慈 濟 大 學 醫 學 系
神 經 內 科 核 心 課 程

EPILEPSY, HEADACHE, AND VERTIGO

Epilepsy

Epileptic Disorders

Definition

Epileptic seizure:
paroxysmal and excessive electrical discharges in the brain

Epilepsy:
A tendency to recurrent epileptic seizures
A condition characterized by recurrent epileptic seizures, unprovoked by any immediate identified cause.

Epileptic syndrome:
An epileptic disorder characterized by a characteristic individual cluster of signs and symptoms customarily occurring together

ILAE OFFICIAL REPORT

A practical clinical definition of epilepsy

Epilepsy is a disease of the brain defined by any of the following conditions

1. At least two unprovoked (or reflex) seizures occurring >24 h apart

2. One unprovoked (or reflex) seizure and a probability of further seizures similar to the general recurrence risk (at least 60%) after two unprovoked seizures, occurring over the next 10 years

3. Diagnosis of an epilepsy syndrome

Epilepsy is considered to be resolved for individuals who had an age-dependent epilepsy syndrome but are now past the applicable age or those who
Have remained seizure-free for the last 10 years, with no seizure medicines for t he last 5 years.

Fisher et al, 2014, Epilepsia.

Etiology of Seizures

Acute

Stroke: ischemic (40% of venous sinus thrombosis has seizure) or hemorrhagic

Metabolic derangement: hypoglycemia, electrolyte abnormalities, renal failure

Infection: CNS infection, sepsis

Head trauma

Drugs: AED noncompliance, withdrawal from alcohol, opiates, benzodiazepines, drug toxicity

Hypoxia/cardiac arrest

Hypertensive encephalopathy (PRES)

Chronic

Epilepsy with breakthrough seizures

Mass lesion: tumor, vascular malformations

Prior cortical CNS lesion: stroke, abscess, dysplasia

Epidemiology

* The incidence of epilepsy has a bimodal distribution with a peak in the first two decades of life (genetic & congenital) and a second peak in later life, over 60 years of age (stroke & tumors)

* 2-5% of the population at age 70years will have had epileptic seizures at some point in their lives

* Most prevalence studies of active epilepsy have found rates between 5 and 10 per 1000 persons in the population

Pathophysiology of epileptogenesis

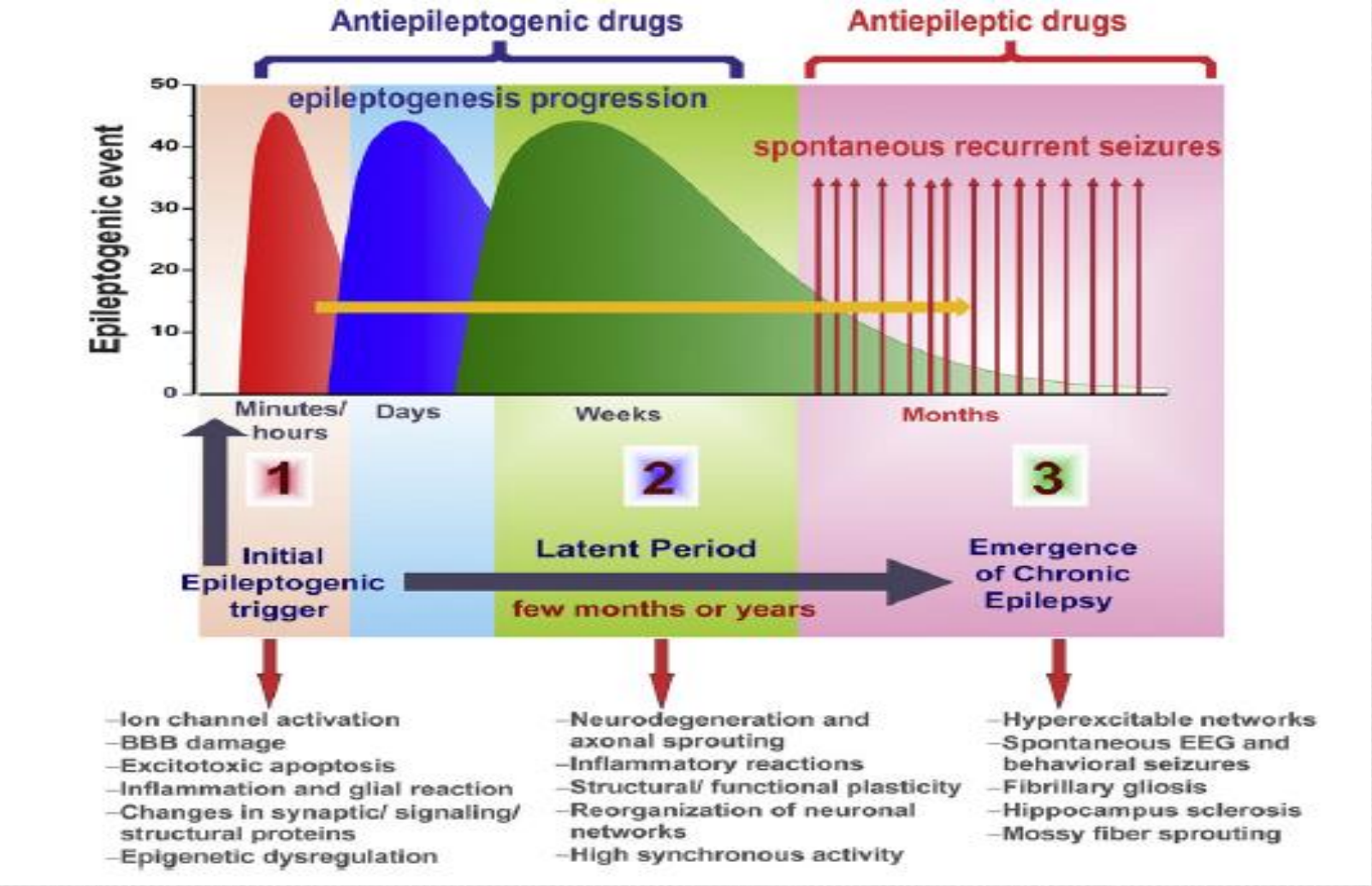
Abnormal Neuronal Excitability

Excitability of Neuronal tissue

Aberrant Neuronal Networks

Synchronization of Neuronal tissue

The process of epileptogenesis



Clossen, Reddy, 2017, Biochim Biophys Acta Mol Basis Dis.

Classification of **Epileptic Seizures**

1981 ILAE Classification

I. Partial seizures (seizures beginning locally)

A. Simple partial seizure

- consciousness not impaired

B. Complex partial seizure

- with impairment of consciousness

II. Generalized seizures

- bilaterally symmetrical and without local onset

III.Unclassified epileptic seizures

- inadequate or incomplete data

I. Partial Seizures

A. Simple partial seizures

1. with motor symptoms
2. with somatosensory or special sensory symptoms
3. with autonomic symptoms
4. with psychic symptoms

B.Complex partial seizures

1. beginning as simple partial seizures and progressing to impairment of consciousness
   a. With no other features
   b. With features as in A.1--4
   c. with automatisms
2. with impairment of consciousness at onset
   a. with no other features
   b. with features as in A. 1--4
   c. with automatisms

C. Partial seizures secondarily generalized

II. Generalized Seizures

A. Absence seizures

1. Typical absence seizures
2. Atypical absence seizures

B. Myoclonic Seizures

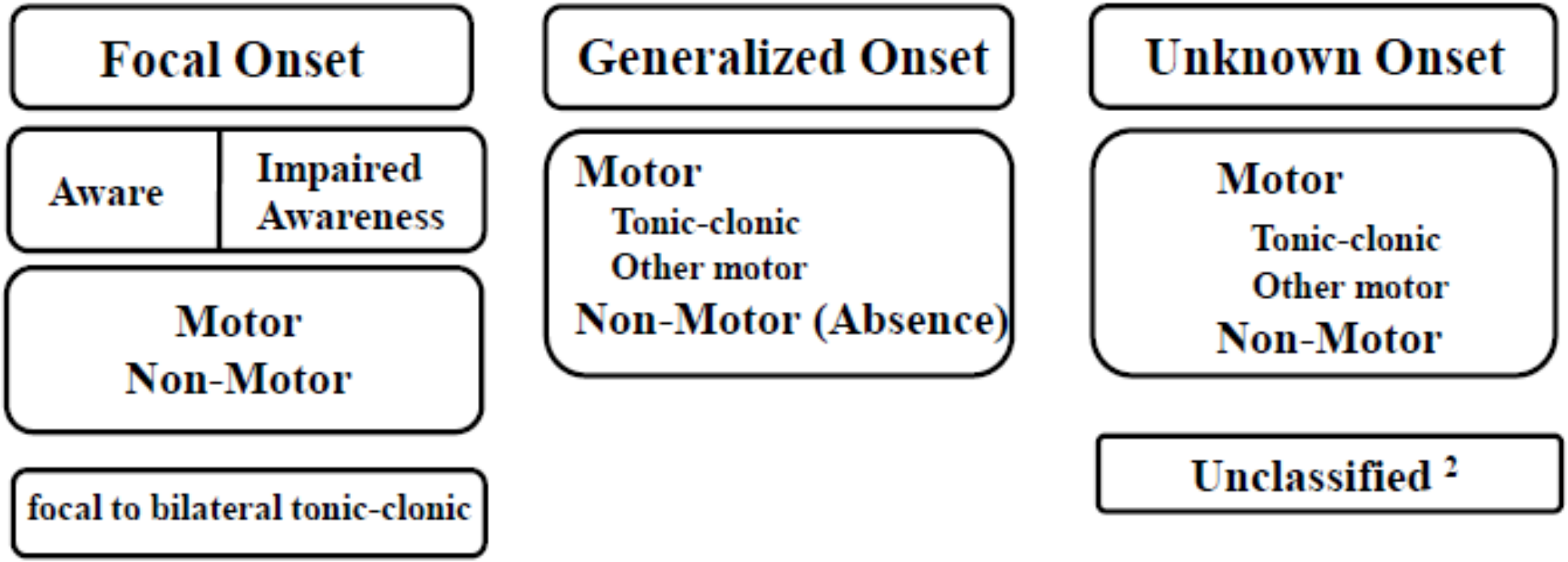
C. Clonic Seizures

D. Tonic Seizures

E. Tonic--Clonic Seizures

F. Atonic Seizures

ILAE 2017 Classification of Seizure Types Basic Version



[1] Definitions, other seizure types and descriptors are listed in the accompanying paper & glossary of terms

[2] Due to inadequate information or inability to place in other categories

From *Fisher et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia doi: 10.1111/epi.13671*

ILAE 2017 Classification of Seizure Types Expanded Version

Focal Onset

Aware	Impaired Awareness

Motor Onset
- automatisms
- atonic[2]
- clonic
- epileptic spasms[2]
- hyperkinetic
- myoclonic
- tonic

Non-Motor Onset
- autonomic
- behavior arrest
- cognitive
- emotional
- sensory

focal to bilateral tonic-clonic

Generalized Onset

Motor
- tonic-clonic
- clonic
- tonic
- myoclonic
- myoclonic-tonic-clonic
- myoclonic-atonic
- atonic
- epileptic spasms[2]

Non-Motor (absence)
- typical
- atypical
- myoclonic
- eyelid myoclonia

Unknown Onset

Motor
- tonic-clonic
- epileptic spasms

Non-Motor
- behavior arrest

Unclassified[3]

1 Definitions, other seizure types and descriptors are listed in the accompanying paper and glossary of terms.

2 These could be focal or generalized, with or without alteration of awareness

3 Due to inadequate information or inability to place in other categories

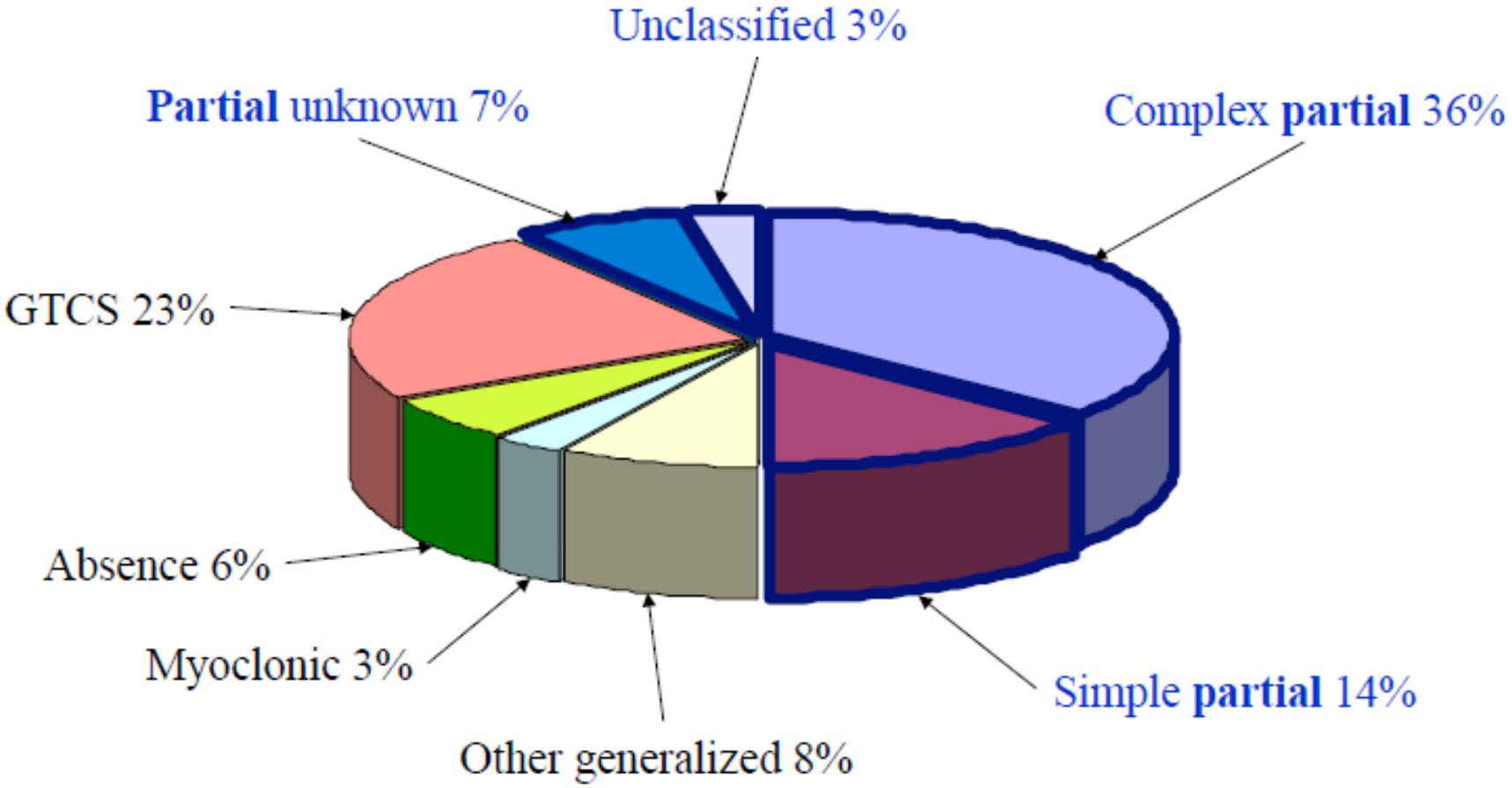
From *Fisher et al. Instruction manual for the ILAE 2017 operational classification of seizure types. Epilepsia doi: 10.1111/epi.13671*

Termiology Changes (ILAE 2017)

O L D T E R M	N E W T E R M
Unconscious (still used, not in name)	Impaired awareness (surrogate)
Partial	Focal
Simple partial	Focal aware
Complex partial	Focal impaired awareness
Dyscognitive (word discontinued)	Focal impaired awareness
Psychic	Cognitive
Secondarily generalized tonic-clonic	Focal to bilateral tonic-clonic
Arrest, freeze, pause, interruption	Behavior arrest

