some clinical clues:

- ***Hypotonic***
- ***Patients may report being aware of their surroundings but unable to see or speak***
- ***Absence of jerks***
- ***No obvious trigger***
- ***May occur while the patient is lying down,***
- ***Typically prolonged, and recurs many of times in a day.***
- ***Eyes are closed during the attack, often with forced eye closure***
- ***Recovery often slow,***
- ***Weeping might occur.***

Gold standard: Attacks of pseudosyncope provoked with a TILT-TABLE TEST, in the absence of low blood pressure and/or altered heart rate

Petersen, et al. QJM 88, 209-213 (1995); van Dijk, et al. J. Cardiovasc. Electrophysiol. 19, 48-55 (2008) Benbadis, et al Epilepsy Behav. 9, 106-110 (2006).