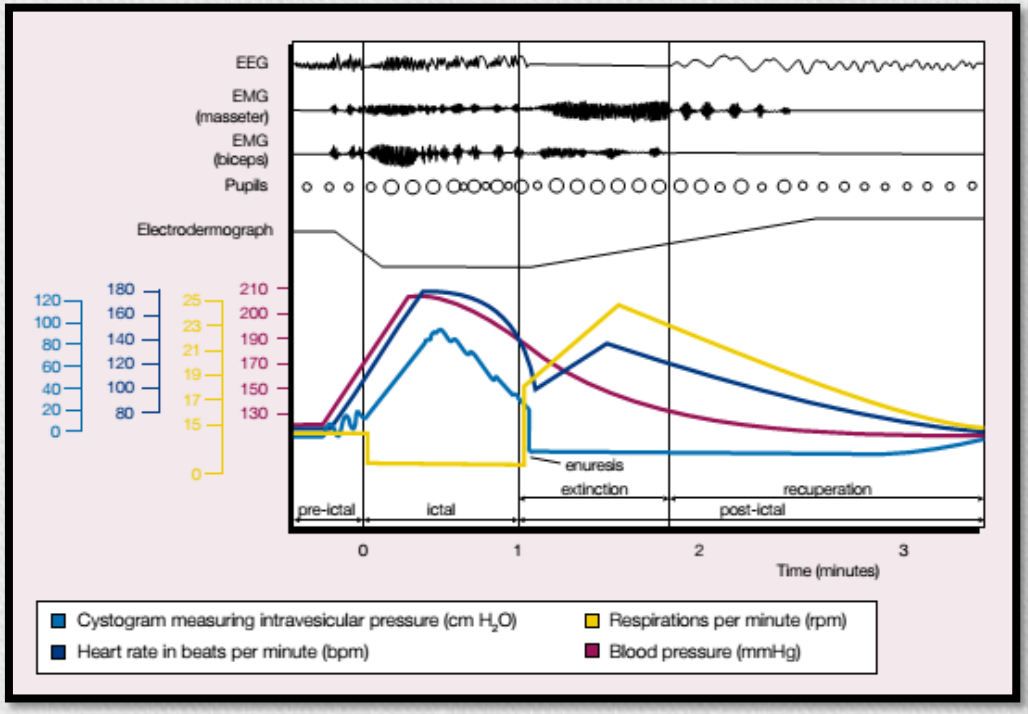
Seizure Types

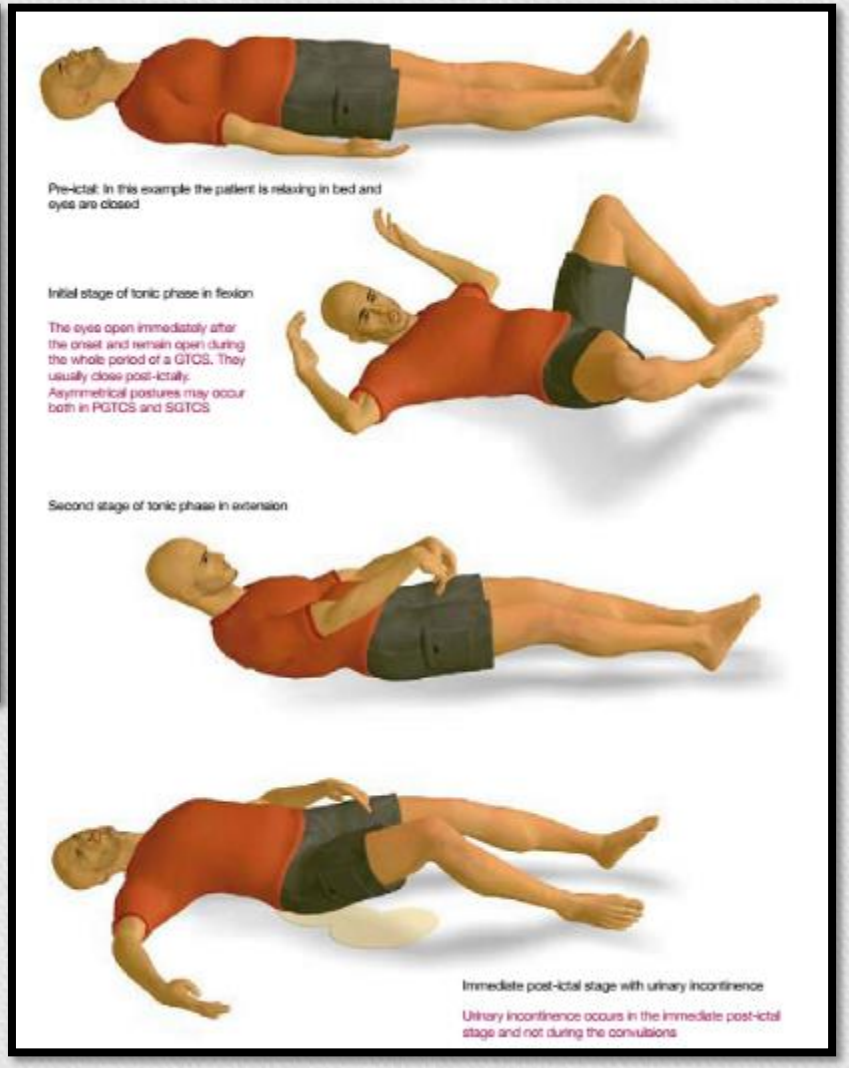
Rochester, 1935-1984



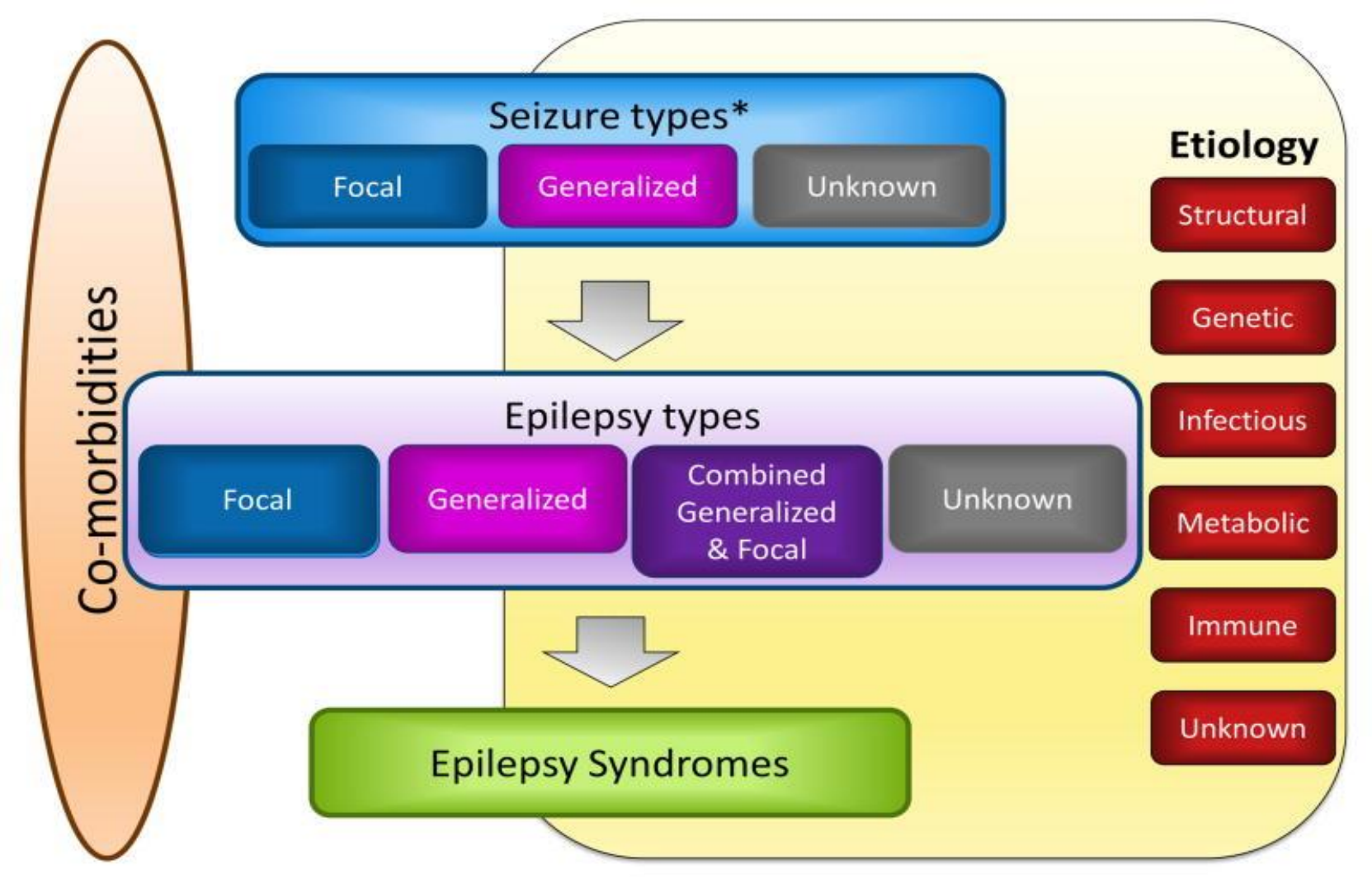
Mayo Clinic Proc 1996;71:576-86

Generalized tonic-clonic seizure (GTCS) [primary or secondary]





Framework for Classification of the Epilepsies



Scheffer et al, 2017, Epilepsia.

Modified Classification of Epileptic Syndromes

(1) Idiopathic epilepsy syndrome (focal or generalized)

e.g. childhood/juvenile absence epilepsy, juvenile myoclonic epilepsy (including generalized tonic-clonic seizures on awakening)

(2) Symptomatic epilepsy syndromes (focal or generalized)

e.g. West syndrome (infantile spasm), Lennox-Gastaut syndrome, temporal lobe epilepsy, frontal lobe epilepsy, posttraumatic epilepsy

(3) Other epilepsy syndromes of uncertain or mixed classification

e.g. febrile seizures, reflex seizures

Juvenile Myoclonic Epilepsy

(Idiopathic Generalized Epilepsy)

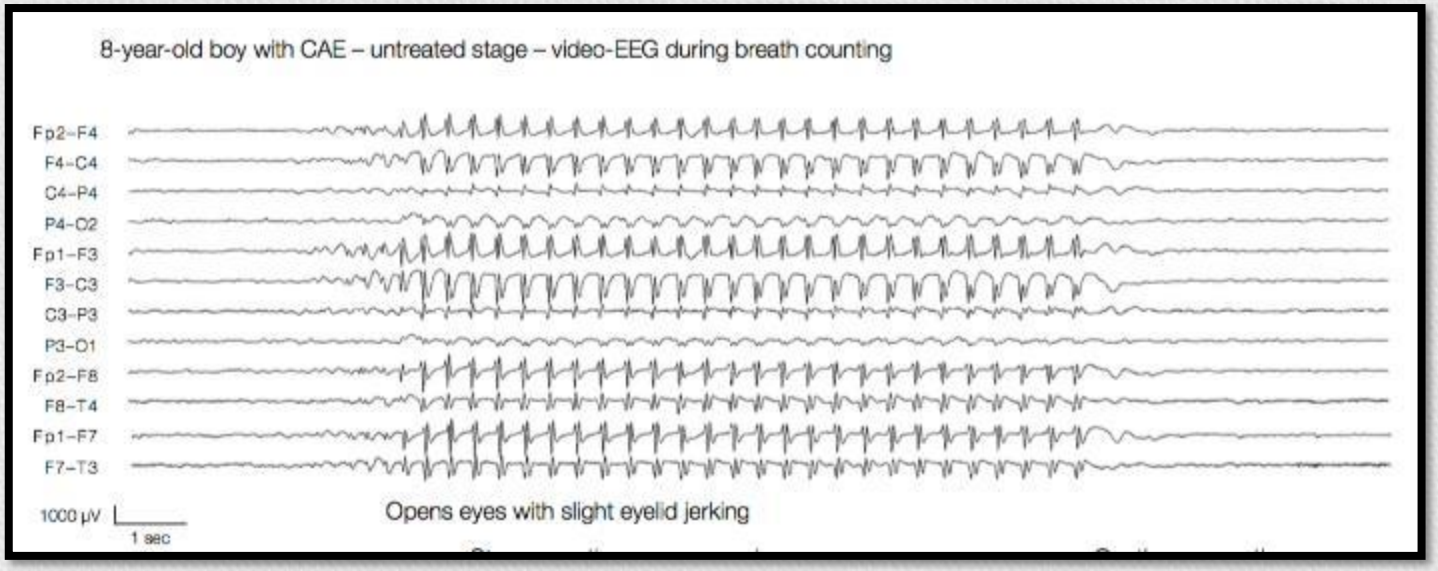
- The most common idiopathic generalized epileptic syndrome
- Age-related onset (8-20 y/o)
- Familial history of epilepsy

Clinical manifestations

- Myoclonic jerks on awakening
- Generalized tonic clonic seizures (rare)
- Typical absences (rare)
- **EEG:** 4-6Hz poly-spike-wave complexes
- **Prognosis:** life-long disorder, mostly controlled well with medication (e.g. valproate)

Childhood Absence Epilepsy (Petit Mal)

- Begins most often between the ages of 4 and 12 years
- Recurrent absence seizures (hundreds of times each day, brief: 4-20 seconds)
- EEG: stereotyped , bilateral symmetric, 3-Hz spike-wave discharge (often induced by hyperventilation)
- GTCS in 30-50% of cases (generalized tonic-clonic seizure)
- Normal in neurologically and intellectually



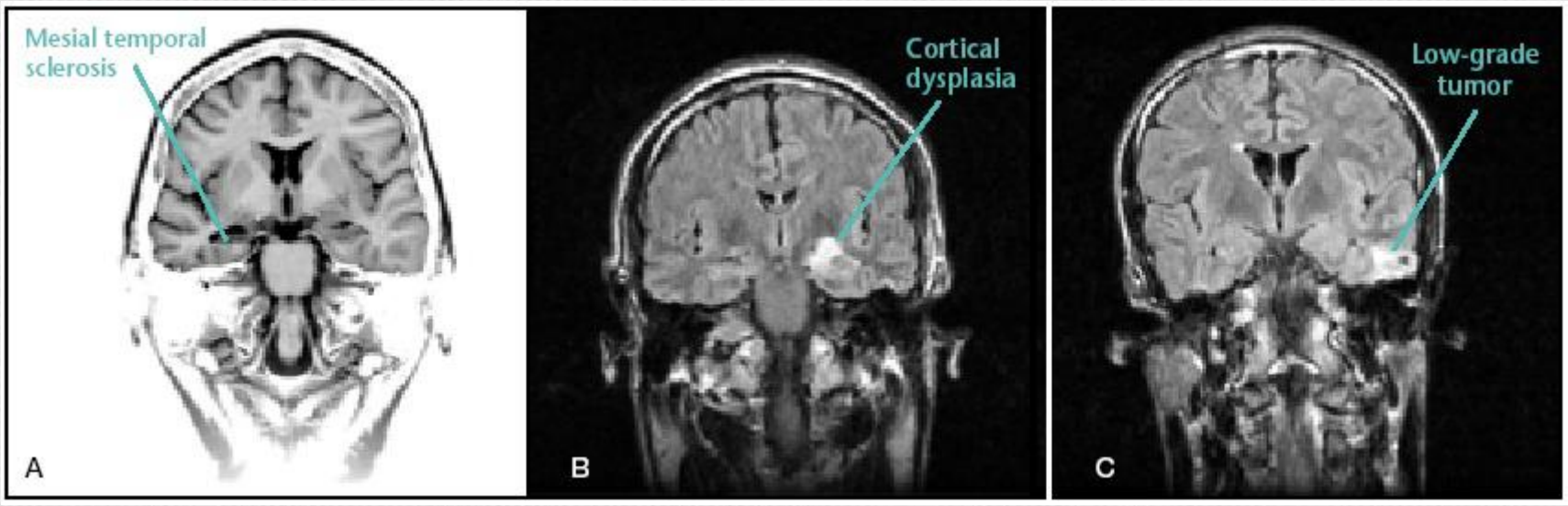
Benign Rolandic Epilepsy (benign childhood epilepsy with centrotemporal spikes (BCECTS))

- Onset: 3-13 Peak: 8-9 Stop: 14-18
- Presentation
  - Often attacks during NREM sleep
  - Hemifacial or oropharyngeal sensori-motor seizure
  - Ictal anarthria
  - Secondary generalized tonic-clonic seizure (GTC)
- Etiology
  - Probable autosomal dominant with age dependency and variable penetrance
  - 15q14
- Treatment: carbamazepine, valproate, phenytoin, etc.
- Prognosis: excellent, adult seizure < 2%

Temporal Lobe Epilepsy

(Symptomatic/probably Symptomatic Epilepsy)

- The most common localized (symptomatic) partial epilepsy of adults
- 2/3 originate from the mesial temporal lobe
- 65% of mesial TLE: hippocampal sclerosis



- History of febrile seizures
- Focal hypometabolism in PET scan
- Unilateral or bilateral temporal lobe spikes on EEG
- Onset frequently in childhood or young adulthood
- Common auras
  - Ascending epigastric aura
  - Experiential symptoms
  - Fear and panic
  - Déjà vu or jamais vu
  - Auditory hallucinations and illusions
  - Olfactory and gustatory hallucinations

Frontal Lobe epilepsy

- Depends on the specific location and spread pathway
- Brief seizures that begin and end abruptly with little or no postictal confusion
- A tendency for seizures to cluster and to occur at night
- Prominent, but often bizarre, motor manifestations (such as asynchronous thrashing or flailing of arms and legs; pedaling leg movement, pelvic thrusting; and loud, sometimes obscene, vocalization)
- Minimal abnormality on scalp EEG
- A history of status epilepticus

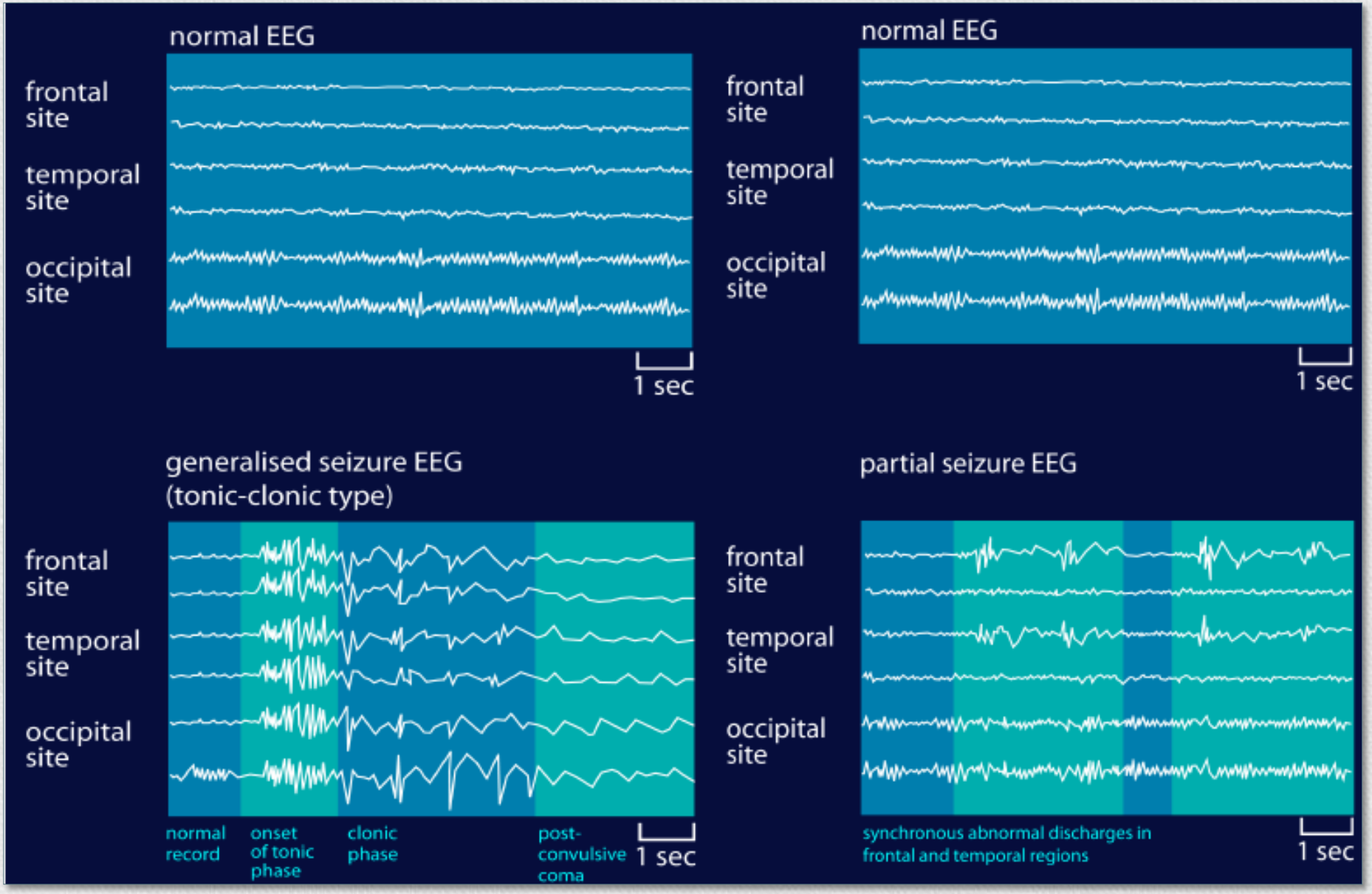
Other Epileptic Syndromes

- **Reflex Epilepsies**
  - Photosensitive seizures (most common, 75-80%): occipital lobe
  - Musicogenic seizures: temporal lobe
- **West Syndrome**
  - Infantile spasm, hypsarryhthmia in EEG, developmental delay
  - Therapy: ACTH, oral steroid, vigabatrin, ketogenic diet
- **Lennox-Gastaut Syndrome**
  - Multiple types of seizures
  - Difficult to treat with antiepileptic drugs
  - Slow spike-wave (<3Hz) in EEG
  - Poor prognosis

Diagnostic tests

Making diagnosis, Localization of epileptogenic region

- **Neurophysiological dysfunction?**
  - Electroencephalography (EEG)
    - Interictal EEG, ictal EEG (extracranial, intracranial)
  - Magnetoencephalography (MEG)
- **Structural disorder?**
  - Brain Imaging
    - Magnetic resonance imaging (MRI)
    - Computed tomography (CT)
- **Functional disorder?**
  - Brain perfusion/metabolic imaging
    - Positron emission tomography (PET)
    - Single photon emission computed tomography (SPECT)


normal EEG
frontal site
temporal site
occipital site
1 sec
normal EEG
frontal site
temporal site
occipital site
1 sec
generalised seizure EEG
(tonic-clonic type)
frontal site
temporal site
occipital site
normal record
onset of tonic phase
clonic phase
post-convulsive coma
1 sec
partial seizure EEG
frontal site
temporal site
occipital site
synchronous abnormal discharges in frontal and temporal regions
1 sec

Differential diagnosis of epilepsy

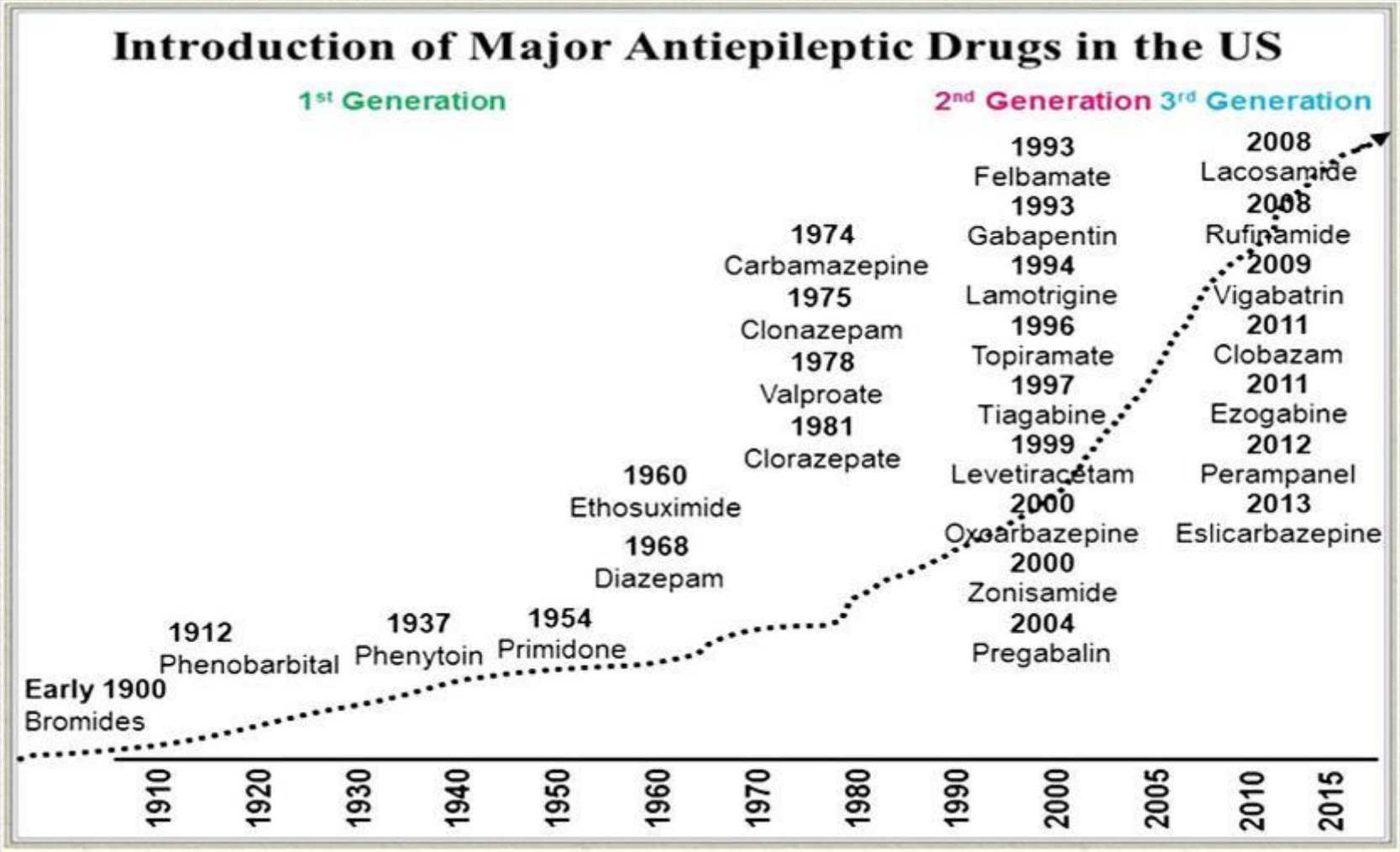
	Differential diagnosis		
	Epilepsy	**Syncope**	**Psychological causes**
Circumstance	Unpredictable	Upright	Observed
	Sleep or wake	Precipitant	Often precipitant
	Occasionally precipitant		
Prodrome/aura	Stereotypical	Presyncope	Dissociation
	Brief	Can be prolonged	Autonomic features
	Evolve		
Event	Stereotypical	Pallor	Slumping
	Brief	Variable semiology	Eyes shut
	Evolve	Brief	Thrashing
		(Non-coordinated jerking can be seen)	Directed violence
			Waxes and wanes
			Prolonged
After the event	Confusion	Unwell	Tearful

Comorbidities in Epilepsy

Somatic	Neurological	Psychiatric	Intellectual/Cognitive	Infectious/Immune	Nutritional/Dietary
Anemia	Cerebral palsy	ADHD	Down syndrome	Encephalitis	Malnutrition
Asthma	Chronic pain	Alzheimer disease	Fragile X syndrome	Glioma	Obesity
Diabetes	Migraine	Autism	Intellectual disability (MR)	Meningitis	
Fibromyalgia	Stroke	Depression	Memory loss	Neurocysticerosis	
Gastrointestinal	Rett syndrome	Schizophrenia			
	Hearing loss	Substance abuse			
	Vision loss	Suicidality			
	Sleep disorders				

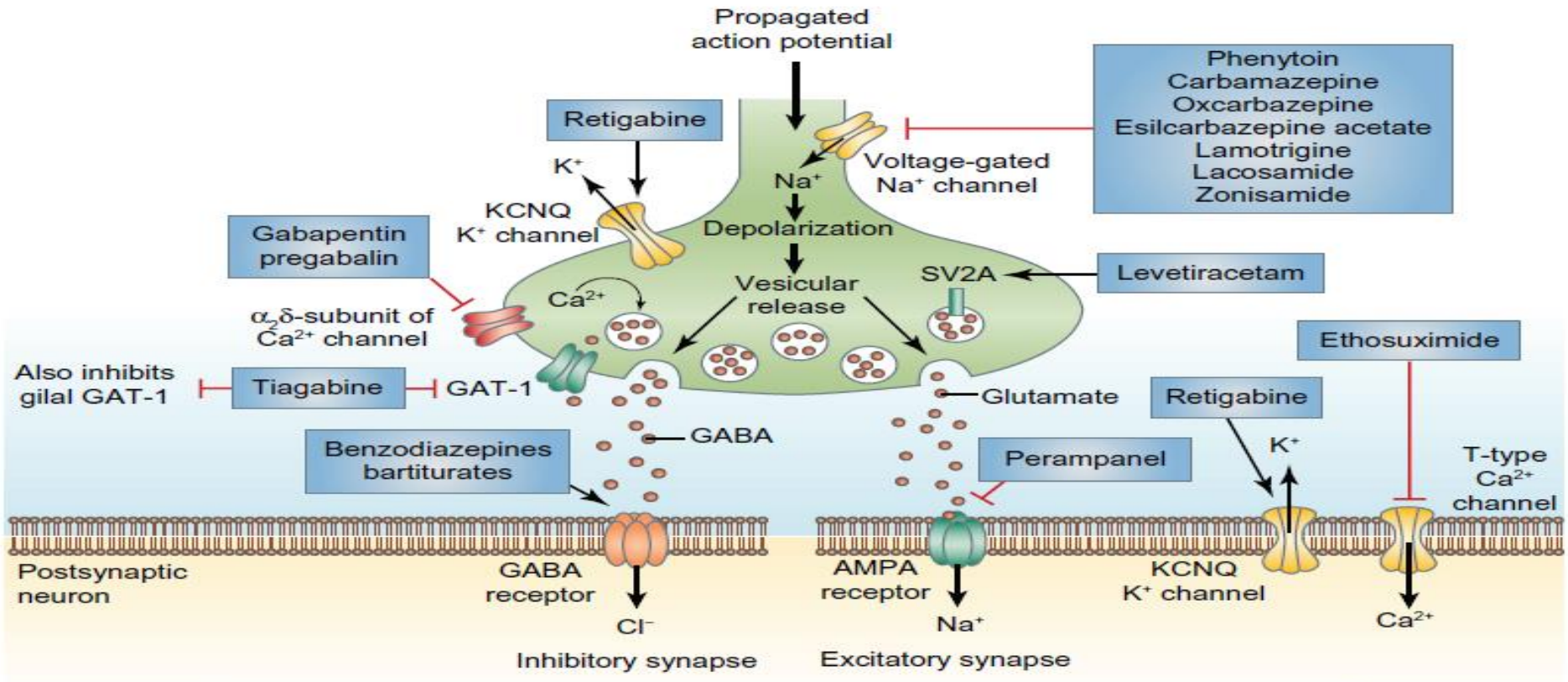
ADHD: attention deficit/hyperactivity disorder; MR: mental retardation.

Update of antiepileptic drugs development



Rudzinski et al, 2016, J Investig Med.

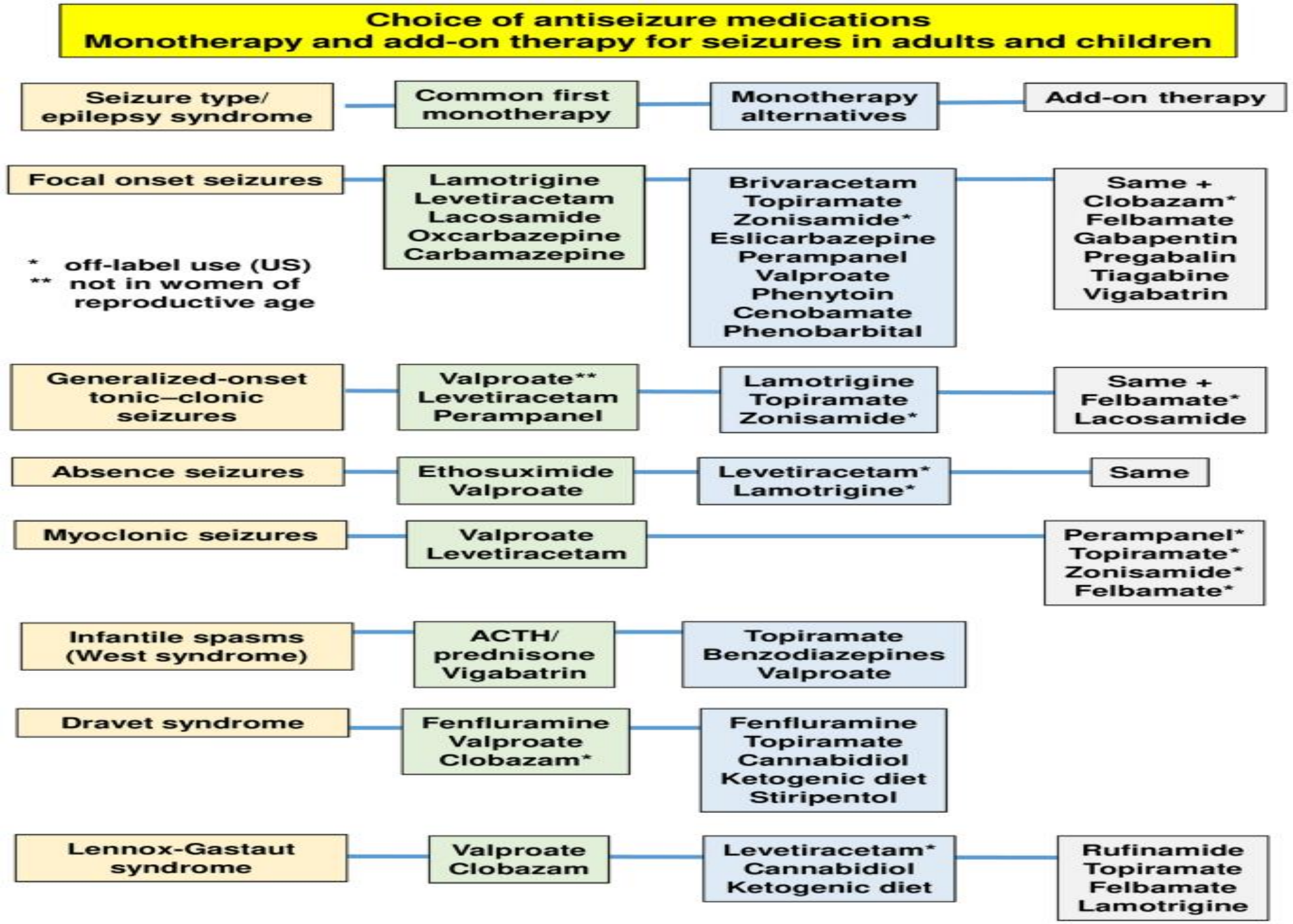
Action of antiepileptic drugs



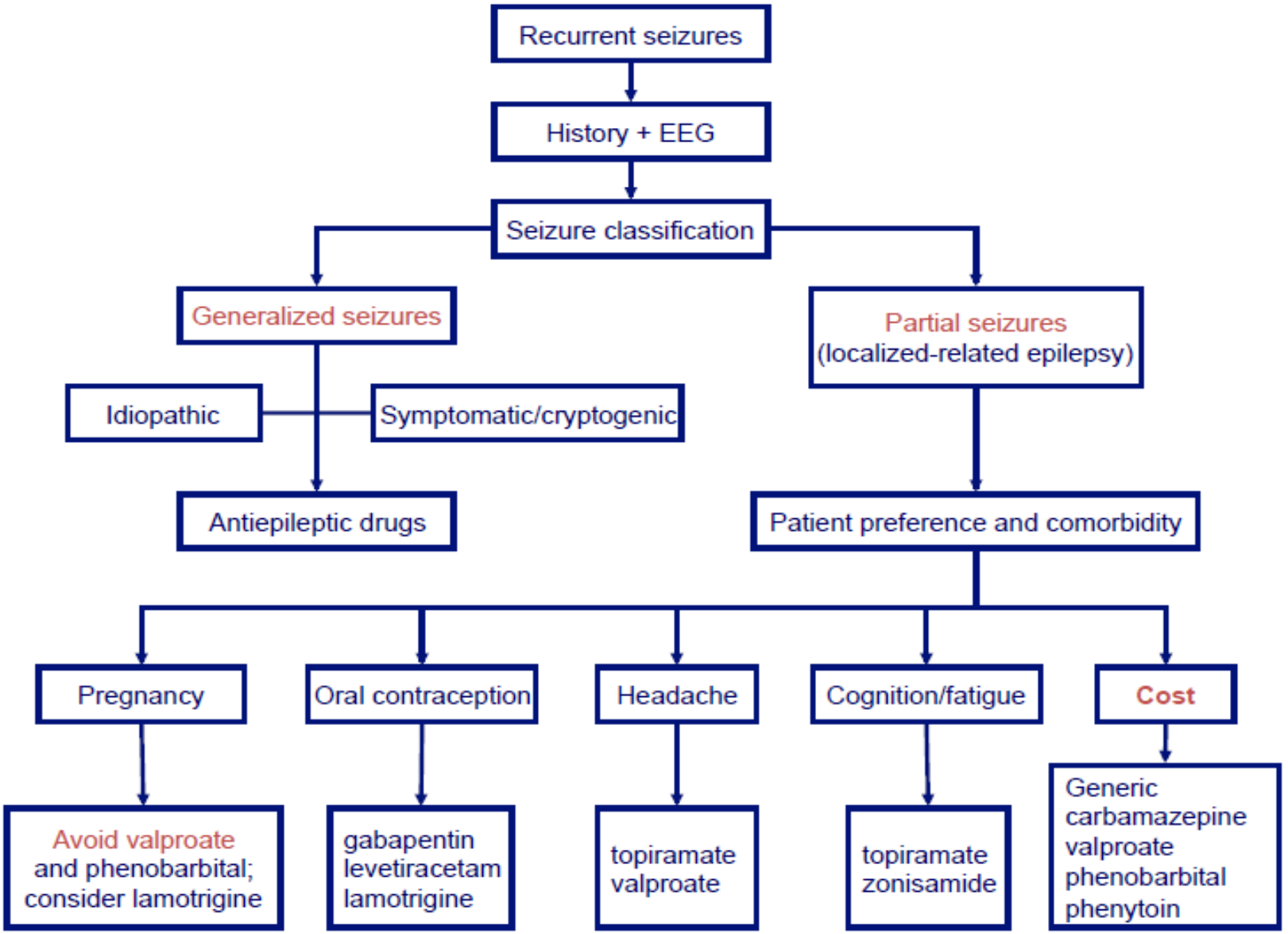
Not illustrated:

- Vigabatrin → ↓GABA degradation and drugs with multiple mechanisms:
- Valproate → ↑GABA turnover, ↓ Na^+ channels, ↓NMDA receptors
- Topiramate → ↓Na^+ channels, ↓AMPA/kainate receptors, ↑$GABA_A$ receptors
- Felbamate → ↓ Na^+ channels, ↑$GABA_A$ receptors, ↓NMDA receptors

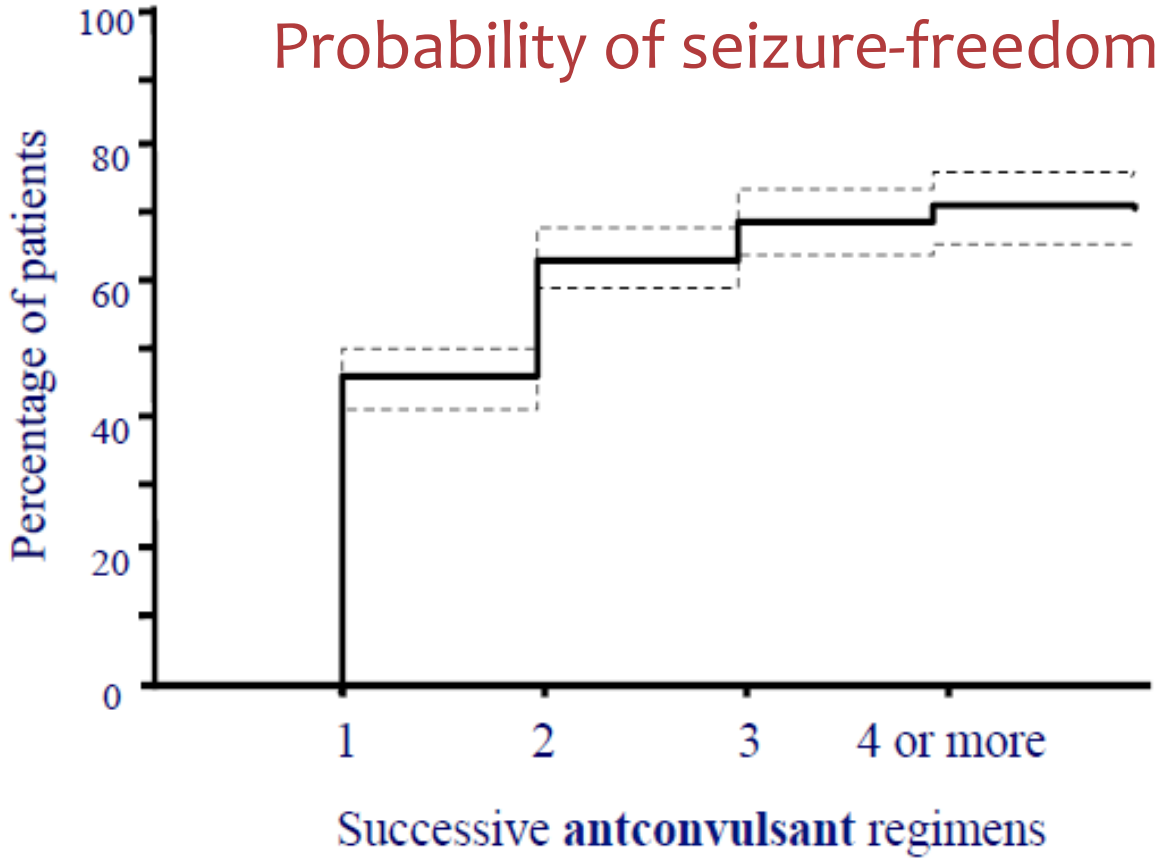
Shih et al, 2013, Ther Clin Risk Manag.



Löscher, Klein , 2021, CNS Drugs.


Recurrent seizures
History + EEG
Seizure classification
Generalized seizures
Partial seizures
(localized-related epilepsy)
Idiopathic
Symptomatic/cryptogenic
Antiepileptic drugs
Patient preference and comorbidity
Pregnancy
Oral contraception
Headache
Cognition/fatigue
Cost
Avoid valproate
and phenobarbital;
consider lamotrigine
gabapentin
levetiracetam
lamotrigine
topiramate
valproate
topiramate
zonisamide
Generic
carbamazepine
valproate
phenobarbital
phenytoin

Outcome of Newly Diagnosed Epilepsy



Neurology 2002;58(Suppl 5):S2-S8

Status epilepticus (SE)

- A condition resulting either from the failure of the mechanisms responsible for seizure termination or from the initiation of mechanisms which lead to abnormally prolonged seizures (after time point t1). It is a condition that can have long-term consequences (after time point t2), including neuronal death, neuronal injury, and alteration of neuronal networks, depending on the type and duration of seizures.

Trinka et al, 2015, Epilepsia

Conceptual definition of SE

Type of SE	Time point 1 (t1) (when seizure is likely to be prolonged)	Time point 2 (t2) (when seizure is likely to cause long-term consequences)
Tonic–clonic	5 min	30 min
Focal with impaired consciousness	10 min	>60 min
Absence	10–15 min	Data inadequate

Trinka et al, 2015, Epilepsia

Clinical classification of SE

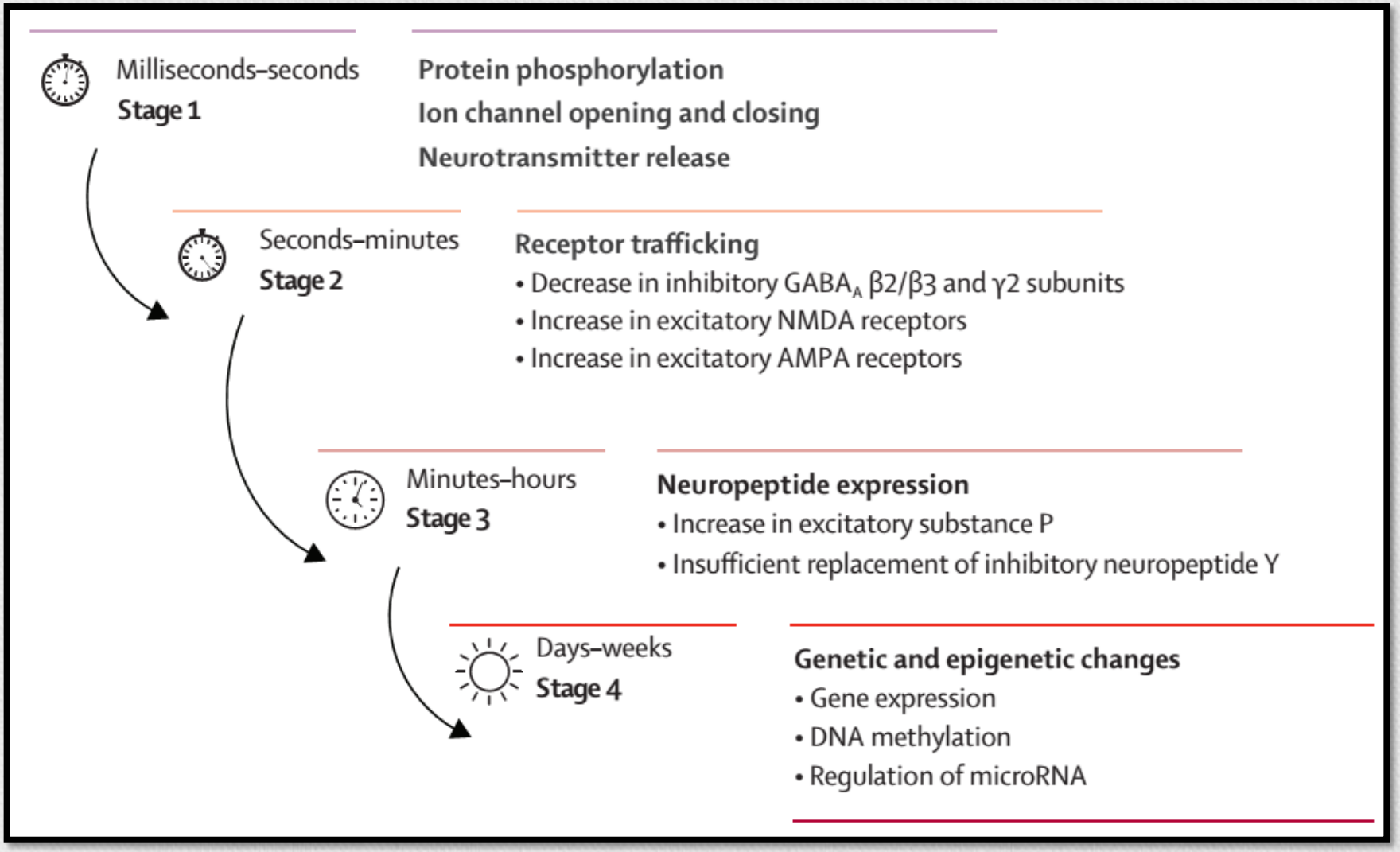
With prominent motor symptoms	Without prominent motor symptoms (Nonconvulsive status epilepticus [NCSE])
a. Convulsive SE (CSE) i. Generalized convulsive ii. Focal onset evolving into convulsive iii. Unknown whether focal or generalized b. Myoclonic SE i. With coma ii. Without coma c. Focal motor SE i. Repeated focal motor seizures ii. Epilepsia partialis continua iii. Adversive iv. Oculoclonic v. Ictal paresis d. Tonic SE e. Hyperkinetic SE	a. NCSE with coma b. NCSE without coma i. Generalized 1. Typical absence 2. Atypical absence 3. Myoclonic absence ii. Focal 1. Without impairment of consciousness 2. Aphasic SE 3. With impaired consciousness iii. Unknown whether focal or generalized 1. Autonomic SE

The frequency and mortality associated with acute and chronic causes of status epilepticus in adults

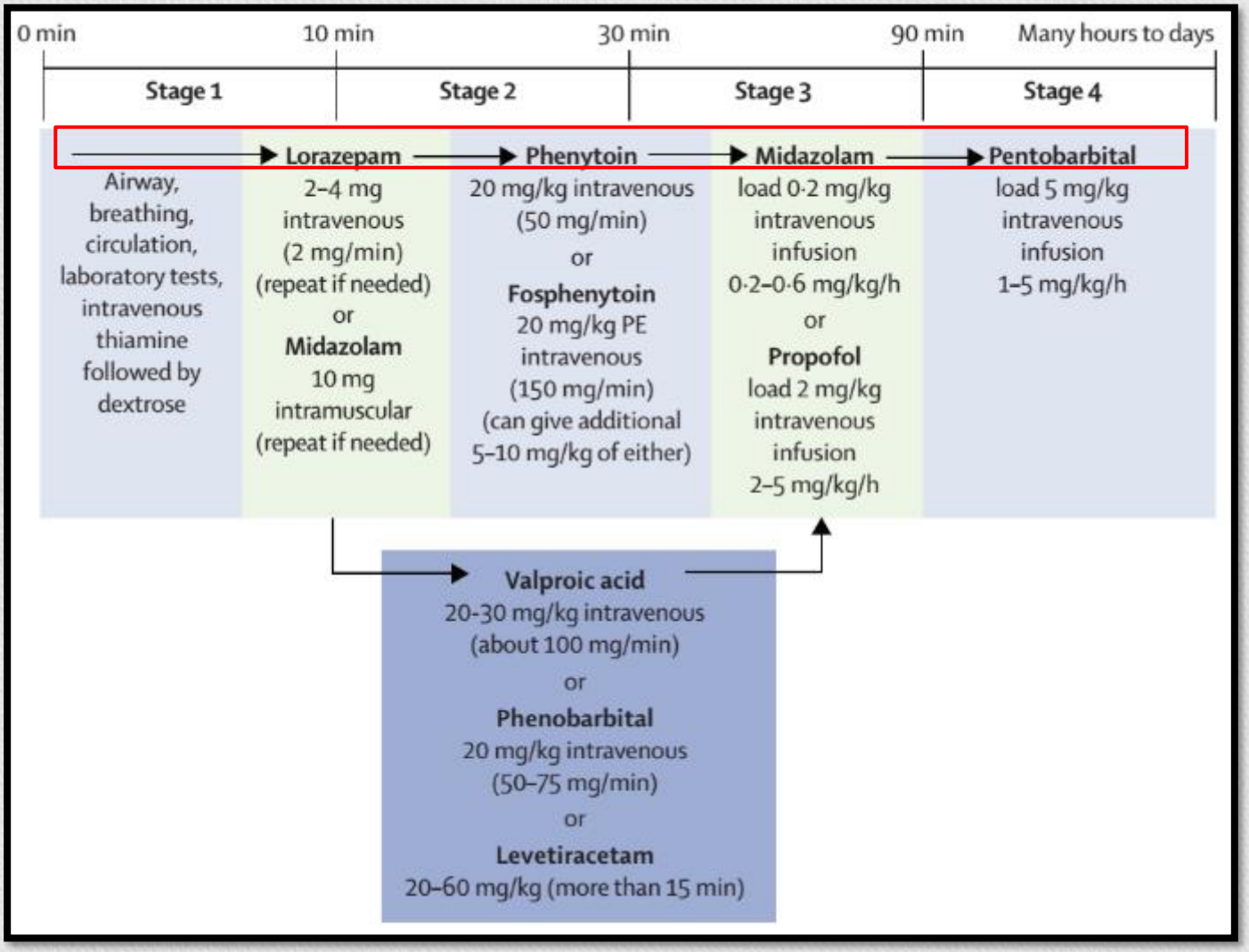
	Frequency (%)	Mortality (%)
Acute		
Stroke	22%	33%
Metabolic abnormalities	15%	30%
Hypoxia	13%	53%
Systemic infection	7%	10%
Anoxia	5%	71%
Trauma	3%	25%
Drug overdose	3%	25%
CNS infection	3%	0%
CNS haemorrhage	1%	0%
Chronic		
Low concentration of anti-epileptic drugs	34%	4%
Remote symptomatic (eg, tumour, stroke, trauma)	25%	14%
Alcohol misuse	13%	20%
Tumour	7%	30%
Idiopathic	3%	25%

Some patients had more than one aetiology.

Cascades of selected mechanism involved in the transition of single seizure to status epilepticus



Treatment of status epilepticus



Headache

Headache

- The most common neurological symptoms
- Most are benign, but the dangerous causes often result in misdiagnosis.
- Symptom severity does not correspond to the potential risk.
- Correct diagnosis is often made by a detailed history taking or physical examinations.

How to approach headache

- Timing
  - Onset
  - Duration
  - Recurrence
- Nature
  - Location
  - Quality
- Triggering factor
- Aggravating factor
- Eliminating factor
- Associated symptoms
- Target examination

Classification

- ICHD-III (international classification of headache disorder, 3rd edition, 2013)
  - Primary headache
    - Migraine
    - Tension-type headache
    - Cluster headache
  - Secondary headache

Migraine

- Most common neurological disorder (15%), particularly in young and middle-aged women (male: 11%; female: 19%)
- An episodic disorder
- Moderate to severe headache, which often affects daily activity
- Often with photophobia, phonophobia, nausea and vomiting
- Often triggered by stress, irregular sleep, or specific food, menses or menopause
- Improved by rest or sleep
- some with "AURA"
- Family history: 70%
- **Unilateral headache is not necessary**

Migraine without aura-ICHD III

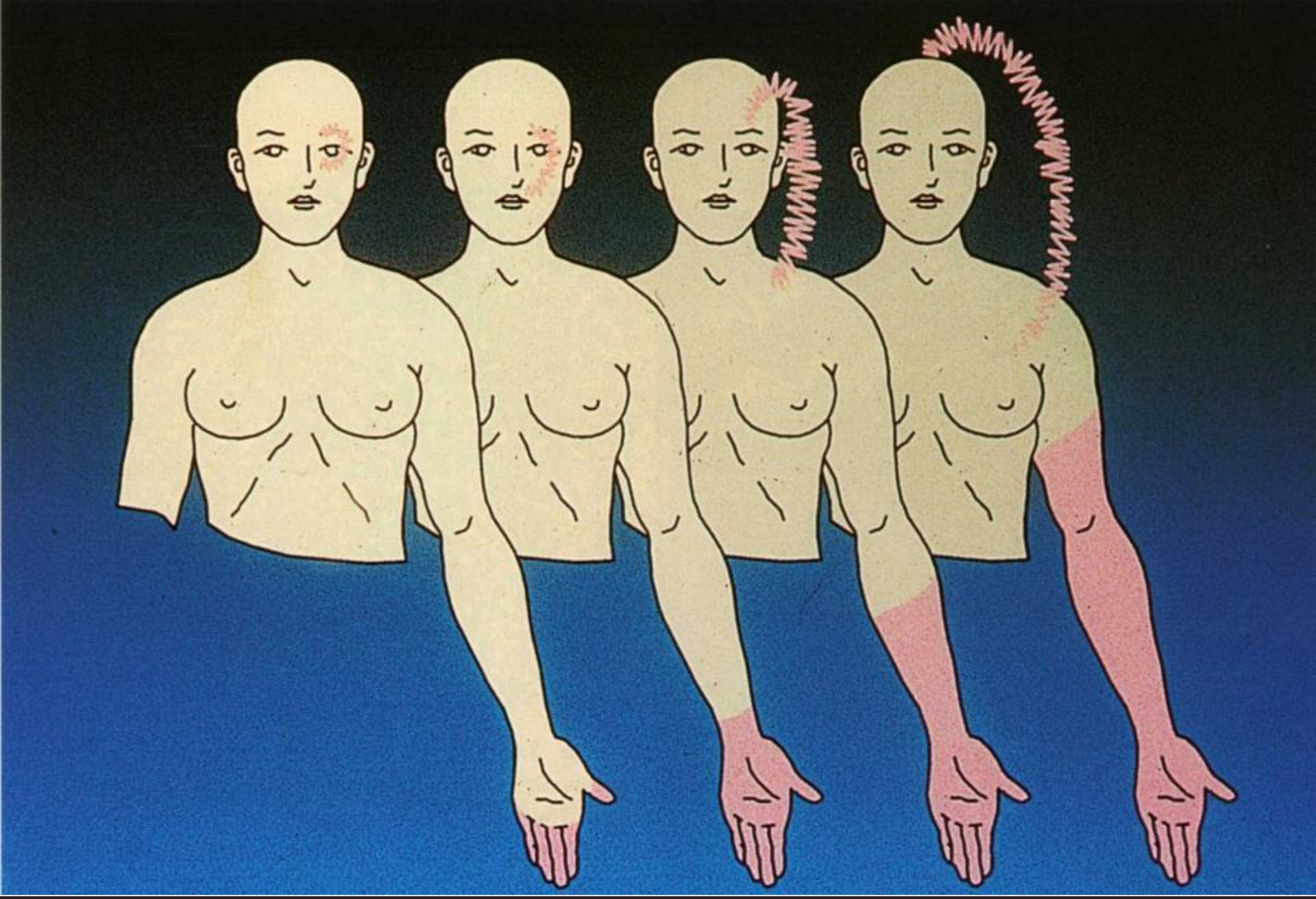
- A. At least five attacks fulfilling criteria B-D
- B. Headache attacks lasting 4-72 hr (untreated or unsuccessfully treated)
- C. Headache has at least two of the following four characteristics:
  - unilateral location
  - pulsating quality
  - moderate or severe pain intensity
  - aggravation by or causing avoidance of routine physical activity (eg, walking or climbing stairs)
- D. During headache at least one of the following:
  - nausea and/or vomiting
  - photophobia and phonophobia
- E. Not better accounted for by another ICHD-3 diagnosis.

Migraine with aura-ICHD III

- A. At least two attacks fulfilling criteria B and C
- B. One or more of the following fully reversible aura symptoms:
  - visual
  - sensory
  - speech and/or language
  - motor
  - brainstem
  - retinal
- C. At least three of the following six characteristics:
  - at least one aura symptom spreads gradually over ≥5 minutes
  - two or more aura symptoms occur in succession
  - each individual aura symptom lasts 5-60 minutes
  - at least one aura symptom is unilateral
  - at least one aura symptom is positive
  - the aura is accompanied, or followed within 60 minutes, by headache
- D. Not better accounted for by another ICHD-3 diagnosis.

Visual Aura

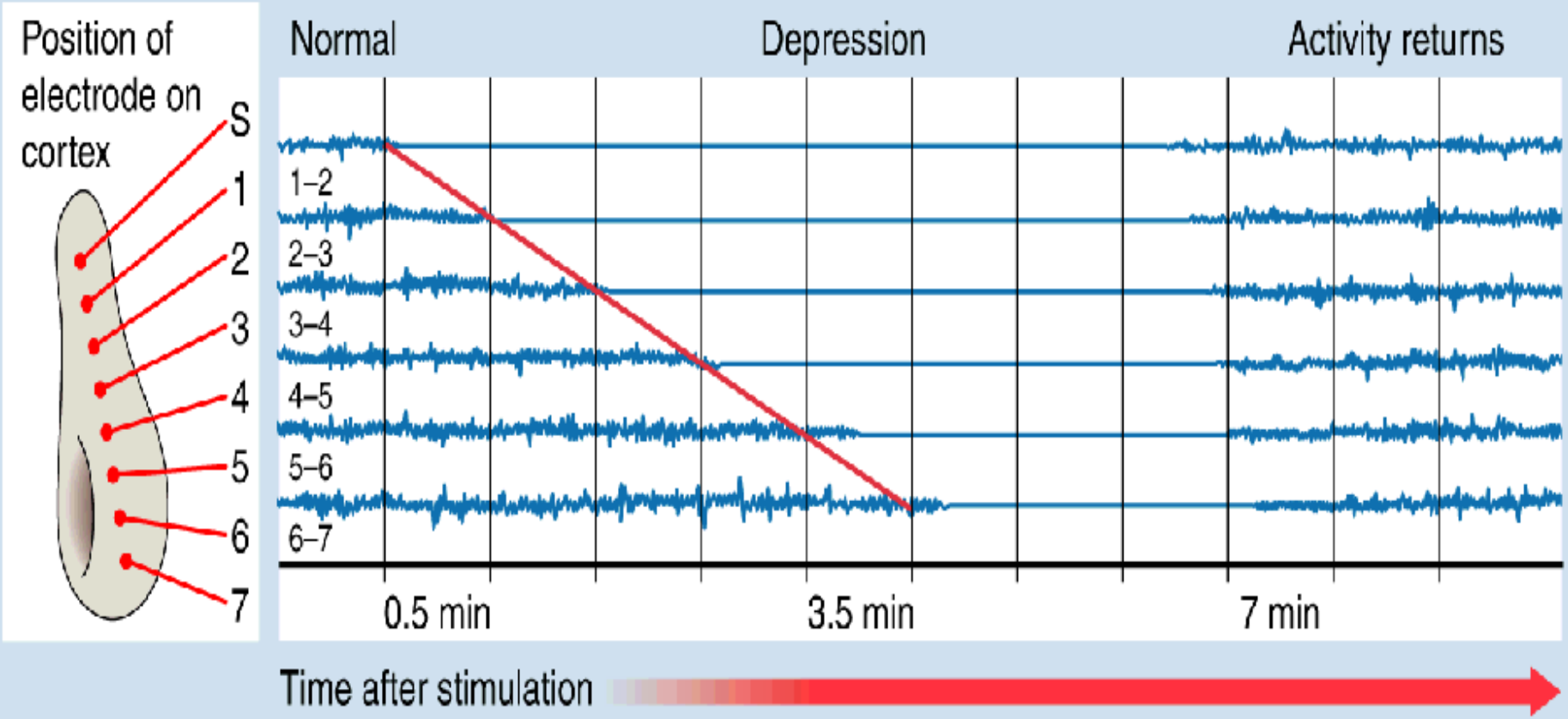
The most common aura



Marching paresthesias, the second commonest aura.

Aura: Cortical Spreading Depression

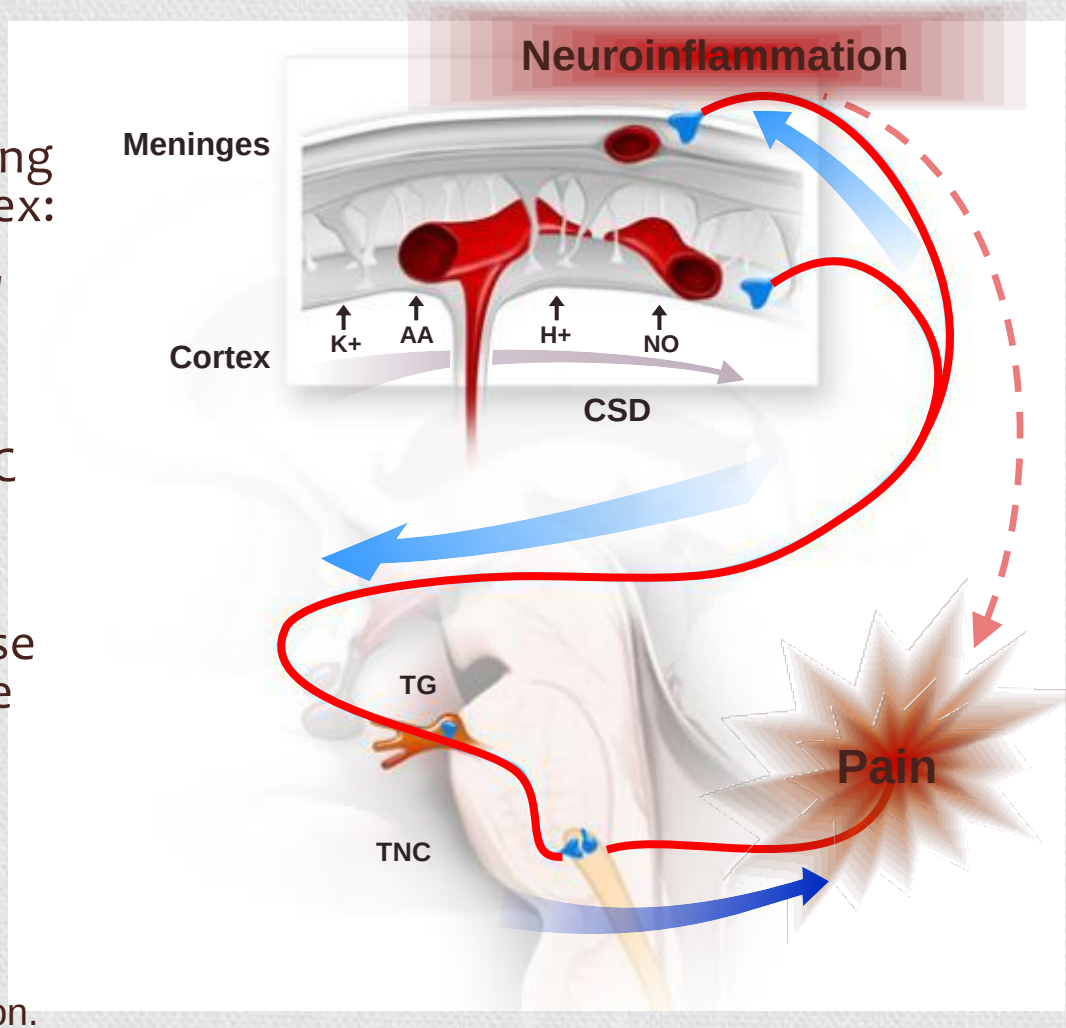
SPREADING DEPRESSION OF LEAO



Courtesy of 活水神經內科Dr.王博仁

Cortical Spreading Depression

- CSD propagates in the cortex
- H+, K+ and other agents, including AA and NO, released in the cortex: *Diffuse toward local blood vessels and* ***depolarize perivascular trigeminal terminals***
- Activate TGS → activate the TNC → **predisposition to pain**
- **Collateral axons** in the TG release proinflammatory peptides in the meninges and their vessels
  - Local inflammatory reaction → more pain?

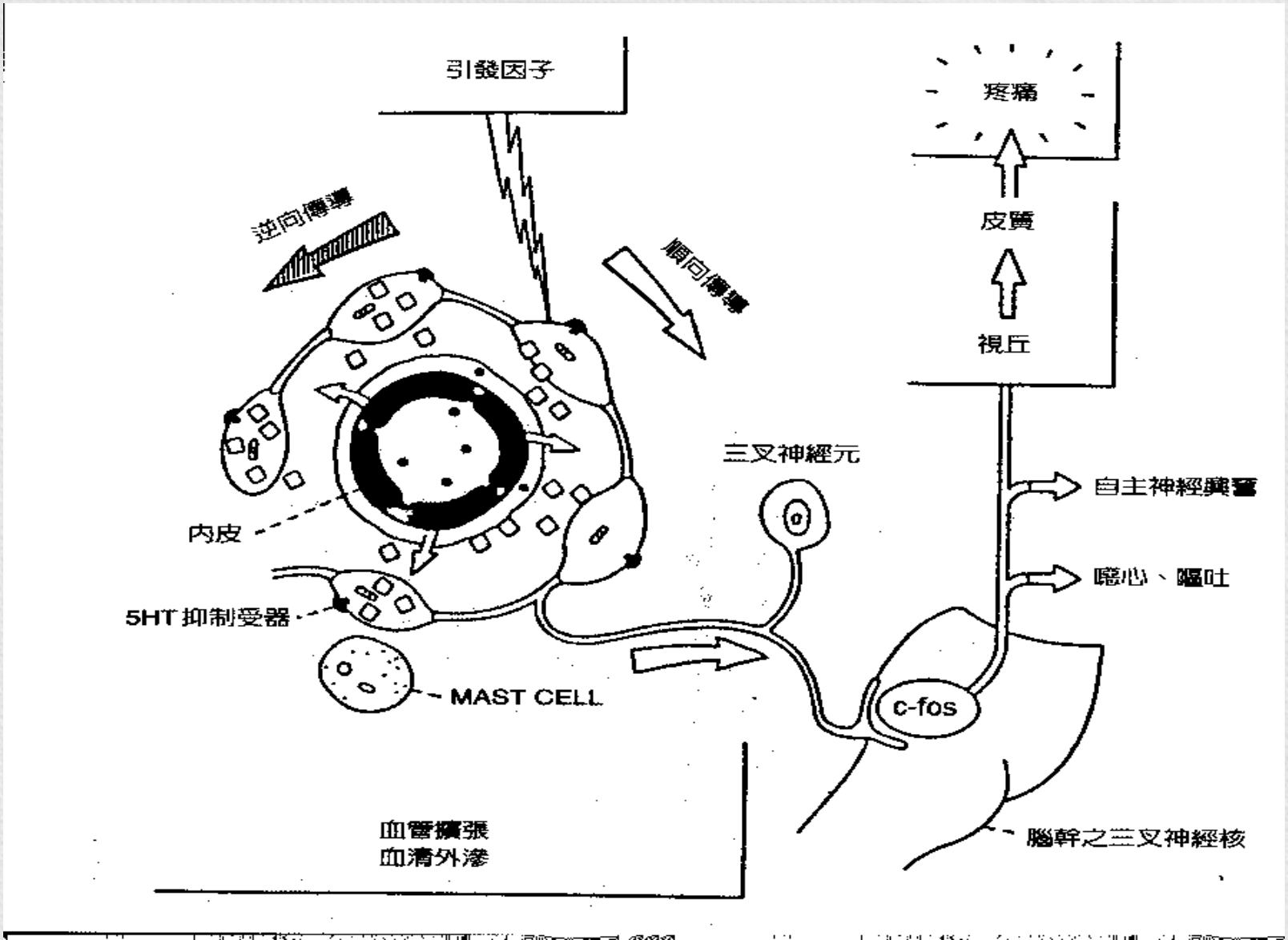


AA = arachidonic acid; TG = trigeminal ganglion.

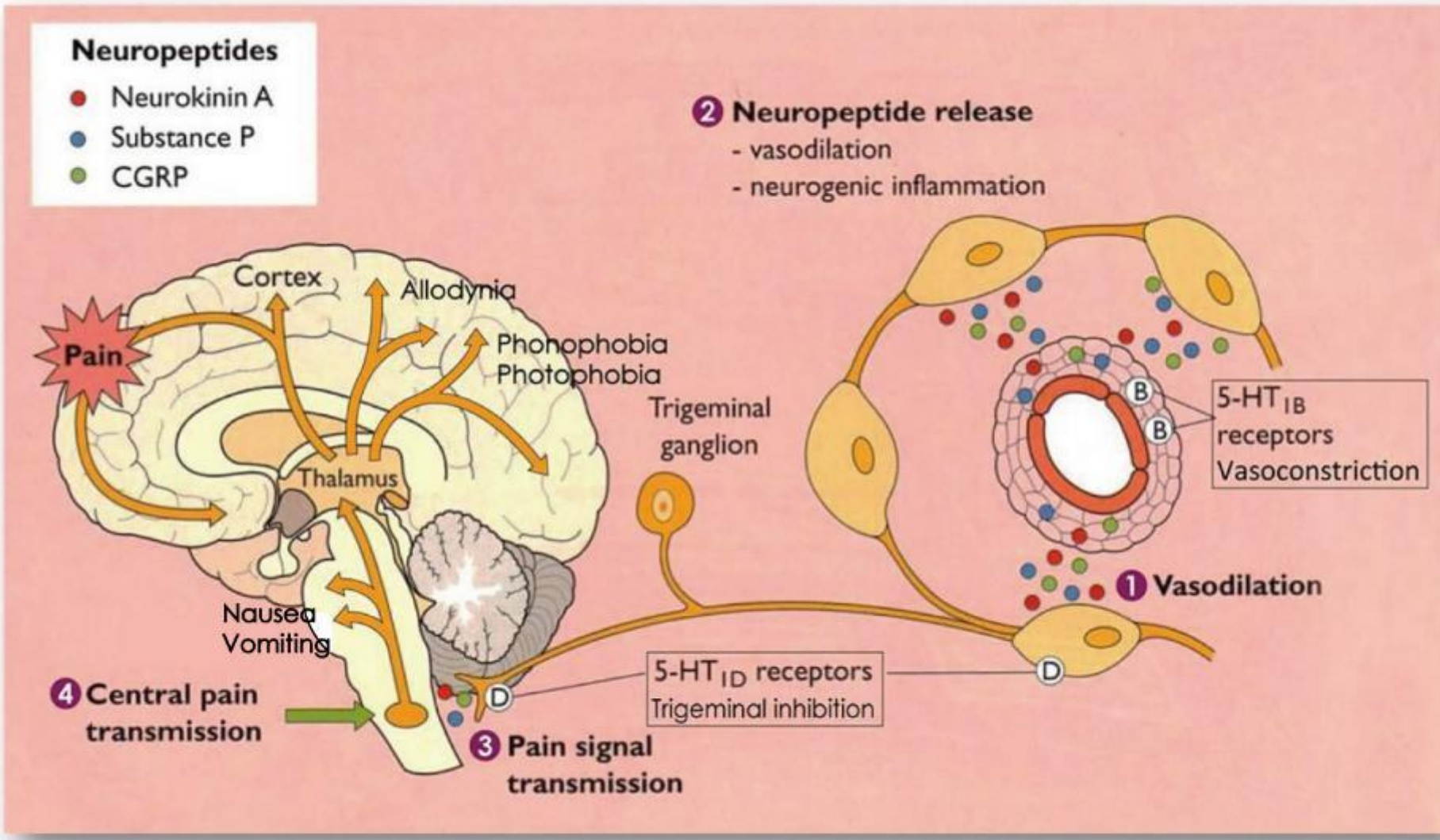
Adapted with permission from Iadecola C. *Nat Med*. 2002;8:110–112; Bolay H et al. *Nat Med*. 2002;8:136–142.

Courtesy of 活水神經內科Dr.王博仁

Trigeminovascular Activation



Central Sensitization



Treatment of Migraine (1)

- Abortive treatment
  - Mild：Acetaminophen
  - Moderate-Severe：NSAID
  - Ergotamine
  - Triptan (5HT $_{1B \text{ and } 1D}$ agnoist): Sumatriptan, Rizatriptan, etc
- Prophylactic treatment
  - β-blocker：propranolol, atenolol
  - Calcium channel blocker：flunarizine, verapamil
  - Antidepressants：amitriptyline
  - Antiepilpetic drugs：valproic acid, topiramate

Treatment of Migraine (2)

Diet

- **Regular diet**; avoid hunger (hypoglycemia)
- Reduce caffeine
- Avoid MSG (monosodium glutamate)
- Reduce Tyramine
  - fermentation food, such as aged cheese, yogurt
  - citrus or overripe fruit
- Reduce alcohol

Treatment of Migraine (3)

- Sleep
  - Regular sleep
  - Sleep deprivation often induces migraine
  - Oversleep may induce “holiday migraine”
- Regular exercise
- Reduce stress

Tension-type headache

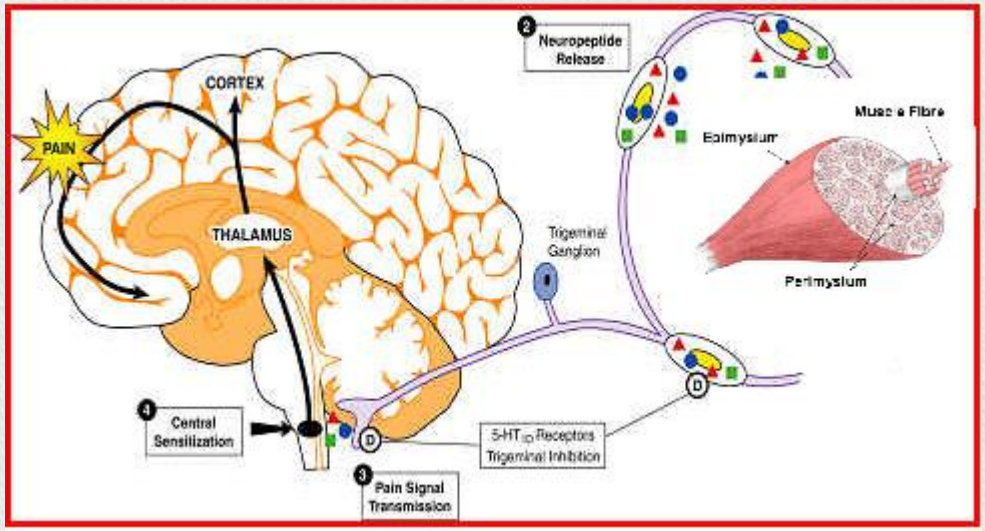
- The most common headache
- Band-like sensation over the head
- Duration: 30 minutes – 7 days
- Severity: mild to moderate
- Not aggravated by physical activity
- No nausea or vomiting
- No photophobia or phonophobia (or only one of them)

Tension-type Headache-ICHD III

- At least 10 episodes of headache fulfilling criteria B-D
- Lasting from 30 minutes to 7 days
- At least two of the following four characteristics:
  - bilateral location
  - pressing or tightening (non-pulsating) quality
  - mild or moderate intensity
  - not aggravated by routine physical activity such as walking or climbing stairs
- Both of the following:
  - no nausea or vomiting
  - no more than one of photophobia or phonophobia
- Not better accounted for by another ICHD-3 diagnosis.

Pathophysiology – Still unknown

- Old theory
- Psychogenic headache?
  - Against: Anxiety/depression are co-morbidities, not necessary conditions.
- Muscle contraction headache?
  - Against: Minimal or no muscle contraction is noted in EMG study.
- New theory
- Trigger point theory
  - Trigger points in myofascial tissue → peripheral sensitization (release inflammatory chemicals) → central sensitization?
- Spectrum theory
  - "mild-degree" migraine?



Trigger points → Peripheral sensitization → Central sensitization

Spectrum theory

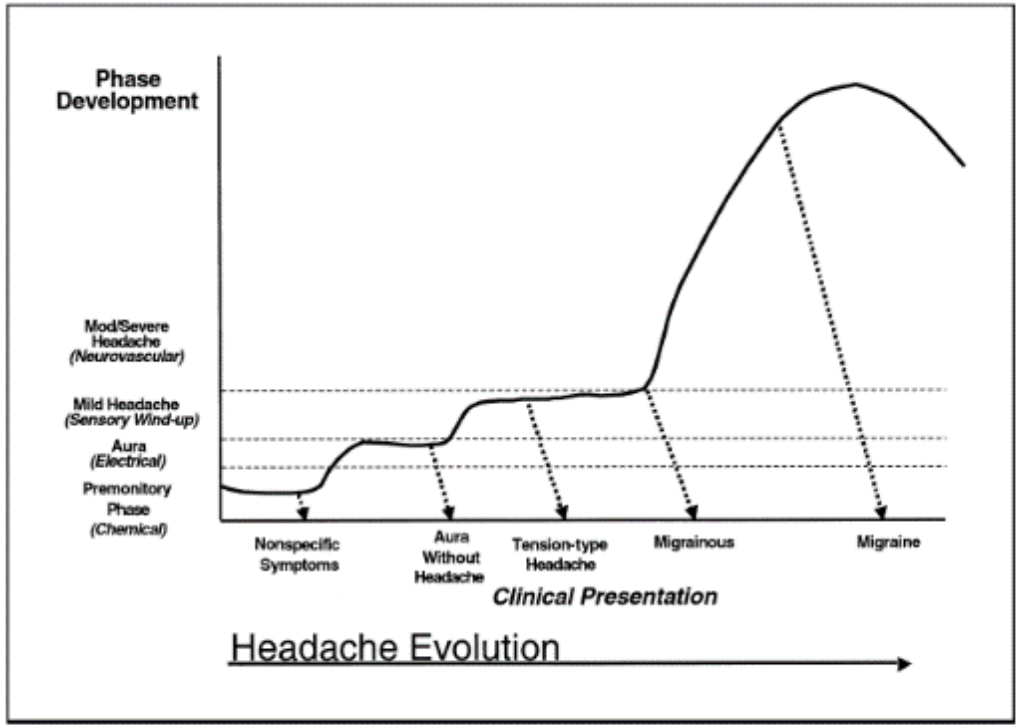
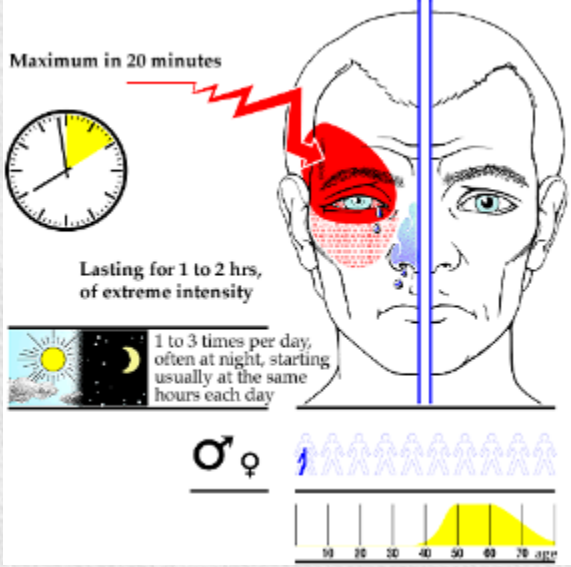


Fig 3.—Clinical evolution of common primary headache disorders.

Therapy of tension-type headache

- Medication
  - Tricyclic antidepressants, SNRI, antiepileptic drugs, muscle relaxants
- Psycho-behavioral therapy
  - Relaxation training, EMG bio-feedback, cognitive behavioral therapy
- Physical therapy

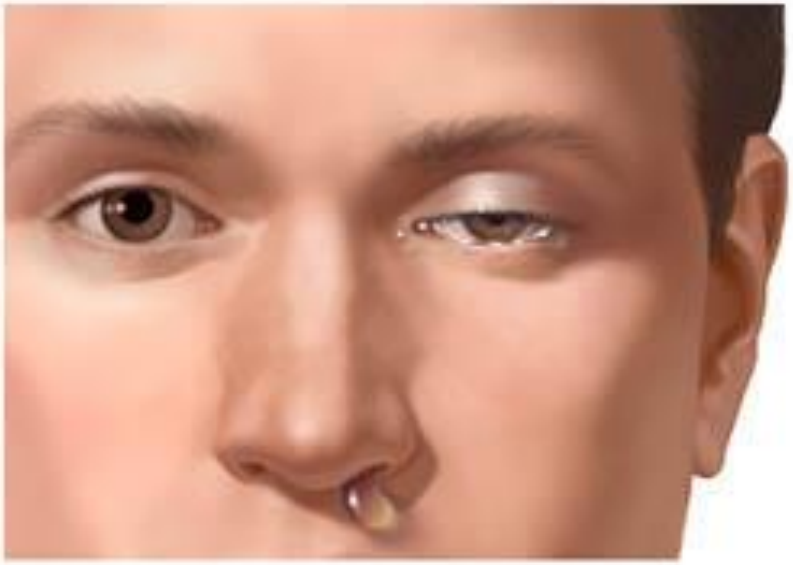
Cluster Headache



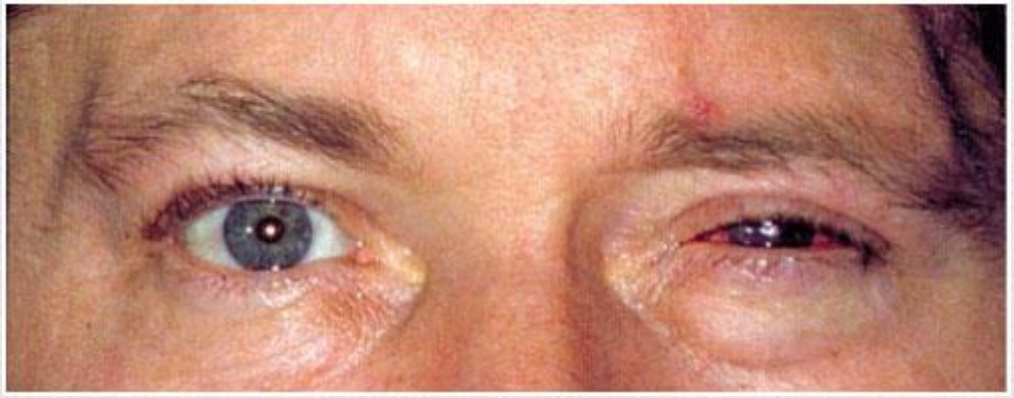
- Male-predominant (5:1)
- Common in young man
- Periorbital pain (severe) with autonomic symptoms/signs
  - Lacrimation, red eye, nasal obstruction
- **Duration:** 15 – 180 minutes
- **Frequency** of attack: daily
- **Cyclic:** 65% of patients have one cycle per year, and each cycle lasts from 2 weeks to 3 months

Cluster Headache-ICHD-III

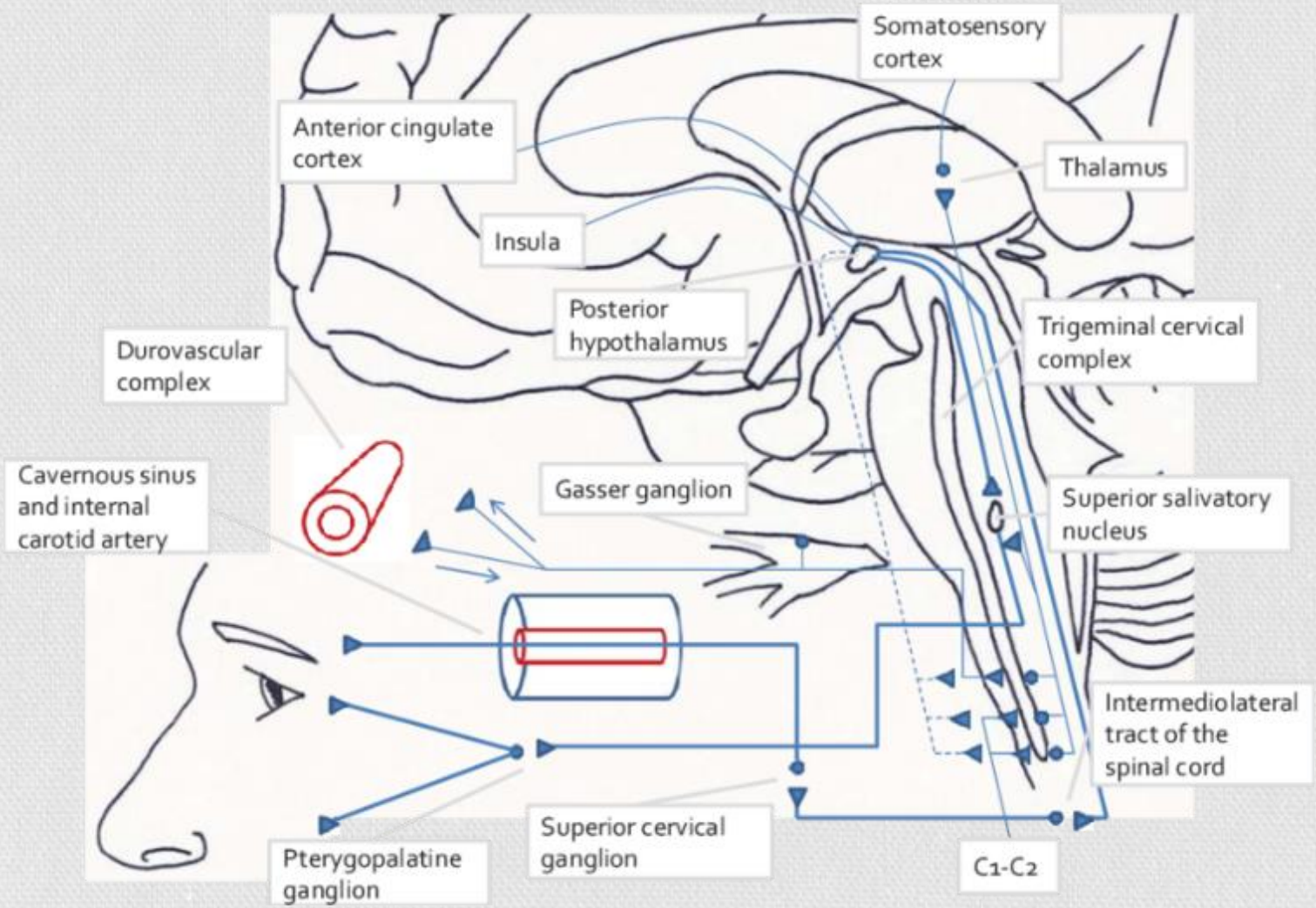
- At least five attacks fulfilling criteria B-D
- Severe or very severe unilateral orbital, supraorbital and/or temporal pain lasting 15-180 minutes (when untreated)[1]
- Either or both of the following:
  - at least one of the following symptoms or signs, ipsilateral to the headache:
    - – conjunctival injection and/or lacrimation
    - – nasal congestion and/or rhinorrhoea
    - – eyelid edema
    - – forehead and facial sweating
    - – miosis and/or ptosis
  - a sense of restlessness or agitation
- Occurring with a frequency between one every other day and 8 per day[2]
- Not better accounted for by another ICHD-3 diagnosis.



Watery eye, drooping eyelid, runny nose



Cluster headache: A trigeminal autonomic cephalalgia

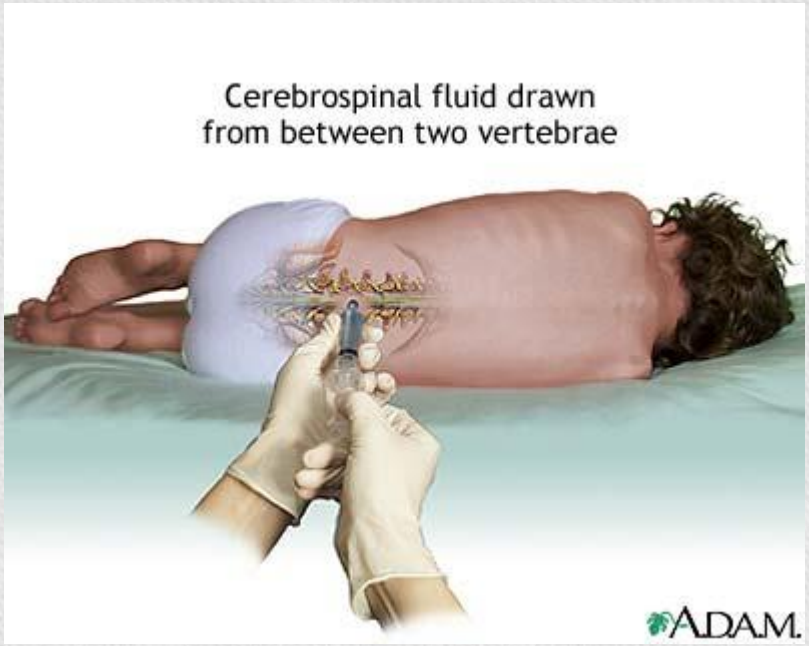


Therapy of Cluster Headache

- Elimination of triggering factors
  - Avoid alcohol, smoking
  - Reduce stress
- Therapy
  - **Oxygen therapy**
  - Verapamil
  - Steroid
  - Lithium
  - Sumatriptan (for acute abortive treatment)

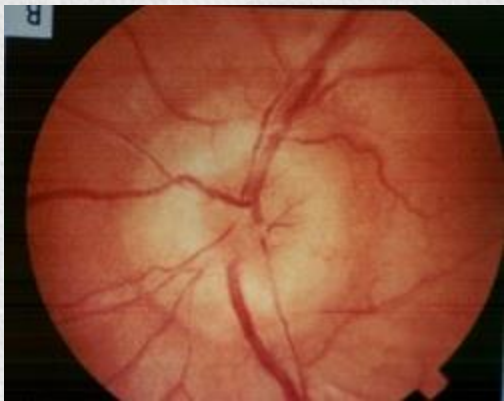
Secondary Headache

- Increased intracranial pressure (IICP)
  - Pressure >=200mmH_2O
- Low intracranial pressure (low ICP)
  - Pressure <=60mmH_2O



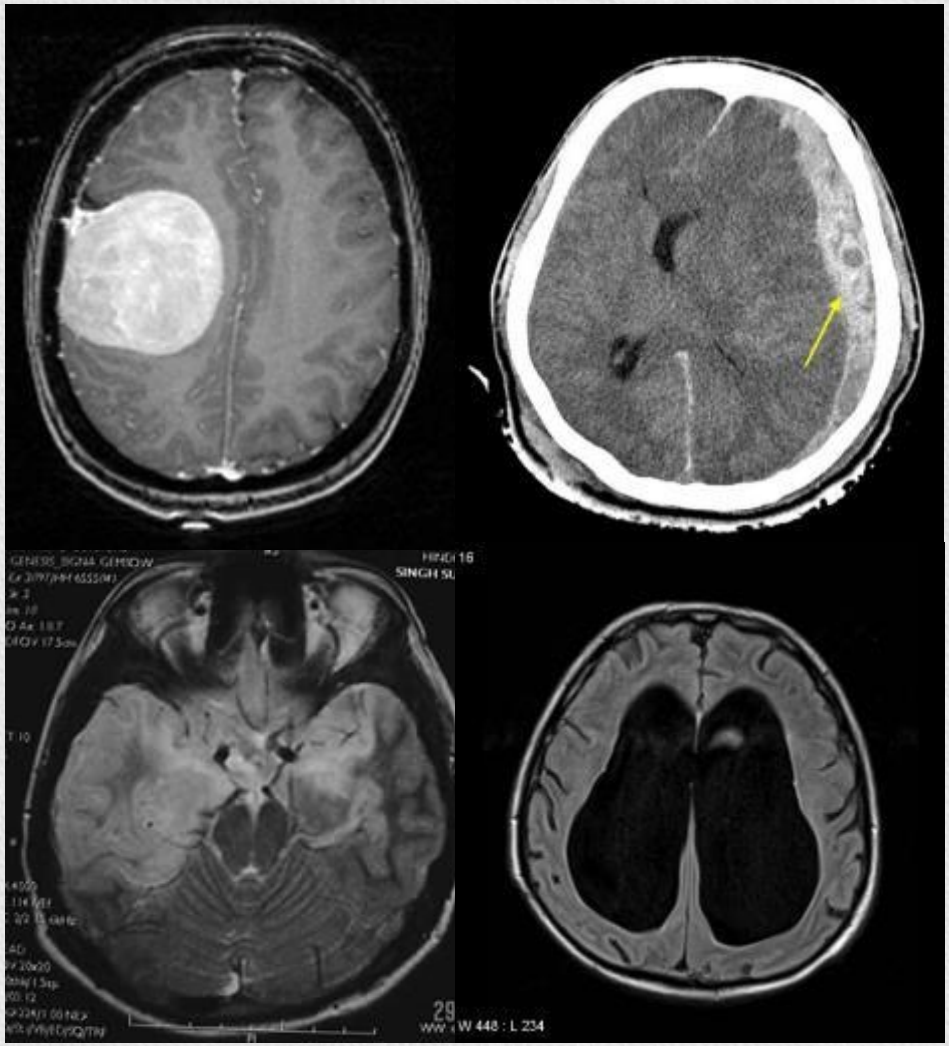
IICP Headache

- Symptoms
  - Headache
    - Morning headache
    - Postural headache (worsened by lying down; improved by sitting up)
    - Valsalva-maneuver headache
  - Projectile vomiting
  - Focal neurological symptoms may be present
- Signs
  - Papilledema
- Cushing's triad
  - Hypertension
  - Bradycardia
  - Abnormal breathing



IICP Headache

- Etiology
  - Tumor
  - Hemorrhage
  - Encephalitis
  - Hydrocephalus
  - Benign intracranial hypertension (pseudo-tumor cerebri)

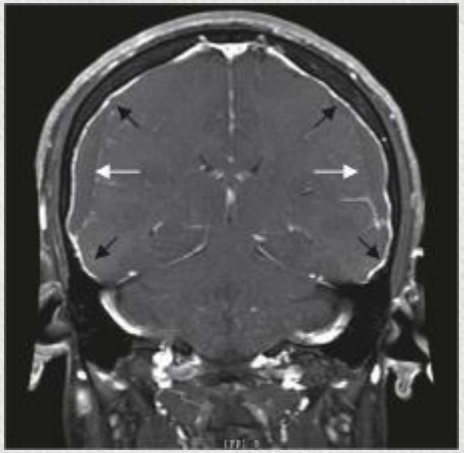


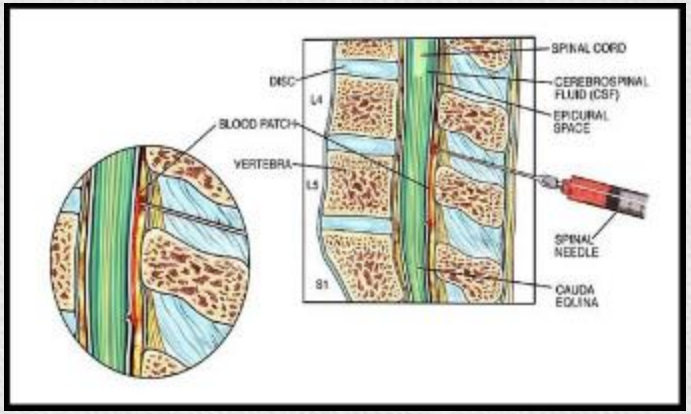
Low-ICP Headache

- Spontaneous intracranial hypotension
  - Postural headache
    - Induced by sitting or standing-up [< 15 mins]
    - Eliminated by lying down
  - Pathogenesis: CSF leakage with unknown cause
    - Most common: cervicothoracic junction, T-spine
  - Brain displacement by CSF reduction
  - MRI: Pachy-meningeal enhancement
- Lumbar puncture headache
  - 12 - 38% after lumbar puncture
  - Related to thickness of needles and repetition of punctures

Therapy of Low-ICP Headache

- Bed rest
- Fluid supplement
- Epidural blood patch





Red Flag Signs of Dangerous Headache

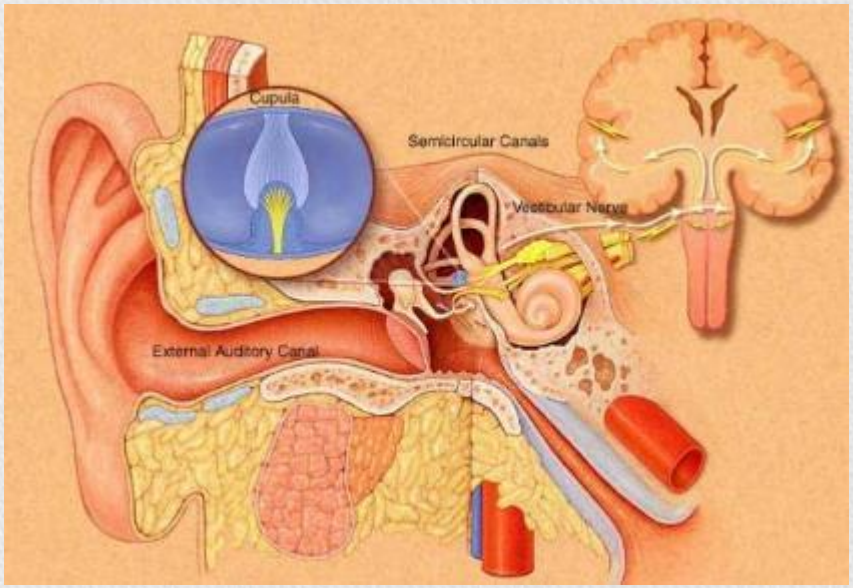
- New-onset headache
- Different from previous usual headache
- Thunderclap headache
- Projectile vomiting
- Persistent progressive headache
- Valsalva-maneuver or cough-induced headache
- New onset of headache in old-age (onset age > 50)
- Associated with fever, conscious disturbance, or other focal neurological signs

Vertigo

Classification of Dizziness

- Vertigo
  - Illusion of movement of the body or surroundings
  - Location: vestibular system
- Presyncope
  - A fainting sensation before syncope
  - Location: cardiovascular system
- Disequilibrium
  - A sensation of unsteadiness when walking
  - Location: nervous system
- Non-specific dizziness
  - Hard to describe
  - Cause: internal medicine, drugs, psychiatry, etc

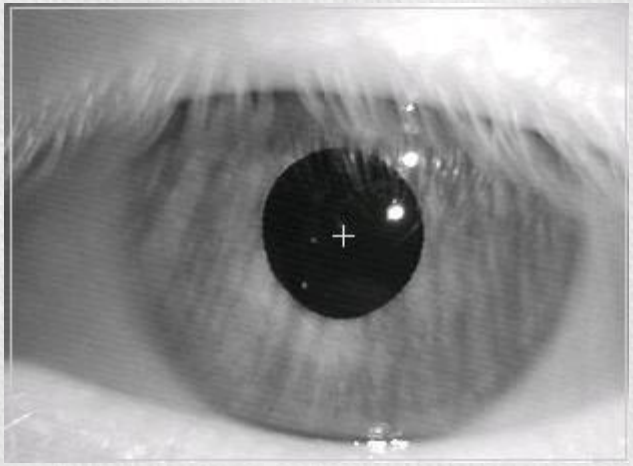
Vestibular System



- Peripheral vestibular system
  - End organs of the inner ear
  - Vestibulo-cochlear nerve (Cranial nerve VIII)
- Central vestibular system
  - Brainstem
  - Cerebellum
  - Vestibular cortex

Disturbance of vestibular system

- Three features
  - vertigo
  - nystagmus
  - unsteadiness
- Peripheral-type vertigo
  - inner ear or CN VIII lesion
- Central-type vertigo
  - brainstem or cerebellar lesion



Classification of Vertigo

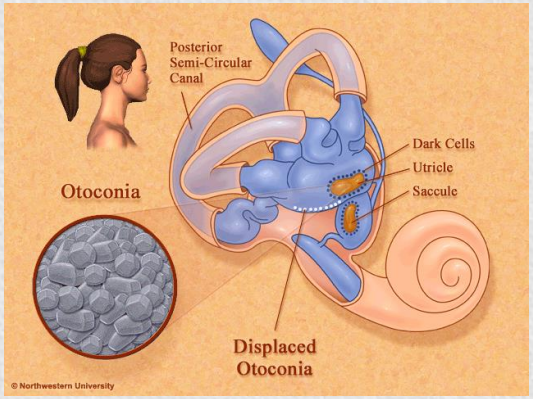
- Episodic vestibular syndrome
  - Recurrent vertigo/dizziness
  - Etiologies
    - Benign paroxysmal positional vertigo
    - Vestibular migraine
    - Meniere's disease
    - Vertebro-basilar insufficiency
- Acute vestibular syndrome (AVS)
  - Acute, only one episode, but prolonged (>= 24 hrs)
  - Etiologies
    - Vestibular neuritis
    - Stroke
- Chronic vestibular syndrome
  - Chronic persistent or progressive
  - Etiologies
    - PPPD
    - Tumor
    - Cerebellar degeneration

Episodic Vestibular Syndrome

Benign Paroxysmal Positional Vertigo (BPPV)

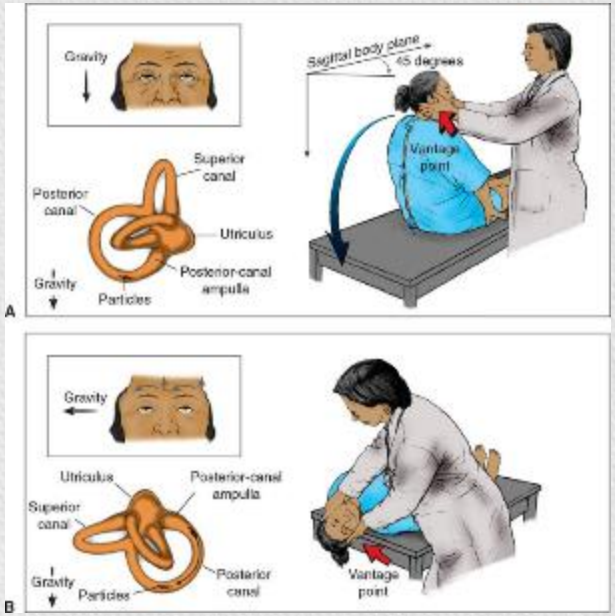
- The most common vertigo (prevalence: 3%)
- Brief vertigo during head position change
  - Lying down or sitting up in bed
  - Rolling over in bed
  - Extending the head to look up or do something
  - Bending over to tie the shoelaces
- Duration: secs to 2 mins (mostly < 30 secs)

Pathophysiology:
Otolith dislodgement into semicircular canal

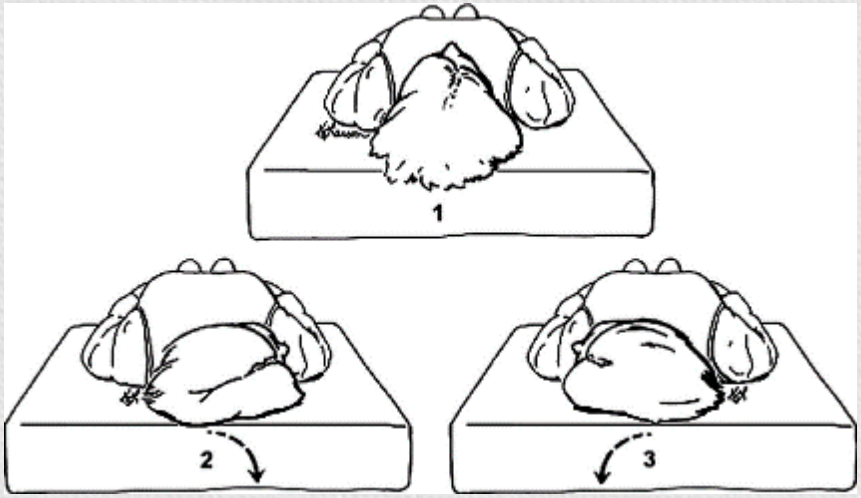


Diagnosis: Dix-Hallpike test & Supine-roll test

- Dix-Hallpike test for posterior-canal BPPV (80%)
- Supine-roll test for horizontal-canal BPPV (20%)



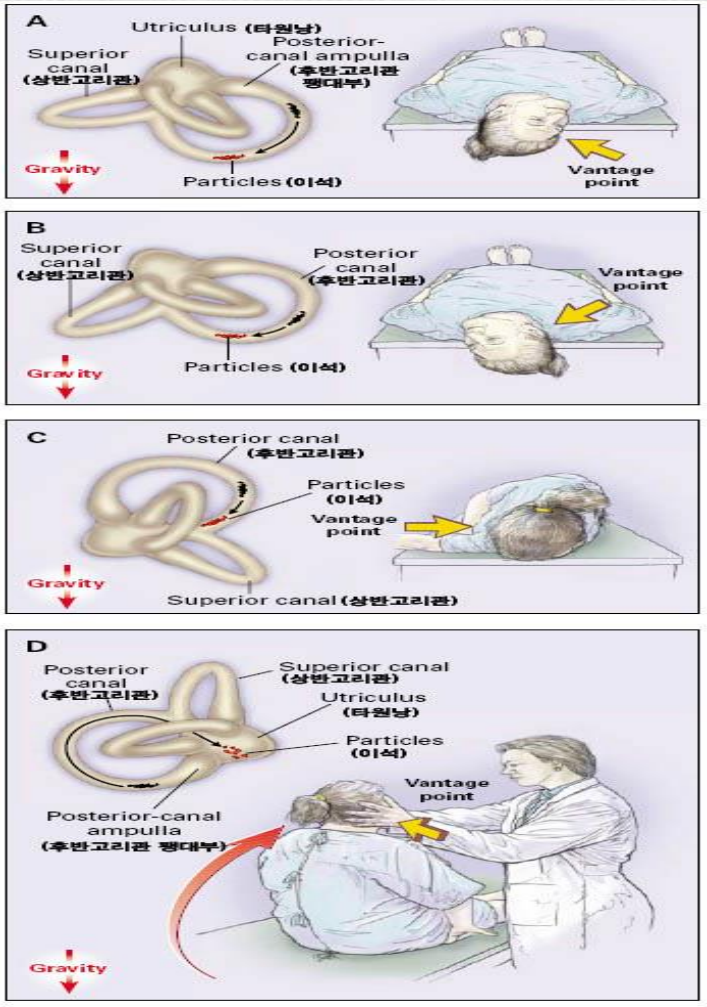
Torsional and upbeat nystagmus



Geotropic nystagmus

Physical therapy of BPPV

- Canalith repositioning maneuver
  - Posterior canal BPPV
    - Epley Maneuver
    - Semont Maneuver
  - Horizontal canal BPPV
    - Barbecue Maneuver
    - Gufoni Maneuver
    - Forced Prolonged Position
- Habituation exercise
  - Brandt-Daroff Exercise



Vestibular Migraine

- The second common vertigo (1%)
- Susceptible population: young to middle-age woman
- Symptoms
  - Recurrent vertigo or dizziness
  - History of migraine
  - Migrainous features during vertigo/dizziness
  - Headache together with dizziness is not necessary (independent in 50%)
- Other features

Diagnostic Criteria of Vestibular Migraine (ICVD/ICHD)

1. Vestibular migraine

A. At least 5 episodes with vestibular symptoms[1] of moderate or severe intensity[2], lasting 5 min to 72 hours[3]

B. Current or previous history of migraine with or without aura according to the International Classification of Headache Disorders (ICHD)[4]

C. One or more migraine features with at least 50% of the vestibular episodes[5]:

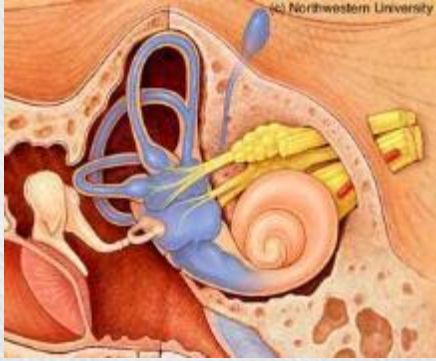
- headache with at least two of the following characteristics: one sided location, pulsating quality, moderate or severe pain intensity, aggravation by routine physical activity
- photophobia and phonophobia[6],
- visual aura[7]

D. Not better accounted for by another vestibular or ICHD diagnosis[8]

Pathophysiology and treatment of vestibular migraine

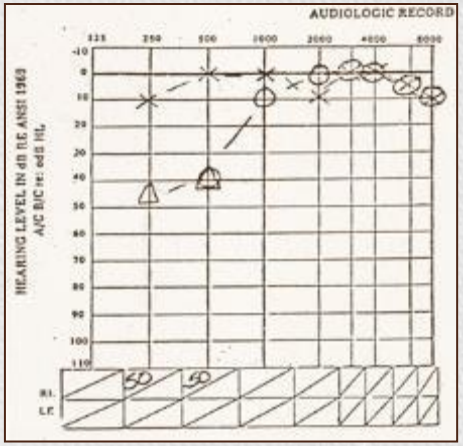
- **Pathophysiology**
- Peripheral Theory
  - Vasospasm of labyrinthine artery
  - Release of neuropeptide in the inner ear
- Central Theory
  - Spreading depression to vestibular cortex, cerebellum or brainstem
  - Serotonin/Norepinephrine-related vestibular hyperexcitability
- Channelopathy (peripheral or/and central)
- **Treatment**
- Acute: vestibular suppressants
- Chronic: the same as migraine prophylactic therapy

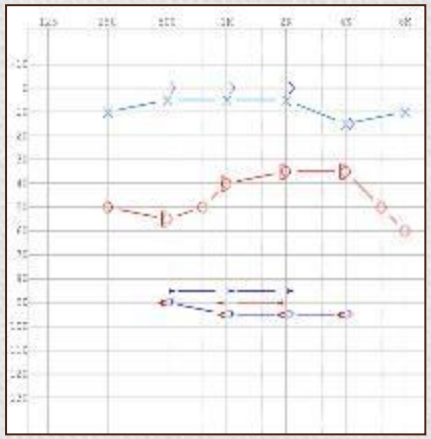
Meniere's Disease



Endolymph Hydrops

- The most famous vertigo, but often over-diagnosed
- Meniere's triads
  - Recurrent vertigo (20 mins – 12 hrs)
  - Tinnitus or aural fullness (before or during vertigo)
  - Hearing loss (before or during vertigo)
- Maybe no auditory symptoms in early stage
- At least subclinical hearing loss one year after onset (low-frequency)
- Progressive sensor-neural hearing loss after middle stage



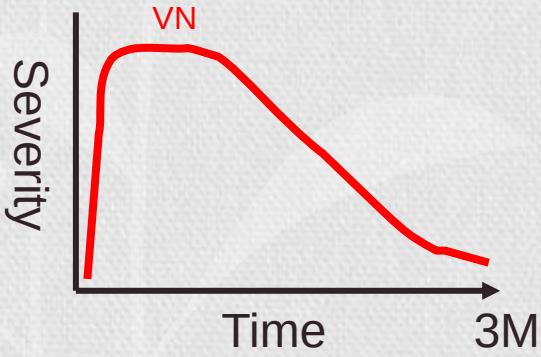


Medical treatment: salt restriction, betahistine and/or diuretics

Acute Vestibular Syndrome

Vestibular Neuritis

- The most common cause of AVS (≈75% of AVS)
- Viral infection damages vestibular nerve (HSV or others)
- Sustained violent vertigo and vomiting
- Vertigo lasts over 24 hours
- No auditory symptoms
- Vertigo → Dizziness and disequilibrium → head motion dizziness
- Standard course: 3 months
- Low recurrence (1.9%)

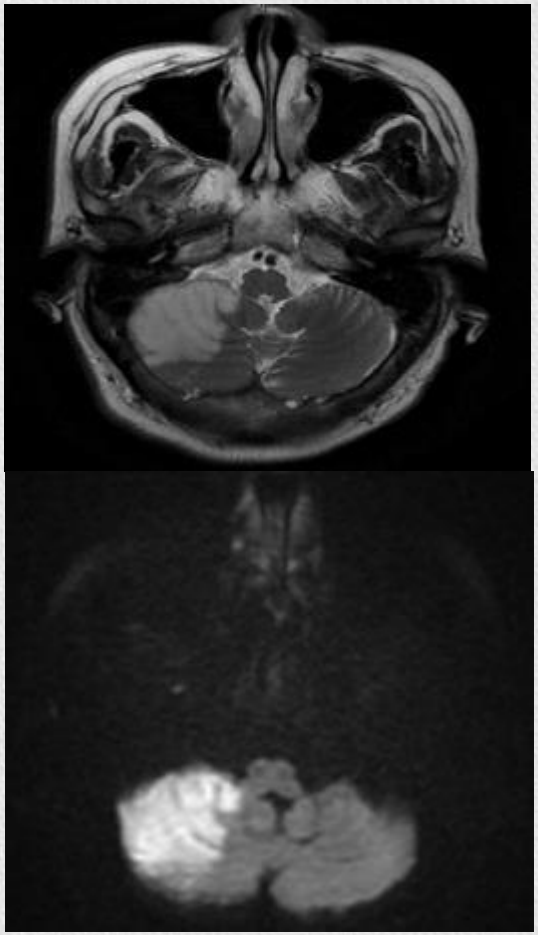


Therapy for vestibular neuritis

- Acute Stage
  - Vestibular suppressants
  - Anti-emetics
  - Short-term steroid treatment
  - Bed rest
- Chronic Stage
  - Vestibular suppressants inhibit central vestibular compensation, and should be stopped as soon as the patients no longer vomit.
  - A gradual program of vestibular rehabilitation improves the central compensation.

Posterior Circulation Stroke

- The second common cause of AVS (15-25% of AVS)
- *Characteristics*
  - *One episode with prolonged symptoms, similar to vestibular neuritis*
  - *Focal neurological signs may be present*
  - *Unsteadiness may be more severe*



Posterior circulation stroke

Risk Factors

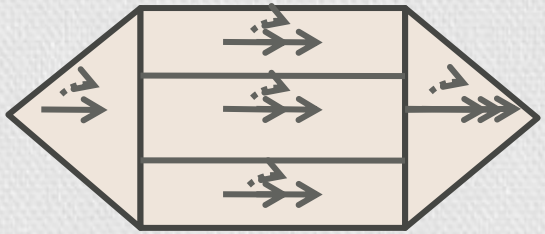
- Old age
- Hypertension
- DM
- Dyslipidemia
- Smoking
- Obesity
- Old strokes
- Heart diseases

Focal neurological signs

- Common focal signs with dizzy stroke
  - Diplopia
  - Dysarthria
  - Dysphagia
  - Focal weakness
  - Focal numbness
  - Limb ataxia
- **> 50% of dizzy stroke patients do not have focal neurological signs!**

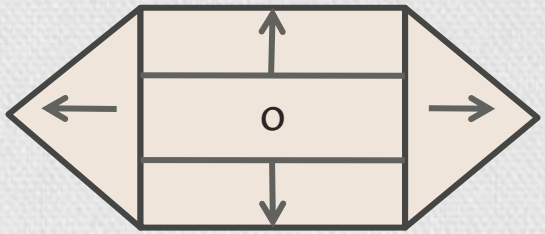
Peripheral VS Central Nystagmus

- Typical peripheral-type nystagmus (e.g. vestibular neuritis)
  - Unidirectional horizontal nystagmus (with some torsional component)
  - Compatible with Alexander's Law

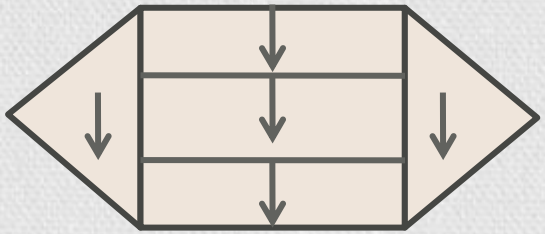


Typical peripheral nystagmus

- Central-type nystagmus (e.g. posterior circulation stroke)
  - Pure vertical nystagmus or pure torsional nystagmus
  - Gaze-evoked nystagmus
  - Other rare forms (periodic alternating nystagmus, seesaw nystagmus, etc)



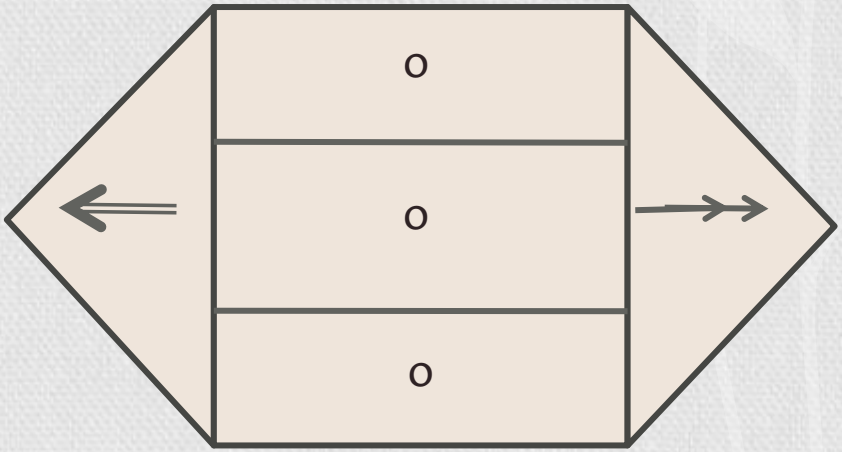
Gaze-evoked nystagmus



Downbeat nystagmus

Brun's nystagmus

- Cerebellopontine (CP) angle tumor
- Peripheral (vestibular) + Central (cerebellar) –type nystagmus
- Gaze away from lesion side → vestibular nystagmus
  - Rapid
  - Small amplitude
- Gaze toward lesion side → cerebellar gaze-evoked nystagmus
  - Slow
  - Large amplitude



Right CP angle tumor

	Peripheral	Central	
Obvious oculomotor signs	0%	32%	0.68 (0.59–0.80)*
Dominantly vertical or torsional nystagmus	0%	12%	0.88 (0.81–0.96)
Oculomotor paralysis (3-4-6, INO, gaze palsy)	0%	21%	0.79 (0.70–0.89)*
Subtle oculomotor signs	4%	100%	0.00 (0.00–0.11)*
Direction-changing horizontal nystagmus	0%	20%	0.80 (0.72–0.90)*

- 60-70% of central-type vertigo presents with "peripheral-type" nystagmus.
- In addition to nystagmus, head impulse test and test of skew deviation help identify central-type vertigo.
  - HINTS = Head Impulse test, Nystagmus, and Test of Skew deviation
  - AVS with normal head impulse test, direction-changing nystagmus, or presence of skew deviation implies central-type vertigo

Peripheral VS Central Vertigo-Summary (1)

Table 1. Features in the history that help distinguish between peripheral and central causes of vertigo

	Peripheral	Central
Imbalance	Mild-moderate	Severe
Nausea and vomiting	Severe	Variable, may be minimal
Auditory symptoms	Common	Rare
Neurologic symptoms	Rare	Common
Compensation	Rapid	Slow

Adapted from Baloh RW, Honrubia V. Clinical neurophysiology of the vestibular system. 2nd ed. Philadelphia: FA Davis; 1990.

Vertigo may be more **severe** in **peripheral** than central vertigo

Peripheral VS Central Vertigo-Summary (2)

Table 2. Differentiation between peripheral and central varieties of spontaneous nystagmus

Type	Appearance	Fixation	Gaze	Mechanism	Localization
Peripheral vestibular	Combined torsional & horizontal	Inhibited	Unidirectional (Alexander's law)	Asymmetric loss of peripheral vestibular tone	Labyrinth or vestibular nerve
Central vestibular	Often pure vertical, horizontal, or torsional	Usually little effect	May change direction	Imbalance in central vestibular tone	Brain stem or cerebellum

Adapted from Baloh RW, Honrubia V. Clinical neurophysiology of the vestibular system. 2nd ed. Philadelphia: FA Davis; 1990.

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